PAIN AND PSYCHOLOGICAL DISTRESS
IN MIDDLE-AGED AND OLDER MARRIED COUPLES

by
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GENERAL TABLE OF CONTENTS

LIST OF TABLES ii
LIST OF FIGURES iv
ABSTRACT v

CHAPTERS

CHAPTER 1: Background and Significance 1-i
CHAPTER 2: Pain and Psychological Distress at the Intra-Individual Level in Middle-Aged and Older Adults 2-i
CHAPTER 3: Pain and Psychological Distress at the Inter-Individual Level in Middle-Aged and Older Married Couples 3-i
CHAPTER 4: Method 4-i
CHAPTER 5: Results 5-i
CHAPTER 6: Discussion 6-i

APPENDICES

APPENDIX A: HRS 1998 Questionnaire Items A-1
APPENDIX B: Details Regarding the HRS “Unfolding-Bracketing” Strategy and the RAND Imputations for HRS Household Income and Wealth Data B-1
APPENDIX C: Variables in the Datafile Used in the Multiple Imputation Procedure C-1
APPENDIX D: Procedures for Fixing the Initial Error Variances in the Dual-Intercept HLM Models D-1
APPENDIX E: General Multilevel Model for the Dual-Intercept Model E-1
APPENDIX F: Sample Multilevel Models for Research Question 7 F-1

REFERENCES R-1
LIST OF TABLES

Table 1  Prior Empirical Studies that Tested Mediation of the 
Relationship between Pain and Psychological Distress by 
Activity Limitation (N = 14)  2-153

Table 2  Summary of Analyses Used to Address Research Questions 
and Hypotheses in Dual-Pain (DP) Couples  4-95

Table 3  Pattern of Husband and Wife Responses to HRS 1998 
Pain Item within Married Couples Initially Eligible for 
Inclusion in the Present Study (N = 5,386 couples)  5-3

Table 4  Characteristics of Dual-Pain Couples and Comparisons with 
Each Other Group  5-5

Table 5  Characteristics of Husbands and Wives in Dual Pain Couples 
and Selected Comparisons to the Relevant Spouse in Other 
Groups  5-11

Table 6  Husbands and Wives in Dual-Pain Couples Compared on Key 
Study Variables & Other Characteristics  5-40

Table 7  Intraclass Correlations Between Husbands and Wives in Dual- 
Pain Couples (N = 423)  5-46

Table 8  Cross-Classification Table of Pain Intensity Ratings of 
Husbands and Wives in Dual-Pain Couples (N = 423)  5-48

Table 9  Correlations Among Model Variables for Husbands in 
Dual-Pain Couples (N = 423)  5-55

Table 10  Correlations Among Model Variables for Wives in Dual-Pain 
Couples (N = 423)  5-56

Table 11  Disease Condition Counts & Top 3 Patterns for Husbands and 
Wives in Dual-Pain Couples (N = 423)  5-61

Table 12  Intra-Individual Predictors of Pain Intensity in Dual-Pain 
Husbands and Wives (N = 423)  5-64

Table 13  Intra-Individual Predictors of Depressive Symptomatology in 
Dual-Pain Husbands and Wives (N = 423)  5-75
| Table 14 | Pain Intensity as an Intra-Individual Predictor of Physical Limitations in Dual-Pain Husbands and Wives \((N = 423)\) | 5-84 |
| Table 15 | Test of Physical Limitations as Mediators of the Intra-individual Relationship between Pain Intensity and Depressive Symptomatology in Husbands and Wives in Dual-Pain Couples \((N = 423)\) | 5-90 |
| Table 16 | (Cross-spouse) Correlations between Individual-level Model Variables of Husbands and Wives in Dual-Pain Couples \((N = 423)\) | 5-108 |
| Table 17 | Test of the Cross-Spouse Relationship of Each Spouse’s Pain Intensity to His/Her Partner’s Depressive Symptomatology and Mediation by the Spouse’s Physical Limitations in Dual-Pain Husbands and Wives | 5-114 |
| Table 18 | Test of Respondent by Spouse Pain Intensity Interaction Effect on Depressive Symptomatology in Dual-Pain Husbands and Wives | 5-124 |
| Table 19 | Final Trimmed Model Predicting Depressive Symptomatology in Husbands and Wives in Dual-Pain Couples \((N = 423)\) | 5-141 |
# LIST OF FIGURES

<table>
<thead>
<tr>
<th>Figure</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Figure 1</td>
<td>Williamson’s Activity Restriction Model (ARM) of Depressed Affect</td>
<td>2-43</td>
</tr>
<tr>
<td>Figure 2</td>
<td>The Disablement Process Model of Verbrugge and Jette (1994)</td>
<td>2-48</td>
</tr>
<tr>
<td>Figure 3</td>
<td>The Illness-Disability Cascade of Kahana et al. (1997)</td>
<td>2-52</td>
</tr>
<tr>
<td>Figure 4</td>
<td>Proposed Conceptual Model Linking Pain and Psychological Distress in Individuals</td>
<td>2-60</td>
</tr>
<tr>
<td>Figure 5</td>
<td>Proposed Conceptual Model Linking Pain and Psychological Distress in Married Couples</td>
<td>3-9</td>
</tr>
<tr>
<td>Figure 6</td>
<td>Sample Derivation Flowchart</td>
<td>4-11</td>
</tr>
<tr>
<td>Figure 7</td>
<td>Estimated Intra-individual Effect of Pain Intensity on Depressive Symptomatology in Dual-Pain Husbands and Wives</td>
<td>5-78</td>
</tr>
<tr>
<td>Figure 8</td>
<td>Path Diagram of the Intra-individual Relationship between Pain Intensity and Depressive Symptomatology in Husbands (Panel A) and Wives (Panel B) in Dual-Pain Couples (N = 423)</td>
<td>5-94</td>
</tr>
<tr>
<td>Figure 9</td>
<td>Estimated Remaining Direct Effect of Pain Intensity on Depressive Symptomatology in Dual-Pain Husbands and Wives</td>
<td>5-98</td>
</tr>
<tr>
<td>Figure 10</td>
<td>Respondent by Spouse Pain Intensity Interaction Effect on Depressive Symptomatology in Dual-Pain Husbands</td>
<td>5-128</td>
</tr>
<tr>
<td>Figure 11</td>
<td>Respondent by Spouse Pain Intensity Interaction Effect on Depressive Symptomatology in Dual-Pain Wives</td>
<td>5-131</td>
</tr>
<tr>
<td>Figure 12</td>
<td>Respondent by Spouse Pain Intensity Interaction Effect for Dual-Pain Husbands in the Presence of Additive Risk Factors</td>
<td>5-146</td>
</tr>
<tr>
<td>Figure 13</td>
<td>Respondent by Spouse Pain Intensity Interaction Effect for Dual-Pain Wives in the Presence of Additive Risk Factors</td>
<td>5-147</td>
</tr>
</tbody>
</table>
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Pain is estimated to affect more Americans than diabetes, heart disease, stroke, and cancer combined. An abundant literature documents the negative consequences of pain for individuals (e.g., psychological distress) and society (e.g., high healthcare costs). Studies also have found elevated psychological distress in spouses of persons with pain, but existing knowledge derives mainly from research with clinical samples (e.g., chronic pain patients and spouses). Little is known about couples living with pain in the community—especially those in mid and later life. Moreover, studies have rarely considered that both spouses might have pain.

This study sought to (a) identify a large sample of community-dwelling, aging couples in which both spouses had pain, in order to (b) examine the impact of each spouse’s own pain, as well as the pain of his/her partner, on the psychological distress of both spouses. Self-reports of pain were examined in 5,386 couples who participated in the 1998 Health and Retirement Study, a representative study of U.S. adults over age 50. In 8% of these couples, both spouses reported pain. These 423 “Dual-Pain” couples comprised the focal sample for this study.
Dual-intercept multilevel models were used to test hypotheses relating pain (pain intensity) to psychological distress (depressive symptomatology) at both the intra-individual and inter-individual (i.e., cross-spouse) levels. Activity limitation (physical limitations—e.g., difficulty climbing stairs or kneeling) was also explored as a potential mediator of these relationships.

Results showed that each spouse’s own pain intensity was positively associated with his/her own depressive symptomatology, though the effect was stronger in wives. This relationship was completely mediated by physical limitations in husbands, but only partially mediated in wives. A cross-spouse effect of pain was found, but only for husbands: Wives’ pain intensity affected husbands’ depressive symptoms, but the nature of the effect depended on husbands’ own pain intensity. No evidence was found for mediation of the cross-spouse effect by physical limitations of the spouse. Findings can inform social work practice and advocacy efforts with aging couples affected by pain. Results also highlight the need for practitioners and researchers to consider pain from a dyadic perspective.
### CHAPTER 1: BACKGROUND AND SIGNIFICANCE

#### TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Topic</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Importance of the Topics and Populations Under Study</td>
<td>1-2</td>
</tr>
<tr>
<td>Pain</td>
<td>1-2</td>
</tr>
<tr>
<td>Pain as a Public Policy Issue</td>
<td>1-5</td>
</tr>
<tr>
<td>The Current Climate of Pain Research and Funding</td>
<td>1-8</td>
</tr>
<tr>
<td>Inadequate Training of Healthcare Professionals</td>
<td>1-11</td>
</tr>
<tr>
<td>Psychological Distress</td>
<td>1-15</td>
</tr>
<tr>
<td>Middle-Aged and Older Adults</td>
<td>1-17</td>
</tr>
<tr>
<td>Marriage as an Important Social Context for the Study of Health and Illness</td>
<td>1-21</td>
</tr>
<tr>
<td>Rationale for This Study</td>
<td>1-25</td>
</tr>
<tr>
<td>Why Study Pain and Depressive Symptoms in Midlife and Older Couples?</td>
<td>1-25</td>
</tr>
<tr>
<td>Why Study Pain and its Relationship to Depressive Symptoms in Midlife and Older Married Couples From a Social Work or Social Welfare Perspective?</td>
<td>1-31</td>
</tr>
<tr>
<td>Pain as a Social Work and Social Welfare Issue</td>
<td>1-31</td>
</tr>
<tr>
<td>The Social Work Response to Pain</td>
<td>1-33</td>
</tr>
<tr>
<td>Study Overview</td>
<td>1-37</td>
</tr>
<tr>
<td>Overview and Research Aims</td>
<td>1-37</td>
</tr>
<tr>
<td>Unique Features of This Study</td>
<td>1-38</td>
</tr>
<tr>
<td>Summary</td>
<td>1-40</td>
</tr>
</tbody>
</table>
CHAPTER 1: BACKGROUND AND SIGNIFICANCE

Who, except the gods, can live time through forever without any pain?
~ attributed to Aeschylus, Greek Dramatist (525 - 456 BC)

Pain pays the income of each precious thing.
~ William Shakespeare, The Rape of Lucrece (n.d.; line 334)

The least pain in our little finger gives [us] more concern and uneasiness than the destruction of millions of our fellow beings.
~ William Hazlitt, American Literature (1829)

Pain as God’s megaphone is a terrible instrument.
~ C. S. Lewis, The Problem of Pain (1940, p. 93)

Pain and its consequences have long been the subjects of philosophers, poets, novelists, and religious thinkers. Pain has the power to grab and hold our attention like few other sensations and experiences can. Moreover, prolonged pain, even if not particularly severe, has the capacity to negatively affect our mood, threaten our sense of well-being, and diminish our overall quality of life. Pain can also disrupt our interactions with others and interfere with our everyday lives.

Although it might be possible to distinguish several different types of pain (e.g., physical, emotional, spiritual), this study was concerned with physical pain, in particular, the physical pain experienced by persons in mid and later life. In addition to exploring their reports of pain and pain intensity, this study examined the relationship between pain and one of its most profound consequences—psychological distress. The relationship between pain and psychological distress was considered within a conceptual framework
that positioned pain within the larger context of health and physical disability, and outlined several possible pathways through which physical problems (such as pain) can impinge on psychological well-being. This framework was used to investigate the *intra-individual* effects of pain—that is, how an individual’s pain affects his/her level of psychological distress. Because the conceptual framework extended beyond the individual level to consider pain within the context of the marital relationship, it also guided the investigation of the *inter-individual* (or *cross-spouse*) effects of pain—that is, how pain in one spouse affects his/her partner’s level of psychological distress.

Specifically, then, this study examined pain within middle-aged and older married couples and the ways in which one spouse’s pain is related to his/her own psychological distress *and* the psychological distress of his/her partner.

This chapter addresses the social welfare significance of this study. Relevant background information is reviewed briefly, in order to provide a sense of the importance of the major topics and populations under study. Data supporting the relevance and timeliness of this study are then presented. Next, the general purpose of this study and its main research aims are described. Finally, this chapter highlights some of the unique features of this study, and provides an overview of subsequent chapters.

**Importance of the Topics and Populations Under Study**

*Pain*

Pain is most commonly defined as, “an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage” (International Association for the Study of Pain, 1979, p. S217; Merskey, 1991). More than 1 in 4 adults age 20 or over in the United States (26%) report having
experienced some type of pain that persisted for more than 24 hours during the past month (National Center for Health Statistics, 2006). Based on these data, pain is estimated to affect more Americans—from 76 million to 100 million adults—than diabetes, heart disease, stroke, and cancer combined (American Pain Foundation, 2007; Institute of Medicine [IOM], 2011).

At any one time in the U.S., an estimated 21.6 million persons are taking analgesics for pain (Meeks, 2000), and there are almost 200 million opioid prescriptions written each year (Kuehn, 2007). More than half (57%) of adults in the U.S. have experienced some period of recurrent or ongoing pain in the past year (Research America, 2003). More than four in ten adults (42%) report experiencing pain on a daily basis, and nine in ten (89%) report suffering from pain at least once a month (Arthritis Foundation, 2002). Moreover, there is consensus that most statistics likely underestimate the extent to which pain is a problem, as many people endure pain without seeking formal help or treatment (Marks et al., 2005).

Although pain may serve important evolutionary functions, such as signaling injury or danger, unrelenting or untreated pain can have deleterious consequences. Yet, despite important advances in medicine and pharmacology in recent decades, evidence suggests that pain remains poorly controlled. It is estimated that 4 out of every 10 people with moderate or severe pain do not get adequate relief (National Institutes of Health, 2001). Pain, especially when it is uncontrolled or under-controlled, is associated with negative effects on individuals, families, and society as a whole.

Pain can interfere with the performance of functional activities, sleep, immune functioning, and productivity. Pain can have a negative impact on an individual’s mood
and sense of well-being. Untreated pain is associated with anxiety and depression, and can interfere with one’s ability to enjoy life (e.g., Roy, Thomas, & Matas, 1984; Smedstad, Vaglum, Kvien, & Moum, 1995). Pain has been shown to be an independent risk factor for suicide—above and beyond its association with depression (Fishbain, 1999). Estimates suggest that as many as 50% of chronic pain patients with severe pain contemplate suicide (Marks et al., 2005). Pain can also have a profound effect on interpersonal relationships, contributing to interpersonal conflict and social isolation (e.g., Roy, Thomas, & Cook, 1996). The myriad negative effects of pain are illustrated in a quote from a single mother suffering from chronic pain induced by neck surgery,

Having this constant pain has taken away all of my independence, it has stripped me of my rights as an adult, as a human, as a woman, as a mother. It has made me have to ask for help that I otherwise would never ask for, it has made me have to wait and do things based on someone else’s time frame, it has made me become a child with an adult’s mind. (American Pain Foundation, 2007)

Pain has the capacity to affect all aspects of one’s quality of life. In fact, pain is imbued with such importance in this regard that questions about pain are included in almost all instruments to assess health-specific and general quality of life (Frytak, 2000). Such instruments are often used as measures of the effectiveness of medical and other healthcare services. Thus, freedom from pain is regarded as a critical factor in determining an individual’s overall quality of life and can serve as a key indicator of the success of a health care program or intervention.

Pain affects families, not just individuals. One study estimated that almost half of all American households (44 million or 43%) have at least one family member who suffers from some type of persistent or recurrent pain (Partners Against Pain [PAP], 2000). Family members of persons suffering from pain are likely to be negatively
affected by that pain. Pain in one family member has been found to result in decreased leisure participation, changes in responsibilities and roles, and changes in emotions and how emotions are expressed within the family (Turk, Flor, & Rudy, 1987).

At a societal level, the economic consequences of pain are enormous. A recent study commissioned by the National Institutes of Health (NIH) estimated that pain costs the U.S. $560 to $635 billion annually (expressed in 2010 dollars, Gaskin & Richard, 2011). The economic cost of pain exceeds the annual cost associated with heart disease ($309 billion), cancer ($243 billion), and diabetes ($188 billion; Gaskin & Richard, 2011). Pain is the most common reason for seeking medical attention in the United States. An estimated 80% of all physician visits in the U.S. are related to pain (Marks et al., 2005). Approximately 45% of Americans will seek medical attention for pain at some point in their lives (Meeks, 2000). Among those already receiving care, pain is associated with increased health care costs. For example, pain in hospitalized patients is associated with increased length of stay and poorer outcomes (NIH, 2003). Pain also exacts a heavy toll on workplace attendance and productivity. Data from the 2002 American Productivity Audit suggest that pain accounts for almost 80% of lost time in the workplace (Stewart, Ricci, Chee, Morganstein, & Lipton, 2003). There are also sizeable costs associated with disability compensation provided to persons who are unable to work because of pain.

**Pain as a Public Policy Issue**

Pain has achieved prominence within the national agenda only fairly recently. The prior decade (2000-2010) was designated the “Decade of Pain Control and Research,” representing only the second congressionally-declared, medically-related
decade in history (after the “Decade of the Brain”). This legislation, H.R. 3244, Title VI, Section 1603, was signed into law by President Clinton in October, 2000. Although it was successful in drawing attention to the need for greater investment in pain research, education, and management (Lippe, 2000), much work remains to be done.

Pain relief is increasingly recognized as a right for all persons, not just those in hospice or palliative care. However, unlike the Patients’ (or Consumers’) Bill of Rights and Responsibilities (President’s Advisory Commission on Consumer Protection and Quality in the Health Care Industry, 1997), there is no federal- or state-mandated patient Pain Care Bill of Rights. Policies and standards affecting pain assessment, management, and education remain fairly localized, and are often agency- and/or organization-specific. For the past 10 years, a number of professional health care groups and pain organizations have advocated for a National Pain Care Policy Act (introduced as H.R. 1863 in 2003). The proposed legislation would, among other things, establish pain care standards in all government-sponsored health care plans and systems, provide funding for professional education and training, and mandate a public awareness campaign on pain management (American Chronic Pain Association, 2004). The National Pain Care Policy Act was re-introduced in 2005, again in 2007, and yet again in 2009. The Act was passed in the House of Representatives (H.R. 756), but was stalled in the Senate (S. 660). Ultimately, several provisions from the proposed legislation—a mandated Institute of Medicine conference on medical and policy issues affecting the delivery of quality pain care, educational initiatives around pain and pain management for health care professionals, and an enhanced research agenda on pain for NIH—were incorporated into the Patient Protection and Affordable Care Act of 2010 (Public Law 111-148).
In the absence of a national policy with explicit standards for pain care, some states have enacted legislation to address the importance of pain management. For example, in 2001 the Medical Board of California mandated that practicing physicians complete continuing education on the topic of pain management as part of their license renewal process (Medical Board of California, 2001). Some large-scale healthcare systems have also adopted pain management guidelines. The concept of pain as “the 5th vital sign” (pioneered by the American Pain Society) has been adopted by the U.S. Department of Veterans Affairs (VA). Within VA health care facilities, pain is measured regularly, along with other vital signs such as temperature, pulse, respiratory rate, and blood pressure (U.S. Department of Veterans Affairs, 2001).

In 2000, the Joint Commission on Accreditation of Healthcare Organizations (JCAHCO) mandated new standards for pain assessment and management. These standards became part of the survey and accreditation process for hospital, ambulatory, long-term, and behavioral health care settings in 2001. A 2004 review suggested that although the standards appear to have improved pain assessment practices in many organizations, the impact on pain management practices has been less clear (Dahl & Gordon, 2004). The widespread proliferation of specialty pain clinics/centers in the U.S. can also be seen as an indicator of the importance of the pain problem and a possible signal of the failure of the traditional health care system to adequately address pain.

Pain has also received increased attention within the popular press. For example, pain has been a featured topic in both Newsweek (May 19, 2003) and Time (February 28, 2005). A five-part series on pain aired on NBC’s Today Show in March, 2005. Locally, in early 2012, the Cleveland/Northeast Ohio public television and radio stations (WVIZ/
PBS and 90.3), in conjunction with area healthcare institutions and support from local foundations, produced a multi-media program entitled, “Body in Pain.” The week-long program included a 1-hour documentary about chronic pain and multiple interviews, panel discussions, and educational resources that explored various aspects of pain, along with recent advances in research and treatment. Part of the increased attention may be the result of the controversy surrounding the risks and benefits of certain medications, including COX-2 inhibitors (e.g., Vioxx, Celebrex) and other pain relief compounds (e.g., Ibuprofen, Naproxin, Oxycontin). Increased media attention may also be a result of the public awareness efforts of various professional and non-profit pain organizations.

The Current Climate of Pain Research and Funding

Pain has been the subject of considerable scientific inquiry over the past 30 to 40 years. Several professional organizations dedicated to the study of pain were founded in the 1970s, and remain active today. Two prominent journals—Pain (published by the International Association for the Study of Pain) and the Journal of Pain (published by the American Pain Society)—provide outlets for the latest interdisciplinary research.

In the past decade, pain has received renewed attention within the national research infrastructure. In 2003, the National Institutes of Health (NIH) re-established the Trans-NIH Pain Consortium in order to help coordinate pain research being conducted in and/or funded by over 20 institutes and centers (National Institute of Dental and Craniofacial Research, 2003). The Consortium was also charged with increasing the visibility of pain research among professionals and the lay public. The NIH effort to revitalize the Pain Consortium signals that pain is worthy of study separate from any one disease or disorder (Porter, 2004). The Consortium has hosted a number of national
conferences on pain research—all of which make clear that pain is a topic of national importance and an area of research in need of increased investment. Under the auspices of the Consortium, the National Institute of Nursing Research (NINR) has begun offering a summer “Pain Methodologies Boot Camp,” designed to stimulate interest in and improve the quality of basic and applied (e.g., behavioral and social scientific) research on pain (NINR, 2011).

The problem in estimating research expenditures related to pain is that pain occurs within the context of numerous diseases and conditions, and its treatment cuts across so many disciplines that the boundaries of pain research are not defined by any institute, division, or agency. Analyses limited to research sponsored by NIH suggest that funding for pain-related research has increased steadily over the past decade. For example, funding for chronic pain conditions has grown from approximately $82 million in FY 1997 to roughly $224 million in FY 2007 (NIH, 2009). Unfortunately, estimates from more recent years are inflated as the result of economic stimulus funds associated with the American Recovery and Reinvestment Act of 2009 (IOM, 2011).

As of June 30, 2012, there were 30 currently active, distinct NIH funding opportunities that included pain or pain-related conditions as an objective or targeted topic of interest (NIH Pain Consortium, 2012). The majority of these relate only indirectly to pain—for example, they focus on a specific disorder in which pain is a prominent feature (e.g., arthritis, cancer), or they list pain as one of several symptoms to target in developing clinical interventions. Fewer than a dozen active funding announcements focus primarily on pain. Most of these address specific chronic pain conditions (e.g., migraine, temporomandibular joint pain, chronic pelvic pain). Several
others are devoted to enhancing the scientific basis of, or designing methodologies to evaluate, complementary and alternative therapies used to treat pain and other symptoms.

Four active NIH funding announcements have a substantive focus on pain that is not limited to a specific disease or chronic pain condition. One is a Neuroscience Research Grand Challenge that hopes to stimulate collaboration between pain researchers and non-pain neuroscientists in an effort to understand the transition from acute to chronic pain. Another is a recently-issued call from the National Institute of Alcohol Abuse and Alcoholism (NIAAA) for studies on the relationship between chronic pain and alcohol use and dependence (PA-11-267). The third announcement is the most broadly-written, and has been re-issued continuously since 2006—“Mechanisms, Models, Measurement, and Management in Pain Research” (PA 10-006). This announcement covers basic, applied, and translational research aimed at understanding pain from the molecular level to social and institutional levels. This grant program is primarily sponsored by the National Institute of Nursing Research, but is also co-sponsored by 10 other NIH institutes, including the National Institute on Aging (NIA). Advocacy efforts by NIA also led to a related, but separate program announcement to address the knowledge gap related to pain in older adults (PA-09-193, “Mechanisms, Measurement, and Management of Pain in Aging: From Molecular to Clinical”).

The Patient Protection and Affordable Care Act of 2010 established an Inter-agency Pain Research Coordinating Committee (IPRCC). This committee is charged with coordinating all research activities within the Department of Health and Human Services (including NIH, but also the Centers for Disease Control, Food and Drug Administration, etc.) and other federal agencies (e.g., Department of Veterans Affairs,
Department of Defense) related to the diagnosis, prevention, and treatment of pain and diseases and disorders associated with pain. After completing a review of research efforts to date, the committee is expected to make recommendations to help direct future pain-related research (IPRCC, 2012). Increased opportunities and funding for pain research are also expected to result from improved attention to patient outcomes through mechanisms such as the Patient-Centered Outcomes Research Institute (IOM, 2011).

**Inadequate Training of Healthcare Professionals**

Despite increased public awareness and increased attention from policy makers and researchers, there is ample evidence that healthcare professionals are inadequately prepared to address the needs of persons in pain. Most medical pain management in the U.S. is delivered within primary care settings. Yet, the majority of professionals in these settings feel unprepared to deliver such care. In a survey conducted with a nationally-representative sample of practicing physicians in the U. S. (stratified by specialty), 63% of physicians indicated that they had not been adequately trained to manage chronic pain (Darer, Hwang, Pham, Bass, & Anderson, 2004). Although one might expect better preparation among those affiliated with academia, given their familiarity with research evidence-based practice, this does not appear to be the case: Of 500 primary care physicians at 12 large academic centers, only 34% reported feeling comfortable treating people with chronic non-cancer pain (O’Rorke, Chen, Genao, Panda, & Cykert, 2007).

Studies suggest that instruction regarding pain and effective pain management is lacking throughout the course of training for the majority of physicians. A recent survey found that only four U. S. medical schools currently require a specific course on pain management (Mezei & Murinson, 2011). Analysis of data collected annually from
graduating medical students in the U. S. showed that the percentage interested in pain medicine as their preferred choice of specialty remained at 0.0% from 2006 through 2010 (AAMC, 2010). When surveyed upon graduation, most medical residents express confidence in their ability to manage most of the conditions they expect to encounter in practice; however, as a group, they feel less well-prepared to deliver certain types of care to persons with chronic conditions—especially pain management and palliative care (Blumenthal, Gokhale, Campbell, & Weissman, 2001). And, even though primary care physicians will serve on the front-line of pain care in the U. S., most primary care residencies offer little formal training in pain or pain management (Bair, 2008).

Although the majority of studies in this area have been done with medical students and physicians, there are selected examples from other disciplines, as well. For example, nurse practitioners and physician assistants working in community health clinics were surveyed about the adequacy of their training in several different topic areas. On a scale of 0 (inadequate) to 4 (excellent), the average rating given to their training in pain management was a disturbing 0.5 (Upshur et al., 2006).

In an attempt to locate data regarding the adequacy of social work training in pain and pain management, this author conducted searches in September, 2012 of three large publication indexes: PsychInfo, Social Work Abstracts, and PubMed. The following criteria were used: (pain, in abstract or title) AND (social work*, in abstract or title) AND (education OR curricul* OR training, in abstract or title). These searches returned 33 publications in PsychInfo, 19 in Social Work Abstracts, and 104 in PubMed. A review of abstracts reduced the number of potentially relevant publications to 45.

After removing 10 duplicates, the remaining 35 publications were reviewed in
greater detail. These publications spanned the period from 1981 to 2012. Most appeared in peer-reviewed journals, although two were doctoral dissertations. Although a majority focused on issues in the U.S., several publications originated in foreign countries (e.g., Canada, Greece, Japan). Roughly half of the publications (16) specifically discussed social worker education/training related to pain. However, most (11) were persuasive pieces in which practitioners argued for improved education and increased involvement of social workers in pain management and palliative care (e.g., Altilio & Otis-Green, 2005; Roy, 1986; Mendenhall, 2003). None of these 11 publications was empirically based, and few cited prior studies that were specific to social work education or training.

Only five empirical studies of social worker education/training in pain were identified. These studies were authored mainly by social work researchers in academia and were published from 1996 to 2009. Four of the five publications dealt with pain only in the context of end of life or palliative care. Three of these studies targeted oncology social workers (e.g., Korcz, 2003; Jones, 2005, and Zebrack, Walsh, Burg, Maramaldi, & Lim, 2008), and the other focused on social workers in hospice settings (Oliver, Wittenberg-Lyles, Washington, & Sehrawat, 2009). Only one study focused more broadly on medical social workers practicing in a range of healthcare settings, including hospitals, rehabilitation clinics, nursing homes, and home care agencies (Sieppert, 1996). Except for the study by Sieppert (1996)—which was conducted in Canada—all studies were conducted in the U.S. The studies by Jones (2005) and Zebrack et al. (2008) did not focus exclusively on pain or pain management; instead, these studies examined the roles and competencies of oncology social workers more generally.

Despite differences across studies with respect to survey methodology, sample
size, and responding social workers’ areas of specialization, practice settings, and patient populations, all five studies found evidence of serious deficits in social workers’ knowledge and skills related to pain and pain management. Among the 212 medical social workers surveyed by Sieppert (1996), the percentage of correct responses on a chronic pain knowledge inventory ranged from 8 to 76%, with a mean of 42%, a median of 44%, and only 7% of respondents answering over 60% of items correctly. Most notably, 90% of Sieppert’s respondents expressed a need for additional training in chronic pain management. Inadequate training in pain assessment was also a common problem reported in Korcz’s (2003) study of 185 oncology social workers.

Even though the 131 pediatric oncology social workers surveyed by Jones (2005) identified pain and symptom management as the most important need of the children and families that they served, they indicated that tasks related to pain and symptom management were the ones for which they felt least prepared. This finding was also echoed in Zebrack et al.’s (2008) survey of over 600 oncology social workers. Although the majority of Zebrack et al.’s respondents (80%) were MSWs and had an average of 11 years of experience (SD = 7.9), 34% indicated that they had little or no competence, and 46% indicated that they were only somewhat competent, in pain assessment and management. On average, the 90 hospice social workers surveyed by Oliver et al. (2009) reported devoting roughly one fifth of their time to pain management issues—particularly the pain-related concerns and questions of family caregivers. However, most felt that they could and should spend more time addressing pain-related issues. The authors concluded that “[hospice] social workers understand their role in pain management and struggle to find the time and tools needed to help them address caregiver concerns related
to pain management” (Oliver et al., 2009, p. 69). It is surprising that such deficits have been observed among those social work professionals who might be expected to display the greatest knowledge and highest levels of preparedness. Although additional research is warranted, these findings raise fundamental concerns about the ability of most social workers to effectively work with clients who have problems with pain.

Psychological Distress

One of the most notable consequences of pain is psychological distress. Despite being the subject of considerable theoretical and empirical attention, there is little consensus regarding the definition of, or the number of dimensions comprising, general subjective or psychological distress (Andrews & Robinson, 1991). Depression is regarded as one of the single best indicators of an individual’s current psychological health (Kessler et al., 2001). Hence, depression is often measured as an outcome variable in social science research focused on psychological distress.

The clinical syndrome of depression represents a constellation of psychiatric symptoms, with sad mood and loss of interest or pleasure in activities as defining characteristics (American Psychiatric Association, 1994). Many leading scholars believe that depression is probably more likely a spectrum of disorders, rather than a distinct, categorical disease (e.g., Lebowitz et al., 1997). From this perspective, depression represents an end point on a continuum that encompasses a range of affective states and symptoms from contentment to melancholia to despondency (Ingram & Siegle, 2002). Although the estimated prevalence of major depressive disorder among adults is fairly low—between 2-5% for current prevalence, and 15-20% for lifetime prevalence (Kessler et al., 1994; Kessler, 2002)—up to 20% of adults report significant depressive
symptomatology on diagnostic screening tools (Kessler, 2002). In the present study, depressive symptoms were used as indicators of psychological distress.

Although depressive symptomatology is just one possible consequence of pain, it is an important consequence to study. Depression is generally regarded as a significant social problem. It is one of the most common psychiatric disorders, and exerts a substantial impact on individuals and society. World Health Organization (WHO) data indicate that depression is currently the fourth leading cause of disability worldwide, and is projected to increase in impact in future years (Lopez, Mathers, Ezzati, Jamison, & Murray, 2006; Ustun, Ayuso-Mateos, Chatterji, Mathers, & Murray, 2004). Depression has been linked to increased risk for mortality and other, non-psychiatric illnesses and disabilities (Bruce et al., 1994; Lebowitz et al., 1997; Wulsin, Vaillant, & Wells, 1999). Depression is also associated with increased risk of suicide (Beutler, Clarkin, & Bongar, 2000). Depressive symptoms contribute to poor quality of life for individuals and impair an individual’s ability to function effectively in social and occupational roles. Depressive symptoms adversely affect the quality of relationships and contribute to conflict and dysfunction in marital and family interactions (Beach & Jones, 2002).

The economic consequences of depression are profound. In the U.S., conservative estimates of the effect of depression on lost productivity in the workplace exceed $33 billion per year (Greenberg, Kessler, Nells, Finkelstein, & Berndt, 1996). Depression is also associated with increased health care utilization and costs. For example, depressed persons, especially those with undiagnosed depression, often seek primary care medical services for vague somatic complaints (Katzelnick et al., 2000).

The personal, social, and economic costs associated with depression make it an
important topic of study. Because of these costs, depression is a prominent topic within the national research agenda. For example, depression was identified as a priority research area in the Center for Disease Control and Prevention’s (CDC) *Research Guide for 2006-2015* (CDC, 2007). Although research on depression and depressive symptomatology is needed across all stages of life, the *Research Guide* specifically mentions the need for research to identify risk factors for depression in older adults. The document also clearly identifies the need for research that continues to explore the complex relationship between physical and mental health.

Depressive symptoms are potentially modifiable and amenable to intervention. Today, numerous pharmacological and psychosocial interventions are available to treat depression. Research that helps to clarify the predictors of depressive symptomatology (e.g., pain) and the mechanisms through which such effects occur may ultimately help to reduce unnecessary emotional distress, as well as the interpersonal and societal burdens imposed by such distress.

*Middle-Aged and Older Adults*

This study focused on married couples in middle age and older adulthood. The ages that define the boundaries of middle adulthood or midlife are ambiguous. Lachman, Lewkowicz, Marcus, and Peng (1994) found that middle age was most commonly identified by laypersons as the period between the ages 40-60; however, some viewed middle-age as starting as young as age 30 and extending until the age of 75. Moreover, the perceived age of onset and end of middle age was positively correlated with the age of the respondent. Some notable scholars in the field of adult development including Schaie and Willis (1996) and Willis and Reid (1999) have defined middle adulthood as
the period between the ages of 35 and 64. These authors justified their categorization scheme on the basis that, by age 35, most people have negotiated key work and relationship tasks of youth (e.g., establishing a career or job, getting married); by age 65, most have reached the official retirement age in the U.S. Others (e.g., Brim, Ryff, & Kessler, 2004) cited similar cultural and social norms as the basis for their designation of midlife as the period from age 40 to age 60. Most scholars have acknowledged that the selection of specific ages to define the onset and end of middle age is arbitrary and that chronological age may be of limited value in defining midlife (e.g., Spiro, 2001; Lachman & Jones, 1997). The present study utilized data from individuals who were over age 50 at the time of data collection. Consistent with the definition of middle age offered by Willis and colleagues, this study refers to respondents between the ages of 51 and 64 as “midlife” or “middle-aged” adults.

Older adulthood is typically viewed as beginning at age 65, mainly owing to the fact that it was the official age of retirement prior to Social Security reforms implemented in the last decade. The older adults in this study encompassed a fairly large portion of the upper end of the human lifespan, ranging from individuals often regarded as the “young-old” (typically defined as ages 65-74) to those frequently labeled the “oldest-old” (typically age 85 and beyond; Spiro, 2001). In this study, the terms “older adults” and adults in “later life” are used to refer to respondents age 65 and older. Sometimes for the sake of convenience, all respondents—in either middle age or older adulthood—are referred to in this document broadly as adults in “later life” or “aging” adults.

There are a host of compelling demographic and social reasons to study persons in middle age and older adulthood. Currently, there are large numbers of persons in these
periods of life, and their numbers are increasing steadily with the aging of the “baby
boom” generation (generally defined as those born from 1946 to 1964). The age group
comprised of persons 50 to 54 grew by 55% during the last decade, making it the group
with the largest percent growth in the U.S. population during this period (Meyer, 2001).
The population of older persons (age 65+) in the U.S. has grown steadily since the turn of
the 20th century, swelling from 3 million in 1900 to almost 35 million in 2000.
Furthermore, the size of the older adult population is projected to double by the year 2050
(Federal Interagency Forum on Aging-Related Statistics [FIFARS], 2000). Much of this
growth will be due to the aging of the baby boomers, the oldest of whom began turning
60 in 2006 and reached the age of 65 in 2011.

Owing to the aging of the baby boomers, increased life expectancy, and stagnant
or declining fertility rates, the U.S. population as a whole is getting older. The median
age of the U.S. population was 29.4 years in 1960, and rose to 35.3 years in 2000 (U.S.
Census Bureau, 2001). In addition, the older population itself is also aging. Among
those over age 65, the proportion over age 75 has grown from 39% in 1980 to 47% in
2000, and is expected to exceed 50% by 2020 (Meyer, 2001). Within the population of
older persons, the fastest rate of growth is occurring in the 85+ age group (U.S. Census
Bureau, 2001). These demographic shifts have important implications for society.

There is ongoing debate about the extent to which increases in life expectancy and
reductions in mortality may result in more persons living longer with greater levels of
illness burden and disability. Although the available evidence suggests that age-specific
rates of frailty and disability are actually decreasing over time (e.g., Manton, 1997), the
aging of the population will most likely result in an increase in the absolute number of ill
and disabled older persons in society (Serow, 2001). This increase will place increased demands on both the formal and informal care networks. It also has important economic implications, as more resources will be needed to meet the acute, chronic, and long-term care needs of a larger and older population (Serow, 2001).

Demographic shifts in the population have, and will continue to have, a large impact on public policy. The growth in the number of older persons, and increases in the proportion of older persons in the population, will continue to affect policies regarding work, retirement, social services, and health care. It is these expected increases in the demands on public pension and health care systems that form the cornerstone of current debates about Social Security and Medicare and Medicaid reform efforts.

Given the large and growing number of middle-aged and older persons, and their current and expected impact on society, there is a need for research on the factors that undermine or limit health and well-being in middle and later life. Although research on old age is growing steadily, research on middle age remains relatively sparse (Brim, Ryff, & Kessler, 2004; Spiro, 2001). Yet, it is important to study midlife, because an individual’s health and functioning during these years may profoundly influence his or her quality of life in later years. People may also be especially motivated during these middle years to make changes that positively affect their health. This motivation may derive from an increased awareness of health, generally, as those in middle age, especially late middle age (after age 50), are confronted with increased mortality and morbidity of peers and family members, the emergence of many chronic and serious diseases, profound hormonal changes, and increased emphasis on health screenings in interactions with health care providers (Merluzzi & Nairn, 2004).
Marriage as an Important Social Context for the Study of Health and Illness

Social science has long recognized that social and cultural structures and processes influence the health and well-being of individuals. Historically, social scientists have studied the ways in which social relationships and sociocultural institutions (e.g., schools, churches, neighborhoods) and conditions affect, and are in turn affected by, individuals. Social work is also in line with this tradition, as it is fundamentally concerned with helping individuals, groups, and communities to resolve social problems and work toward social justice and optimal social functioning. Yet, empirical studies in most of the social sciences rarely move beyond the individual level of analysis. Much social science research remains focused on individuals; social and cultural factors are considered attributes of individuals, rather than properties of the larger system or context.

The Office of Behavioral and Social Science Research (OBSSR) at NIH has argued that in order to advance knowledge, social science research must focus more attention on social context—specifically on the social and interpersonal factors that influence health, the mechanisms through which such influence occurs, and the consequences of health and illness on the contexts in which individuals are embedded (OBSSR, 2001). Scholars working within different substantive areas of social science, including gerontology, child welfare, and criminal justice, to name just a few, have also become increasingly concerned with constructing (or revising) theoretical and conceptual models that incorporate different layers of the social context into explanations for problems at the individual and collective levels (see Institute of Medicine [IOM], 2001,
and NIH/National Research Council, 2001, for examples). Testing of more complex, multilayered conceptual models has been helped by the development and availability of specialized statistical software during the past two decades. Hence, researchers have empirical, theoretical, and methodological tools to help them adopt an increasingly multilayered approach to the study of health and well-being. Such an approach recognizes that the experience and course of individual health and well-being are influenced by multiple layers or levels of the social context.

From an intervention or practice standpoint, social contexts are important to understand because contexts themselves can be targeted for health-related interventions. Health promotion and prevention programs, for example, might be aimed at cities, neighborhoods, schools, workplaces, or families. Furthermore, interventions must be adapted to fit the context in which they are implemented (OBSSR, 2001). For example, a workplace intervention program will differ markedly from one designed for families.

Intimate partnerships, especially marriages, constitute unique social contexts. The marital unit forms the core of the family system and serves as the basic unit of human interaction. Despite a decline in the popularity of marriage in recent decades, households comprised of married couples (both with and without children) still constitute a sizeable proportion (48%) of all households and a majority (73%) of all family households (i.e., households comprised of persons related to each other by birth, marriage, or adoption) in the U.S. (Lofquist, Lugaila, O’Connell, & Feliz, 2012). However, when data for the present study were collected, married couple households accounted for the majority (52%) of all households and an even greater proportion (77%) of all family households (Fields & Casper, 2001). Thus, the marital relationship constitutes an important social
context for a large proportion of the population.

The social environment provided by the marital relationship has particular salience for the health and well-being of the individual spouses. A wealth of research has established that being married has consistently positive effects on physical health; further, these effects do not simply reflect biases due to the selection into marriage (e.g., Lillard & Waite, 1995). Research has also established that marriage has generally positive effects on emotional or psychological well-being (e.g., Marks & Lambert, 1998). The benefits of marriage for health and well-being may be due to any number of reasons, including increased economic resources, the availability of social support, the provision of emotional intimacy, and the promotion of healthy behaviors (Hughes & Waite, 2002).

By the same token, married individuals may also be subjected to the potentially negative effects of marriage on health and well-being. First, married individuals may share a lifestyle or an environment that places them at increased risk for certain health conditions. For example, unhealthy eating behaviors, such as overconsumption and emotional eating, have been found to be significantly correlated between spouses and are related to the development of obesity (Provencher, Perusse, Bouchard, Drapeau, Bouchard, Rice et al., 2005). Second, individuals in marriages characterized by conflict, dysfunction, or abuse may have reduced well-being attributable to the interpersonal marital environment (Beach & O’Leary, 1986; Whisman & Beach, 2001). Third, and most importantly, studies have shown that the poor health and/or reduced well-being of one spouse can negatively affect the health and/or well-being of the other spouse. For example, spousal caregivers of persons with Alzheimer’s disease or other disabilities often report poorer physical and emotional well-being (e.g., Biegel, Sales, & Schulz,
The positive and negative effects of one spouse’s characteristics or behaviors on his/her partner’s health or well-being can be either direct or indirect. A direct effect might be observed when a health condition or event in one spouse directly affects the well-being of his/her partner. In one study for example, a significant number of wives whose husbands had suffered a recent heart attack displayed a high level of psychological distress immediately following the event, and many continued to display symptoms of distress for up to one year afterward (Ebbesen, Guyatt, McCartney, & Oldridge, 1990).

Alternatively, effects can be largely indirect. An indirect effect might be observed when one spouse’s health-related behavior change prompts a behavior change in his/her partner, which results in a change in the partner’s overall health. In this instance, one spouse’s behavior change has an effect on the other partner’s health through its effect on the partner’s own health-related behavior. For instance, some studies have shown that when one spouse makes a positive health change, like quitting smoking, increasing exercise, or getting a flu shot, this is likely to spur his/her partner into doing the same, often resulting in positive changes in the health and well-being of the partner (e.g., Falba & Sindelar, 2007).

The behavior or characteristics of one partner could potentially have both direct and indirect effects on the other spouse. For example, attempts by a wife to regulate a husband’s diet may influence his health directly by promoting consumption of more healthy foods and perhaps also indirectly by increasing his feelings of responsibility and obligation toward his wife, thereby prompting him to exercise after work. Of course,
scholars studying such health monitoring (e.g., Rook, 1995) or health-related social control (e.g., Tucker, 2002) note that the wife’s efforts could potentially have very negative effects on her husband’s health-related behavior and overall well-being. Regardless of the specific nature or direction of effects, these examples make clear that marriage is a social context that has significant implications for research on the health and well-being of the individual partners. Yet, many observe that the influences of such social contexts on the health and well-being of those in mid and later life have been relatively understudied (e.g., Hughes & Waite, 2002).

Rationale for This Study

Why Study Pain and Depressive Symptoms in Midlife and Older Couples?

Earlier sections of this chapter established that pain, psychological distress (viz., depressive symptomatology), midlife and older adults, and the marital context are all topics or populations of importance in contemporary society. Each of these topics and populations was central to the current study: An examination of pain and the relationship between pain and depressive symptomatology in middle-aged and older married couples. Literature specific to each topic and population is reviewed in detail in Chapters 2 and 3. The current section outlines five primary reasons for studying pain and its relationship to depressive symptomatology in married couples in midlife and old age.

First, pain has been relatively understudied in middle-aged and older persons. Pain in general has been subject to considerable research since the early 1970s. Yet, relatively little research has been devoted to the prevalence and consequences of pain in midlife and older persons. A review by Farrell (1995) found that less than 1% of published papers on pain focused on the aging population. Although the situation has
improved somewhat since that review, scholars in this area routinely lament the relative paucity of research on the pain experiences of middle-aged and older adults (e.g., American Geriatrics Society [AGS], 2002; Gibson & Weiner, 2005; Hadjistavropoulos et al., 2007; Reid et al., 2011). Indeed, a more recent analysis found that out of roughly 23,000 pain-related publications in 2007 indexed in Medline, only 641 (or 2.8%) were relevant to aging (Gagliese, 2009).

Pleasures are always children,  
pains always have wrinkles.  
~ Joseph Joubert, Notebooks (1796)

Second, available evidence substantiates Joubert’s observation that pain is often aging’s companion. Most epidemiologic studies suggest that the overall prevalence of pain peaks in middle age, and then decreases thereafter (Leveille et al., 2001). However, the prevalence rates for specific types of pain often vary by age. For example, migraine (Crook, Evans, & Funkenstein, 1989) and low back pain (Wright, Barrow, Fisher, Horsley, & Jayson, 1995) have been found to peak in middle age, while musculoskeletal pain and pain in the extremities have been found to peak in later life (e.g., Brattberg, Parker, & Thorslund, 1997). Age is clearly a risk factor for many of the illnesses and conditions that can be associated with pain, including arthritis (osteoarthritis, rheumatoid arthritis), circulatory diseases, and malignancies (Helme, 1999).

Significant numbers of community-dwelling older persons suffer from pain. Epidemiologic studies suggest that anywhere from 36-86% of community-dwelling older persons have pain-related problems (Miller & Talerico, 2002). For many older persons, pain is a routine part of life. For example, 52% of community-residing Medicare beneficiaries reported experiencing pain on a daily basis (Sawyer, Bodner, Ritchie, &
In a national poll, 46% of adults age 65 and older indicated that they suffered from significant pain at least several times a week (Clarus Research Group, 2009). Estimates vary widely due to differences in sampling and definitions of pain. Ancillary data also suggest that pain is common among aging persons. Among community-dwelling adults in the U.S., use of prescription analgesics (including prescription-strength non-steroidal anti-inflammatory drugs [NSAIDs] and opiate-derivatives) has been shown to increase steadily with age. Additionally, women age 75 and older have the greatest prevalence of opiate analgesic use (7%)—more than twice the average rate for all adults (3.4%; Paulose-Rose et al., 2003).

Pain prevalence rates specific to those in middle-age are lacking in most of the literature, as middle-aged persons are usually just incorporated into estimates for “adults.” Most epidemiological estimates put the prevalence rates for any pain among community-dwelling adults of all ages anywhere between 60 and 89% (Arthritis Foundation, 2000; Ruehlman, Karoly, Newton, & Aiken, 2005). Thus, pain appears to affect anywhere from a sizeable minority to a majority of midlife and older persons living in the community—clearly, these are significant numbers when extended to the population level. Moreover, when the overall aging of the U.S. population is considered, the projected numbers of midlife and older Americans likely to be affected by pain become even more sizeable.

Third, numerous studies link pain to depressive symptoms in midlife and older adults (e.g., Herr, Mobily, & Smith, 1993; Peat & Thomas, 2009; Reyes-Gibby, Aday, & Todd, 2007; Weiner et al., 2003); however, the mechanisms through which pain affects depressive symptomatology are not well understood. Very often, pain and its relationship
to psychological distress are studied in isolation from the broader context of the health of the individual. Models of health and disability (e.g., Pope & Tarlov, 1991; Verbrugge & Jette, 1994) suggest that declines in various aspects of health can create pathways to physical impairments and disabilities, which can in turn compromise psychological well-being. Yet, there have been few attempts to apply such models to the study of pain and psychological distress in the general population, much less the population of older adults. The extent to which there may be differences in such pathways or processes related to gender or other demographic and background variables is also unknown.

The relationship between pain and depressive symptoms is especially important to study among middle-aged and older persons. Depression is one of the more common psychiatric conditions affecting persons in mid and later life, and is associated with many negative consequences. The World Health Organization ranked depression as the single most burdensome chronic condition in the world among people in the middle years of life (Murray & Lopez, 1996). Unfortunately, depression remains under-recognized and largely undertreated in older persons (Blazer, 2003; NIH Consensus Panel on Depression in Late Life, 1992). In older adults, depression is associated with increased health care costs, and increased morbidity and mortality from medical illness and suicide (Schneider & Olin, 1995). Research that helps to clarify the link between pain and depressive symptomatology may help to reduce unnecessary psychological distress and related consequences for those in later life.

*Fourth, pain occurs in a social context and affects families, not just individuals.* According to NIH (2003), the social context of pain has received minimal attention from researchers. Yet, estimates suggest that almost half of all U.S. households (44 million)
have a family member who suffers from recurrent pain (PAP, 2000). Studies have also shown that others in the family can be negatively affected by one member’s pain. Spouses seem especially vulnerable, with numerous studies documenting elevated levels of psychological distress (including depressive symptoms) in spouses of persons with pain (e.g., Flor, Turk, & Scholz, 1987; Romano et al., 1989).

In addition to having a direct effect on the emotions of the spouse, pain can lead to changes in couple and family functioning that can be a source of stress and negative emotions for both members of the marital dyad (e.g., Payne & Norfleet, 1986; Roy, 1988). There is also evidence that a spouse’s reaction to a partner’s expression of pain can have a powerful influence on how the partner experiences pain, his/her future expressions of pain, and his/her emotional and functional well-being (e.g., Roy, 2001). Unfortunately, most studies of pain within the context of marriage have been limited to chronic pain patients and their spouses—a population dominated by younger couples with an identified “patient” seeking treatment at a specialty pain clinic.

Yet, many persons in late middle age and older adulthood who experience pain will do so within the context of a marital relationship. The marital relationship is one of the most important and common social contexts for aging persons. Sizeable majorities of persons in both mid and later life are married and reside with their spouse (67% of those age 55-64, and 54% of those age 65 and over; U.S. Census Bureau, 2010). When data for the present study were collected, these percentages were somewhat higher (70% of those age 55-64, and 55% of those age 65 and over; Fields & Casper, 2001).

Changing patterns of longevity and mortality have resulted in more couples surviving into old age (Hagestad, 1988). Within marriages of extended duration, there is
increased need for accommodation and adjustment to changes that occur both within and outside of the family (Riley, 1983). With increased age comes increased risk for illness and physical disabilities. So, aging couples must often confront the challenges associated with the chronic health conditions of one partner or both partners. These challenges can be stressful and lead to conflict and distress in the couple. Indeed, the illness of a spouse is a common reason why older couples enter into marital therapy (Meunier, 1994).

Given the prevalence of pain among middle-aged and older persons, as well as the demographic likelihood that these persons will be in long-term, committed relationships, the scarcity of research on the experience of pain within midlife and older couples is somewhat startling. There is a need to extend the research conducted with couples sampled from chronic pain clinics to couples living with pain in the community, as well as those who are in their middle and later years. Unanswered empirical questions include whether pain in one spouse in these middle-aged and older couples has an effect on the psychological distress of his/her partner and, if so, what are the potential mechanisms through which this occurs. It also is important to explore the extent to which these mechanisms might differ by characteristics such as gender, age, and length of marriage.

Lastly, the limited research on pain in married couples has generally ignored the possibility that each member of the dyad could be suffering from pain. Evidence has established that, at any given time, pain can be experienced by more than one household member. The Partners Against Pain study estimated that 15 million U.S. households have two or more family members who suffer recurrent pain (PAP, 2000).

Given the extent to which pain is commonly experienced by those in middle and older adulthood, it is reasonable to assume that many of these multiple-pain-sufferer
households are comprised of aging spouses. To date, no published study has specifically examined aging couples in which both partners experience pain. Such couples constitute a unique social context in which to examine the impact of each spouse’s own pain, as well as the pain experienced by his/her partner, on the depressive symptomatology of both spouses.

*Why Study Pain and its Relationship to Depressive Symptoms in Midlife and Older Married Couples From a Social Work or Social Welfare Perspective?*

**Pain as a Social Work and Social Welfare Issue**

In addition to prevalence data suggesting that pain is common among aging persons, the demographic imperative associated with the burgeoning populations of middle-aged and older persons, and evidence of the personal and societal costs associated with pain, there are additional reasons why pain (and its relationship to depressive symptomatology) should be considered a critical social welfare issue. Pain—like several other health conditions—is subject to social stratification. Women tend to be overrepresented within certain pain conditions, such as fibromyalgia and temporomandibular joint disorders (NIH, 2001). A number of studies also point to serious disparities in the distribution of pain across racial and ethnic groups. For example, higher rates of self-reported pain have been observed among members of certain racial groups, such as African Americans and those of Hispanic origin (e.g., Green et al., 2003). And, as previously noted, many types of pain are also more common in later life.

Good pain assessment and management requires specialized education and training of all health care providers. However, most health care professionals receive little, if any, specific training in the assessment or treatment of pain (Gloth, 2004; NIH,
Consequently, pain often is overlooked or inadequately assessed by health care providers. Poor pain assessment is common even in populations known to experience high levels of pain, such as cancer patients (e.g., Zaza & Bain, 2002). In addition, there are documented racial/ethnic (e.g., see review by Cintron & Morrison, 2006), gender (see review by LeResche, 2011), and age-related (e.g., Herr et al., 2004) disparities in the extent to which patients are queried about their pain.

Available evidence further suggests that pain is frequently untreated or undertreated. Unfortunately, this circumstance disproportionately affects the most vulnerable and underserved populations, including older adults, women, racial and ethnic minorities, and persons with low socioeconomic status (e.g., Cintron & Morrison, 2006; Institute of Medicine, 2002; Unruh, 1996). Several studies have shown that age itself is a risk factor for the under-treatment of pain (Bernabei et al., 1998; Cleeland et al., 1994; Jones, Johnson, & McNinch, 1996; Morrison & Siu, 2000). The study by Jones et al. (1996), for example, showed that older adults with long-bone fractures were less likely to receive appropriate pain medication than were their younger counterparts with similar injuries.

Some vulnerable populations also lack access to appropriate pain medication. For example, pharmacies located in low income, urban neighborhoods often do not stock commonly-prescribed pain medications (Morrison, Wallenstein, Natale, Senzel, & Huang, 2000). In addition, although most public and private health care insurance plans (including Medicare and Medicaid) will cover pain treatment associated with an acute episode or hospitalization, the treatment of chronic pain is generally not covered (American Chronic Pain Association, 2004). Thus, much of the health care cost associated with persistent pain is borne by individuals, placing a heavy burden on those
It is unclear whether disparities in the experience, assessment, and treatment of pain exist because of structural and institutional forces, such as health care policies, practices, or biases, or because of genetic, biological, or sociocultural differences in pain sensitivity and expression. It is clear, however, that personal and cultural attitudes and beliefs of both the individuals experiencing pain and those treating them also play a role in the lack of adequate pain assessment and treatment. Some individuals may be reluctant to admit pain, believing that it shows personal weakness. Others may simply accept that they must learn to live with a certain amount of pain (Ferrell, 1995). Finally, fear of dependence and addiction among patients, families, and professionals can also serve as powerful barriers to adequate pain relief. Such barriers may be especially pronounced in certain vulnerable populations.

The widespread prevalence, pervasive under-treatment, and myriad individual and societal consequences of pain position it as a public health and social problem of major significance. In an interview for the *NASW News*, NASW (National Association of Social Workers) Executive Director Elizabeth Clark identified pain as a “social-justice issue with a major role for social workers.” (O’Neill, Sept. 2003, p. 4)

**The Social Work Response to Pain**

As a profession, social work has done little to respond to the problem of pain. In 2003, O’Neill observed that virtually nothing on pain was available within curricula in schools of social work and that there was a lack of continuing education directed to social workers on the topic of pain (O’Neill, 2003). The situation appears to have changed little in the past decade (see Chapter 6 for details).
Few published empirical studies of pain—even those addressing health disparities in pain or the effects of pain on various social contexts—have been authored by social work scholars. One notable exception is Ranjan Roy, a social work researcher at the University of Manitoba. For decades, Roy has researched and written about the impact of chronic pain on the family (especially spouses) and the epidemiology of chronic pain in various age groups, including older adults. Several social workers have also achieved prominence for their clinical work, advocacy, and education efforts related to pain management. For example, Terry Altilio, ACSW (Coordinator of Social Work for the Department of Pain Medicine and Palliative Care, Beth Israel Medical Center, New York City) is regarded as an international leader in palliative care. Yvette Colon, PhD, ACSW (Director of Education and Internet Services, American Pain Foundation, Baltimore, Maryland) is a noted lecturer and clinician in oncology social work and end-of-life care. For the most part, however, social work is noticeably underrepresented within national and international dialogues on pain.

Yet, opportunities for social work abound. As O’Neill (2003) pointed out, “no profession has claimed in a major way the psychosocial aspects of pain management” (p. 4). Because of the key roles that social workers play within healthcare and social service systems, they can expect to encounter individuals in pain. There are numerous ways in which social workers can help to address the psychosocial consequences of pain and assist in pain management. These include educating individuals and families about pain and pain management, advocating on behalf of underserved populations for improved pain assessment and treatment, and helping to design and deliver non-pharmacological and psychotherapeutic interventions for those affected by pain. Several authors have
called attention to the lack of psychosocial interventions developed and/or validated for use with older persons suffering from pain (e.g., Park & Hughes, 2012; Sorkin & Turk, 1995). Surely, social work could play an instrumental role in developing and testing interventions for this population. Helping people to adapt to their life circumstances is a key task in social work practice, and their assistance may be welcomed by aging persons adapting to the changes wrought by pain and illness in themselves and their families.

In addition to facilitating access to healthcare services, social workers can be instrumental in referring patients and families to appropriate self-help resources, encouraging exercise and other self-care strategies, and addressing noncompliance with medical and self-care regimens (Altilio, 2007; Subramanian & Rose, 1988). Given their training in advocacy and strategies to affect social change, social workers are ideally-suited to help draw attention to needed changes to pain-related policies and practices within institutions and healthcare systems. Social workers’ commitments to social justice and a strengths-based, person-centered approach to care mean that they can offer a valuable perspective to multidisciplinary teams caring for persons and families confronting pain (Mendenhall, 2003).

Pain does feature prominently in the standards for professional social work practice in end-of-life and palliative care (NASW, 2004). These standards direct social workers to develop knowledge and understanding of the “physical, psychological, and spiritual manifestations of pain” (p. 18) and skills in assessing “the impact of problems such as pain…” (p. 19). They also demand that social workers acquire competence in delivering a range of psychosocial interventions that can help to alleviate pain and discomfort at end of life. It is unfortunate that social work’s role in helping individuals
with pain has received formal attention only within the context of care for the dying. Clearly, social workers’ knowledge and skills in advocacy, resource identification, system navigation, individual and family counseling, and the delivery of psychosocial interventions could benefit persons suffering from pain and its sequelae throughout the life course. Altilio has written extensively about the “landscape of opportunity” for social work involvement in the management of both acute and chronic pain in persons of all ages (e.g., Altilio, 2007, p. 44).

Social workers bring a unique, holistic perspective to the study of compelling social problems, mainly because of the “person in environment” perspective that tends to define social work practice (Barker, 2003; Kondrat, 2008). When studying issues related to health and illness, social workers are likely to embrace a multidimensional, biopsychosocial perspective; hence, the contributions of family, sociocultural, and environmental factors are often featured in research emanating from a social work or social welfare perspective. Social work researchers have a long history in the study of mental illness, including the study of depression and depressive symptomatology; they have made notable contributions to the study of health and healthcare, as well (Berkman & Harootyan, 2003). Social work has also achieved prominence within the gerontological research community (Gardner & Zodikoff, 2003). Social workers are thus highly qualified to study pain and its relationship to psychological distress within the context of the aging marital dyad. Social workers also bring unique values and perspectives to the research enterprise that may facilitate the conduct of translational research in this area. Having a professional duty to enhance individual and societal well-being may prompt social work researchers to design studies with a strong potential to inform clinical
practice. In addition, social workers’ commitment to social justice may foster more widespread dissemination of study findings and increased efforts to use empirical findings to guide the development of relevant public policy.

Study Overview

Overview and Research Aims

The overarching goal of this investigation was to examine pain and its relationship to psychological distress in middle-aged and older married couples. This was accomplished through a secondary analysis of a large, national survey of community-dwelling persons in mid and later life—the Health and Retirement Study (HRS). Although HRS was not designed as a study of married couples, investigators capitalized on the presence of spouses and partners within household sampling units in order to increase sample size. Thus, in households in which a respondent reported that he/she was married (or living as married), an attempt was made to obtain an interview with his/her spouse or partner. Couple-level participation rates were high and data from these couples provided a unique context in which to study pain and its relationship to psychological distress. This study had four general aims:

1. To examine reports of pain in a national sample of community-dwelling, middle-aged and older married couples, and to compare the characteristics of couples in which both spouses reported pain to those of couples in which only one, or neither, spouse reported pain. All subsequent aims were specific to these “dual-pain” couples.

2. At the intra-individual level, to determine (a) the demographic and health-related characteristics associated with reports of pain in these spouses; (b) the
relationship between each spouse’s pain and his/her own psychological distress; and (c) whether this relationship is mediated by activity limitation.

3. At the inter-individual level, to investigate (a) the covariation between spouses’ reports of pain, activity limitation, and psychological distress; (b) the relationship between each spouse’s pain and the psychological distress of his/her partner (i.e., cross-spouse effect); and (c) whether this cross-spouse relationship is mediated by the spouse’s activity limitation.

4. To evaluate whether the measured levels of, or the relationships between or among, key study constructs (i.e., pain, activity limitation, psychological distress) vary by gender.

**Unique Features of This Study**

Several unique features position this study to make a contribution to the existing literature. First, this study focused on a unique, and heretofore relatively invisible, population—those midlife and older married couples in which both partners report experiencing pain. Second, these couples were drawn from a diverse, national sample of married, middle-aged and older adults. Given that pain has been less well-studied in older populations, this study can contribute to the existing body of knowledge regarding the pain experienced by aging individuals. As the study sample consisted of community-dwelling midlife and older adults, it may also provide important information regarding pain as it more naturally occurs in real-world settings.

Pain and depression are most often studied within the context of a medical model, and one which emphasizes the individual. However, this study adopted a more interpersonal, social-contextual perspective. Specifically, this study considered both the
intra-individual and inter-individual (i.e., cross-spouse) effects of pain on psychological distress (specifically depressive symptoms). By utilizing data drawn from married dyads in which both partners reported pain, this study was able to examine the impact of each partner’s own pain, as well as the pain experienced by his/her spouse, on the depressive symptomatology of both partners. Finally, this study examined both the direct effects of pain and possible indirect (i.e., mediated) effects of pain on psychological distress, thereby replicating and extending previous research.

According to the most recent NIH (2009) general funding announcement on pain research (PA-10-006), even basic epidemiologic and descriptive information about pain remains under-developed. This study sought to contribute knowledge relevant to several areas outlined in that announcement: pain in special populations, including the elderly; the interplay of physical, behavioral, social (and familial), and environmental factors in the incidence, prevalence, and correlates of pain; demographic and other risk factors associated with the occurrence and maintenance of pain; the impact of pain on an individual’s physical and mental health; and health disparities in the incidence, severity, and consequences of pain (PA-10-006, Research Objectives). The 2009 NIH funding announcement specific to aging (PA-09-193) identified numerous gaps in research on pain in older adults. Those relevant to the current study include improved estimates of the prevalence of pain, especially with respect to age-related diseases or conditions; the impact of co-morbid disease conditions on pain in older adults; social and demographic factors associated with disparities in pain among the aged; the effect of pain on physical, psychological, and social functioning in older persons; and improved understanding of the behavioral, psychological, and social responses to pain in order to better target
interventions for older adults (PA-09-193, Research Objectives). The aims of this study were thus closely aligned with several national priorities in pain research.

Summary

In summary, this study addressed two topics of fundamental importance to social welfare—pain and psychological distress. Furthermore, this investigation was conducted within populations that have been significantly understudied—middle-aged and older married couples. As outlined above, the study of pain and psychological distress (specifically depressive symptomatology) in midlife and older married couples is both relevant and timely. Moreover, study aims fit closely with several priorities outlined within the national agenda for pain research.

The next two chapters—Chapters 2, 3—present the theoretical and conceptual frameworks guiding this study and review the existing literature. Chapter 2 reviews what is known about pain and its relationship to depressive symptomatology in midlife and older adults at the intra-individual level. Chapter 3 synthesizes information about pain and its relationship to depressive symptomatology within aging married couples (i.e., at the inter-individual level). Specific research questions and hypotheses are presented at the end of each of these chapters. Chapter 4 describes the methods employed in this investigation and provides an overview of the data analysis strategy. The results of these analyses are presented in Chapter 5. Finally, Chapter 6 discusses the main findings from this study and offers specific directions for social work practice, education, and public policy. The limitations of the study are also reviewed in Chapter 6, along with suggestions for enhancing existing theory and developing future research related to the experience of pain within couples in mid and later life.
# CHAPTER 2:
PAIN AND PSYCHOLOGICAL DISTRESS AT THE INTRA-INDIVIDUAL LEVEL IN MIDDLE-AGED AND OLDER ADULTS

## TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Background Information About Pain</td>
<td>2-1</td>
</tr>
<tr>
<td>Definitions and Terminology</td>
<td>2-1</td>
</tr>
<tr>
<td>Pain Terminology</td>
<td>2-1</td>
</tr>
<tr>
<td>Pain as Conceptualized in This Study</td>
<td>2-7</td>
</tr>
<tr>
<td>Pain Prevalence in Middle-Aged and Older Persons</td>
<td>2-9</td>
</tr>
<tr>
<td>How Common Are Reports of Pain in General?</td>
<td>2-10</td>
</tr>
<tr>
<td>How Common Are Reports of Chronic or Persistent Pain?</td>
<td>2-11</td>
</tr>
<tr>
<td>Background Information About Psychological Distress</td>
<td>2-14</td>
</tr>
<tr>
<td>Definitions and Terminology</td>
<td>2-14</td>
</tr>
<tr>
<td>Prevalence of Depressive Symptomatology in Middle-Aged and Older Adults</td>
<td>2-17</td>
</tr>
<tr>
<td>Correlates of Depressive Symptomatology in Mid and Later Life</td>
<td>2-19</td>
</tr>
<tr>
<td>Depressive Symptomatology as Conceptualized in This Study</td>
<td>2-23</td>
</tr>
<tr>
<td>The Relationship Between Pain and Psychological Distress</td>
<td>2-24</td>
</tr>
<tr>
<td>Evidence of a Relationship</td>
<td>2-24</td>
</tr>
<tr>
<td>Conceptual Issues in Studying This Relationship</td>
<td>2-27</td>
</tr>
<tr>
<td>Theoretical Models for Studying the Relationship Between Pain and Psychological Distress</td>
<td>2-40</td>
</tr>
<tr>
<td>Dominant Theories of Pain and of Depressive Symptomatology</td>
<td>2-40</td>
</tr>
<tr>
<td>Theoretical Foundations for a Proposed Conceptual Model</td>
<td>2-41</td>
</tr>
<tr>
<td>The Activity Restriction Model of Depressed Affect</td>
<td>2-42</td>
</tr>
</tbody>
</table>
Similar Concepts in the Chronic Pain Literature ....................... 2-43

Limitations of the ARM ............................................................. 2-45

Models of Disability and the Disablement Process .................... 2-48

Pain in the Illness-Disability Cascade ........................................ 2-52

An Extension of the Kahana et al. (1997) Model ................. 2-57

Proposed Conceptual Model Linking Pain and PsychologicalDistress
at the Intra-Individual Level ............................................................. 2-59

Review of the Literature Specific to the Proposed Conceptual Model ............ 2-69

Factors Related to Pain and Pain Intensity ................................... 2-72

Background Characteristics ............................................................... 2-72

Gender ........................................................................................ 2-72

Age ................................................................................................ 2-85

Race and Ethnicity .................................................................. 2-90

Health-Related Characteristics ....................................................... 2-104

Disease Conditions ................................................................. 2-104

Body Weight ............................................................................ 2-108

Summary: Factors Related to Pain and Pain Intensity .................... 2-114

Major Pathways in the Proposed Conceptual Model ...................... 2-118

Review of Paths A, B, and C .......................................................... 2-119

Path A: The Relationship Between Pain and Depressive
Symptomatology ......................................................................... 2-120

Path B: The Relationship Between Pain and Activity
Limitation ...................................................................................... 2-131

Path C: The Relationship Between Activity Limitation
and Depressive Symptomatology ................................................ 2-138

Summary of Evidence for Paths A, B, and C .............................. 2-142
CHAPTER 2: PAIN AND PSYCHOLOGICAL DISTRESS AT THE INTRA-INDIVIDUAL LEVEL IN MIDDLE-AGED AND OLDER ADULTS

This chapter introduces the theoretical and conceptual frameworks that helped to guide the current study. It also provides a review of the literature with respect to pain and psychological distress in middle-aged and older adults. The focus in this chapter is on pain and its relationship to psychological distress at the intra-individual level. The subsequent chapter will address pain and psychological distress at the inter-individual or couple level (i.e., within the marital dyad in later life).

This chapter begins by providing some background information concerning pain terminology and reviewing prevalence data for pain in the populations of interest. Next, the terminology and prevalence of psychological distress, in particular depressive symptomatology, in middle aged and older adults is reviewed. This is followed by an overview of the literature linking pain and psychological distress. Theoretical frameworks that depict the relationship between pain and psychological distress and that position pain within a cascade of physical disability are presented, followed by an integrated conceptual model that was developed to guide the current study. The relevant empirical literature is then reviewed systematically, with an eye toward identifying gaps in existing knowledge. Finally, research questions and hypotheses concerning pain and its relationship to psychological distress at the intra-individual level are presented.

Background Information About Pain

Definitions and Terminology

Pain Terminology

Despite having been the subject of decades of research, “pain remains a poorly
understood, highly complex, and less well-studied phenomenon” (Ibrahim, Burant, Mercer, Siminoff, & Kwoh, 2003, p. 472). There are, of course, problems in studying pain. Because of its inherent subjectivity, pain can be assessed only indirectly. Pain assessment is highly dependent on an individual’s ability to communicate (either verbally or non-verbally) something about his/her subjective experience of pain. Research also suggests that the sensation of pain must go through many layers of internal processing before it may be recognized, reported, and/or expressed. Most pain scholars believe that the perception and expression of pain are influenced by a complex interplay of sensory, motivational, cognitive, affective, and behavioral factors (Yehuda & Carasso, 1997).

Pain Characteristics or Dimensions

The definition of pain offered in Chapter 1, “An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage” (International Association for the Study of Pain, 1979, p. S217; Merskey, 1991), makes it clear that pain has both sensory and emotional/affective dimensions. The sensory dimension concerns the sensation of pain, or how pain is sensed, felt, or experienced in the body. The emotional or affective dimension of pain is more difficult to explicitly define, but it is known commonsensically to all those who have experienced it. As Aydede (1995) put it most eloquently and briefly—“pains hurt” (p. 3). That is, pain is accompanied by an immediate negative affect that has motivational properties. Pain is unpleasant, annoying, hurtful, and bothersome and invokes a general desire to end it, avoid it, or reduce it. Even though most pain scholars have long recognized that these two dimensions exist and that they can be readily disassociated (for example, in patients who have experienced certain brain injuries or
surgical procedures or through the effects of various drugs), the affective dimension of pain is still subject to continued debate and has caused researchers and theorists the most consternation, presumably because of its inherently subjective and evaluative nature. As a result, most efforts to further define, classify, and characterize pain tend to focus mainly on the sensory dimension of pain.

Pain researchers have identified several sensory characteristics or dimensions of pain. The most commonly discussed and researched characteristics include frequency of pain, pain intensity, site/location (or type) of pain, and pain duration and/or temporal patterning (e.g., Von Korff & Dworkin, 1989; Turk & Melzack, 2001). Each of these is discussed briefly below.

Frequency concerns how often pain is experienced. Frequency may be assessed in terms of a count of pain episodes within a specific time frame (e.g., past day, month, or year) or with more vague quantifiers (e.g., rarely/some of the time, often).

Intensity refers to a sensory quality of pain in terms of its magnitude or strength. Some also argue that a judgment of pain intensity involves the affective/ motivational properties of the pain as well—that is, pain that is more intense is also inherently more distressing or bothersome (Melzack & Torgerson, 1971). Pain intensity has been identified as one of the most critical domains of pain assessment (e.g., American Geriatrics Society, 2002; Turk & Melzack, 2001). Intensity is commonly used interchangeably with the term “severity;” in fact, many pain intensity scales use the word “severe” (or a close variant or synonym) to anchor the rating scale. There are some researchers, however, who employ the terminology “pain severity” to represent a combination of pain characteristics, such as a summed rating of pain intensity and
duration (e.g., Von Korff & Dworkin, 1989). Because of the potential conceptual confusion created by using these terms interchangeably, the current study primarily uses the term “pain intensity.” However, in describing the results of other studies, “pain severity” is used if it was used by the study authors (and if it is accompanied by a definition or explanation of the measure used in the study).

Pain can also be distinguished in terms of the anatomical region, location, or site that is primarily involved in the pain. The site can be specific (e.g., jaw, neck, lower back, shoulder, foot pain), or relatively general (e.g., headache, joint pain, peripheral or central). Some researchers advocate assessing an individual’s experience of pain in each site (e.g., Urwin et al., 1998). Pain can also be characterized into more general types that often reference some underlying cause)—e.g., pain that is neuropathic (nerve), visceral (internal organs), or somatic or nociceptive (associated with real or impending tissue damage). Sometimes pain sites (and even underlying causes) are used to classify people with different “pain conditions”—e.g., arthritis pain, diabetic neuropathy, TMJ pain, back pain, migraine pain. Some (e.g., American Pain Society, 2003; Cleeland et al., 1994) prefer to categorize pain more broadly; for example, malignant and non-malignant (or benign) pain. Malignant pain typically refers to pain associated with cancer, while non-malignant pain encompasses most other pains.

Most pain scholars differentiate between acute pain and chronic pain. This distinction is both important and relevant to the current study. Acute pain and chronic pain are most often distinguished on the basis of the duration of the pain, but definitions commonly reference other characteristics or dimensions of pain as well. In general, acute pain has a distinct onset, usually implies the existence of some underlying injury or
illness, and is of short duration (American Pain Society [APS], 1999). Estimates of
duration vary, but most experts seem to agree that pain lasting less than 3 months is acute
(Fillingim, 2005). Examples of pain usually regarded as acute include post-surgical pain,
pain following an injury, and experimentally-induced pain (such as pain from a cold-
pressor task).

Chronic pain, on the other hand, is pain that has continued over a prolonged
period of time. Various definitions have been offered for chronic pain, both specific
(e.g., 6 months or more) and relative (e.g., pain that persists beyond the usual course of
an acute illness or injury, or past the time of normal healing; APS, 1999; Bonica, 1953).
Although many definitions set the temporal criterion for chronic pain at six months
(International Association for the Study of Pain [IASP], 1994), some specify a three
month threshold (e.g., Andersson, Eljertsson, Leden, & Rosenberg, 1993; National
Institute of Arthritis and Musculoskeletal and Skin Diseases [NIAMS], 2006). In broad
epidemiological surveys (e.g., Nutrition and Health Examination Survey [NHANES]),
chronic pain has often been operationalized as pain lasting one month or more (e.g.,
Magni et al., 1990). Other distinguishing features highlighted in some definitions include
a less well-defined onset and/or inciting stimulus for chronic pain. Some scholars also
distinguish chronic from acute pain on the basis that the pain no longer serves an adaptive
warning function and/or has a negative impact on a person’s quality of life (Fillingim,
2005). Some definitions lump recurrent acute pain under the category of chronic pain
(e.g., APS, 1999), while others locate recurrent pain at about the midway point on an
underlying continuum of pain duration (e.g., Dworkin, Von Korff, & LeResche, 1992).

The American Society of Anesthesiologists offers a definition of chronic pain as
“persistent or episodic pain of a duration or intensity that adversely affects the function or well-being of the patient, attributable to any nonmalignant etiology” (American Society of Anesthesiologists Task Force on Pain Management, Chronic Pain Section, 1997, p. 995). Note that this definition explicitly excludes cancer pain. In the medical literature, the terms “persistent” and “chronic” are used somewhat interchangeably.

The American Geriatrics Society (AGS) eschews the use of the term “chronic pain” because of negative connotations and stereotypes that exist, particularly among older persons. They recommend instead the term “persistent pain” in order to better describe the experience of older patients with recurrent pain and to foster improved communication with these patients (AGS, 2002). AGS guidelines on the management of persistent pain in older persons define persistent pain as, “A painful experience that continues for a prolonged period of time that may or may not be associated with a recognizable disease process” (AGS, 2002, p. S205). Reference to a specific time frame is notably absent in this definition; pain that is of prolonged duration, and/or that recurs at intervals over months or years, can be considered persistent pain. So, too, can pain associated with a chronic disease process (such as arthritis) or a terminal disease (such as cancer). The AGS definition is thus more inclusive of, and more consistent with, older persons’ experiences of pain. For middle-aged and older persons who live in the community, many of whom never contemplate seeking care at a specialty pain clinic, arbitrary distinctions regarding the duration of pain may be largely irrelevant, though.

Various groups of pain researchers have investigated other characteristics or dimensions of pain. Some researchers prefer to characterize pain as tractable (i.e., treatable) or intractable (e.g., Loser, 2001). Other scholars differentiate between pain that
has an organic cause involving observable tissue damage versus pain that has no known organic cause (e.g., Cox, Chapman, & Black, 1978). There are also inventories of adjectives describing different sensory qualities or clinical features of pain—e.g., “shooting,” “stabbing,” “burning,” “aching,” “wrenching” (McGill Pain Questionnaire [MPQ], Melzack, 1975).

Despite receiving considerable attention from pain scholars, the various dimensions of pain have not always been clearly distinguished in prior research. Moreover, even experts in pain research do not have a shared understanding regarding the number of core, or relevant, dimensions of pain (Craig & Hadjistavropoulos, 2004; Turk & Melzack, 2001a; Von Korff, 2001). As a consequence, it is common to see a mixing of nomenclature (e.g., site/location, cause, and sensory qualities) in published reports—both within and across various scholarly journals devoted to research on pain.

**Pain as Conceptualized in This Study**

This study involved a secondary analysis of data drawn from a survey of community-dwelling middle-aged and older persons—The Health and Retirement Study (HRS). HRS uses a vaguely-worded item to assess the presence of pain in respondents: “Are you often troubled with pain?” This item does not directly assess the duration of pain and uses an extremely vague quantifier for frequency (“often”). Using the broad definition of persistent (or chronic) pain offered by AGS, it is *possible* that the pain reported by HRS respondents could be classified as persistent. HRS does not, however, include information about the duration of the pain experienced by respondents, nor does it specify a time frame. Therefore, pain reported by respondents in HRS is referred to generally in this document as “pain,” and phrases such as “pain reports” or “reports of
pain” are used to reduce repetitiveness in the text. Because of the ambiguity surrounding whether or not the HRS pain item measures chronic or persistent pain, studies of chronic pain were included in the literature review, especially when such studies have been conducted in the populations of interest—i.e., middle-age and older adults.

In HRS, individuals who endorsed the “troubled by pain” item were asked a follow-up question regarding the average intensity of their pain. Thus, pain intensity is the only dimension of pain available for use in the current study. This is not unusual in broad, community-based surveys. Although there exist several comprehensive pain batteries (e.g., MPQ; West Haven-Yale Multidimensional Pain Inventory [WHYMPI; Kerns, Turk, & Rudy, 1985]), those instruments were developed primarily for use in research with, and treatment of, chronic pain patients. Such instruments assess key components of pain in great depth in order to develop and evaluate the effectiveness of medical and behavioral interventions. However, they are impractical for use in large-scale studies of community-dwelling persons, most of whom are not likely to seek care at specialty pain clinics. Information regarding other dimensions of pain—for example, site/location, underlying cause, sensory qualities—is not available in HRS.

When summarizing the results of prior research or existing theoretical models, the language employed by the authors of those studies or models is used (e.g., “persistent pain,” “chronic pain,” or “pain,” depending on the definitions used in the study). In addition, the term “type(s)” of pain is used to broadly summarize findings related to pain locations, sites, causes, kinds, and sensory qualities. To the extent that research findings appear to be limited to a specific pain dimension, however, the distinctions and nomenclature employed by the study author(s) is preserved in reviewing those results.
Pain Prevalence in Middle-Aged and Older Persons

Prevalence estimates for pain in middle-aged and older populations are extremely difficult to compare across studies. There are three general sources of difficulty. First, there are inconsistencies in how pain is defined and measured. Slight variations in the operationalization in survey questions (e.g., “any pain,” “pain lasting more than one week,” “pain that was troubling,” “pain lasting 3 months or more”) often result in large discrepancies in prevalence estimates (Jones & Macfarlane, 2005). Studies also differ widely in the time periods used (e.g., pain in the “past year,” “past month,” or “past week”), as well as the type of pain assessed (e.g., back pain, joint pain).

Second, the samples used to derive prevalence estimates vary greatly. Some studies use samples of middle-aged and older persons attending specialty pain clinics; others sample patients from other clinical settings (e.g., outpatient rehabilitation clinics, primary care practices, home health care). To get a sense of the diversity of prevalence estimates, consider a review by Miller and Talerico (2002). These authors reviewed 80 published reports of pain and pain problems in older adults (defined as those over age 65). They found that prevalence estimates for pain in older adults ranged anywhere from 36-86%. Prevalence estimates varied according to study setting, country or geographic region, and the operationalization of pain (e.g., chronic pain vs. any pain). The most accurate estimates come from broad, epidemiologic studies of community-dwelling adults. However, assessments of pain within these studies are usually very limited—mainly because in these studies, depth of content is sacrificed in favor of breadth.

Finally, there are differences in how studies have defined and classified adults of different ages. Estimates derived from studies that group all adults over a certain age (for
example, all over age 50 or all aged 65 and over) into a category of “older adults,” are
difficult to compare directly with estimates from studies that create age groups based on
decades (e.g., 61-70, 71-80) or even periods (e.g., 45-64, 65-84, 85 and over) of life.

**How Common Are Reports of Pain in General?**

Notwithstanding the aforementioned difficulties, what do studies tell us generally
about how common pain is among middle-aged and older adults? When questions about
pain are *not* restricted to “chronic” or “prolonged” pain, prevalence estimates tend to be
fairly high—with most studies reporting estimates between 66% and 86%, depending on
the specific population under study, the type of sampling done, and the time period for
reporting pain. For example, a random sample of 2,449 community-dwelling adults in
the United States found that 72% reported experiencing *any* pain over the previous 6
months (Ruehlman, Karoly, Newton, & Aiken, 2005).

These estimates seem fairly consistent with estimates obtained in international
studies. For example, in a mail survey of randomly sampled community-dwelling adults
in Sweden, fully 66% reported experiencing recent pain or discomfort in any of 10
specific body locations of any duration or level of intensity (Brattberg, Thorslund, &
Wikman, 1989). A mail survey of adults aged 50 and over assigned to 3 primary care
practices in England found that 66% of respondents reported pain (i.e., “pain lasting for 1
day or longer in any part of your body”) during the past month (Thomas, Peat, Harris,
Wilkie, & Croft, 2004).

When samples are restricted to “older” adults, estimates of pain prevalence tend
to be slightly higher. For example, in a large, epidemiological study of over 3,600 older
adults (≥ age 65) living in rural areas in Iowa, Mobily et al. (1994) found that 86%
reported experiencing any pain in the year prior to the study. In addition, more than half of the sample (59%) reported multiple pain complaints. Joint, or musculoskeletal, pain was the most common type of pain reported in the sample. In a sample of community-dwelling older persons (≥ age 65) in Ontario, Canada, 73% reported musculoskeletal pain during the past two weeks (Scudds & Robertson, 2000). A series of studies conducted in Winnipeg by Roy and colleagues (reported in Roy, Thomas, & Cook, 1996) found that the majority (70%) of community-dwelling elderly subjects (≥ age 65) report living with pain on a regular basis. In a large sample of adults over age 72 living in Florida, Kahana et al. (1997) found that 82% reported experiencing some pain in the past year.

Existing epidemiologic evidence suggests that the prevalence of pain peaks in the 65-85 year-old age group, then decreases with age (Leveille et al., 2001; Mobily et al., 1994). However, it is unclear whether findings regarding lower prevalence estimates among some of the oldest adults might result from the under-sampling of those who live in institutional settings. Ferrell (1995) estimated that between 45% and 80% of nursing home residents suffer from pain. Studies of pain among the oldest old (i.e., those over age 85) are also underrepresented in the literature (Miller & Talerico, 2002).

**How Common Are Reports of Chronic or Persistent Pain?**

As might be expected, prevalence estimates for “chronic” or “persistent” pain among middle-aged and older adults are generally lower than estimates for reports of any pain. However, these estimates also tend to vary greatly, depending on the definition(s) used to solicit pain reports, the specific population(s) studied, and the type of sampling done. In general, there is relatively little research focused on chronic pain in later life general (Roberto & Gold, 2001).
Estimates of chronic or persistent pain among “older” adults (the definition of which varies by study) tend to range between roughly 20-25% and 50-60% (American Geriatrics Society [AGS], 2002; Ferrell, 1991). A random sample of community-dwelling older adults (age 65 or older) in Australia found that 56% reported experiencing “persistent” pain in the past year (Bradbeer, Helme, Yong, Kendig, & Gibson, 2003). In the Kahana et al. (1997) study of older adults in Florida, almost one-quarter of the total sample (23%) reported being in pain always or most of the time. When questions about pain are restricted to a particular site or type of pain, estimates tend to be lower. For example, persistent hip pain (“significant hip pain on most days” during the prior 6 weeks) was reported by only 14% of over 6,000 community-dwelling older adults (age 60 and over) surveyed in NHANES-III from 1988 to 1994 (Christmas et al., 2002).

Available evidence suggests that the prevalence of chronic or persistent pain increases with advancing age, but whether the peak prevalence occurs in middle-age or older age is a matter of ongoing debate (see Jones & Macfarlane, 2005, and Pickering, 2005, for discussion). Some studies suggest that the prevalence of chronic pain peaks in
middle-age and decreases thereafter (Andersson et al., 1993; Cook et al., 1989). Other studies (e.g., Bradbeer; Crook et al., 1984; Helme & Gibson, 2001) have found peak prevalence rates for chronic pain occurring in those over age 65.

When the rates of chronic pain among older adults are compared to those of younger adults, older adults appear to experience greater chronic pain. For example, a population-based study in Denmark found that older adults (age 67 and older) were 3.9 times more likely to report suffering from chronic pain (defined as pain lasting 6 months or more) than were young adults (age 16-24) (Eriksen, Jensen, Sjorgren, Ekholm, & Rasmussen, 2003). However, there are few comparative studies between older and middle-aged adults and between different age groups within older adult samples. In addition, existing research tends to lack comparability in terms of the kinds or types of pain studied—some specifically examine musculoskeletal pain, others look only at low back pain, and others examine pain of all types. Additional data regarding the relationship between age and chronic or persistent pain are reviewed later in this chapter.

As evidenced in the report of studies above, definitions applied in labeling pain as “chronic” have varied widely. In the face of such variability, however, there is also mounting evidence to suggest that chronic pain is, itself, quite persistent. For example, in a study of over 1,600 community-dwelling adults aged 25-74 in Sweden, 55% of the sample reported chronic pain (defined as “persistent or regularly occurring pain lasting for at least 3 months”). In responding to a follow-up question, almost the entire subsample (roughly 90%) reported that their pain had lasted for at least 6 months (Andersson et al., 1993). Moreover, short- and long-term follow-up studies with this sample document that the majority of the subsample was still experiencing chronic pain.
years later—for example, 85% of subjects who initially reported chronic pain also reported chronic pain 12 years later (Andersson, 2004). Studies of community-based samples in other countries have documented “persistence rates” for chronic pain of anywhere from 40-85%, depending on the length of the follow-up period and the type of pain assessed. The average duration of pain reported by the community-dwelling elderly subjects in the Winnipeg studies (reported in Roy, Thomas, & Cook, 1996, and cited previously) was approximately 11 years. Taken together, these data suggest that a fair amount of the pain that is reported by community-dwelling middle-aged and older adults can reasonably be considered “chronic” or “persistent.”

**Background Information About Psychological Distress**

**Definitions and Terminology**

This study examined depressive symptomatology as an indicator of psychological distress. This study did *not* assume that depressive symptoms were equivalent to a clinically-diagnosed depressive disorder (e.g., major depressive disorder, dysthymia). Clinically-relevant depressive symptoms include depressed mood, loss of interest or pleasure in activities, sleep disturbance, unintentional weight loss/gain, fatigue/loss of energy, feelings of worthlessness or guilt, diminished concentration, and suicidal ideation or recurrent thoughts of death (American Psychiatric Association, 2000).

There is growing interest in constellations of depressive symptomatology that may fall below the threshold required for diagnosis of a depressive disorder, which numerous scholars have labeled “subthreshold” (e.g., Hybels, 2001; Judd, Schettler, & Akiskal, 2002), “subclinical” (e.g., Schneider, Kruse, Nehen, Senf, & Heuft, 2000), or “minor” (American Psychiatric Association, 2000) depression. Most of these labels refer
in some way to depressive symptomatology that does not meet the strict criteria required for a clinical diagnosis of a depressive disorder (e.g., not having enough symptoms, not having a “core” symptom, not having symptoms for the required amount of time, etc.). Although not currently an accepted clinical diagnosis, an appendix in the latest version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR; American Psychiatric Association, 2000) does offer a “research diagnosis” of minor depression—defined by the presence of depressive symptoms that are fewer in number and accompanied by less severe impairment in functioning (vs. major depression). Such constellations are referred to hereafter as minor depression.

A focus on depressive symptomatology that may not rise to the level of diagnosable depressive disorder is consistent with a perspective advocated by some prominent experts in the study of depression. These scholars argue that there is considerable continuity between minor depressive states or symptoms and clinical depressive states, such as a major depressive episode (e.g., Clark, Beck, & Alford, 1999; Judd & Akiskal, 2002). There is substantial empirical support for such etiologic continuity. A 12-year, prospective study of a large cohort of patients initially diagnosed with major depressive disorder observed a stepwise linear relationship between increased levels of depressive symptomatology (from asymptomatic, to subthreshold or subsyndromal depressive symptoms, to minor depression or intermittent depression, to major depressive episode) and worsening psychosocial and general functioning in the same patients over time (Judd, Akiskal, Schettler et al., 2000). In other studies, individuals with a prior history of depressive symptoms (even if just a few symptoms or symptoms of mild severity) have been shown to be at higher risk for developing a
subsequent depressive episode (e.g., Flett, Vredenburg, et al., 1997; Howarth, Johnson, Klerman, et al., 1992). For individuals treated for a major depressive episode, the presence of minor depressive symptoms places them at increased risk for recurrence and increased severity of future episodes (Judd, Akiskal, Maser et al., 1998). Recent studies have also established that persons with minor depression can respond positively to standard treatments for major depression, including selected medications and psychotherapy (Judd et al., 2004; Cuijpers & Van Straten, 2007), leading some to suggest that the treatment of subthreshold depression may eventually reduce the incidence of major depression (Cuijpers, 2004).

Even if subclinical levels of depressive symptomatology do not go on to develop into a major depressive episode, they still have important implications for individual well-being and for public health. Depressive symptoms (in the absence of major depression) have a fairly high prevalence in the community. For example, while the lifetime prevalence rate for major depressive disorder for all adults is estimated at about 15.8%, an additional 10% are estimated to experience minor depression (Kessler, Zhao, Blazer, & Schwartz, 1997). Overall, prevalence rates for subclinical levels of depressive symptomatology (variously defined) range from 2% to 23% in community-based settings and from 4% to 17% in medical settings (Pincus, Davis, & McQueen, 1999; Banazak, 2000). Thus, the number of persons affected with subclinical depression is considerably larger than the number of persons with diagnosable depressive disorders.

Depressive symptoms often go unrecognized and untreated by health care professionals (Sartorius, 2003), and many individuals are reluctant to seek help for such symptoms, especially from mental health professionals (Henderson, Pollard, Jacobi, &
Untreated depressive symptoms at subthreshold levels have been associated with negative psychological, social, and vocational sequelae (e.g., Judd et al., 2002). In one study, individuals with minor depression had 51% more disability days than did individuals with diagnosed major depressive disorder (Broadhead, Blazer, George, & Tse, 1990). The presence of even minor levels of depressive symptomatology has also been linked to increased utilization of costly health care services, including emergency room visits and primary care doctor visits (e.g., Johnson, Weissman, & Klerman, 1992).

There is also reason to believe that depressive symptoms can be a marker of poor emotional health in general. Studies suggest that individuals who score relatively high on self-report depression symptom inventories are experiencing considerable psychological distress and may, in fact, have additional and/or other diagnosable psychopathology (e.g., other mood disorders, anxiety disorders, substance use disorders; Fechner-Bates, Coyne, & Schwenk, 1994). And, even at subclinical levels, depressive symptomatology can be regarded as an indicator of poor general affective and/or psychosocial functioning (Gottlib, Lewinsohn, & Seeley, 1995).

Prevalence of Depressive Symptomatology in Middle-Aged and Older Adults

The issue of subclinical depressive symptomatology is of considerable relevance to research involving middle-aged and older adults. In general, studies have found that rates of diagnosable depressive disorders decline in later life. For example, the Epidemiological Catchment Area (ECA) study documented rates of major depression of less than 1% among those age 65 and older and 2% among those age 45 to 64, compared to a rate of 3% among those age 18 to 44 (Robins & Reiger, 1991). Yet, when the focus
is broadened to include minor depression, prevalence rates are typically much higher, particularly among adults in later life. For example, one ECA site found that an additional 27% of older adults had “prominent” levels of depressive symptomatology (Blazer, Hughes, & George, 1987). The Longitudinal Aging Study Amsterdam documented a one-month prevalence rate of 13% for minor depression among middle-aged and older community-dwelling adults (age 55-85; Beekman et al., 1995).

Judd and Kunovac (1998) analyzed all ECA data in order to examine patterns of depressive symptomatology across the age spectrum. They found that the one-month point-prevalence of major depressive episode and dysthymia peaked in young adulthood (26% for each disorder among those ages 25-34), declined fairly steadily into late middle-age (11% and 13% in those ages 55-64), and reached the lowest rates among older adults (6% and 8% among those age 65 and older). A markedly different pattern was observed for subclinical levels of depression: the prevalence rates for both minor and subthreshold depression increased in later life, with an even more dramatic up-tick among those age 65 and older. Additionally, within the oldest age group (age 65+), minor depression occurred at more than twice the rate (13%) and subthreshold depressive symptoms occurred at roughly 3 times the rate (18%) of major depression. These findings, combined with similar findings from other studies, prompted Judd and Akiskal (2002) to characterize subthreshold and minor depressive symptoms as the “modal presentation of depressive illness” in elderly patients (p. 236).

When viewed from a perspective that acknowledges the relevance of subclinical symptomatology, depressive symptoms can be thought of as having a curvilinear, u-shaped relationship to age. Depressive symptom levels are typically high in young
adulthood, lower in middle age, and then increase in older adulthood from roughly the sixth (or seventh) decade on (Gatz & Hurwicz, 1990; Gatz, Johansson, Pedersen, Berg, & Reynolds, 1993; Kessler, Foster, Webster, & House, 1992). Increased depressive symptomatology levels in older adults are not simply due to increased somatic complaints that accompany aging. Studies have found that both negative affect and somatic symptoms increase with age (e.g., Gatz et al, 1993; Kessler et al., 1992).

Correlates of Depressive Symptomatology in Mid and Later Life

Analyses of data from several large epidemiological studies of community-dwelling adults have found that the correlates of subclinical depressive symptomatology (variously defined) are generally the same as those for diagnosable depressive disorders (Beekman et al., 1995; Hybels, Blazer, & Pieper, 2001; Van den Berg, Oldehinkel, Brilman, Bouhuys, & Ormel, 2000). In addition to age, the main correlates of depressive symptomatology include various aspects of health, gender, social support, socioeconomic status, psychological factors, and familial and personal history.

Some of the age differences observed in the prevalence of depressive symptomatology are thought to result from the increased prevalence of physical health problems in later life. Poor physical health (measured in a variety of different ways) has been found to be one of the strongest, most consistent predictors of depressive symptomatology (e.g., Okun, Stock, Haring, & Whitter, 1984; Cole & Dendukuri, 2003). Elevated depressive symptoms have been observed in persons with coronary heart disease, cerebrovascular disease, diabetes, and dementia (see review by Satariano, 2006), as well as in arthritis, lung disease, and cancer (e.g., Penninx et al., 1996). Rates of subclinical depressive symptomatology have been found to be positively associated with
an increased number of chronic disease conditions in adults of all ages (Kessler et al., 1997), and in older adults specifically (e.g., Van den Berg et al., 2000). Fair or poor self-rated health has also been associated with higher depressive symptomatology among community-dwelling older adults (Reid & Planas, 2002; Stallones, Marx, & Garrity, 1990). Depressive symptomatology has also been linked to the existence and extent of various types of activity limitation, such as limitations in physical functioning, Activities of Daily Living (ADLs), and Instrumental Activities of Daily Living (IADLs; e.g., Berkman et al., 1986; Blazer et al., 1991; see also Satariano, 2006, for review).

Gender differences in depressive symptomatology in mid and later life generally conform to the differences seen earlier in the life course, with women displaying higher levels of depressive symptomatology than men (Blazer, Burchett, Service, & George, 1991; Kessler, 2000). Findings are mixed regarding whether the size of these gender differences changes with increased age. Some studies have found that women evidence greater depressive symptomatology than men even in the oldest age groups (e.g., Kessler, Foster, Webster, & House, 1992). Other investigations suggest that the size of the gender difference tends to narrow in later ages (e.g., Barefoot, Mortensen, Helms, Avlund, & Shroll, 2001).

Life events have been associated with depressive symptomatology among adults of all ages, presumably through intervening stress-based processes. Research by Nolen-Hoeksema and colleagues (e.g., Nolen-Hoeksema & Ahrens, 2002) has demonstrated that although the specific types of life events that confront young, middle-aged, and older adults may be different, the relationships between these events and depressive symptomatology are similar. For example, although employment status and caregiving
status varied by age group, those who were unemployed or providing care to an ill relative had higher levels of depressive symptomatology than their peers, regardless of age. A variety of different life events (alone and in combination) have been associated with increased depressive symptomatology, even after controlling for the effects of other known predictors such as age, poor health, and socioeconomic status (e.g., Glass, Kasl, & Berkman, 1997).

Being in a close relationship and perceiving strong social support have been shown to be a buffer against the development of depressive symptoms in the face of various stressors and negative life events (e.g., Krause, 1986, 1987; Lin, Dean, & Ensel, 1986). Strong social support has also been found to be directly related to lower depressive symptomatology (e.g., Lin, Dean, & Ensel, 1986). Lack of, loss of, and/or low levels of social support have all been linked to the development of depressive symptomatology in various studies (e.g., Beekman et al., 1995; Blazer et al., 1991; Blazer, Hughes, & George, 1992; Hybels et al., 2001). Both positive and negative social interactions in, as well as satisfaction with, key social relationships have also been associated in predictable ways with depressive symptomatology levels in mid and later life (Okun & Keith, 1998; Nolen-Hoeksema & Ahrens, 2002).

Depressive symptomatology in middle-aged and older adulthood has also been linked to a variety of indicators of socioeconomic status (SES). Lower levels of education, lower income, lower occupational status, and perceived financial inadequacy have been associated with higher levels of depressive symptomatology in numerous studies (e.g., Blazer et al., 1991; Bromberger, Harlow, Avis, & Kravitz, 2004; Kessler & Cleary, 1980). Explanations for the effects of SES include the possibility that lower SES
increases exposure to other risk factors for depressive symptoms (e.g., negative life events, ill health) and/or that lower SES decreases the availability of, or access to, beneficial resources (e.g., social support) that might buffer the effects of other risk factors (Satariano, 2006). Differences in SES have also been shown to explain some, though not all, of the higher levels of depressive symptomatology observed in African American and Hispanic populations (as compared to non-Hispanic Whites) in mid and later life (e.g., Bromberger et al., 2004; West, Reed, & Gildengorin, 1998).

A variety of psychological constructs (e.g., personal control, mastery, self-efficacy, personality characteristics, cognitive styles, and coping strategies) have also been studied in relation to depressive symptomatology in middle age and older adulthood. In general, empirical evidence supports the importance of most of these constructs. The diversity of these constructs and the breadth of the supporting literatures make it impractical to attempt a review of this literature in the present context; however, it should be noted that, for the most part, these constructs have been conceptualized as intervening mechanisms, invoked to help explain observed relationships between depressive symptomatology and other variables, including some of those mentioned above (e.g., gender, age, SES). The issue of intervening mechanisms (or mediation) in relation to depression is addressed at a later point in this chapter.

A personal history of mood disturbance or depressive disorder (e.g., Beekman et al., 1995; see also review by Djernes, 2006) has been associated with depressive symptoms in later life. Although a familial association for depressive disorders has been documented in some population-based studies, it is generally believed that any genetic susceptibility is of decreased relevance in explaining depressive symptoms in later life.
(Satariano, 2006). However, a different sort of familial association is a topic of growing interest to researchers—the association of depressive symptomatology between married partners in later life (e.g., Sigel, Bradley, Gallo, & Kasl, 2004). This phenomenon is of major interest in the current study and is addressed at length in Chapter 3.

**Depressive Symptomatology as Conceptualized in This Study**

The data source used for this study (HRS 1998) does not contain enough information about the duration and severity of depressive symptoms to yield a diagnosis of a depressive disorder. The HRS survey includes a modified version of a commonly-used measure of depressive symptoms: the Center for Epidemiologic Studies-Depression Scale (CES-D; Radloff, 1977). In order to simplify the measure for administration to older adults, HRS researchers shortened the number of items (from 20 to 8), and restructured the response categories to indicate the presence or absence of these symptoms “much of the time” during the past week. The eight items assess a range of depressive symptoms, including depressed mood, lack of positive affect, and somatic/vegetative symptoms. Moreover, the items cover the major types of symptomatology that define a diagnosis of major depression or dysthymia (Steffick, 2000).

Throughout this document, terminology such as “depressive symptoms” or “depressive symptomatology” is used when referring to the symptoms reported by the respondents in HRS. Such language is also used when summarizing existing studies that have focused specifically on depressive symptoms. Terms such as subthreshold and minor depression will generally be avoided, as experts have yet to agree on a shared definition (or operationalization) of these terms (e.g., Cuijpers & Smit, 2004; Chaturvedi, 2004). Moreover, studies of pain and depressive symptomatology have not yet focused...
attention specifically on subthreshold levels of depressive symptomatology.

Unfortunately, existing studies of the relationship between pain and depressive symptomatology have often used “depressive symptoms” and “depression” interchangeably, and have conceptualized and operationalized the terms in ways that overlap or are inconsistent. In reviewing the existing literature, it was therefore necessary to include some studies of “depression” or “major depression” in relation to pain. Such studies were included if they addressed any of the general aims of this study (as described in Chapter 1), or if they have focused on middle-aged and/or older adults.

The Relationship Between Pain and Psychological Distress

Evidence of a Relationship

Pain is related to the entire continuum of psychological distress, including a variety of clinically-diagnosable psychological disorders such as anxiety and mood disorders (e.g., Sartorius et al., 1993), and a host of negative affective symptoms and states such as sadness, anger, worry, fear, and tension (e.g., Affleck, Tennen, Urrows, & Higgins, 1991; Riley, Robinson, Wade, Myers, & Price, 2001; Zautra, Hamilton, & Burke, 1999). Of the various types of psychological distress, depressive symptomatology and depression have, thus far, received the most attention from researchers.

Evidence regarding the relationship between pain and depressive symptomatology or depression that is specific to middle-aged and older adults is reviewed in depth later in this chapter. The purpose of this section is to highlight some of the research that has established a link between pain and depressive symptomatology or depression. There are two basic lines of evidence that support a relationship between pain and depressive symptomatology or depression: (a) rates of depression or levels of depressive
symptomatology are elevated in persons with pain, (b) pain is more commonly reported by persons with depression or those with higher levels of depressive symptomatology.

The relationship between pain and depressive symptomatology has been studied most extensively by scholars interested in chronic pain. Among chronic pain researchers, there is general agreement that rates of depression are higher among patients with chronic pain than among patients without pain (Fishbain, Cutler, Rosomoff, & Rosomoff, 1997). For example, it is estimated that over half of chronic pain patients meet standardized diagnostic criteria for a depressive disorder (Fishbain et al., 1997). Bair, Robinson, Katon, & Kroenke (2003) conducted an extensive review of the literature on pain and depression/depressive symptomatology published between 1966 and 2002. Bair et al. identified 15 studies that documented the percentage of chronic pain patients who met criteria for major depression. Estimates in these studies ranged from 2% to 100%, with an average prevalence rate across studies of 52%—quite consistent with the Fishbain et al. (1997) estimate. Clearly then, chronic pain patients display high rates of depression.

Even if chronic pain patients do not meet standard diagnostic criteria for a depressive disorder, studies suggest that sizeable numbers of patients experience significant depressive symptomatology. The observed range for significant depressive symptoms among pain patient samples is extremely varied, ranging from a low of 18% to a high of 70% in published studies. Estimates depend on the clinical characteristics of the sample (e.g., patients at intake vs. those already in treatment), the measure of depressive symptomatology or depression used, and the gender composition of the sample. Two examples illustrate this diversity. Ahern, Adams, and Follick (1985) found that 70% of their 117 predominantly male chronic low back pain patients scored in the
distressed range on the Depression subscale of the Minnesota Multiphasic Personality Inventory (MMPI). In contrast, only 18% of 51 predominantly female pain patients in the Thomas and Roy (1989) study scored in the “clinically depressed” range on the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Modck & Erbaugh, 1961).

Elevated rates of depression and relatively high levels of depressive symptomatology have also been observed in adults who are not specifically labeled chronic pain patients. Portenoy, Ugarte, Fuller, and Haas (2004) interviewed over 1,300 adults (age 18 or over, identified through a nationwide probability sample) who reported “frequent or persistent pain” during the previous 3 months. Thirty-five percent of the sample endorsed a statement indicating that they had experienced “depressed mood” as a result of their pain—a sizeable figure, given this community-based sample. A critical review of the literature on cancer pain concluded that strong evidence exists to support a relationship between pain and psychological distress (mainly depressive symptoms) in cancer (Zaza & Baine, 2002). Persons with other disease conditions in which pain is a frequent symptom (e.g., rheumatoid arthritis, osteoarthritis, osteoporosis) have also been shown to have higher depressive symptomatology levels than those observed in the general population (Campbell et al., 2003).

Similar findings have emerged from studies of community-dwelling adults who have not been pre-selected on the basis of their pain status or having some painful disease condition. For example, community-dwelling older adults who report experiencing pain display greater depressive symptomatology than do those who report being pain-free (e.g., Roy & Thomas, 1988; Williamson & Schulz, 1992a). Approximately 18% of adults who reported a history of frequent musculoskeletal pain in the NHANES study met
a commonly-used cut-off for “probable depression” (Magni, Caldieron, Rigatti-Luchini, & Merskey, 1990). In their review, Bair et al. (2003) calculated an average prevalence estimate of 18% for current major depression in persons with pain living in the community. They also noted considerable variability across studies, with prevalence rates ranging from 5% to 22%.

Pain is more common in persons who are diagnosed with depression or who have higher levels of depressive symptomatology. Studies show that depressed people are more likely to report pain than are non-depressed people. The review by Bair et al. (2003) identified 14 studies that reported the prevalence of pain complaints among persons with depression. Pain complaints were documented in 15% to 100% of patients in these studies, with an average prevalence rate across studies of 65%. Although the majority of studies were based on samples of psychiatric inpatients and outpatients, three studies of patients seen in primary care settings documented pain complaints in 43%, 59%, and 69% of patients with depression. This phenomenon has also been found in studies comparing the pain reports of chronic pain patients with and without a diagnosis of depression. In one study, chronic pain patients who met criteria for diagnosis of major depression reported pain of significantly higher intensity than did those who were not diagnosed as depressed (Keefe, Wilkins, Cook, Crisson, & Muhlbaier, 1986).

**Conceptual Issues in Studying This Relationship**

Most existing research, then, shows a clear link between pain and depressive symptomatology or depression, although neither the nature of this link nor reasons for it have yet to be fully explained. Van Houdenhove and Onghena (1997) articulated five distinct etiological hypotheses concerning the relationship between chronic pain and
depression: (1) Depression is a direct consequence of chronic pain (the “consequence” hypothesis); (2) Chronic pain causes depression through some psychological or physiological mediating factor—e.g., cognition, coping style, social factors, or some genetic, biological, or psychological vulnerability (the “mediation” hypothesis); (3) Chronic pain and depression have a common pathogenic basis (the “common pathogenesis” hypothesis); (4) Depression causes pain (the “antecedent” hypothesis); and (5) Depression perpetuates or exacerbates pain once it develops (the “exacerbation” hypothesis). One additional etiological hypothesis (which here is labeled #6) has been identified as the “scar” hypothesis (Fishbain, Cutler, Rosomoff, & Rosomoff, 1997; Skevington, 1994). The scar hypothesis suggests that a predisposition to depression increases the likelihood of depression developing after the onset of pain. Note that the labels for each hypothesis above were applied by the author of this dissertation and were drawn from an amalgam of published discussions on the topic (e.g., Fishbain, Cutler, Rosomoff, & Rosomoff, 1997; Skevington, 1994; Van Houdenhove & Onghena, 1997).

The history of research on pain and its relationship to depression has been punctuated by several published literature reviews. Some of these reviews (Bair et al., 2003; Van Houdenhove & Onghena, 1997) have already been mentioned above. Other influential reviews include an early review by Romano and Turner (1985), one by Von Korff et al. (1996), and one by Craig (1999). Another review (also mentioned previously) by Fishbain, Cutler, Rosomoff, and Rosomoff (1997) deserves special comment. This review resulted from a workshop sponsored by the NIH; as such, it became the definitive resource for publications on the topic. And, although the Bair et al. review is more current (published in 2003), the Fishbain et al. review is still cited with
great frequency and appears to still be the dominant resource in the field. There are likely several reasons for this, including: (a) the extensive (83 studies) and detailed nature of the review by Fishbain et al.; (b) the inclusion of topics slightly removed from the central pain-depression relationship in the Bair et al. review (e.g., the influence of pain presence on physician recognition of depression); and (c) the location of, and the population of researchers targeted by, each publication (*Clinical Journal of Pain* for Fishbain et al. vs. *Archives of Internal Medicine* for Bair et al.). In addition, the Fishbain et al. (1997) review evaluated the existing literature with respect to several of the competing hypotheses articulated above, while the Bair et al. (2003) review offers no assessment of the literature with respect to competing hypotheses. Bair and colleagues instead embrace a more pragmatic stance in their review—demonstrating that since pain and depression co-occur, they should be treated by physicians as comorbid conditions.

The empirical evidence for each of the six hypotheses regarding the pain-depression relationship is reviewed briefly below. These hypotheses, as laid out by Van Houdenhove and Onghena (and others), were originally intended to be specific to chronic pain and depression. However, they are easily extended to the broader constructs of pain and depressive symptomatology. This review uses the broad terms “pain” and “depression,” unless study findings (or implications) were related to a more specific construct. This review also incorporates observations culled from existing reviews.

There is a considerable body of evidence that lends support to the “consequence” hypothesis (#1). The existence of a relationship between pain and depression or depressive symptomatology has been documented in a large number of correlational and descriptive studies (several of which were mentioned earlier). There are also several
retrospective studies of pain patients that provide evidence to support this hypothesis (e.g., Arena, 1985). However, convincing evidence must really come from longitudinal studies that have helped to establish directionality in the relationship between pain and depression. Unfortunately, rigorous longitudinal studies of pain and depression are fairly rare. One of the strongest examples is a study of community-based adults with rheumatoid arthritis ($N = 243$) by Brown (1990), who collected self-reports of pain and depressive symptomatology every six months over a period of three and one-half years. Covariance structure modeling revealed positive cross-wave effects for pain on depressive symptomatology during the last 3 waves (12 months) of the study, even after controlling for the influence of prior levels of depressive symptomatology. He also tested alternate models, including a model of the cross-wave influence of depressive symptomatology on pain—a model that did not offer a good fit to the observed data. In general, his findings offer support for the influence of pain on depressive symptomatology (specifically, an exacerbation of depressive symptomatology over time).

Although the review by Fishbain et al. (1997) appears to have overlooked the above study by Brown, it identified three other longitudinal studies that found evidence of a temporal relationship between pain and depression. These include two studies of chronic pain patients (e.g., Holroyd, 1993; Rains, 1993) and one study of women with menstrual pain (Bancroft, 1995). In total, Fishbain et al. identified 15 studies with findings related to the consequence hypothesis; of these, they determined that all 15 (100%) offered some level of support for the hypothesis that depression emerges as a consequence of pain.

Van Houdenhove and Onghena (1997) also concluded that solid empirical
evidence exists to support the consequence hypothesis. However, they called attention to the fact that the bulk of these studies do little to explain why only a portion of chronic pain patients go on to develop depression or significant depressive symptomatology. Their observation can also be applied beyond the chronic pain population, as well.

Some chronic pain scholars have argued that pain results in depression, but only after it has lasted an extended length of time (e.g., Sternbach, 1974). However, based on their review of the existing literature through the early 1990s, Von Korff et al. (1996) concluded that there was compelling evidence that psychological distress (especially depression) emerges fairly early in the natural history of a pain condition. Across available studies, there appeared to be no conclusive evidence that the duration of a pain problem (i.e., its chronicity) was systematically associated with psychological distress.

The “mediation” hypothesis (#2) characterizes several different lines of research that appear to have developed to explain the underlying process(es) through which depression develops in persons with chronic pain. In some ways, such studies represent a logical extension of the consequence hypothesis; although, to be clear, these explanations do not address the issue of why depression develops (as noted by Van Houdenhove & Onghena, 1997)—they attempt to address the mechanism by which it does.

A fair number of studies have addressed the mediation hypothesis. However, these studies have varied widely in terms of which mediating or explanatory process has been tested. The most researched explanatory processes to date include: Cognitive factors (such as beliefs about pain, perceptions of pain, and cognitive coping processes—e.g., reappraisal); behavioral factors (e.g., low levels of activity, loss of social reinforcement); interpersonal relationships and social roles (e.g., relationship
dissatisfaction, lack of social support, reinforcement of sick role behavior); the inhibition of anger (or other negative affect); the effects of pain treatments; and genetic, biological, or developmental-psychological vulnerabilities to depression within chronic pain patients. Note that, although tested as mediators in prior research, some of the above constructs could potentially play moderating roles in the pain-depression relationship.

The review by Van Houdenhove and Onghena (1997) concluded that the evidence for mediating factors such as inhibited anger or negative affect, negative reactions to poor pain treatment, and developmental-psychological vulnerabilities is relatively weak. For example, although some studies have found a relationship between childhood physical or sexual abuse and depression in chronic pain patients (e.g., Goldberg, 1994; Taylor, Throtter, & Csuka, 1995), there are several possible explanations for this association. The Fishbain et al. (1997) review focused more on cognitive or cognitive-behavioral mediators (e.g., cognitions or perceptions related to pain, coping styles). That review concluded that 5 of 6 studies in this area showed support for cognitive mediation. The other reviews (Romano and Turner, 1985; Van Houdenhove & Onghena, 1997; Von Korff et al., 1996) reached similar conclusions about the existence of good support for cognitive or cognitive-behavioral mediators.

All of the reviews except Fishbain et al. (1997) cited some evidence supporting the existence of mediating variables related to family relationships (e.g., quality of relationships, marital satisfaction). However, most noted the need for further study in this area. This author’s review of the literature published after these reviews (discussed in Chapter 3) suggests that there is now considerably more evidence in support of marital factors as mediators in the pain-depression relationship—especially marital satisfaction.
and spousal reactions to patient pain.

Discussions regarding proposed mediating mechanisms are often confusing. Many authors—both the original authors of studies designed to test such effects, and the authors conducting the literature reviews—confuse mediating processes (i.e., mechanisms through which an effect occurs) with moderating processes (i.e., a third variable that affects when or for whom the effect occurs). This, along with the diversity of proposed mediating factors, makes it difficult to generate any substantive conclusions about hypothesis #2.

The hypothesis that both pain and depression result from some shared biological or neurological pathology (#3) has received some support from existing empirical research. With the current emphasis on brain-behavior relationships, this is probably one of the most actively pursued areas of chronic pain research today. Van Houdenhove and Onghena (1997) noted that this explanation seems most applicable to individuals whose chronic pain has no known, organic cause. There is mounting evidence of neurobiological abnormalities that appear in both chronic pain and depression, including low levels of certain neurotransmitters and hypo- or hyper-secretion of specific hormones (Bair et al., 2003). In addition, some antidepressants have demonstrated a therapeutic effect on chronic pain, even in patients who do not appear to have depressed mood; however, researchers have yet to fully explain how such medications work in the context underlying biological pathway. Fishbain et al. (1997) address this notion of a possible shared neurobiological pathway in their review of the “scar” hypothesis (see below).

Interestingly, Van Houdenhove and Onghena (1997) also included models of shared psychodynamic processes and underlying personality traits under hypothesis #3.
They noted, however, that it is extremely difficult to validate these models empirically. The existing empirical evidence cited in support of such models (e.g., common affective deprivation, narcissistic injury, and feelings of loss/guilt; or similar levels of neuroticism or susceptibility to dysphoric symptoms) is largely correlational and can be subject to many different interpretations (Van Houdenhove & Onghena, 1997).

Over the past several decades, many different iterations of the “antecedent hypothesis” (#4)—that depression is a cause of chronic pain—have been offered, mainly within the clinical literature on chronic pain. Some have argued that pain may be a symptom of depression, especially in older adults in whom depression may often present as a series of somatic complaints (e.g., Katona, 1994). Others have suggested that chronic pain is actually “masked depression” (Blumer & Heilbronn, 1982), a symptom of underlying depression, or a somatization of affective distress that would emerge as pain through some hysterical conversion or hypochondriacal mechanism (e.g., Van Houdenhove, Verstraeten, Onghena, & De Cuyper, 1992).

Although such explanations have enjoyed some popularity among more psychodynamically-oriented professionals (especially those who treat patients with no diagnosed physiological basis for their chronic pain), many prominent pain researchers and clinicians have actively disputed these explanations. A review by Craig (1999) concluded that there is no empirical basis for the notion that an underlying affective disorder is responsible for the pain reported by most chronic pain patients—even those who do not have an identified, organic cause for their pain. Merskey (1999) also pointed out that hysterical conversion and somatoform disorders are very rare, and that the available empirical evidence does not support the broad application of these explanations.
to most chronic pain patients.

Patrick Wall—one of the world’s leading authorities on pain and co-author of one of the most influential texts, *The Challenge of Pain* (Melzack and Wall, 1982)—has summarily dismissed the claims of his psychodynamic colleagues. As recently as 1999, Wall wrote: “I have not seen a scrap of convincing evidence that the mood and attitude create the pain.” (p. 7). Another notable figure in pain research, psychologist Kenneth D. Craig, has also argued against the notion that underlying anxiety or depression constitutes the primary cause of pain, rather than being secondary to the pain:

Outside of anecdotal reports by clinicians, there seems to be no conclusive evidence that severe emotional distress can trigger new pain or reinstate old pain in the absence of some sort of physical pathology. (Craig, 1999, p. 298)

The review by Fishbain et al. (1997) identified 13 studies with findings related to the antecedent hypothesis. Of these 13 studies, Fishbain et al. concluded that 9 offered no support for the hypothesis, 3 offered some support, and 1 offered mixed results. The mixed study was a 3-year follow-up study of HMO enrollees by Von Korff, LeResche, and Dworkin (1993). Von Korff et al. found that depressive symptoms were linked to the development of headache and chest pain, but were not associated with back, abdominal, or temporomandibular (TMJ) pain. Furthermore, these authors also found that the presence of any pain condition at baseline was a more consistent predictor of developing a new pain condition at follow-up than was either the severity or chronicity of depressive symptoms. The Von Korff et al. (1993) study is one piece of evidence cited by scholars who argue that depression is linked to migraine headaches. However, Merskey (1999)
contends that the association between depressive episodes and increased frequency of migraine headaches is less strong than is often intimated in the existing literature.

There is some evidence that depression can perpetuate or exacerbate pain once it develops (#5). For example, there is evidence that depression can intensify the perception of pain. Affleck et al. (1991) found that, after controlling for disease state and disability status, depressive symptoms led to increased reports of pain severity in rheumatoid arthritis patients over a period of days. Another study of rheumatoid arthritis patients found that depression was related to an exacerbation of pain (Parker et al., 1988). Van Houdenhove and Onghena (1997) cited only one study in support of this hypothesis, and it was a study that offered evidence suggesting the existence of neurobiological pathways with pain-facilitating properties (Geisser et al., 1993).

The exacerbation hypothesis appears to have more conceptual than empirical support. For example, most pain scholars do believe that there are feedback loops between pain and depression. Even Patrick Wall (1999) has acknowledged that pain-produced anxiety, fear, depression, and obsessive preoccupation do influence attention and behavior, possibly making pain worse or harder to bear. Although Von Korff et al. (1996) concluded in their review that the evidence favors the pain-depression direction, they acknowledged that psychological distress may serve to further amplify pain and may negatively affect an individual’s capacity to adapt effectively to pain, thereby creating a feedback loop between depression and pain. Craig (1999) also conceded that heightened emotional distress (perhaps induced by pain) can precipitate the development of disease through over-activation and repeated activation of physiological stress mechanisms, which can, in turn, cause pain.
Merskey (1999) regards hypothesis #5 as offering a compromise between hypotheses #1 and #4 above: He suggests that, instead of depression causing chronic pain, it is more likely the case that psychological factors (i.e., mood states, personality characteristics, coping styles) exacerbate pain that already exists. In this explanation, it is not depression per se, but associated symptoms, cognitions, and behaviors that perpetuate or exacerbate pain. Eich, Rachman, and Lopatka (1990) offered one explanation of how this might happen. In an experimental study, they found that pain induced negative affect, which in turn interfered with access to memories of pleasant events and promoted retrieval of more negative or unpleasant events. The authors reasoned that these relationships may result in a vicious cycle of pain increasing negative affect, which in turn increases recall of negative events and decreases recall of pleasant events, which in turn increases unpleasant affect, which in turn may help to perpetuate pain.

On first inspection, it would seem that the “scar” hypothesis (#6) closely resembles hypothesis #3, the shared pathogenesis hypothesis articulated by Van Houdenhove and Onghena (1997). However, it differs in that the scar hypothesis focuses almost exclusively on the notion of an underlying genetic predisposition to recurrent depression. The Fishbain et al. (1997) review identified 12 studies with findings related to the scar hypothesis: 9 offered some support for the hypothesis, while 3 did not. A series of studies by Magni and colleagues (e.g., Magni, 1987) found that more chronic pain patients (especially those with migraine and irritable bowel syndrome) have first degree biological relatives with depressive disorders than controls; however, several other studies have not found such differences. Another group of studies documented that chronic pain patients had a higher prevalence of depression prior to the onset of their pain
than did matched control subjects (e.g., Atkinson et al., 1991). The conclusions of the Fishbain et al. review regarding the scar hypothesis aligned with those of other reviewers (e.g., Van Houdenhove & Onghena, 1997): A predisposition to depression or a prior episode of depression may increase the likelihood of depression after pain onset in some chronic pain patients.

With the exception of Bair et al. (2003), which offers no conclusion, prior reviews have generally concluded that the empirical evidence favors the consequence hypothesis (#1) over the antecedent hypothesis (#4). With respect to the other hypotheses, most reviews have indicated the need for additional research. Fishbain et al. (1997) also offered an important observation about the studies that have examined the mediation hypothesis: “the direction of the relationship between pain and depression was for all studies from pain to depression. Thus, these six studies also are consistent in supporting the consequence hypothesis.” (p. 120).

A review by this author of more recently published studies does not contradict the conclusions offered in prior reviews. The bulk of existing research appears to support the consequence hypothesis. This interpretation is also shared by others. For example, Marks et al. (2005) stated in an authoritative Health Psychology textbook, “Existing research on pain appears to suggest that depression and negative mood states in general are more likely outcomes of pain than causes.” (p. 76; emphasis added by the present author). Additional evidence has accrued in recent years with respect to the mediation hypothesis, as well. Several experimental and observational studies have sought to demonstrate reciprocal effects—suggesting that those who are already depressed may focus more on pain and report more pain. Thus, support for the exacerbation hypothesis
has also grown. However, to expand Fishbain et al.’s (1997) observation: Studies that examine the mediation hypothesis or the exacerbation hypothesis generally begin with the premise that pain causes depression; thus, studies that offer support for these hypotheses also offer implicit support for the consequence hypothesis as well.

Efforts to characterize the relationship between pain and depressive symptomatology can also be informed by findings from the general literature on the relationship between physical health and mental health. It is well-recognized that physical health and illness are some of the strongest predictors of an individual’s psychological distress. There is evidence of some reciprocal effects between physical health and mental health, especially depression (e.g., Penninx, Leveille, Ferrucci, van Eijk, & Guralnik, 1999). However, the strongest, most immediate negative effects (after controlling for other confounding variables) have been observed for the effect of physical illness on mental health. This has been demonstrated in studies in community-based samples (e.g., Aneshensel, Frerichs, & Huba, 1984) and those drawn from clinical settings (e.g., Hays, Marshall, Wag, & Sherbourne, 1994).

The current study addressed hypotheses #1 (consequence) and #2 (mediation)—the hypotheses with the most empirical support and the ones most endorsed by experts in the field. Because this study was cross-sectional in nature, it could not definitively address the antecedent hypothesis (#4), nor could it provide evidence to support or refute hypothesis #5 (exacerbation). In addition, since the data for this study were drawn from a community-based survey comprised mainly of self-reports of current or recent experiences, neither the common pathogenesis hypothesis (#3) nor the scar hypothesis (#6) could be examined.
Despite voluminous research on depression and depressive symptomatology, experts still note the need for further study. In particular, there is a need to “study both risk factors and mechanisms of risk” for depression (Ingram & Siegle, 2002, p. 99). Available evidence clearly suggests that pain is a risk factor for the development of depression and depressive symptoms. It is therefore important to develop and empirically test conceptual models that outline some of the mechanisms or processes through which pain may affect depressive symptomatology.

**Theoretical Models for Studying the Relationship Between Pain and Psychological Distress**

**Dominant Theories of Pain and of Depressive Symptomatology**

Several theoretical models exist to guide the study of pain in general; however, most of these models focus on the internal processing of pain-related information, such as the sensation and perception of pain. The dominant theoretical model in this regard is the gate control theory of pain (Melzack & Wall, 1965, 1983). The gate control theory posits a multifaceted interaction between physiological, psychological, and behavioral inputs and mechanisms in the spinal cord and brain (i.e., the “gate”) that regulates whether and how internal and external stimuli are perceived and experienced as pain.

The gate control theory has greatly advanced the study of pain and has offered compelling explanations for how and why people may vary in response to the same painful stimulus. More recent work by Melzack (1999) has replaced the gate metaphor with the concept of a neuromatrix and neural networks. Although the theories of Melzack and Wall do highlight the importance of psychosocial and cultural variables in the pain experience, most of the emphasis has been on the role of these variables in the
perception and processing of pain. Neither the gate control theory nor theories about a
neuromatrix gives direct attention to the psychosocial consequences of pain or to the
specific relationship of pain to psychological distress or depressive symptomatology.

Dominant theories of depression are equally lacking in attention to the role of
pain in the development of depression or depressive symptomatology. Cognitive theories
of depression (viz., Beck, 1987) generally posit that depression results from, and is
maintained by, dysfunctional cognitions such as overgeneralization, arbitrary inference,
magnification of the negative aspects of a situation, and minimization of positive aspects
of a situation. Most cognitive-behavioral models of depression integrate some aspects of
Beck’s theory along with concepts derived from learning theory, operant conditioning,
and respondent conditioning (e.g., Burns, 1999; Ellis, 2001; and Lewinsohn, Hoverman,
Teri, & Hautzinger, 1985). To date there has been no overarching model developed
from either a cognitive or a cognitive-behavioral perspective that specifically addresses
the psychosocial consequences of pain or the relationship of pain to depression. The
absence of such a model is even more notable, given that cognitive-behavioral therapy is
used as the standard form of psychotherapeutic treatment in chronic pain clinics.

In summary, neither of the dominant theoretical paradigms in the study of pain or
depression provides explicit guidance for examining the relationship between the two
constructs. Although both recognize the general contribution of physical health to mental
health and vice versa, the specific role of pain has yet to receive much direct attention.

*Theoretical Foundations for a Proposed Conceptual Model*

The current study was based on the premise that, in general, pain has a causal
relationship with depressive symptomatology (i.e., the “consequence” hypothesis).
Attempts to understand and explain this relationship (especially in light of sometimes conflicting empirical findings) have led to the development of theoretical perspectives that embrace the mediation hypothesis (i.e., that pain causes depressive symptomatology through some mediating mechanism). Existing theories in this area, however, are not well-developed. The lack of theory in this area has been noted by others, including most of the authors who have published reviews of the literature in this area (e.g., Fishbain et al., 1997; Van Houdenhove & Onghena, 1997).

In the absence of solid theoretical models in this area, a conceptual model was developed specifically for use in this study. Three theoretical frameworks—the “Activity Restriction Model of Depressed Affect,” models of disability or disablement, and a model of pain within the “Illness-Disability Cascade”—helped to inform the proposed conceptual model. Each of these frameworks is considered below.

**The Activity Restriction Model of Depressed Affect**

The activity restriction model of depressed affect (ARM) was developed by Gail Williamson and colleagues (e.g., Williamson & Schulz, 1992a; Williamson, 2000b). The model, depicted in Figure 1, proposes that the relationship between pain and depressive symptomatology is best explained by pain’s capacity to restrict normal activities. That is, pain is hypothesized to affect depressive symptoms primarily through its effect on such “activity restriction.” The ARM thus proposes that the effect of pain on depressive symptomatology is mediated by activity restriction.

Williamson and colleagues have conducted studies with a variety of samples (e.g., breast cancer patients, general cancer patients, community-dwelling older adults, and children with sickle cell disease). The majority of these studies lend some support to the
activity restriction model of depressed affect (ARM). Most provide support for the proposition that activity restriction \textit{at least partially} mediates the relationship between pain and depressive symptomatology. Several of these studies are reviewed in detail later in this chapter, along with other studies that have used Williamson’s ARM.

In some publications, Williamson and colleagues (e.g., Williamson & Schulz, 1995; Williamson & Shaffer, 2000) describe the ARM as applying to illness in general, not just to pain. That is, illness and chronic diseases are also linked to depressive symptoms by virtue of the fact that they create restrictions on one’s ability to engage in routine activities. In fact, in some publications (e.g., Williamson & Shaffer, 2000), “physical illness” is added to the model, in a position parallel to that of pain.

\textit{Similar Concepts in the Chronic Pain Literature}

The concept of “activity restriction” has received some attention independent of Williamson’s work. Several pain scholars have speculated that activity restriction (or a closely related construct) serves as mechanism or pathway through which pain may lead to depressive symptomatology. For example, Ross and Crook (1995) stated:

\begin{quote}
It seems reasonable, however, to hypothesize that activity restriction associated with declining mobility and increasing dependence in later life
\end{quote}
may account for a significant portion of the association between pain and depression among older adults. (p. 11)

These authors later went on to say, “It seems reasonable to suggest that the decreased functioning associated with advancing age may be a result not only of disability, but also of pain” (p. 15). Gamsa (1990) offered a similar argument, suggesting that pain influences depressed affect primarily through its effects on “functional limitations” or difficulties in conducting routine activities and functions. Earlier, Crisson and Keefe (1988) argued that severe pain may interfere with participation in life activities, which leads to decreased opportunities for social rewards and decreased feelings of mastery and control, and this is what leads to depression. Some scholars also rely on the impact that pain has on activities and/or the level of functional disability associated with pain to help establish the “chronicity” of the pain condition (e.g., Marks et al., 2000).

Early on, Turk, Rudy and colleagues proposed a cognitive-behavioral mediation model of pain and depression (Rudy, Kerns, & Turk, 1988; Turk & Rudy, 1986). Originally, these authors were concerned with how and why some chronic pain patients developed major depression. Their model integrated concepts from both behavioral and cognitive theories regarding the development and maintenance of depression (e.g., Beck, 1967; Rehm, 1977). In general, the model proposed that depression develops in some chronic pain patients as a result of a sustained reduction in activities, reduced receipt of social and personal rewards, a host of maladaptive cognitions including declines in perceptions of personal control and mastery and attributions of helplessness. Although Turk, Rudy and colleagues never specifically labeled any mediating construct “activity restriction,” their theory does align closely with Williamson’s ARM.
Limitations of the ARM

Several limitations are associated with Williamson and colleagues’ ARM and associated empirical studies. First, at a conceptual level, “activity restriction” is defined very broadly. This broad definition makes it difficult to operationalize the concept in empirical tests of the theory. Indeed, even Williamson herself has operationalized “activity restriction” in markedly different ways across different studies. In early studies, Williamson and colleagues operationalized activity restriction as limitations experienced across a broad array of Activities of Daily Living (ADLs—e.g., eating, dressing, personal hygiene), Instrumental Activities of Daily Living (IADLs—e.g., shopping, preparing meals), and social, recreational, and leisure activities. They measured this restriction with a scale that they had developed—the Activity Restriction Scale (ARS; Williamson & Schulz, 1992b). To be sure that their study results were not specific to the ARS, Williamson and colleagues later conducted studies that used alternate existing measures and found similar results.

Second, when framed and operationalized so broadly, activity limitation need not be caused directly by pain. For example, difficulty with dressing might be due to shortness of breath, lack of dexterity, or impaired fine motor control—not to pain. Yet, Williamson and colleagues generally interpret such difficulties as resulting from pain.

Third, it is conceptually difficult to distinguish Williamson’s construct of “activity restriction” from other more commonly referenced constructs related to physical capacities and limitations. In their early work, Williamson and colleagues (e.g., Williamson & Schulz, 1992a) identify their interest in “functional disability” or “functional impairment,” and use these terms interchangeably with activity restriction. In
later work, however, Williamson and colleagues resort almost exclusively to the language of activity restriction or “restriction of normal activities” (e.g., Williamson & Schulz, 1995; Williamson, 2000a). Thus, it is never really clear how activity restriction relates to these other constructs. It is only in some later writings that Williamson attempts to re-establish the interchangeability between activity restriction and functional disability. She also attempts to draw some distinctions between the ARS measure (a more “subjective” measure of “perceived activity restriction”) and traditional ADL/IADL measures (more “objective” measures of how much help people need with different tasks; Williamson, 2000b, p. 59)—however, these distinctions are not always clear.

In addition, the Williamson ARM suffers from some broader conceptual limitations. One limitation is that the relationship between pain and physical illness is never really addressed. Another is that the exact mechanism whereby activity restriction leads to depressive symptoms is not well articulated. Finally, in light of the accumulated knowledge regarding different aspects of physical health and the interplay between physical and mental health, especially within the gerontological literature, the ARM model seems overly simplistic. The model simply aggregates various types of functioning—ranging from fundamental physical abilities (e.g., fine motor skills) to basic functional tasks (e.g., ability to dress oneself) to more complicated functional tasks that require physical abilities as well as cognitive and social skills (e.g., shopping). By combining all of these different elements into a single construct, the model creates a “black box” problem that does little to help elucidate the specific mechanism(s) through which pain can lead to depressive symptomatology. The model seems to describe, more than explain, a series of empirical relationships observed between measures of pain,
“activity restriction,” and depressive symptoms. It would be more desirable to distinguish different aspects of functioning in order to determine which are instrumental in the pain-depressive symptom relationship. Relatedly, although Williamson and colleagues have proposed and tested several factors that may influence the interrelationships depicted in the model (e.g., age, the number of chronic diseases), these factors have never been incorporated into the ARM model. Thus, the ARM really has not evolved much over time.

It is worth briefly considering whether or not the ARM could be incorporated into a more encompassing theoretical framework in order to address some of the aforementioned limitations. Williamson occasionally made vague reference to stress and coping and/or stress-process paradigms, suggesting that pain and illness are “stressors” to which an individual must adapt (Williamson & Schulz, 1992a; Williamson & Shaffer, 2000). With respect to pain specifically, Williamson and Schulz (1992a) identified two related challenges—dealing with the pain itself, and dealing with the constraints that pain imposes on one’s routine activities. In this context, depressive symptoms (and poor mental health, generally) can then be regarded as the outcome of poor adaptation. However, concepts from existing stress and coping theories, such as appraisal and coping styles (e.g., Lazarus & Folkman, 1984), are not included in the ARM or in writings about the ARM; nor are concepts from stress-process models, such as primary and secondary stressors, role strains, mastery, social support, and other resources (e.g., Pearlin & Schooler, 1978; Pearlin, Mullan, & Semple, & Skaff, 1990). To be fair, Williamson’s ARM has certainly played a pivotal role in focusing attention on the importance of pain and the role of restrictions or limitations in activity, especially within research on older
adults. The limitations just noted in the ARM may help to explain the relatively sparse attention that Williamson’s work, and the ARM in general, has received within the past few decades.

Is it possible that another theoretical framework could be tapped to help address some of the main conceptual limitations associated with Williamson’s ARM? Are there available frameworks that more clearly distinguish between certain aspects of physical functioning and more readily incorporate the influence of various demographic, background, and other factors? Broader theoretical frameworks of health and physical functioning—especially those that outline the process of disability or disablement—seem to hold promise in this regard.

**Models of Disability and the Disablement Process**

Theoretical models of disability and disablement emerged from the recognition that disability, especially disability associated with aging, tends to develop in a sequential manner. Nagi (1965, 1991) and later Verbrugge and Jette (1994) delineated a theoretical pathway of disablement, moving from pathology to disability. Figure 2 illustrates the steps in the main pathway of the Verbrugge & Jette (1994) disablement process model:

*Figure 2. The Disablement Process Model of Verbrugge and Jette (1994)*

*Note. Adapted from Verbrugge and Jette (1994).*
The Verbrugge & Jette (1994) model proposes that the disablement process begins with some sort of pathological condition—most often a biochemical or physiological abnormality that results from disease or injury (alternatively, it could represent a congenital or developmental condition). Pathology can be either acute or chronic. This pathological condition may lead to impairment—the second stage in the pathway. Impairments are defined as dysfunctions, losses, or “significant structural abnormalities” that occur in specific body systems, such as the musculoskeletal, cardiovascular, or metabolic systems (Verbrugge & Jette, 1994, p. 3). Impairment associated with a specific pathology can occur in a primary system, as well as in secondary locations. For example, chronic lung disease primarily produces impairments in the lungs, but can also produce impairments in the heart and other systems.

Functional limitations represent the third stage in the disablement pathway. They are conceived broadly as restrictions or difficulties in performing basic physical and mental tasks or activities. As Nagi (1991) noted, functional limitations occur at the level of the organism as a whole. They are limitations in generic abilities of the body and mind, and are specific to one’s age and sex group. Examples include overall mobility, discrete physical motions, and central cognitive and emotional processing functions.

The fourth stage is disability, and involves difficulty doing activities in any domain of human activity (typical to one’s age and sex). As these activities usually involve some socially defined or desired roles and behaviors, disability has a situational or social component. Disability is also ultimately determined by the level of “fit” or match between the demands of the environment and the capabilities of an individual. Verbrugge and Jette (1994) note that although commonly measured in terms of Activities
of Daily Living (ADLs) and Instrumental Activities of Daily Living (IADLs), disabilities can occur in other domains, including hobbies/leisure, recreation, and service activities.

As delineated by Verbrugge and Jette (1994), the disablement pathway can be influenced by a host of intra- and extra-individual factors, including demographic characteristics, biological predispositions, personal lifestyle factors, medical access and intervention, and external supports. These factors can influence the disablement process by acting as risks, buffers, and/or exacerbating agents. One critical feature of this theoretical pathway is that difficulties in basic physical functioning precede difficulties in role and social functioning. The disablement process may also lead over time to more global outcomes, such as reduced quality of life. Although Verbrugge and Jette (1994) did not offer much elaboration of the possible extensions of the pathway, they did specifically identify depression as one possible outcome of the disablement process.

Progression through the pathway is not inevitable. So, for example, Nagi (1991) argued that not all impairments lead to functional limitations, and not all functional limitations lead to subsequent disability. In addition, impairments can arise in the absence of active pathology, and disability can occur in the absence of a specific functional deficit (Pope & Tarlov, 1991). Nor is the pathway solely unidirectional, as feedback loops are possible (Verbrugge & Jette, 1994). Secondary conditions and dysfunctions can also develop, and new disablement processes can emerge and co-occur in the same individual. Nonetheless, the pathway is the most direct way through which pathologies and their associated impairments lead to disability (Nagi, 1991).

Nagi (1969) mentioned pain within his discussion of impairments, noting that impairments can vary in terms of “the degree of pain” (p. 11) with which they are
associated. In later writings (e.g., Nagi, 1979, 1991), he mentioned pain along with loss of structural integrity and pathological findings as factors that can signal the presence of impairments and underlying pathology. Pain is also conceptually similar to other impairments discussed by Nagi, such as muscle loss, weakness, and endurance.

Pain is addressed only indirectly within the Verbrugge & Jette (1994) framework. In offering an example of how a secondary condition and dysfunction can occur, Verbrugge and Jette discussed a woman with “painful arthritis [who] may restrict her recreational walking [that] eventually reduces her cardiopulmonary function…” (1994, p. 7). In this example, pain is identified as a characteristic or feature of a chronic condition. The authors also mentioned “vertebrogenic pain syndrome” as an example of a diagnosed pathology (Verbrugge & Jette, 1994, p. 3). Thus, in the Verbrugge and Jette model and the Nagi model, pain is most readily conceptualized as an impairment. Some chronic pain conditions (e.g., fibromyalgia) could alternatively be conceptualized as pathological conditions.

Verbrugge & Jette (1994) argued that the disablement process influences global quality of life outcomes, such as life satisfaction and general well-being. Verbrugge (1990) and Verbrugge & Jette (1994) outlined a number of possible mechanisms whereby the disablement process can influence quality of life. Functional impairment and resulting disability can negatively affect an individual’s sense of autonomy, compromise one’s sense of self, and create feelings of helplessness. Moreover, they can result in loss of valued roles and can disrupt one’s social relationships. These effects, in turn, can threaten general well-being. Although not explicitly labeled “activity restriction,” the concepts of limited or reduced capacity to perform basic tasks (functional impairments)
and/or to engage in or complete routine activities (disabilities) are central elements in the Verbrugge & Jette (1994) model of the disablement process.

**Pain in the Illness-Disability Cascade**

There have been a few formal attempts to position pain within the disablement process. Kahana, Kahana, Namazi, Kercher, and Stange (1997) explicated an “illness-disability cascade” that built upon the disablement process outlined by Nagi and Verbrugge and colleagues. This model is depicted in Figure 3.

*Figure 3. The Illness-Disability Cascade of Kahana et al. (1997)*

![Illness-Disability Cascade Diagram](image_url)

*Note.* Adapted from Kahana, Kahana, Namazi, Kercher, & Stange (1997, p. 186).

The Kahana et al. framework more clearly outlines the role of pain in the disablement process and outlines linkages among the various elements of the model. They also identify several quality of life end-points in the cascade, including social functioning and various dimensions of psychological well-being (e.g., life satisfaction, self-esteem). In the figure, pain and depression are presented in boldface, underlined type, as
they were the primary focus of the empirical study reported in Kahana et al. (1997).

By positioning pain within a cascade from chronic illness to disability to reduced psychological well-being, Kahana and colleagues hoped to move toward increased understanding of the mechanisms underlying the relationship between more “subjective” elements of health (such as the experience of pain) and more “objective” elements of health (e.g., psychological morbidity; Kahana et al., 1997, p. 187). Kahana et al. defined physical impairments as organ-level dysfunctions that are reflected in deficits in sensory functioning (e.g., vision deficits), physical functioning (e.g., limited ability to reach), and discomforts (e.g., pain, dizziness). They also expanded the conceptualization of disability to include both limitations in personal activities (ADLs, IADLs) and social activities (e.g., maintaining interactions with family/friends, social participation, volunteering). The ultimate outcome of the “cascade” is psychological well-being, conceptualized broadly to include not only depressive symptoms/depression and negative affect, but also other diverse aspects of psychological well-being (e.g., life satisfaction). The model identifies different types of chronic illness that serve as antecedents to pain and other impairments. It also specifies several important demographic and social-structural characteristics (e.g., age, gender, income) that can influence the cascade.

For researchers interested in explaining the relationship between pain and psychological distress, the Kahana et al. (1997) model makes several contributions. First, it clearly highlights pain and places it within a larger conceptual framework of physical health and the disablement processes. Second, it extends the disability “cascade” to a psychological outcome. In this way, physical health status and functioning are linked to psychological well-being and, more importantly, to psychological distress (e.g.,
depressive symptoms, negative affect). Third, the Kahana model clearly specifies one mechanism through which pain can lead to psychological distress, via the development of physical and social disabilities. In other words, personal and social disabilities serve as mediators between pain and psychological well-being.

Although Kahana et al. (1997) do cite the findings of Williamson and colleague’s empirical work, their model does not explicitly draw upon Williamson’s ARM (e.g., Williamson & Schulz, 1992a). However, by emphasizing pain’s role in the disablement process and relating physical health to mental health outcomes via constructs of reduced physical functioning and disability, the Kahana et al. model incorporates several key elements and relationships outlined in the ARM. Furthermore, by distinguishing between personal disability (i.e., physical functioning—e.g., IADLs and ADLs) and social disability (i.e., social functioning—e.g., social activity performance), the Kahana et al. model indirectly helps to identify some different elements of the activity restriction “black box” – thereby addressing a clear conceptual limitation of Williamson’s ARM.

Still, researchers interested in using the Kahana et al. (1997) model to study pain in relation to depressive symptomatology are confronted by several conceptual limitations. First, in addition to considering pain to be an impairment, the model also considers performance deficits (i.e., difficulties in basic physical functions like mobility difficulties or difficulties reaching) as impairments. Thus, the model essentially equates functional limitations, as outlined in the Verbrugge and Jette (1994) model, with physical impairments, despite conceptual and empirical efforts to distinguish these constructs by others (e.g., Jette, Branch, & Berlin, 1990; Nagi, 1991; Verbrugge & Jette, 1994). This limitation is compounded further in the Kahana et al. (1997) empirical test of the
model—the study did not include any indicators of these performance deficits (e.g.,
difficulties in basic physical functions). Thus, the relationships between difficulties in
physical functioning and pain, subsequent disability, and outcomes are unknown.

Second, the Kahana et al. (1997) model emphasizes the constructs of personal and
social disability as the intervening mechanisms in the relationship between pain and
psychological well-being. Personal disability was defined as difficulty in performing
IADLs and ADLs. Social disability was defined as “restriction or limitation in
performance of customary and valued social activities.” (p. 188). There are two
shortcomings associated with an exclusive emphasis on disability as the intervening or
mediating variable in the relationship between pain and psychological well-being. One
shortcoming is that disability is a complicated construct that poses problems both
conceptually and operationally. The construct of disability has been the focus of
considerable debate across a variety of fields (Nagi, 1991; Pope & Tarlov, 1990). Many
authors distinguish disability from other physical disablement constructs (e.g.,
“functional limitations,” “physical impairments”) by noting that disability necessarily
involves interaction with the demands and resources in the environment (e.g., Lawton,
1983; Pope & Tarlov, 1990; Verbrugge & Jette, 1994). Thus, disability can be influenced
by a variety of factors, including the use of assistive devices, cognitive limitations,
gender-specific social roles, limited exposure to certain task demands, social and
instrumental support, and the availability of specific types of assistance (e.g.,
transportation). Kahana herself has, in other work, discussed the importance of the “fit”
or congruence between the person and the task demands of the environment (e.g.,
Kahana, 1974, 1982). Because disability results from an interaction of the person and the
Given this conceptual confusion, models that emphasize disability should attempt to account for at least some of the environmental and task demands, as well as external resources that can influence disability; yet, these other constructs are rarely incorporated into conceptual models. Building on the conceptual confusion (or perhaps because of it), the ways in which disability is typically operationalized often results in contamination of the disability construct by many of the concepts mentioned above. For example, most measures of ADLs and IADLs (e.g., ability to dress, cook) do not measure pure physical ability, but a combination of physical and cognitive abilities (Willis, 1996). Social roles, personal preferences, task demands, the physical environment, and available support can also shape ADL and IADL performance (Jette, 1996).

The second shortcoming of focusing exclusively on disability as a mediating construct in the relationship between pain and depressive symptomatology (in particular) is that the resulting empirical research cannot adequately defend against the possibility that depressive symptomatology caused the disability, and not the reverse. This is especially true when disability is operationalized in traditional fashion, resulting in measures that tap some of the other factors that influence disability. For example, in assessing social disability, Kahana et al. (1997) measured the frequency with which individuals engaged in a variety of valued social activities, including participation in hobbies, leisure pursuits, and social events. Two distinguishing features of depression—anhedonia and lack of positive affect—are often signaled behaviorally by loss of pleasurable engagement with the environment, disinterest, and withdrawal (Watson & Kendall, 1989). Thus, decreased involvement in hobbies and leisure activities, as well as
lack of social participation, easily could be consequences of depressive symptomatology, rather than the cause of it.

To be fair, this limitation is not exclusive to the Kahana et al. model; it can be attached to both Williamson’s ARM and the Verbrugge and Jette (1994) models. However, both of these other models incorporate separate constructs (or at least additional indicators) that reflect more basic physical limitations or impairments. The causal direction of the relationship between depressive symptoms and these basic physical limitations (e.g., difficulty walking, bending, climbing stairs) may be less debatable than the direction of the relationship between depressive symptoms and, say, ADLs or social disability.

An Extension of the Kahana et al. (1997) Model

The Kahana et al. (1997) model of pain in the disability cascade has not been widely used, nor has it been cited with much frequency even within the relatively sparse literature on pain and depressive symptomatology among older adults. One notable exception is a paper by Bookwala, Harralson, and Parmelee (2003). These authors attempted to integrate Williamson’s ARM into a broader theoretical framework of physical disablement. They developed a conceptual model that incorporated elements of both Williamson and colleagues’ ARM and Kahana et al.’s (1997) disability cascade model, and used the model to study pain, functioning, and quality of life.

The Bookwala et al. (2003) model is based on a central tenet of Williamson’s ARM—i.e., that a restriction of activities mediates the relationship between pain and depressed affect. The model incorporates Kahana et al.’s (1997) distinction between physical and social disability (now labeled physical and social functioning). Bookwala et
al. also expanded the model to encompass one additional quality of life outcome—perceived global health. The authors proposed that the relationships between pain, physical and social functioning, and physical well-being (i.e., perceived global health) mirror those that exist in the relationships between pain, physical and social functioning, and psychological well-being. In their model, physical and social functioning are hypothesized to mediate the relationship between pain and global physical health, just as they are thought to mediate the relationship between pain and psychological health.

The Bookwala et al. (2003) model represents a major advance theoretically, in that it formally marries Williamson and colleagues’ ARM to a more comprehensive conceptual framework focused on the role of pain in the disablement process (i.e., Kahana et al., 1997). However, the Bookwala et al. model shares some of the same limitations that confront each of its component frameworks. In particular, there are difficulties associated with how Bookwala et al. define and operationalize physical and social functioning. The physical functioning construct encompasses a broad range of basic physical tasks (e.g., walking, bending) as well as IADLs (e.g., household tasks) and ADLs (e.g., self-care). Measures of social functioning are similarly broad, assessing participation in various social, recreational, and leisure activities (e.g., attended a senior center, took a walk). Thus, the Bookwala et al. (2003) model, like Williamson’s ARM, also relies on a very broad definition of physical functioning, making it difficult to disentangle components of the mediating construct. Like the Kahana et al. (1997) model, the Bookwala et al. (2003) model also has a relatively heavy emphasis on disability, especially in terms of how social functioning is operationalized. One additional limitation is that, as it is currently developed, the Bookwala et al. (2003) model is specific
to osteoarthritis pain. That is, osteoarthritis pain is thought to lead to detriments in physical and social functioning, which in turn compromise both physical and psychological well-being. Although it might be possible to extend the model to other types of pain or other populations, this has not yet been done.

Like the Kahana et al. model, the Bookwala et al. (2003) conceptual model has not received much attention within the literature exploring the relationship between pain and depressive symptomatology. This lack of attention may be due, in part, to some of the aforementioned limitations. In addition, both models were derived from, and published within, the field of aging, and this may have further limited their exposure to pain researchers. The specific findings of both studies are reviewed in detail later in this chapter; however, it should be noted that, although some aspects of each model were supported by the authors’ published findings, neither model represented a good fit overall to the observed data. Finally, although it is commendable that both the Bookwala et al. (2003) and the Kahana et al. (1997) models go beyond depressive symptomatology as an outcome, this extension may be a bit premature, given the current state of knowledge. Considerable gaps remain in the knowledge base regarding the relationship between pain and depressive symptomatology and the processes through which this relationship is created and maintained.

*Proposed Conceptual Model Linking Pain and Psychological Distress*

*at the Intra-Individual Level*

Figure 4 offers a proposed conceptual model for studying the impact of pain on psychological distress in individuals. The proposed model builds upon all three of the conceptual frameworks discussed above—the activity restriction model of depressed
Figure 4. Proposed Conceptual Model Linking Pain and Psychological Distress in Individuals
affect (e.g., Williamson & Schulz, 1992a), models of disability or disablement (e.g., Nagi, 1965; Verbrugge & Jette, 1994), and the model of pain in the disability cascade (Kahana et al., 1997). The model is not specific to persons in mid and later life, nor is it specific to married persons. Rather, the model was applied to midlife and older spouses within the context of the current study. This model will be expanded to the couple or inter-individual level in the next chapter (Chapter 3).

In recognition of the importance of framing the investigation of pain-depression mediation hypotheses within a broader theoretical paradigm related to health and disability (as noted by Kahana et al., 1997, and Bookwala et al., 2003), the proposed model incorporates elements of the disablement process. Consistent with the main disablement pathway outlined by Nagi (1975) and Verbrugge and Jette (1994), chronic health conditions are proposed to contribute to pain, and pain is thought to be a major contributor to difficulties in physical functioning. Pain is also proposed to limit or interfere with usual activities, consistent with Williamson’s notion of activity restriction (Williamson & Schulz, 1992a). Consistent with the Kahana et al. (1997) cascade model, the proposed model highlights the role of pain within the disablement process and explicitly links pain to psychological functioning. Although several dimensions of psychological distress could be integrated into the proposed model, the model specifically focuses on depressive symptomatology.

Williamson’s ARM and subsequent models of pain within the disablement process (e.g., Kahana et al., 1997; Bookwala et al., 2003), refer to “pain” in the abstract and appear to conceptualize pain as a unidimensional construct. Theoretically, various psychological dimensions of pain (e.g., presence/absence, intensity, duration) could be
associated with distress, and could be explicitly studied using the model. However, only one dimension of pain is identified in Figure 4—pain intensity, i.e., the magnitude or level of pain. Intensity was the only measure of pain routinely administered in HRS, and thus was the only dimension considered in the present study. Note that intensity is the dimension most commonly assessed in studies of pain (Jensen & Karoly, 2001), and is considered by many to be the most salient dimension (e.g., Turk & Melzack, 2001).

In the model, pain is proposed to affect psychological distress directly (Path A). Pain is also expected to have an indirect effect on psychological distress. Drawing on Williamson’s activity restriction model, pain’s indirect effects are expected to occur primarily through (i.e., be mediated by) activity limitation. These indirect effects are represented in Figure 4 by the combination of Paths B and C. In the proposed model, activity limitation can be defined generally as difficulty that an individual experiences in performing his/her usual activities.

Although a variety of constructs could potentially function as mediators in the relationship between pain and depressive symptomatology (e.g., coping styles, social support), the proposed model’s emphasis on activity limitation is generally consistent with existing conceptual models (e.g., Williamson’s ARM; Verbrugge & Jette, 1994). It is also congruent with a sizeable body of existing empirical research (some of which was discussed earlier) that has established poor physical health as a major risk factor for depression and/or depressive symptomatology. Moreover, as will be made clear in the literature review that follows, focusing on activity limitation provides an opportunity to marry two seemingly independent avenues of research that have emerged within the fields of gerontology (e.g., Kahana et al., 1997; Bookwala et al., 2003) and chronic pain
“Activity limitation” was preferred over the label “activity restriction” (the term proposed by Williamson and colleagues in the ARM) for two reasons. First, the latter term has not been clearly defined within applications of the ARM. Two scales appear to have been developed around Williamson’s (largely implied) definition of activity restriction—Williamson’s own Activity Restriction Scale (ARS) and the Groningen Activity Restriction Scale (GARS, Suurmeijer et al., 1994). Although the ARS has been used in much of Williamson’s own work and the GARS has been used in some international studies (e.g., Kempen et al., 1996), neither scale has been widely adopted. The lack of adoption may be at least partially attributable to item (and conceptual) overlap with other, well-validated measures of ADLs, IADLs, and physical impairment.

Second, the term “activity restriction” seems to have been adopted by some gerontological researchers to characterize more intentional or volitional restriction of activities—for example, actions that an aging person might take in order to avoid accidents or injuries, such as falls (e.g., Deshpande, Metter, Lauretani, Bandinelli, Guralnik, & Ferrucci, 2008; Fletcher & Hirdes, 2004). Although pain could provide an impetus for restricting one’s activities, there are undoubtedly a host of other reasons to do so, including fear, medical advice, legal proscription, and budgetary concerns. In addition, most would agree that many of the physical difficulties associated with pain do not result from an exercise of individual choice or will.

Although activity limitation could be conceptualized as a multidimensional construct, the proposed model is primarily concerned with activity limitation of a physical nature. Thus, physical limitations are defined as difficulties in performing basic
physical functions such as lifting, walking, stooping, reaching, etc. This definition is thus consistent with the concept of “functional limitations” offered by the Nagi (1991) and the Verbrugge and Jette (1994) models of the disablement process. Selected physical limitations have also been included (albeit intermittently) as indicators of activity restriction in Williamson & Schulz’s ARM studies (e.g., Williamson & Schulz, 1992a). Physical limitations occur relatively early in the course of a disease or in the aging process (Verbrugge, 1990). Although they underlie and have an impact on functioning in broader spheres of life (e.g., vocational, social, ADL, IADL functioning), they are not specific to any one task or area of functioning. As specified in the proposed model, pain intensity is hypothesized to be a primary contributor to these physical limitations, which are then hypothesized to be associated with greater depressive symptomatology.

The term “physical limitations” was selected for three reasons. First, the term was selected to reflect a primary interest in physical functioning, rather than other areas of functioning (e.g., cognitive, sensory) which might also interfere with an individual’s ability to perform complex tasks or activities. Second, physical limitations was selected as the most parsimonious—yet conceptually clear—term from among possible alternatives, including “physical functional limitations” (e.g., Lichtenstein, Dhanda, Cornell, Escalante, & Hazuda, 1998), “physical disabilities” (e.g., Guccione et al., 1994), “lower [or upper] extremity disability” (e.g., Volpata et al., 2003). Third and finally, the term was preferred over “functional limitations” because of the conceptual confusion that commonly surrounds the latter term. Within the gerontological literature, functional limitations refer almost exclusively to difficulties in performing ADLs and IADLs (e.g., Beckett et al., 1996; Mendes de Leon et al., 1999). Even when authors specifically define
functional limitations as difficulties in basic physical abilities, audiences tend to ignore this definition and assume that the term references limitations in (instrumental) activities of daily living. It should be noted that Verbrugge occasionally used the term physical limitations to distinguish difficulties in basic physical abilities from other types of limitations or disabilities (e.g., Verbrugge & Lepkowski, 1989).

There are both conceptual and practical reasons for focusing specifically on physical limitations. As will be discussed later in this chapter (in the literature review section), Williamson herself has suggested that it may be the earlier limitations in functioning and losses of ability that most clearly lead to the development of depressive symptoms in the face of pain. By the time a person loses functioning in terms of IADLs or ADLs, the damage (so to speak) has already been done, or perhaps the person has already come to accept (even expect) such losses (e.g., Williamson & Schulz, 1992a, 1995; Williamson, 2000b). By conceptually limiting the construct to only basic physical limitations, the present study was able to examine this suggestion empirically. Although at a conceptual level, IADL and ADL functional disabilities could be (and have been—e.g., Williamson & Schulz, 1992a) classified as different types of “activity limitation” that could mediate the pain-depression relationship, a focus on physical activity limitation helps the proposed model avoid some of the shortcomings associated with a focus on disability (see critique of models by Kahana et al., 1997, and Bookwala et al., 2003).

Conceptual and practical support for an emphasis on physical limitations can also be found in the study design and methods utilized in the data source used for the current study—the Health and Retirement Study. HRS includes both middle-aged and older adults (generally over age 50). An analysis of HRS panel data (NIA, 2007) suggested
that HRS participants demonstrate the same trends that have been observed elsewhere regarding the preservation of functional abilities well into old age and the “compression of morbidity” among those in old age (e.g., Manton et al. 2006). ADL and IADL limitations are reported by very few middle-aged participants and relatively low numbers of older HRS participants. From a conceptual standpoint, such prevalence patterns have generated interest in studying changes in physical functioning that occur earlier in life—often in middle age—and that precede the development of ADL and IADL limitations (e.g., Fried et al., 1997; Verbrugge et al., 1999). From a practical standpoint, adequate testing of any mediation model requires sufficient range and dispersion of scores on the mediating construct—characteristics more likely to be observed in the physical limitations reported by HRS respondents than in their reports of ADL and IADL impairments. Thus, HRS data provided a tangible (and practical) opportunity to examine physical limitations as a conceptually distinct mediating construct in the relationship between pain and psychological distress.

Activity limitation has been of interest within the chronic pain literature for many years. Here—in contrast to the gerontological literature—activity limitation is most often conceptualized as the direct result of pain. However, different scholars have defined and labeled the construct in different ways. This construct has been labeled “pain-related disability” (Pollard, 1984), “pain interference” (e.g., Kerns, Turk, & Rudy, 1985), and “pain impact” (e.g., Flor, Turk, & Rudy, 1989). Chronic pain researchers have also disagreed about the number and types of indicators that should be used to measure the construct. This has led many to develop their own measures (e.g., Pain Disability Index [PDI; Pollard, 1984]; Pain Interference Scale, Kerns et al., 1985). Upon inspection, most
of these measures contain very similar content—i.e., the extent to which pain interferes in various spheres of life (e.g., physical, social, occupational). In publications, however, chronic pain researchers tend to use the name of the scale to refer to the construct—a practice that promotes confusion both within and outside the field. Many researchers studying disease conditions in which pain is a prominent feature have followed a similar path. Various tools have been developed to assess the impact of pain (and other symptoms of the disease) on daily tasks or different areas of functioning. For example, studies of persons with rheumatoid or osteoarthritis often utilize the Arthritis Impact Measurement Scales (AIMS; Meenan, Gertman, & Mason, 1980).

The proposed conceptual model has several unique advantages over existing models. First, pain features prominently in the proposed conceptual model. Pain is worth considering on its own, not just in relation to its role as a symptom of illness or disease. Research shows that pain reports are not always directly correlated with objective evidence of disease. For example, radiographic evidence of hip joint damage in osteoarthritis patients is often only weakly correlated with reports of pain (e.g., Birrell et al., 2000). Pain can also occur in the absence of a detectible disease or illness. In one study, over 20% of adults over age 65 who reported recent musculoskeletal pain stated that they did not know the cause of their pain (Scudds & Robertson, 2000). The proposed conceptual model also goes further, to focus specifically on pain intensity—a dimension of pain that has received relatively limited attention within studies of the relationship between pain and depressive symptomatology.

Second, the model makes clear that pain can affect psychological distress directly (Path A—e.g., higher pain intensity can lead to greater depressive symptomatology) and
indirectly (Paths B & C—e.g., higher pain intensity can lead to greater depressive symptoms through increased physical limitations). While prior models (e.g., Kahana et al., 1997) have focused mainly on the mediating role of disability in the relationship between pain and depressive symptoms, the proposed model considers the possible mediating role of more basic limitations in physical abilities (e.g., difficulties lifting, bending, stooping, pushing)—limitations likely to occur earlier in the disablement process and at younger ages (Verbrugge, 1999). Existing models (e.g., Williamson’s ARM) generally have not identified specific dimensions of activity limitation.

Third, as depicted in the model, background characteristics and health-related characteristics can influence all elements of the model, including the relationship between pain and depressive symptomatology. Drawing on Verbrugge & Jette’s (1994) suggestion that other factors that can influence the disablement process, the proposed model includes one primary, health-related risk factor that has been demonstrated to increase risk of pain and activity limitation (and sometimes also depressive symptoms)—body weight, especially overweight and obesity. Evidence of these relationships is reviewed later in this chapter. Also consistent with Verbrugge & Jette (1994), the proposed model makes clear that the effect of disease conditions on depressive symptomatology is not expected to be wholly mediated by pain. Disease conditions can contribute to physical limitations through mechanisms other than pain—for example, breathing difficulties associated with chronic lung disease can lead to problems walking, and circulation problems associated with cardiovascular disease can produce muscle weakness leading to difficulties with climbing stairs. Disease conditions may also contribute to psychological distress directly or through mechanisms not included in the
model. In addition, the model allows for investigation of both potential direct and indirect effects of demographic characteristics such as gender, age, or SES. Thus, the proposed conceptual model both builds on the strengths of existing models and empirical evidence and offers some advantages over existing models.

Review of the Literature Specific to the Proposed Conceptual Model

This section reviews the empirical literature related to two general relationships depicted in the conceptual model presented in Figure 4. These two relationships have been relatively understudied, and limitations within the existing literature base provide ample opportunities for contribution. These relationships were selected as foci for the current study because knowledge development in these areas has the potential to advance both the theoretical and practical understanding of the experience of pain within married couples. The first relationship concerns the primary etiological factors associated with pain—in particular, the background and health-related characteristics thought to be related to pain intensity. Research regarding these etiological factors is reviewed first. The second subsection reviews evidence concerning the second, and central relationship under study—the relationship between pain intensity and psychological distress (viz., depressive symptomatology). In addition to summarizing evidence that supports the proposed conceptual model, this review also highlights gaps in the existing knowledge base—specifically emphasizing those gaps that were addressed in the present study.

Pain intensity was the focal dimension considered in the present study. In some areas, however, research specific to pain intensity is fairly sparse. In addition, existing measurement tools and nomenclature often confound intensity with other dimensions of
pain. Where necessary, this review is supplemented with study findings related to pain generally—i.e., pain, broadly writ, to include reports of pain and other closely-related or ill-defined characteristics of pain (e.g., severity, frequency, number of pain sites, etc.).

This review emphasizes empirical studies conducted in populations that most closely resemble the focal sample for the present study—i.e., middle-aged and older, community-dwelling couples in which both spouses have pain. This author is unaware of any large-scale study that has specifically sampled mid and later life couples and that has also collected detailed and comparable information about pain from each spouse. Thus, the majority of studies included in this review are based on data collected from individuals who have been sampled without regard to marital status.

That said, this review does include roughly a dozen studies that have been conducted with samples comprised solely of married (or partnered) persons with pain. Such studies are challenging to identify because the nature of study samples is not always clearly conveyed in published titles and abstracts. Most of these studies emanate from larger research projects focused on married couples in which one partner is the identified “patient” or “person with pain.” As such, most samples have been drawn from clinical settings, with the majority comprised of patients in chronic pain treatment programs or outpatients in specialized medical practices (e.g., rheumatology or orthopedics). A limited number of studies have recruited community-dwelling married couples in which one partner suffers from pain. Research teams tend to carve up study data into small units for publication. As a result, some publications focus on the “patients,” others focus on the (non-patient) spouses, and, more rarely, some consider data from the couple. Different amounts and treatments of missing data, along with the publication of results
based on preliminary samples (i.e., recruitment not yet completed), make it difficult to pinpoint those studies that represent unique contributions to the literature.

Note that, although the samples in these studies are comprised of married persons with pain, these persons are generally not married to each other. These studies are therefore more appropriately characterized as studies of unrelated married individuals (generally referred to hereafter as studies of married “individuals” or married “persons”). Where available, findings from the studies of married persons with pain are compared with those from studies using samples selected without regard to marital status.

Because so few studies of community-dwelling adults in mid and later life have included detailed questions about pain, this review also draws on evidence obtained from studies conducted with related populations. Studies of chronic pain patients were specifically included in this review—a decision buttressed by evidence and observations presented at the beginning of this chapter suggesting that much of the pain experienced by midlife and older adults can be considered persistent or chronic in nature. Studies conducted with samples of persons with diseases in which pain is assumed to be prominent feature (e.g., osteoarthritis, cancer) are also included. Studies of both chronic pain patients and (clinical or community) samples of persons with painful diseases are directly relevant to the present study because such studies have more often examined the predictors and correlates of pain intensity, whereas studies with community-dwelling populations have most often examined predictors and correlates of the presence of pain. Where possible, this review highlights differences and/or limitations in findings that appear to be related to the sampling or recruitment strategies used (e.g., types of settings, eligibility and exclusion criteria), or to the criteria used to establish the presence and/or
chronicity of pain. Special attention is also paid to any apparent differences linked to other methodological features (e.g., measures, statistical techniques, control variables).

In the review, occasional reference is made to the findings of experimental or laboratory-based studies. This is generally limited to instances in which more controlled studies: (a) would help to establish a compelling rationale for further investigation of a particular construct or relationship, (b) appear to contradict existing observational/correlational research; or (c) would provide a useful context from which to interpret the results of observational studies.

Factors Related to Pain and Pain Intensity

This subsection is concerned with the exogenous factors in Figure 4 found to predict or be associated with pain, especially pain intensity. Existing empirical data are summarized to provide support for the inclusion of these factors in the conceptual model. This review provides a foundation for the research questions and hypotheses associated with Study Aim 2 (to determine which demographic and health-related characteristics are associated with pain intensity in the focal sample of dual-pain [DP] couples) and Study Aim 5 (to evaluate gender differences). The review is organized around the two major groupings presented in Figure 4: Background characteristics are reviewed first, followed by health-related characteristics. An overall summary concludes the section.

Background Characteristics

Gender

Because of interest in gender differences and the central role of gender in the study of married couples, the existing literature regarding gender and pain is reviewed in greater depth than the literatures relating pain to other background characteristics. A
summary and critical analysis of the literature concerning pain and gender is provided. This summary is especially relevant because, as will be seen, many of the same issues raised in the review of studies of gender differences in pain and pain intensity also apply to research on other background and health-related factors.

Prior to considering evidence of gender differences specific to pain intensity, it is important to establish that, in general, women are more likely to report pain than are men. Experimental data suggest that, compared to men, women have lower pain thresholds (i.e., they respond to painful stimuli sooner) and exhibit less pain tolerance (i.e., they endure painful stimuli for a shorter time) than men (see reviews by Berkley, 1997; LeResche, 1995). Women also report more naturally-occurring pain. Based on a review of over 100 epidemiological studies, Unruh (1996) concluded that women are more likely than men to experience a variety of recurrent pains common to both genders (e.g., headache, oral/facial, and musculoskeletal pain). This pattern has been found in more recent population-based studies, as well. In the 2003 National Health Interview Survey of U.S. adults, significantly more women than men reported joint pain in the past 30 days (34% vs. 28%; National Center for Health Statistics [NCHS], 2006).

Gender differences in reports of pain have also been documented specifically in community-dwelling middle-aged and older adults. Miller and Talerico (2002) observed that, across studies of persons over age 65, there was a clear pattern of women reporting more pain than men. Although effect sizes have varied, and not all studies have been consistent, the majority of studies to date have found significant differences in reports of pain by gender. In one large Canadian study of adults age 70 and over, 59% of women and 48% of men reported experiencing any bodily pain in the past month (Scudds &
Similar findings have emerged in studies of different types of pain among older adults in the U.S., including abdominal, headache, and joint pain, including pain in the hips (e.g., Christmas et al., 2002) and knees (e.g., Andersen, Crespo, Ling, Bathon, & Bartlett, 1999). One notable exception has occurred in studies of back pain in later life, in which no consistent gender pattern has been observed (LeResche, 2000). Few studies have focused on middle-aged adults exclusively, however, gender differences have generally been found in studies that have included both midlife and older adults. For example, among the over 13,000 adults age 51 and over who participated in HRS 2000, more women reported being “often troubled by pain” (31%) than did men (25%; Reyes-Gibby, Aday, Todd, Cleeland, & Anderson, 2007).

Women are also more likely to experience chronic or persistent pain (LeResche, 2000; Unruh, 1996), and to report multiple pain sites (LeResche, 2000). Perhaps as a result, women tend to be over-represented in specialty pain clinics (Robinson, Riley, & Meyers, 2000). Gender differences in chronic or persistent pain also appear to extend across cultures. In a 15-country World Health Organization (WHO) study, the odds of women reporting persistent pain were roughly 70% greater than those of men (Gureje, Von Korff, Simon, & Gater, 1998). Nearly 25% of women reported persistent pain, as compared to 16% of men, suggesting that gender differences in persistent pain may not be unique to Western cultures. Gender differences of similar magnitude have also been observed in the reports of chronic pain among community-dwelling samples of midlife and older adults (e.g., Christmas et al., 2002; Saastamoinen et al., 2005).

Evidence suggests that gender differences in reports of pain are not simply a function of women being more susceptible to—or more likely to be diagnosed with—
certain chronic pain conditions (e.g., temporomandibular, i.e., jaw, disorders, migraines, and fibromyalgia; LeResche, 1999) or diseases in which pain is a prominent symptom (e.g., irritable bowel syndrome, Ringel, Sperber, & Drossman, 2001; rheumatoid arthritis, Helmick et al., 2008; knee and hand osteoarthritis, see meta-analysis by Srikanth, Fryer, Zhai, Winzenberg, Hosmer, & Jones, 2005). Even among persons with the same condition, women are more likely than men to report pain. For instance, women with osteoarthritis are more likely to report pain than are men with osteoarthritis (e.g., Davis, 1981). Similar findings have been reported in diseases such as cancer and multiple sclerosis (Unruh, 1996). Furthermore, gender differences in reports of disease-related pain have been shown to persist, even when researchers control for disease severity, as has been done in studies of osteoarthritis pain (e.g., Felson et al., 1995).

Beyond gender differences in reports of pain, there is a growing body of evidence showing that women report higher levels of pain intensity than do men (e.g., Unruh, 1996; Berkley & Holdcroft, 1999; Fillingim, 2000; Fillingim et al., 2009; Roger et al., 2009). Evidence is most strong in laboratory studies where, for example, women have been found to rate equivalent stimuli as being of greater intensity or severity than men (see reviews by Berkley, 1997, and Fillingim et al., 2009).

Gender differences in pain intensity ratings have been observed in community-based studies of adults, as well. In the U.S., intensity ratings for several different types of pain (headaches, general musculoskeletal pain, pain in specific joints) have been found to vary by gender. For instance, 10% of adult females age 18 and over rated their joint pain as “severe,” as compared to 7% of males (NCHS, 2006). However, intensity ratings for some types of pain in the general population—especially low back pain—have not shown
consistent gender differences (Unruh, 1996).

Gender differences have also been found in the intensity of chronic or persistent pain reported by adults living in the community. In a population-based study of persistent pain among adults in Sweden, more women rated their pain at the maximum level of intensity than did men (24% vs. 16%; Andersson et al., 1993). Among community-dwelling adults reporting chronic pain in Canada, the U.K., and other European countries, females generally report more severe pain than their same-aged male peers (e.g., Breivik, Collett, Ventafridda, Cohen, & Gallacher, 2006; Smith et al., 2001; VanDenKerkhof et al., 2003).

Gender differences in pain intensity have also been observed in studies of middle-aged and older adults living in the community. Among community-dwelling midlife and older adults reporting pain, women tend to endorse greater levels of pain intensity than do men. In the HRS 2000, middle-aged and older women were more likely to report that their pain was severe than were similarly-aged males (21% vs. 17%; Reyes-Gibby et al., 2007). Data from later waves of HRS have shown similar differences in reports of moderate and/or severe pain (Covinsky Lindquist, Dunlop, & Yelin, 2009; Shi et al., 2010). In a Canadian sample, 68% of women over age 70 reported at least moderate pain intensity, compared to only 60% of their same-age male peers (Scudds & Ostbye, 2001). Krueger and Stone (2008) reported that the average pain intensity ratings of midlife and older U.S. women were significantly higher than those of their male counterparts.

Gender differences in pain intensity ratings have also been found in some, though not all, clinical populations. For example, Idler (1993) found that being female had one of the strongest (positive) relationships with the level of musculoskeletal pain (a
combined measure of amount and intensity) reported by adult rehabilitation outpatients. In group-matched samples of rheumatoid and osteoarthritis patients, the average daily joint pain ratings of women were 72% greater than those of men, regardless of arthritis type (Affleck et al., 1999). Note, however, some studies of patients with rheumatoid arthritis have not found significant differences in ratings of pain intensity by gender (e.g., Smedstad et al., 1995). Although some studies of pain among cancer patients suggest that females report greater pain intensity than males (e.g., Cleeland et al., 1994; Pud, 2011), findings from other studies have failed to find evidence of gender differences (e.g., Edrington et al., 2004; Turk & Okifuji, 1999).

In studies of chronic pain patients, findings regarding gender differences in pain intensity have been more variable. Although some studies have documented greater pain intensity in females (e.g., Hasvold & Johnson, 1993; Krogstad et al., 1996), some studies have actually found higher ratings of pain intensity for male patients (e.g., Marcus, 2003). Still others have found no differences in the pain intensity ratings of male and female chronic pain patients (e.g., Koegh, McCracken, & Eccleston, 2005; Robinson et al., 1998; Turk & Okifuji, 1999).

Studies conducted with samples comprised of married individuals with pain have rarely focused on gender differences in the pain experience. Nonetheless, a number of authors have reported comparisons of the pain intensity ratings of married men and women. To date, such comparisons have been limited to the male and female “patients” or “persons with pain” in these studies, not their spouses. Thus, such studies only provide evidence regarding gender differences among unrelated married individuals.

In studies of married individuals with pain, gender comparisons have yielded
inconsistent results. The strongest evidence of comes from an analysis of baseline data collected from 168 married patients with osteoarthritis of the knee (Keefe et al., 2000). In this largely midlife and older adult sample, women rated their osteoarthritis-related knee pain (on the AIMS pain intensity/severity scale) significantly higher than did men. A similar difference was found on a measure of observed pain behavior (e.g., stiffness, slowness, interrupted movement) during a sequence of physical tasks, suggesting that gender differences may not be limited to verbal self-reports.

A majority of studies involving samples of married persons with pain have not found significant gender differences in ratings of pain intensity. Negative findings have been reported in studies of married patients recruited from pain clinics (e.g., Burns, Johnson, Mahoney, Devine, & Pawl, 1996; Cano, Johansen & Geisser, 2004), as well as in studies of married persons with chronic pain recruited from the community (e.g., Cano, Mayo, & Ventimiglia, 2006; Gauthier, Thibault, & Sullivan, 2008). There appear to be some differences between these studies and the study by Keefe et al. (2000) in which gender differences were found. First, many non-supporting studies had fairly small samples—ranging from 58 in Gauthier et al. (2008) to 127 in Burns et al. (1996). As some of these studies also had unequal cell sizes (e.g., 43% to 62% female), low statistical power may have contributed to a lack of significant results. Second, as compared to a mean age of 61 ($SD = 10.6$) in the Keefe et al. (2000) sample, participants in most non-supporting studies were generally younger, with the mean age of samples ranging from 41 ($SD = 8.6$) in Burns et al. (1996) to 54 ($SD = 13.1$) in Cano et al. (2006). Finally, the samples in many non-supporting studies were comprised of persons with “chronic pain;” some were patients at chronic pain clinics, while others self-identified as
“chronic pain sufferers” in response to a request for study volunteers from the community. Although those in the Keefe et al. (2000) study had, on average, suffered from knee osteoarthritis for over a decade ($M = 11, SD = 10.7$), they were not expressly labeled as persons with “chronic pain.”

**Summary and critical analysis.** Overall, a majority of studies has found that women are more likely to report pain and to report pain of greater intensity than men. Although these findings are far from uniform, evidence of such gender differences has been found in laboratory experiments of induced pain, in several large population-based studies, studies of clinical populations with diseases in which pain is a prominent feature, and research on chronic pain patients. Gender differences in reports of pain and pain intensity have been documented in studies involving adults of all ages, as well as in studies of middle-aged and/or older adults specifically.

It is worth noting that the empirical data are at odds with some commonly-held beliefs regarding gender and pain. In a sample of married couples, Nurofen (1989) found that the majority of both wives (86%) and husbands (64%) endorsed the belief that women are better able to tolerate pain than men. A study of single adults found a smaller but substantial proportion of both genders (66% of females, 33% of males) believed that women handle pain better than men (Bendelow, 1993). Despite such widely-held beliefs, the bulk of empirical data suggests that women appear to be more sensitive to pain, are more likely to report experiencing pain, and generally report pain of greater intensity than men. A host of biological, psychological, and social factors have been hypothesized to explain observed gender differences (see Berkley, 1997, and Fillingim et al., 2009).

Evidence of gender differences in reports of pain and pain intensity is strongest in
laboratory-based studies, where gender differences have been observed across a range of painful stimuli (e.g., see reviews by Fillingim et al., 2009; and Riley, Robinson, Wise, Myers, & Fillingim, 1998). However, even in experimental studies, the size of observed gender differences tends to vary across pain induction techniques, the nature and characteristics of the population(s) studied, and various aspects of the experimental setting (Berkley, 1997; Fillingim & Maixner, 1995; Riley et al., 1998). Thus, even within the relatively circumscribed domain of experimental pain, it can be difficult to draw firm conclusions about the magnitude of gender differences in pain and pain intensity.

A sizeable minority of studies have not found evidence of gender differences in pain or in pain intensity. Compared to gender differences in reports of pain, findings have generally been more equivocal with respect to pain intensity or severity (Fillingim et al., 2009). There appears to be more diversity of findings regarding gender differences within clinical samples and also samples of persons with “chronic pain” (determined in various ways), both in clinical and community settings.

Lack of consistency in findings may be attributable to three primary sources of variation among studies: (a) differences in how pain and pain intensity have been operationalized; (b) differences in the age ranges of the populations studied; and (c) the extent to which other explanatory factors have been controlled. Each of these issues is explored next.

First, studies have differed widely in terms of how pain and pain intensity have been defined and measured. Some studies have operationalized pain very globally (e.g., “any pain in the past month”), while other studies have been concerned with more specific types of pain (e.g., “hip pain”). Many large epidemiological studies in the U.S.
ask only about specific types of pain, thereby limiting the generalizability of observed
gender differences. Studies have also varied greatly in terms of the specified time frame
(e.g., “pain in the past 3 months” vs. “any pain in the past year”), frequency (e.g., “pain
most days”), and nature of requested pain reports (e.g., “any significant hip pain”).

Measures of pain intensity, as well as the mode of administration (e.g., interview vs.
questionnaire), have also been sources of variation across studies. Existing studies have
also varied in the extent to which they have focused on, and the ways in which they have
defined and measured, “chronic” pain.

Although findings with respect to gender do not appear to vary systematically
according to either the definitions or the measures pain intensity used, the fact that
relatively few studies have employed multiple measures of pain intensity makes the
influence of methodological factors difficult to assess. The few studies that have
compared the responses of males and females across multiple measures in a single
sample have also yielded inconsistent findings. For example, in a small sample of 68
osteoarthritis patients, female patients reported significantly greater pain intensity on one
measure (a visual analogue scale), but not on two other measures (a rating of pain
severity across several different body positions and a composite measure of pain types
and severity; Creamer, Lethbridge-Cejku, & Hochberg, 1999). In a large population-
based study in Norway, females with chronic pain (≥ 3 months) gave significantly higher
ratings than their male counterparts for current pain intensity and worst pain intensity;
however, ratings of pain intensity at its least did not differ by gender (Rustoen et al.,
2004). Yet, in a recent study of cancer patients, females rated their current and least pain
intensity higher than males, but did not differ in their ratings for worst or average pain
intensity (Pud, 2011). In one review, Miller and Talerico (2002) concluded that pain intensity had been measured with such inconsistency in studies of adults over age 65 that conclusions regarding gender differences may be suspect. Unfortunately, the situation appears to have changed little since that review.

Second, the age range of the populations studied may be a factor contributing to the inconsistency in findings. There is some suggestion that the relationship between gender and pain may vary across the life cycle, at least for some types of pain. Migraine headache and temporomandibular pain (jaw pain) are more common in women throughout the lifespan, but peak for both sexes at the onset of middle-age (roughly age 40-50) and decline thereafter (LeResche, 2000). In contrast, joint pain prevalence tends to be similar in young adult men and women and increases in both genders with age; however, in old age, the prevalence curves diverge and older women report substantially more joint pain than older men (LeResche, 2000).

Studies of pain intensity limited to samples of older adults have been somewhat less consistent with regard to finding gender differences than have studies of samples with broader age ranges. This has led some to suggest that, along with the overall prevalence of some types of pain (e.g., joint pain in Magni et al., 1990), the gender disparity in pain prevalence and intensity may actually start to decrease in very old age. In their study of daily pain, Krueger and colleagues (2008) observed that while middle-aged and older women reported higher levels of pain intensity than their same-aged male peers, younger women actually reported slightly lower average pain intensity ratings than men of similar age. But, findings have been far from consistent, and definitions of “young,” “middle,” “old,” and “very old” age have varied widely across studies. In
another study, ratings of arthritis-related knee pain were highest among individuals ages 50 to 64 and 65 to 74, regardless of gender; however, among those age 75 and over, men rated their knee pain more severe than did women (Jinks et al., 2002).

Third, existing studies have varied in the extent to which they have controlled for other demographic and health-related characteristics (e.g., living arrangements, number and types of disease conditions). Many studies have examined only bivariate associations between gender and reports of pain or pain intensity (e.g., NCHS, 2006; Creamer et al., 1999), or have adjusted only for age (e.g., Mobily et al., 1994). Thus, it is possible that other factors may explain differences that appear, on the surface, to be gender-based. Bradbeer, Helme, Yong, Kendig, and Gibson (2003), for example, failed to find gender differences in either reports of any pain or reports of moderate to severe pain among older adults in the community once they controlled for factors like widowhood and living alone. And, although females age 51 and over in HRS 2000 were more likely to report severe pain than males (as reported above), gender was not a significant predictor once background and health status variables (e.g., number of chronic diseases, education) were taken into account (Reyes-Gibby et al., 2007). Similar findings have been reported in some clinical samples, including patients with rheumatoid arthritis (Smedstad et al., 1995). Yet, in other studies, gender differences in reports of pain or pain intensity have remained, even after controlling for background and health-related variables (e.g., Idler, 1993; Smith et al., 2001). Such diverse findings suggest the need for additional research and highlight the importance of examining gender differences at both the bivariate and multivariate level.

Prior studies conducted within the context of marriage merit additional
discussion. Although findings in samples of married persons with pain have been mixed, the bulk of studies have generally not found evidence of gender differences in pain intensity. At present, it is unclear whether such mixed findings across studies might be attributed to differences in sample size or composition. What is clear, however, is that the literature base concerning gender differences in married persons is meager. Moreover, the findings from existing studies may have limited applicability to the present investigation, as samples in prior studies have been comprised of individuals who were married, but not to each other. In contrast, the present study examined gender differences between spouses within married couples—i.e., between husbands and wives.

The current study sample also differed in other important ways from those in prior studies. For example, HRS respondents were not sampled as couples, nor were they identified as patient-caregiver dyads. In contrast to the samples used in previous investigations, respondents in this study derived from a national sample of community-dwelling middle-aged and older adults. Additionally, although those comprising the focal sample in the current study all reported having pain, they were not sampled from the community on the basis of such pain reports, nor were they required to have specific diseases or conditions (e.g., osteoarthritis, fibromyalgia, low back pain, etc.). Most importantly, because all of the husbands and wives in this focal sample reported pain and because respondents were married to each other, this meant that each respondent was married to a spouse who also had pain. Given the lack of comparability between the current study and prior studies of married—but unrelated—individuals, these prior studies receive relatively limited attention throughout the rest of this review.

Many of the aforementioned issues also characterize the literature concerning
other background and health-related predictors of pain and pain intensity. This is partly the result of overlap in the evidence base—many empirical studies provide evidence related to several different demographic and/or health-related variables of interest. In order to reduce repetition, less attention is paid to differences across studies in the operationalization of pain/pain intensity, the age range of samples, and the use of control variables. The bulk of the discussion about these (and other identified issues) is instead presented later, within an overall summary of this body of literature.

Age

As noted in the review of prevalence data earlier in this chapter, pain is relatively common among middle-aged and older adults. One reason is that pain is often tied to the existence of chronic illness, and several chronic diseases (e.g., arthritis, Type II diabetes) are more prevalent among adults in mid and later life (e.g., Anderson & Felson, 1988). Given that more than 80% of older adults have at least one chronic health problem, and 69% have three or more chronic conditions (Hoffman & Rice, 1996), it seems reasonable to expect that pain reports would increase with age. The frequency with which older adults suffer some painful acute illnesses (e.g., shingles), sustain injuries from falls, and undergo surgical procedures also suggests that reports of pain should increase with age.

Despite these general expectations, empirical studies of the relationship between age and reports of pain have provided contradictory results. Findings have been especially varied among studies of community-dwelling adults. Some studies document increased pain reports with increased age. For instance, reports of some types of pain (e.g., back, hip, knee, neck) have been found to increase with age, even within older adulthood (e.g., Urwin et al., 1998). Reyes-Gibby et al. (2007) also observed a steady
increase in reports of pain across the 4 age groups represented in HRS 2000 (i.e., 51-64, 65-74, 75-84, and 85 and over). Other studies have found decreased pain reports with increased age. For instance, a population-based study in Sweden found an overall decrease in reports of pain with age, with those in the oldest age group (65-84) reporting less pain than those in other age groups (Brattberg et al., 1989). Other studies have found no evidence of age differences in reports of pain. For example, low back pain was reported by similar proportions of midlife (31% of those aged 45-64) and older adults (30% of those age 65 and over) in the 2000 NHIS (Schoenborn, 2004).

Myriad nonlinear patterns have also been reported. Consider reports of pain lasting more than 24 hours in the past month from NHANES 1999-2002 (NCHS, 2006): Those in middle-age (age 45-64) were most likely to report pain (30%), followed by younger adults (age 20-44; 25%), and then older adults (age 65 and over; 21%). Krueger and Stone (2008) observed a steady increase in reports of pain with each decade up to age 40, followed by a plateau extending through most of older adulthood, and then a dramatic increase among individuals in their 90s. Studies of community-dwelling adults with chronic pain have also yielded inconsistent findings. Studies have variously documented that reports of chronic pain decline (e.g., Toblin et al., 2010), increase (e.g., Currie & Wang, 2004; Magni et al., 1993), show a leveling-off (e.g., Hardt et al., 2008; Rustoen et al., 2005), or follow a bell-curve (e.g., Breivik et al., 2006) with increased age.

Age differences in pain intensity have been less well-studied than have age differences in reports of pain. Existing studies have provided conflicting results. All manner of relationships between age and pain intensity have been reported—increased pain intensity with increased age, no age-related differences in pain intensity, decreased
pain intensity with increased age, as well as some nonlinear relationships. To illustrate the diversity of findings, a sample of studies is presented below.

Population-based studies in Sweden and in Europe have generally found that older adults (with old age defined as starting anywhere from age 50 to age 75) report greater pain intensity for many types of musculoskeletal and arthritis-related pain, as compared to adults of younger ages (e.g., Brattberg et al., 1996; Jakobsson et al., 2003; Perrot et al., 2009). Similar findings have been reported in some U.S. studies of adults with arthritis pain (e.g., Knight et al., 2010). A pattern of increased pain intensity ratings with age has also been observed *within the older adult population* itself. For instance, within a sample of roughly 2,500 older adults (age ≥ 70) who reported daily pain, the average rating of pain intensity increased steadily by decade (Scudds & Ostbye, 2001). A number of studies with chronic pain patients have also reported higher pain intensity ratings with increased age (see review by Tunks et al., 2008).

However, a sizable number of studies have not found evidence of age differences in pain intensity (e.g., Gagliese & Melzack, 1995; Harkins et al., 1994; Huskisson, 1983; Lavsky-Shulan et al., 1985). Numerous studies comparing older and younger chronic pain patients have reported no significant age differences in pain intensity ratings (e.g., Edwards, 2006; Herr et al., 1993). Studies of pain intensity among community-dwelling adults with chronic pain have been limited, but existing evidence suggests little systematic variation related to age (e.g., Gagliese & Katz, 2003). Despite observing an age difference in the prevalence of chronic pain in the community-dwelling adults in their study, Rustoen et al. (2005) reported that age was not a significant predictor of ratings of current, least, or worst pain intensity. Several studies of pain (not specified as chronic) in
samples limited to older adults residing in the community have also failed to find age differences in pain intensity. Although they found fewer reports of back and joint pain among the oldest adults in the Iowa Established Population for Epidemiologic Studies of the Elderly (EPESE), Mobily et al. (1994) found no evidence of age-related differences in ratings of pain intensity. Once other demographic and background variables were controlled, Bradbeer et al. (2003) found no difference in pain intensity by age in their sample of community-dwelling older adults (age 65 and over). Leveille and colleagues (2005) also found no evidence of age differences in the levels of pain reported by the older adults in their sample (age 72 and over).

Some studies have even found a pattern of decreasing pain intensity associated with increasing age. This finding has been reported in community-based studies of adults of all ages (e.g., Leveille et al., 1998). A study of community-dwelling older women also found an inverse relationship between knee pain intensity ratings and age (Lamb et al., 2000). That all women in this sample had been selected on the basis of having an existing “disability” (difficulties in ADLs, IADLs, mobility, and upper extremity functioning) makes this finding even more compelling. An inverse relationship between pain intensity ratings and age has also been documented in some clinical samples, including patients with chronic pain (e.g., Turk, Okifuji, & Scharff, 1995) and those with cancer (Cheung et al., 2010; Pickering, 2005). Cano and colleagues (2006) also reported a negative correlation between age and pain intensity ratings in their sample of community-dwelling married individuals with chronic musculoskeletal pain; theirs is the only known study of community-dwelling married persons with pain that clearly reported findings with respect to age differences in pain intensity.
A few studies of pain among community-dwelling adults have documented non-linear or otherwise unusual relationships between age and pain intensity. In the study by Krueger and Stone (2008) reported above, the average intensity of daily pain episodes increased steadily with age up until middle age (roughly age 45), then plateaued until it started to increase again around age 75. Among persons in the community with chronic neurological pain, the average pain intensity rating of older adults (over age 60) was significantly higher than that of younger adults (under age 40); however, the average pain intensity rating of those in middle age did not differ significantly from that in either group (Molton et al., 2008). Lastly, age-related patterns of pain intensity have sometimes varied by type of pain. In one Canadian study, pain severity was found to decrease with age for neck pain, but not for other types of musculoskeletal pain (Cassidy et al., 1998).

The myriad findings regarding possible age-related changes in pain intensity (and in the report of pain, generally) have prompted considerable debate among pain researchers. Anatomical, physiological, biochemical, and psychological changes associated with aging have all been suggested as possible explanations for the various age-related patterns that have been observed. Although one might hope for clarification from laboratory-based studies, experimental research on the effects of aging on pain has offered little solid guidance. The bulk of experimental research on changes in the pain threshold in older persons generally suggests either no change, or a slight increase, in the pain threshold with aging (Gibson, 2004). Some age-related changes (e.g., declines in neurotransmitters involved in pain processing, prior experience with pain) are also thought to contribute to decreased sensitivity to pain with aging (Gibson, 2004). Taken together, such evidence suggests that older adults should report less (and less intense)
pain than younger persons. There is, however, disagreement regarding whether the findings from experimental studies of pain can be generalized to individuals with pain in the community. Moreover, some age-related changes in physiological and neurological processes (e.g., decreased responsiveness of the pain-inhibitory system with age, scarring of neural pathways with repeated stimulation) might actually suggest increased sensitivity to pain with increasing age (Pickering, 2005).

Limitations in the existing literature make it difficult to draw definitive conclusions about whether age differences exist in the pain intensity reports of adults in mid and later life. Additionally, the extent to which findings regarding age differences in pain intensity can be applied to married spouses in later life is unclear, as prior research has generally not examined such effects. If expectations regarding age differences in reports of pain intensity are drawn primarily from prior studies with samples and measures most similar to those in the current study (e.g., community-dwelling adults in mid and later life who report pain), then pain intensity ratings can be expected to increase with increasing age (e.g., Reyes-Gibby et al., 2007; Scudds & Ostbye, 2001). However, the evidence also suggests that age differences may be reduced when other variables are controlled (e.g., Bradbeer et al., 2003; Rustoen et al., 2005).

Race and Ethnicity

Numerous scholars have argued convincingly that various aspects of culture can influence pain perception and experiences. One of the first was Zborowski (1969), who conducted an ethnographic study of pain among male VA patients from different ethnic and religious backgrounds (e.g., Italian, Jewish, Irish). His study illustrated how cultural traits and values (e.g., orientation to time, attitudes toward emotional expressiveness,
attitudes toward violence, and the value placed on suffering) influenced individual perceptions of, and responses to, pain. Whereas Zborowski studied differences in pain among early European immigrants, contemporary studies have focused on more recent immigrant and minority groups (e.g., African American, Asian, Hispanic/Latino). Typically, such groups have been identified on the basis of race and/or ethnicity.

**Definitions of race and ethnicity.** Historically, race has been regarded as a biologically- or genetically-determined characteristic. Different races have been distinguished on the basis of blood relationships, heredity, or common descent, as well as distinct combinations of physical features, especially skin color (Edwards, Fillingim, & Keefe, 2001). Ethnicity is viewed by many as the broader construct (Edwards, Fillingim, & Keefe, 2001; Markus, 2008). Ethnic distinctions tend to be based on shared cultural characteristics, such as language, customs, belief systems, religion, and history (Montague, 1942). However, physical and cultural characteristics are often mixed together in both the self-identification and the social categorization of racial and ethnic groups (National Research Council, 2004). Race and ethnicity are thus largely socially-determined—i.e., defined by, and in relation to, the larger society, as well as by group members themselves.

The classification system used in statistics reported by the U.S. government recognizes five major races—White, Black or African American, American Indian or Alaska Native, Asian, and Native Hawaiian or other Pacific Islander. An additional category, “Other race,” is sometimes also included in reports. Ethnicity (i.e., of Spanish, Hispanic, or Latino origin) is most often conceptualized as a characteristic that cuts across racial categories (U.S. Census Bureau, 2007). The term “Hispanic/Latino” is often
used as an umbrella term, grouping together individuals of diverse heritage and various countries of origin (e.g., Mexico, Puerto Rico; Grieco & Cassidy, 2001).

**Race and ethnicity as conceptualized in the current study.** Both race and ethnicity were considered in the current study. The racial identification question in HRS 1998 used response categories similar to those used by the U.S. Census Bureau. Because a limited number of respondents identified themselves as Asian, American Indian, or of other race, these respondents were grouped together into an “Other race” category. Thus, respondents in the current study were initially grouped into three racial categories: White, Black, and Other. Given the recognized acceptability of both sets of terms (e.g., American Psychological Association, 2001), “Black” and “African American,” and “White” and “Caucasian,” are used interchangeably throughout this document. Additionally, the term “non-White” is used to refer collectively to those respondents in either the Black or Other race category. In HRS, all respondents were queried regarding possible Hispanic ethnic heritage. Consequently, respondents of any race could also identify as Hispanic or Latino. Although additional information was collected about each Hispanic/Latino respondent’s country of origin (e.g., Mexico, Puerto Rico, Cuba), these subcategories yielded sample sizes too small to use in the current study. Throughout the remainder of this document, the term “Hispanic” is used to refer broadly to all respondents who identified as Hispanic or Latino, regardless of their country of origin.

**Race and ethnicity in studies of pain.** Existing research on pain in middle-aged and older persons has largely ignored the potential for racial and/or ethnic differences. The oversight is particularly pronounced in research focused on adults in later life. Few studies of pain in older persons have included minority participants, and even fewer have
explicitly analyzed racial and/or ethnic minority differences. Given evidence of culturally-based variations in the experience of health and illness (e.g., McElroy & Jezewski, 2000), the lack of research on racial and ethnic differences in pain is striking.

Most investigations into the pain experiences of persons of different racial and/or ethnic groups have been either single-race studies or comparisons limited to two racial categories. Much of the study of racial differences in pain has taken place in the laboratory, where participants (usually younger adults) are subjected to various procedures designed to induce acute pain. Such studies have been largely limited to comparisons of Whites and Blacks with little, if any, consideration of ethnic heritage. A smaller group of studies has compared the experiences of White and Black patients with respect to acute pain (usually post-operative or procedural pain). Studies of chronic pain patients have also focused mainly on differences between Blacks and Whites (e.g., Green, Baker, Smith, & Sato, 2003).

Population-based studies of racial and ethnic differences in pain have also been dominated by comparisons between Blacks and Whites, or between Whites and non-Whites (see review by Edwards & Helme, 2005). With notable exceptions (e.g., Avis, Ory, Matthews, Schocken, Bromberger, & Colvin, 2003), few studies have included persons of other racial backgrounds. Most nationally-representative studies (including HRS, the data source for the current study) lack sufficient numbers to explore differences involving other races. When studies have specifically included (or recognized) ethnicity, most have either compared all persons of Hispanic ethnicity to (non-Hispanic) Whites and Blacks (e.g., Portenoy, Ugarte, Fuller, & Haas, 2004) or used the largest subgroup of Hispanic respondents (e.g., Mexican American) as the group with whom non-Hispanic
Whites and non-Hispanic Blacks are compared (e.g., NCHS, 2006). A limited number of studies have compared sub-groups of persons of Hispanic heritage—e.g., Magni, Rossi, Rigatti-Luchini, and Merskey, (1992) compared the pain reports of Mexican, Puerto Rican, and Cuban Americans; however, such comparisons are generally only possible within large studies fielded specifically within the Hispanic population (e.g., Hispanic EPESE; Markides et al., 1999).

On the whole, the available evidence suggests that persons of minority race and/or ethnicity are more likely to report pain than those of White race. This finding has been observed samples of younger chronic pain patients (e.g., Green, Baker, Sato, Washington, & Smith, 2003), as well as patients in mid and later life (e.g., Green, Baker, Smith, & Sato, 2003). Similar findings have been reported in a number of comparative studies of chronic pain in the community (see review by Moore & Brodsgaard, 1999), as well as in studies of persons with painful disease conditions, such as arthritis (e.g., Creamer et al., 1999). There are considerably more data to indicate differences between African Americans and Whites, but this is largely because Hispanics have so rarely been included in comparative studies. Existing evidence suggests that persons of Hispanic ethnicity are more likely to report pain than are non-Hispanic Whites (e.g., Hernandez & Sachs-Ericsson, 2006). However, findings have not always been consistent. For example, an analysis of pain reports in HRS 2000 documented higher rates of pain among Hispanics, as compared to both Whites and Blacks, but no differences were seen between Whites and Blacks (e.g., Reyes et al., 2007). In contrast, a study by Hardt et al. (2008) found Hispanics (Mexican Americans) were less likely to report chronic, “non-fleeting” pain in most body locations than either Blacks or Whites.
The evidence base regarding racial and ethnic differences in pain intensity is more limited. In general, both laboratory and clinical studies have found that Blacks report greater pain intensity than do Whites (see reviews by Edwards, Fillingim, & Keefe, 2001, and Green, Anderson, & Baker, 2003). Edwards, Doleys, Fillingim, and Lowery (2001) conducted one of the few studies to examine both naturally-occurring chronic pain and experimental pain within the same group of individuals—Black and White chronic pain patients referred to an interdisciplinary pain clinic. With respect to their chronic pain condition, Black patients reported greater pain intensity (pain intensity reported hourly averaged over 2 days) than did White patients. In response to an experimental pain stimulus, Black patients displayed lower pain tolerance (i.e., greater sensitivity) than did White patients. Moreover, these findings remained after controlling for gender, surgical history, and the use of pain medication. The difference in pain tolerance observed by Edwards, Doleys et al. (2001) was also consistent with the findings of several prior experimental studies (e.g., Edwards & Fillingim, 1999; Zatzick &Dimsdale, 1990).

Racial differences in pain intensity ratings have been observed in heterogeneous samples of persons with chronic pain, including some samples of older adults. In one study comparing older (over age 50) Black (N = 164) and White (N = 1,906) chronic pain patients, Blacks reported significantly higher pain intensity levels (over 3/4ths of a point higher on a 7-point present pain intensity scale) than did their White peers (Green, Baker, Smith, & Sato, 2003). Significantly higher pain intensity ratings among Blacks (as compared to Whites) have been documented in other samples of chronic pain patients (e.g., McCracken et al., 2001), as well as samples limited to different types of chronic pain (e.g., low back pain in Chibnall et al., 2005; orofacial pain in Riley & Gilbert, 2002).
Studies of chronic pain patients have rarely had large enough subsamples of Hispanic patients to permit reliable statistical comparison. A few published studies do exist, however. For example, Hispanic and African American patients have been found to report greater intensity of chronic pain arising from spinal injury (Selim et al., 2001), migraine headache (Burke-Ramirez, Asgharnejad, Webster, Davis, & Laurenza, 1999), and (in women) gynecological disorders (Harlow & Stewart, 2003).

Studies of racial differences in pain intensity among persons not identified as chronic pain patients have been limited. The majority of studies have focused on persons with chronically-painful diseases, especially arthritis. Although available evidence suggests that the prevalence of arthritis is significantly lower in Hispanics and Blacks than in Whites, some studies suggest that arthritis-related pain and its consequences may be more pronounced in minority populations. In one recent study, the odds of Blacks and Hispanics reporting severe joint pain (at or above a 7 on a 0-10 scale) were 80 to 90% greater than those of Whites (Bolen et al., 2010). Other studies of osteoarthritis have documented higher pain intensity/severity ratings among Hispanic and Blacks, as compared to Whites (e.g., Creamer, Lethbridge-Cejku, & Hochberg, 1999). Studies with samples limited to Blacks and Whites with osteoarthritis have typically found that Blacks report higher pain intensity (e.g., Golightly & Dominick, 2005), although some report that this finding is limited to specific joints. For example, in the Johnston County Arthritis Study, racial differences have been found for ratings of knee pain intensity (e.g., Allen et al., 2009), but not for pain in the hip (e.g., Jordan et al., 2009). Few studies have examined racial and/or ethnic differences in the pain intensity associated with other disease conditions. Studies of pain intensity among rheumatoid arthritis (e.g., Jordan,
Lumley, & Leisen, 1998) and cancer (e.g., Payne, Medina, & Hampton, 2003) patients often restrict samples to one gender, and results with respect to racial and/or ethnic differences have been inconsistent.

Racial and/or ethnic differences in pain intensity have been examined in several population-based samples of community-dwelling adults. For example, Portenoy, Ugarte, Fuller, & Haas (2004) examined racial/ethnic differences in reports of “frequent or persistent pain” in a 2002 population-based, telephone survey of U.S. adults, with proportional over-sampling of Hispanics. More than three-quarters (76%) of all participants reported experiencing pain of moderate to severe intensity during the past week. Among those reporting pain, Whites rated their usual pain intensity (from 0-10, with 10 labeled “worst you can imagine”) significantly lower ($M = 5.6, SD = 2.3$) than did either African Americans ($M = 6.1, SD = 2.7$) or Hispanics ($M = 6.4, SD = 2.5$). In their analysis of HRS 2000 data, Reyes-Gibby et al. (2007) found that proportionally more Hispanic (27%) and non-Hispanic Black (26%) respondents reported severe pain than non-Hispanic White respondents (17%). Non-Whites also were significantly more likely to report moderate or severe pain than Whites in the 2004 wave of HRS (Covinsky et al., 2009). Similar results documented in a large, multi-site sample of community-dwelling middle-aged (42-52) American women: The odds of scoring in the “impaired” range (in 25%tile) on a measure of body pain were significantly higher for Black (OR = 1.51) and Hispanic (OR = 3.87) women, as compared to White women (Avis et al., 2003). However, inconsistent findings have also been observed; for instance, minority racial status (non-White vs. White) was not related to level of musculoskeletal pain intensity among urban-dwelling adults (Idler, 1993).
The reasons for observed racial and/or ethnic differences in reports of pain and pain intensity remain unclear. Various explanations have been offered. Some focus on differences in health care access and quality, implicating disparities in the assessment and management of pain (e.g., Todd, Lee, & Hoffman, 2003). Others cite differences across races and cultures in willingness to report and endure pain and differences in styles of coping with pain (e.g., Green et al., 2003). Still others attribute observed differences to underlying differences in the risk factors associated with painful conditions and/or pain (e.g., Allen et al., 2009).

Support for this latter suggestion can be amassed from the results of several studies. One methodologically rigorous study of chronic pain intensity was conducted by Edwards, Moric, Husfeldt, Buvanendran, and Ivankovich (2005): Using a large pool of patients seeking treatment at a specialty pain clinic, the authors compared subsamples of Hispanics, Blacks, and Whites (n = 97 each group) matched on gender, age, education level, employment status, pain site, and pain duration. Once the racial/ethnic groups were matched in terms of these potential confounding variables, there were no statistically significant differences on measures of pain intensity. In a similar study of community-dwelling adults with chronic pain, Ruehlman, Karoly, and Newton (2005) created age- and gender-matched samples of non-Hispanic Blacks and Whites (n = 214 each group). The authors found no significant differences between the groups in terms of ratings of pain intensity. Some studies of community-dwelling persons with pain that is not expressly labeled “chronic” have also documented that racial and ethnic differences in pain intensity are reduced and/or eliminated in multivariate analyses (e.g., Allen et al., 2009; Reyes-Gibby et al., 2007; McIlvane, 2007). Together, these findings suggest that
racial and ethnic differences in pain severity might be explained by differential
vulnerability to chronic diseases, and differences in socioeconomic status and access to
health care. In some studies, however, multivariate controls have not diminished racial
and ethnic differences in pain intensity (e.g., Avis et al., 2003).

In summary, the bulk of available evidence suggests that racial and ethnic
differences exist in the experience of pain. For the most part, persons of minority race
and/or ethnicity appear to report more pain and pain of greater intensity than do Whites.
There is not yet enough evidence to draw conclusions regarding differences in pain
experiences between African Americans and Hispanics. Additionally, there is growing
evidence to suggest that racial and/or ethnic differences in pain and pain intensity may be
reduced once other important background and health-related variables are controlled
(e.g., Reyes-Gibby et al., 2007; Ruehlman et al., 2005).

Socioeconomic Status

Few prior studies of pain among middle-aged and older adults have specifically
examined the relationship between socioeconomic status (SES) and the experience of
pain. If SES has been measured, it often has just been reported with other descriptive
information about the sample. Unfortunately, this is true of the larger body of pain
research as well, including studies of chronic pain patients and pain among persons with
disease conditions in which pain is a frequent concomitant (e.g., arthritis, cancer).

Most knowledge about the relationship between socioeconomic status (SES) and
pain derives from research focused on other relationships. For example, studies of the
relationship between pain and depression may control for the influence of SES on
depression. Or, as just discussed, SES has recently been included in studies of racial and
ethnic differences in pain. The task of summarizing findings regarding the relationship between SES and pain is thus challenging. This task is further complicated by the fact that, when SES is included in studies of pain, the construct has operationalized in various ways. As existing data are fairly limited, a sampling of findings is reviewed for several major indicators of SES used in prior research.

**Education.** Available evidence suggests that education is related to both pain reports and pain intensity. In general, lower education has been associated with an increased likelihood of reporting pain. This pattern has been observed in community-based samples of adults of all ages (e.g., NCHS, 2006), as well as middle-aged adults (e.g., Avis et al., 2003) and those in midlife and older adulthood (e.g., Reyes-Gibby et al., 2007). The relationship between educational attainment and pain intensity is generally negative, but the strength and significance of this relationship appears to vary depending on whether analyses control for other variables. So, for example, in a small sample of 68 osteoarthritis patients, years of formal education was significantly negatively related to two different measures of pain intensity (Creamer, Lethbridge-Cejku, & Hochberg, 1999). However, education was no longer significant in multivariate models that controlled for other factors (e.g., race, age, radiographic indicators). A similar inverse relationship was also found between educational attainment and knee pain intensity among community-dwelling adults (Hannan, Anderson, Pincus, & Felson, 1992), an association that remained significant even after controlling for other known risk factors for knee pain (age, obesity, prior knee injury, etc.). The authors noted that the relationship appeared especially strong among those with very low education (e.g., less than eighth grade).
However, not all studies document a significant relationship between education and pain. For instance, education was not a significant predictor of the onset of persistent pain in a World Health Organization-funded study of primary care patients in 15 different countries (Gureje et al., 2001). As these authors also controlled for the location of the primary care clinic (i.e., country), it is possible that variability associated with education may have been captured by the location of care. Education level was also not related to level of musculoskeletal pain (a mixed construct of amount and intensity) in a sample of adults attending an outpatient rehabilitation clinic (Idler, 1993).

**Income.** Income has also been studied in relation to pain, although studies have more often examined pain reports than pain intensity. One early community-based study found that among adults, pain was more likely to be reported by those with lower income than those with higher income (Taylor & Curran, 1985). More recent epidemiological survey data (from NHANES 1999-2002) similarly showed that adults with incomes below the poverty threshold were more likely to report pain than were those with higher incomes. Reports of pain—of any kind—that had lasted for more than 24 hours in the month prior to the interview were significantly more common among those with family incomes below the poverty threshold than for those with family incomes of 200% or more of poverty (30% vs. 25%; NCHS, 2006). Analyses of NHIS study data from 2002-2004 revealed a similar pattern with respect to reports of joint pain. In addition, those below the poverty line were almost twice as likely to report that their joint pain was “severe” than were those in the highest income category (NCHS, 2006).

The association between income and pain has been found to persist, even when other demographic or background variables are controlled. A study by Edwards, Doleys
et al. (2001) using NHIS data found that both race/ethnicity and income were significant in multivariate models predicting reports of pain. Those with lower income were more likely to report pain, regardless of race. The Avis et al. (2003) study of community-dwelling middle-aged women also examined a slightly different conceptualization of income—perceived income adequacy. After adjusting for ethnic group and education, women who had low perceived adequacy of income (i.e., found it somewhat hard or very hard to pay for basic necessities) reported greater pain than women who reported no trouble paying for basic necessities.

**Summary regarding SES.** In summary, the preponderance of evidence suggests that socioeconomic status (as measured by any of several indicators, including education and income) is related to pain reports. In general, lower SES has been associated with increased reports of pain, including reports of any pain, persistent or chronic pain, and pain associated with disease conditions, such as arthritis. Considerably fewer studies have examined SES in relation to pain intensity. Most prior studies have found lower SES (specifically lower income and education) to be related to greater pain intensity, although the significance of this relationship has sometimes varied depending on whether or not (and which) other predictors have also been included in statistical models. In numerous other studies, indicators of lower SES remained significantly associated with greater pain intensity, even after controlling for other demographic and background variables (e.g., Cote et al., 2000; Smith et al., 2001). Variation across studies in the indicators used to measure SES and in how these measures are treated in analyses (i.e., as interval, ordinal, or categorical variables) also make it difficult to assert that the association between SES and pain intensity is linear; however, the available evidence
does suggest that a general linear trend exists.

Prior studies of pain have tended to make use of only one SES indicator. There are some notable exceptions, however (e.g., Avis et al., 2003; Hannan et al., 1992). The majority of these studies suggest that different indicators of SES may be independently related to pain. Several other studies provide additional evidence to support this idea. For example, in their study of pain among community-based adults in the U.S., Krueger and Stone (2008) found that lower income and less education were each associated with more reports of daily pain and higher average pain intensity. The authors reasoned that this socioeconomic status-pain gradient was probably related to occupational status, as those in blue collar occupations reported higher pain intensity ratings during work periods versus non-work periods, and also higher intensity ratings than did white collar during both work and non-work periods. The study by Portenoy et al. (2004) found that low income, low education, unemployment were each independently related to reports of persistent or chronic pain; moreover, each indicator remained statistically significant, even after controlling for the effects of the other two. Multiple indicators of SES—education, blue collar occupation, and financial strain—were also found to independently predict greater pain intensity in a population-based study of adult women in Sweden (Jablonska, Soares, & Sundin, 2006). Whether or not multiple indicators of SES are used, existing evidence does justify including SES in studies of pain intensity.

In most pain research, SES has been conceptualized and measured as an individual-level construct (i.e., an individual’s education, occupation, income). A slightly different approach was adopted in the current study. Three indicators of SES were used in this study: education, income, and wealth. Education was conceptualized as
an individual-level construct. Because the sample was comprised of married couples, income and wealth were conceptualized as household level constructs. Note that this distinction was also consistent with how these constructs were measured in HRS.

The mechanism whereby SES influences reports of pain and pain intensity is not clear. Lower SES may convey risk for pain via increased exposure to occupational or environmental hazards that lead to injury or that increase susceptibility to certain diseases. Having low SES may also negatively affect an individual’s ability to access and effectively utilize health care services, including treatment for pain. Also, rather than conceptualizing the mechanism as being one of excess risk tied to lower SES, it is also possible that higher SES conveys benefits to individuals when faced with pain. For example, studies of persons with rheumatoid arthritis have found that individuals with higher education (especially graduate-level education) engage in more beneficial self-care activities than do persons with other levels of education (e.g., Katz, 1998). Wealth might operate in a similar manner. Regardless of the specific mechanism of effect, evidence suggests that SES is an important construct to examine in studies of pain.

**Health-Related Characteristics**

*Disease Conditions*

In general, middle-aged and older adults who report a greater number of disease conditions are more likely to report pain and to report pain of higher intensity than those with fewer conditions. In HRS 2000, respondents with more disease conditions were significantly more likely than those with fewer conditions to report pain (Reyes-Gibby et al., 2007). An increasing number of chronic diseases was associated with a steady increase in the number of respondents reporting severe pain. Additionally, in a
multivariate model including significant sociodemographic and background variables (e.g., insurance status, education, psychological distress), the number of chronic diseases remained a significant predictor of severe pain (Reyes-Gibby et al., 2007).

In a sample of older adults enrolled in a clinical trial evaluating treatments for depression, Karp et al. (2005) found that greater chronic medical burden (as measured by a geriatric index) was moderately related to higher levels of pain (as measured by the SF-36 bodily pain subscale [Ware & Sherbourne, 1992]—a mixed scale of frequency, intensity, and interference). Bennett et al. (2002) studied a sample of 225 older adults drawn from two large health maintenance organizations. They found that greater pain on the SF-36 bodily pain scale was positively related to the number of medical conditions ($r = .38$, $p < .01$).

Numerous prior studies have documented a connection between a greater number of disease conditions and greater pain intensity among adults in general (e.g., Jablonska et al., 2006), as well as in older populations specifically (e.g., Leong et al., 2007). A positive relationship between number of disease conditions and pain intensity has also been observed within samples comprised solely of persons with pain. For example, Ayis and Deppe (2009) identified a subset of participants with hip or knee pain at the outset of the Somerset and Avon Survey of Health, a community-based cohort study in the U.K. Among the 1,072 subjects with chronic hip and/or knee pain at baseline, ratings of pain intensity were positively associated with the number of reported health conditions.

Within the literature, far more studies have examined comorbid or multiple disease conditions in relation to reports of pain than pain intensity. Findings from some studies suggest that there may a threshold effect with respect to the number of disease
conditions and reports of pain—for example, Bryant et al. (2007) found that reports of chronic pain were elevated only among those having three or more disease conditions (vs. those with no conditions). Other studies have observed a more linear relationship, with increasing numbers of chronic diseases positively associated with a steady increase in pain prevalence (e.g., Ruehlman et al., 2005). The existence of comorbid disease has also been linked to increased reports of pain among persons with defined disease conditions, such as cancer (e.g., Given et al., 2001). This phenomenon has even been observed for comorbid conditions that fall within the same category of disease. For instance, Picavet and Hoeymans (2004) found that the pain reports of those with one of several musculoskeletal disease conditions (e.g., rheumatoid arthritis, osteoarthritis, gout) were significantly increased among those who also suffered from one (or more) of the other musculoskeletal diseases.

**Specific disease conditions.** Musculoskeletal problems (e.g., pain, stiffness, aching) are the most commonly reported symptoms experienced in daily life among both middle-aged and older persons. In both age groups, diseases of the musculoskeletal system (osteoarthritis most often, but also rheumatoid arthritis, and osteoporosis) are listed as the number one cause of these daily symptoms (Verbrugge 1986).

In a large epidemiologic study, Idler (1993) found that diseases with demonstrated mortality risk (e.g., heart disease, stroke, diabetes, high blood pressure, cancer) had no independent association with level of musculoskeletal pain (after controlling for their symptoms and effects on functional disability). However, diseases more commonly associated with morbidity, but not mortality, risk (e.g., arthritis, hip/spine fracture, gout) maintained an independent, positive relationship with level of musculoskeletal pain.
Cancer pain is also becoming more common among middle-aged and older persons, largely because rates of cancer have been increasing in these populations. Available evidence suggests that cancer-related pain is not limited to advanced, metastatic, or end-stage cancer. Van den Beuken-Van Everdingen et al. (2007) conducted a comprehensive review of the cancer literature over the last 40 years. Based on prevalence rates pooled across various high-quality studies, these authors estimated that approximately 50% of all cancer patients experience significant pain associated with their disease. Of course, pain reports are highest among those with advanced, metastatic disease (64%); however, reports are also high among patients undergoing curative therapies (59%), and are relatively common among those who have completed curative treatment (33%). In cancer, pain intensity levels also tend to vary according to the primary tumor site, the extent of disease, and the treatments used. In a randomly selected sample of inpatient and outpatient breast, ovarian, colon, and prostate cancer patients, 63% reported the presence of pain and 43% of those rated their pain as moderate to severe (Portenoy & Hagen, 1990). Another study found 67% of a mixed group of cancer outpatients reported experiencing recent pain, and 36% described their pain as severe (Von Roenn, Cleeland, Gonin, Hatfield, & Pandya, 1993).

There has been limited study of pain intensity in relation to other disease conditions. More often, studies have explored the conditions that appear to be associated with reports of pain. Reyes-Gibby et al. (2007) found that all 7 disease conditions queried in HRS (except cancer) were associated with increased reports of pain, with the highest prevalence of reported pain observed among those with lung disease (44%) and arthritis (39%). Covinsky et al. (2009) reported similar findings using HRS 2006 data,
although they found that even cancer was associated with increased odds of reporting significant pain (i.e., moderate or severe pain). Even long-term survivors of cancer display increased odds of reporting pain, as compared to those without a history of cancer (e.g., Reyes-Gibby et al., 2006; Townsend, Ishler, Bowman, Rose, & Peak, 2009).

Beyond arthritis and cancer, however, there is little consensus regarding the disease conditions most consistently associated with pain. Chronic pain was reported by roughly 27% of community-dwelling older adults (≥ 60) with no doctor-diagnosed disease condition within the San Louis Valley Health and Aging Study (Bryant et al., 2007). In contrast, at least 50% of participants with at least one chronic disease condition (e.g., arthritis, diabetes, cancer, osteoporosis, high blood pressure). In addition, reports of chronic pain reached upwards of 70% for persons with arthritis and any other conditions. In multivariate analyses, however, only arthritis and diabetes maintained an independent relationship with reports of chronic pain. Another study of community-dwelling older adults documented greater prevalence of pain among those with high blood pressure, arthritis, and congestive heart failure, but not among those with diabetes or myocardial infarction (Buchman et al., 2010).

Body Weight

A variety of health risk factors could potentially be related to pain reports and pain intensity among middle-aged and older adults. All health-related habits, behaviors, and lifestyle choices are candidates, including body weight, alcohol consumption, substance use/abuse, cigarette smoking, and lack of exercise and/or a sedentary lifestyle. Of these factors, body weight appears to have the most sizeable and consistent relationship to pain. Body weight (especially excess body weight) has become an
increasingly important priority for public health education and health promotion efforts at
the federal, state, and local levels; for example, see Healthy People 2020 (U. S.
Department of Health and Human Services, 2010).

Both excess body weight and very low body weight have been associated with a
variety of health problems. Although body weight is typically conceptualized and
measured as an interval level variable, researchers often classify individuals into four
general categories: (1) those who are underweight; (2) those of normal (or approximately)
normal weight; (3) those who are overweight; and (4) those who are obese (NIH &
National Heart, Lung, and Blood Institute [NHLBI], 1998; World Health Organization
[WHO], 2000). Guidelines are also available to further distinguish among obese
individuals—Class I, Class II, and Class III (extreme) Obesity (e.g., NIH/NHLBI, 1998).

Few studies have specifically focused on the relationship between body weight
and reports of pain; fewer still have examined body weight in relation to pain intensity.
Much of the knowledge about the relationship between body weight and pain comes from
research devoted to other topics—for example, studies documenting myriad ill-health
effects of obesity, or studies of gender and physical functioning that include body weight
as a covariate. There are also relatively few studies that document a relationship between
body weight and pain in middle-aged and older samples. Four basic lines of research
justify additional attention to body weight in studies of pain.

First, research shows that persons with chronic pain (or conditions associated with
recurrent pain) tend to have more extreme body weight levels than do individuals without
pain. For example, women with fibromyalgia (a chronic pain condition) have been
shown to have higher average body weight than women in the general population (Yunus
et al., 2002). A similar relationship has been observed in some studies of persons suffering from disease conditions in which pain is a prominent feature. For example, a sample of over 200 older persons (mean age = 70) reporting osteoarthritis knee pain was found to have higher average body weight than a comparison sample of older persons without osteoarthritis knee pain (Brandt et al., 2000).

Second, persons with extreme body weight have higher rates of pain and/or report higher pain intensity levels than persons of normal weight. For instance, reports of pain among obese persons pursuing weight loss programs are very common (e.g., over 50% in Barofsky, Fontaine, & Cheskin, 1998), especially in comparison to the prevalence of pain reported by matched controls of normal weight (Hafner, Watts, & Rogers, 1987).

Third, epidemiological studies of community-dwelling populations (i.e., those not selected on the basis of extreme body weight or the presence of chronic pain) have found an association between body weight and pain. Hitt et al. (2007) recently conducted a survey about pain in the general adult population in the southeastern United States. Those at or above the obese range of body weight had significantly greater odds of reporting moderate or severe pain that occurred at least monthly than those who were of normal weight or underweight. Furthermore, the odds of reporting such pain increased steadily as the level of obesity increased. For example, compared to only 6% of normal and underweight respondents, 10% of Class I and II obese and 14% of Class III obese respondents reported experiencing severe pain. Those who were overweight (but not obese) did not show any increased risk for moderate or severe pain. Analysis of data collected from over 150,000 adults in the 1999 Behavioral Risk Factor Surveillance Survey, also found a significant, positive relationship between body weight and reports of
joint pain (Heo, Allison, Faith, Zhu, & Fontaine, 2003).

A relationship between obesity and pain has also been found in samples of community-dwelling middle-aged and older adults. In the Avis et al. (2003) study of community-dwelling middle-aged women, obese women were more likely to score in the impaired range on a scale of pain severity than were those of normal or low weight. No statistically significant differences were observed in comparisons involving overweight women. A study of over 3000 older adults (over age 65) found that obese men and women were more likely to report severe or very severe pain than their normal-weight peers. This relationship was reduced slightly, but still was still significant after controlling for demographic variables, chronic diseases, and other risk factors (Yan et al., 2004). Similar results have been found other studies of older adults (e.g., Buchman et al., 2010), as well as those in midlife (e.g., Han, Tijhuis, Lean & Seidell, 1998).

Some community-based studies have even observed a more linear relationship between body weight and pain—extending beyond the effects associated with obesity. Linear relationships have been observed for reports of several different types of pain, including severity of migraine headache (e.g., Bigal, Liberman, and Lipton, 2006), reports of upper abdominal pain (e.g., Delgado-Aros et al., 2004), and reports of back pain (e.g., Brown, Dobson, & Mishra, 1998). A roughly linear relationship was found between body weight and reports of musculoskeletal pain in community-dwelling older adults (e.g., Andersen, Crespo, Bartlett, Bathon, & Fontaine, 2003). However, such findings have sometimes been limited by gender—e.g., in the Framingham Disability Study (age ≥ 72), musculoskeletal pain was associated with higher body weight in women, but not in men (Leveille, Zhang, McMullen, Kelly-Hayes, & Felson, 2005).
Studies of the relationship between low body weight and pain are rare, and results have been inconsistent. Some studies have found low body weight to be a risk factor for pain (e.g., Eisman et al., 2004; Kerr, Frank, Norman, Wells, & Neumann, 2001). However, other studies have found underweight persons to be less likely to report pain than those of normal weight (Brown et al., 1998; Leboeuf-Yde, Kyvik, & Bruun, 1999).

Fourth, and finally, some prospective studies have yielded compelling evidence of a causal relationship between body weight and pain reports and/or pain intensity. Although limited in number, these studies suggest that having high body weight at younger ages places individuals at risk for both the onset of pain later in life and increased pain intensity over time. Lake, Power, and Cole (2000) found that high body weight, especially in the obese range, among women in young-adulthood (age 23) was associated with the onset of chronic back pain by age 33. In a Swedish study of adults with a history of knee injury or knee osteoarthritis, both overweight and obesity were risk factors for worsening knee pain over the course of a two-year period, even after controlling for characteristics such as age and gender (Paradowski, Englund, Lohmander, & Roos, 2005). In a three-year prospective study of a large sample of adults age 50 and over in the United Kingdom, obesity was a strong predictor of the onset of severe knee pain among those who reported no knee pain at baseline—obese individuals had more than a three-fold increase in the risk of severe knee pain, as compared to those of normal weight (Jinks et al., 2006).

Looking across these four basic lines of research, a substantial body of evidence has established a relationship between body weight and increased reports of pain and also (although far less voluminous) greater pain intensity. The evidence appears strong for
body weight in the obese range, and is more moderate for body weight in the overweight range. At present, the evidence suggesting a relationship between pain and low body weight is relatively weak and inconclusive. In general, findings with respect to body weight seem to apply across different types of pain. It is also interesting to note that the relationship between obesity and pain does not appear to be limited to pain in weight-bearing joints. In the Hitt et al. (2007) study, reports of moderate and severe pain among obese persons were high for weight-bearing joints and lower body sites (e.g., hips, legs, knees, feet), as might be expected; however, elevations were observed in most other body sites (e.g., head, mouth, shoulder, stomach, arm), as well.

A relationship between body weight and pain reports—and, to a lesser extent, pain intensity—has also been demonstrated across a variety of clinical and community-based samples. And, although not all studies have controlled for the presence of other variables, the results of studies that have done so suggest that the observed relationships between body weight and pain are robust (e.g., Avis et al., 2003; Yan et al., 2004). There has been some speculation that the effect of body weight may be more pronounced in women, especially in arthritis (see Felson & Zhang, 2004 for a discussion of this issue), but study findings have been inconsistent. Proportionally more studies of body weight and pain have been conducted with general adult or middle-aged populations than with older adults. That said, a sizeable number of studies of body weight and pain have been done in samples of arthritis patients, or have focused specifically on those reporting musculoskeletal pain within large, epidemiological studies. And, as musculoskeletal pain is the most common type of pain reported among midlife and older adults, these findings should generalize to most population-based studies of aging adults. Overall, this body of
research suggests that studies of pain in midlife and older adults should examine body weight as a possible predictor of pain intensity.

**Summary: Factors Related to Pain and Pain Intensity**

As noted earlier (under the topic of gender), several limitations exist within the existing literature concerning the factors associated with pain and pain intensity. Study findings in many areas have often been inconsistent, even contradictory. Variability in the definition and measurement of key constructs (including pain and pain intensity), the ages and age-ranges of samples, and the identification and use of covariates may have contributed to the inconsistency of results observed across studies.

Definitions and measures of pain and pain intensity have varied widely across studies. Consider, for example, the definitions and decision rules used to characterize pain as “chronic.” This diversity is most pronounced among studies that have attempted to measure chronic pain in the general population. Studies have varied in terms of the required duration of pain—for example, pain lasting “at least 1 month out of the last 12” (Magni et al., 1990), “at least 3 months” (Elliott et al., 2002), “6 months or more” (Munce & Stewart, 2007). Other studies have specified additional criteria—for example, that pain had been experienced in the past month, and/or several times in the past week, and/or at the time of the survey. Some have even required that pain reports be of a certain level of intensity or severity (e.g., pain of moderate severity or higher in Breivik et al., 2006) or that reported pain be “non-minor” (i.e., lasted for ≥ 24 hours and was “not fleeting” in Hardt et al., 2008). It is unclear whether the use of differing definitions and restrictions in identifying chronic pain have influenced the results of studies involving gender, race/ethnicity, age, or body weight comparisons in pain intensity ratings.
Measures of pain intensity have also varied across studies. Although in general findings do not seem to be dependent on the measure of pain intensity that is used. For example, body weight was shown to be significantly related to three different measures of knee pain severity among hospital outpatients with osteoarthritis of the knee (Creamer, Lethbridge-Cejku, & Hochberg, 1999). Significant relationships have even been observed across studies in which the same measure of pain intensity was treated differently in statistical analyses. For example, Brown et al. (1998) found a relationship between increased body weight and the bodily pain subscale of the SF-36 coded as an interval-like measure, while both Avis et al. (2003) and Han et al. (1998) used cut-offs to identify high scores on the same subscale and also found significant relationships with increased body weight.

There are two other factors related to the definitions and measurement of pain that are worth noting. First, across this vast literature base, there is a fair amount of inconsistency in terms of the labels and descriptions given to specific items, scales, and subscales that have been used to measure different dimensions of pain. This is true even when different studies have used the same measure. For instance, the Pain Rating Index of the McGill Pain Questionnaire has been variously described as a measure of overall pain, pain severity, and pain intensity. In addition, authors often used the labels provided by the scale/item’s authors, but provide no information about the content of the measure. Although many readers are likely familiar with many of the most commonly used pain measures, this approach can mask important details about specific scales or items. For example, the “Bodily Pain” scale of the SF-36 has been reported in some studies as a measure of pain intensity or severity (e.g., Bingefors & Isacson, 2004). However, the
scale consists of two items that measure distinctly different dimensions of pain—intensity
and interference. Second, there is a tendency to use very generic language in discussing
measures that appear to assess different and/or multiple dimensions of pain, especially
when summarizing results. For example, some authors report findings with respect to
“greater severity of pain” or “higher levels of pain,” when the measure was a count of the
pain locations (e.g., Leveille et al., 2005). This can create conceptual confusion among
readers and promote the mischaracterization of study findings by subsequent authors.

The background and health-related constructs of interest have also been measured
in various ways across studies. This is most apparent within research on body weight,
where, for example, there is ongoing controversy regarding the most appropriate measure
of body weight. The majority of existing studies involving body weight and pain have
measured body mass index (BMI), a measure of body weight relative to height
(NIH/NHLBI, 1998). However, many studies apply the “one-size-fits all” NIH/NHLBI
guidelines to create categories of body weight, some studies have used national norms
and cutoffs to standardize respondents’ body weights by age and gender (e.g., Lamb et
al., 2000). Some studies have even further distinguished categories of obesity within the
obese range (e.g., Hitt et al. (2007). Other studies have not created categories, opting
instead to use BMI as an interval-like measure in analyses (e.g., Brown, Dobson, &
Mishra, 1998). Because many more studies have created categories than have used BMI
as an interval measure, it is difficult to fully evaluate any differences related to this
particular issue.

Definitions of certain age groups (e.g., “young” and “old”) have varied across
studies. Considering just one example, studies of the effects of older age in relation to
pain have variously examined individuals at or above age 60 (Molton et al., 2008), 65 (Bonnewyn et al., 2009), 70 (Reyes-Gibby, 2002), and 72 (Leveille et al., 2005). In the midst of such diversity, it is hard to draw firm conclusions from the existing literature.

One recurring theme throughout this body of literature is that the effects of some background or health-related characteristics are sometimes reduced or eliminated when other variables are controlled in multivariate models. This is especially true in studies of differences by age and by race. For example, Black and Hispanic adults reported daily pain episodes of higher intensity than did Whites, multivariate analyses found that age, income, and education were the strongest predictors of pain intensity and race/ethnicity was no longer significant (Krueger & Stone, 2008). However, even among studies that have controlled for the same explanatory variables, findings have not been consistent. For example, race was still a significant predictor of differences in pain intensity ratings among chronic pain patients after controlling for SES (Edwards, Doleys et al., 2001).

Findings from the study by Avis et al. (2003) suggest that the choice of control variables may influence results. In a series of separate logistic regression analyses, these authors found that the inclusion of body weight (especially obesity) along with some other lifestyle variables (e.g., physical activity level) accounted in large part for the observed difference in pain levels reported by Blacks and Whites (but did not account for Hispanic vs. White differences). At present, it is unclear the extent to which some of the differences observed in prior research may be due to unmeasured, and/or uncontrolled, third variables such as socioeconomic status. In studies that have initially found age differences in reports of pain, such differences have often been diminished when other variables are controlled (e.g., Bradbeer et al., 2003).
It is also possible that the relationship between many of these background and health-related characteristics may be more complex than the simple main effects that have thus far been the focus of much research. For example, some studies have suggested that observed differences between Hispanics and Whites may be gender-specific (e.g., Christmas et al., 2002). Other studies have suggested that gender differences in reports of pain and pain intensity may disappear among adults of very advanced age (e.g., Fillingim et al., 2009).

Within this body of literature, relatively little attention has been given specifically to the predictors of pain intensity. Studies more often have focused on identifying factors that distinguish those who report pain, or a certain level thereof. This is especially true of research within middle-aged and older adult populations. The literature base concerning the demographic and health-related factors associated with pain in married persons in mid and later life is also sparse. Moreover, it is unclear whether the findings from existing studies are applicable to the married couples examined in the present study.

**Major Pathways in the Proposed Conceptual Model**

The second aim of this study (Study Aim 2) was to examine the relationship between an individual’s pain and his/her psychological distress, in particular, his/her depressive symptomatology. The empirical evidence reviewed in this section provided a basis for the research questions and hypotheses developed around this aim. One major focus of the current study was to determine the extent to which the intra-individual relationship between pain and psychological distress is mediated by activity limitation—specifically, by physical limitations.

The first part of this review is organized around each of the main pathways
involved in this proposed mediated relationship. It begins with a summary of the evidence showing a direct relationship between pain and depressive symptomatology (Path A). Then, evidence regarding the relationship between pain and activity limitation (especially physical limitations, as highlighted in the proposed conceptual model) is reviewed (Path B). This is followed by a review of studies showing a relationship between the activity limitation and depressive symptomatology (Path C). Because each of these component pathways (i.e., Paths A, B, and C) is examined as part of tests for mediation, the review of evidence concerning each individual path is intentionally brief.

The second part of this review contains a more detailed analysis of studies that have empirically tested the activity limitation mediation hypothesis. To avoid redundancy, findings from those mediation studies were excluded from the review of evidence for the individual pathways. Note, however, that a majority of those studies have also provided evidence in support of the proposed pathways.

**Review of Paths A, B, and C**

In reviewing the evidence pertaining to the individual pathways, emphasis is placed on research within the population that comprised the focal sample of the current study—i.e., middle-aged and older, community-dwelling, married couples in which both spouses have pain. However, because of a lack of research conducted with this specific population, the review also draws on evidence accumulated from studies of other, related populations. Consequently, the review incorporates relevant studies conducted with samples of (a) middle-aged and/or older persons drawn from the community without regard to pain levels, disease presence, or marital status, (b) chronic pain patients, and (c) persons with specific painful diseases (e.g., arthritis, cancer). In addition, although
priority was given to studies that employed similar conceptual and operational definitions of the key constructs in the proposed conceptual model, it was sometimes necessary to include studies in which these constructs were more broadly defined. For example, studies that utilized cutoff scores on depressive symptom scales, clinical measures of depression, or more generalized indicators of psychological distress, are sometimes included in the review alongside studies that employed measures of depressive symptomatology. There is considerable overlap in the strengths and limitations of the empirical literatures associated with the three paths. Thus, these issues are addressed within an overall summary at the end of this subsection.

Path A: The Relationship Between Pain and Depressive Symptomatology

A previous section of this chapter outlined several lines of research that supported the existence of a relationship between pain and depression and/or depressive symptomatology. Consequently, this review concentrates on studies showing that depressive symptomatology is positively associated with pain intensity.

A sizeable body of literature documents a positive association between pain intensity and depressive symptomatology. This relationship has been observed in studies with a wide range of samples drawn from a variety of settings. Amid this diversity, most studies have found a statistically significant, often sizeable, positive relationship between ratings of pain intensity and increased levels of depressive symptomatology. Although effect sizes have not always been reported, available estimates of the strength of this bivariate relationship have covered a wide range. For instance, a modest association ($r = .17$) was observed between the pain severity ratings of 127 married patients with chronic musculoskeletal pain and their scores on the Beck Depression Inventory (Burns
et al., 1996). A relationship of more moderate strength was reported between cancer patients’ current pain intensity and depressive symptomatology, as measured by the CES-D ($r = .35, n = 85$; McMillan, Tofthagen, & Morgan, 2008). Finally, in a longitudinal study of 243 outpatients with rheumatoid arthritis, within-wave correlations between latent pain severity and CES-D scores were fairly strong, ranging from .45 to .59 (Brown et al., 1990). In general, bivariate effect size estimates of Path A have been in the low to moderate range (i.e., $r = .20$ to .45).

The relationship between pain intensity and depressive symptomatology has been examined in well over a dozen studies with samples comprised of married persons. Most studies have sampled married patients at specialty pain clinics (e.g., Cano, Weisberg, & Geisser, 2000; Geisser et al., 2000; Goldberg et al., 1993; Pence et al., 2008). A few prior studies have focused on married persons with painful conditions, such as arthritis (e.g., Druley & Stephens, 2003). Participants in other studies have been drawn from community-dwelling couples recruited on the basis of (at least) one spouse suffering from chronic musculoskeletal pain (e.g., Gauthier et al., 2008). Most prior studies have documented a positive relationship—at least at the bivariate level. Despite having small to moderately-sized samples—e.g., less than 60 in Gauthier et al. (2008) to over 150 in Cano et al. (2000)—most prior studies have reported bivariate associations in the small to moderate range. Relationships of similar strength have also been reported in studies in which psychological distress was assessed more broadly (e.g., in Cano et al., 2006, “psychosocial disability” was measured using the Sickness Impact Profile [SIP; Bergner, Bobbitt, Carter, & Gilson, 1981]). Again, studies to date have been limited to samples comprised of unrelated, married individuals.
Studies of chronic pain patients (sampled without regard to marital status) have consistently documented a positive relationship between pain intensity and depressive symptomatology (e.g., Bates et al., 1993; Faucett, 1994; Geisser et al., 2000; Haythornthwaite et al., 1991; Johnson et al., 1993; Keogh et al., 2006; Turner & Romano, 1984). The link between increased pain intensity and greater depressive symptomatology has been observed in most subgroups of chronic pain patients, including those with chronic back pain (e.g., Sullivan, Reesor, Mikail, & Fisher, 1992), neck pain (Radanov, Sturzenegger, & Di Stefano, 1995), headaches and/or migraine (e.g., Holroyd et al., 2000), and fibromyalgia (e.g., Gormsen, Rosenberg, Bach, & Jensen, 2010). Evidence of Path A has also been found in studies of chronic pain conditions that are most prevalent in midlife and older adults, such as post-herpetic neuralgia (e.g., Haythornthwaite, Clark, Pappagallo, & Raja, 2003). Studies of chronic pain patients have generally reported bivariate effect sizes for Path A in the small to moderate range (see reviews by Banks et al., 1996; Haythornthwaite & Benrud-Larson, 2000; and Tunks et al., 2008).

A positive relationship between pain intensity and depressive symptomatology has been documented in clinical samples of persons not specifically identified as chronic pain patients. Correlations in the moderate range (e.g., $r = .31$ to $.48$) have been reported in most studies of patients with rheumatoid arthritis (e.g., Covic et al., 2000; Hagglund, Haley, Reveille, & Alarcon, 1989; Smedstad et al., 1995; Waltz, Kriegel, & Van’T Pad Bosch, 1998). Studies of patients with osteoarthritis have generally reported small to moderate bivariate effect sizes (e.g., Breedlove, 2004; Rosemann et al., 2007; Scopaz et al., 2009). Pain intensity has also been linked to depressive symptomatology in the context of cancer. A review by Zaza and Baine (2002) found that greater pain intensity
was associated with increased psychological distress in 14 of 19 studies published between 1980 and 2000. In these studies, psychological distress was most often assessed via measures of depressive symptomatology or mood disturbance, and effect sizes were generally moderate. Several more recent studies of cancer pain have also found evidence of Path A (e.g., McMillan et al., 2008; Mystakidou et al., 2007). Finally, pain intensity has been linked to depressive symptoms in other clinical samples, including neurology outpatients (Williams, Jones, Shen, Robinson, & Kroenke, 2004) and patients with multiple sclerosis (see review by Arnett, Barwick, & Beeney, 2008) or Hepatitis C (e.g., Morasco et al., 2010).

Although many studies have examined pain in relation to depression and/or depressive symptomatology in samples of community-dwelling persons, few have investigated pain intensity. This may be at least partially attributable to the lack of attention given to pain in broad social surveys, especially in the United States. Measures of psychological distress, depression, and depressive symptomatology have also varied widely across such studies; moreover, when used in the general population, many of these measures yield skewed data. As a result, researchers often have been limited to examining one or both constructs at a very broad level. So, for example, many more studies have examined the presence of pain—often further limited by location (e.g., back pain) or associated with a specific disease condition (e.g., osteoarthritis)—in relation to depressive symptomatology than have examined pain intensity or severity. Research also has focused more on the presence of high levels of distress—often defined by cut-points on established scales of depressive symptomatology, such as the CES-D or BDI.

Consequently, volumes of research document a relationship between reports of pain and
“depression,” “depressed mood,” or “elevated” depressive symptomatology in community-dwelling adults. Studies of pain in midlife and older adults are especially likely to be represented in this category (e.g., Onder et al., 2005; Reyes-Gibby et al., 2002; Reyes-Gibby et al., 2005). For example, Reyes-Gibby and colleagues found a significant association between reports of trouble with pain and scoring as “depressed” (endorsing 4 or more items on an 8-item CES-D) in a representative sample of community-dwelling adults over age 70 in the U.S. (i.e., the AHEAD study).

There have been few in-depth studies of pain in the general population (i.e., with sizeable samples drawn using complex sampling procedures). The few studies that exist have focused mainly on identifying community-dwelling persons with “chronic pain.” Although not common, a handful of these studies have examined pain intensity in relation to depressive symptomatology. Ohayon and Schatzberg (2010), for instance, queried a representative sample of over 3,000 adults in California about their experiences of pain in several body sites (e.g., back, neck, arms, abdomen). Reported pain was classified (post hoc) as chronic if it was present at the time of the interview, had occurred at least three days per week for at least the past three months, and was of at least mild intensity. While the authors were most interested in the association between chronic pain and clinically-diagnosable major depression, they reported a significant modest positive correlation ($r = .25$) between overall pain intensity (summed intensity ratings across all sties) and depressive symptom severity in the total sample.

One common strategy used by researchers has been to infer the existence of pain—often “chronic pain”—from data contained in large health and social surveys regarding specific diseases, or conditions, in which pain is a common symptom. So, for
example, reports of back problems, frequent headaches, arthritis, and gastrointestinal conditions have been examined as sources of chronic pain within large studies of community-dwelling persons throughout Europe (e.g., Ohayon & Schatzberg, 2003), Australia (e.g., Bonnewyn et al., 2009), Canada (e.g., Carroll et al., 2000; Munce & Stewart, 2007), and the U.S. (e.g., McWilliams et al., 2003). Since most of these surveys routinely assess or screen for depression, several researchers have examined these symptom/screening data in relation to these chronic pain conditions. Many of these studies have yielded compelling evidence of an association between these pain conditions and levels of symptomatology that may signal depression in community-based samples. Ohayon and Schatzberg (2003), for example, examined the health problems reported by a representative sample of over 18,000 adults who participated in a study of sleep habits and behaviors fielded in five western European countries. While those who reported any health condition (painful or not) had increased odds of having major depression (diagnosed via clinical interview), the odds were significantly greater among those reporting chronically painful physical conditions (CPPC).

Researchers have also made use of the (often limited) pain information collected within studies of health and/or aging targeting certain subgroups of community-dwelling adults (e.g., middle-aged persons in the MIDUS study or HRS; persons with physical disabilities; persons of Hispanic origin; women). Pain intensity or severity data have sometimes been available in these studies, yet researchers often have collapsed response categories prior to analysis. Thus, several studies of midlife and/or older adults have observed a significant relationship between reports moderate/severe pain and depressive symptomatology (e.g, Herrick et al., 2004; Scudds & Ostbye, 2001) or “significant”
levels of depressive symptomatology (e.g., Covinsky et al., 2009).

Some “quasi-linear” relationships have also been reported in community samples, although findings often have been limited by pain type—for example, Weiner and colleagues (2003) reported a significant linear trend between CES-D scores and levels of severity of low back pain reported by older adults in the Health ABC study. A similar finding was reported for knee pain intensity in the Women’s Health and Aging Study (Lamb et al., 2000). A few studies have reported correlations of low to moderate size between pain intensity/severity and depressive symptomatology. For example, statistically-significant, within-wave correlations of .11 to .12 were found in a community study that oversampled physically-disabled adults (Gayman, Turner, & Cui, 2008). Slightly larger effect sizes have been reported in studies comprised of community-residing aging persons with specific disease conditions (often arthritis), including samples recruited from the community (e.g., Sale, Gignac, & Hawker, 2008), as well as those in which a subset of respondents was culled from larger, community-based samples—for example, a correlation of .30 was found between pain severity and depressive symptomatology in a sample of 235 older adults with arthritis that was drawn from a larger (non-arthritis), representative sample (Tsai, 2005).

A sizeable minority of studies has not found evidence of a significant bivariate relationship between pain intensity and depressive symptomatology. Several early studies with samples comprised of chronic pain patients reported a lack of significant findings (e.g., Harkins & Price, 1992; Kerns & Turk, 1984; Thomas & Roy, 1989; Von Korff et al., 1992). The association between pain intensity and depressive symptoms has not reached significance in several clinical samples, including a few studies of patients
with advanced cancer (e.g., Mystakidou et al., 2006; Rustoen et al., 2005), and some studies of osteoarthritis patients (e.g., Dekker et al., 1993). It should be noted that several of these studies had fairly small samples (e.g., $n = 58$ in Dekker et al., 1993; $n = 30$ in Kerns & Turk, 1984). Interestingly, few studies of community-dwelling adults (including aging adults) have reported that the bivariate relationship between depressive symptomatology and pain intensity was not significant. One study of a small cohort ($n = 79$) of community-dwelling adults with ADL limitations found that the development of significant depressive symptomatology was not associated with reports of pain intensity; it was, however, associated with a measure of pain frequency (Livingston et al., 2000). However, the (non-)findings in community-based studies may reflect (a) the small number of studies that have examined (or reported on) this specific relationship; or (b) a potential “file drawer” problem.

Thus, evidence from a variety of studies with markedly different populations attests to the covariation of pain intensity and depressive symptomatology. Moreover, available evidence indicates that this relationship is surprisingly robust. This relationship has been evaluated in a multivariate context in a substantial number of prior studies. Although some weakening in relationship strength was observed in some studies (e.g., Smedstad et al., 1995; Tsai, 2005), others reported that it remained statistically significant, with little diminution in strength. In fact, in several studies, pain intensity or severity displayed the strongest, or one of the strongest, relationships with depressive symptomatology. Such findings have been reported in studies of both married chronic pain patients (e.g., Cano et al., 2000) and chronic pain patients not selected on the basis of marital status (e.g., Geisser et al., 2000); clinical samples of persons with osteoarthritis
(e.g., Rosemann et al, 2007) and rheumatoid arthritis (e.g., Wolfe & Hawley, 1993); and in some studies of community-residing adults (including aging adults) with chronic pain or pain in specific locations (e.g., Carroll et al, 2000; only among those reporting chronic back pain in Currie & Wang, 2004; knee pain in Sale, 2008). This pattern has also been reported in several studies in which one or both constructs were examined more broadly (e.g., Covic et al., 2006; Ohayon & Schatzberg, 2003).

The list of covariates included in past studies is vast and diverse. Studies have typically included age, gender, and SES (most often income and/or education); race and/or ethnicity and marital status have also been included in some studies. The inclusion of health-related characteristics other than pain intensity/severity has depended largely on the nature of the sample. Studies of persons with specific disease conditions, such as arthritis or cancer, have typically included one or more measures of disease severity and/or progression (e.g., disease markers in x-rays or blood tests, location and stage of cancer). Several of these studies observed that pain intensity was more strongly related to depressive symptomatology than were clinical indicators of disease severity (e.g., Smedstad et al., 1995; McAlindon, Cooper, Kirwan, & Dieppe, 2003). Numerous disease-specific studies have also controlled for disease duration, but it is difficult to determine whether these questions assessed disease duration or duration of pain.

Studies of chronic pain patients often have included additional pain information, such as duration, frequency, and/or location(s); select studies have also controlled for employment status, involvement in litigation, and/or current treatment. Most studies of pain in community-dwelling persons have included some indicator of the presence and/or severity of disease conditions, although the nature of these indicators has varied across
studies. Ohayon & Schatzberg (2003), for example, controlled for non-painful
diseases/conditions, effectively illustrating the unique impact of chronically painful
physical conditions. Others have included a count of disease conditions/comorbidities
(e.g., Currie & Wang, 2004; Tsai, 2005). Depending on the specific aims of a study,
variables such as cognitive appraisals, coping strategies, marital functioning, significant
life events, social support and functioning, personality styles, self-rated health, health and
lifestyle indicators (e.g., body weight, exercise), and other dimensions of psychological
well-being may also have been incorporated into multivariate models.

In spite of observing a significant bivariate relationship, a number of studies have
reported that the relationship between pain intensity and depressive symptomatology was
not significant in a multivariate context. This pattern has been observed in several
studies of chronic pain patients (e.g., Pence et al., 2008; Tan et al., 2005), as well as a
study of chronic pain reported in a sample of primary care patients (Karoly et al., 2006).
A recent study of over 500 community-dwelling midlife and older adults with
osteoarthritis of the hip and/or knee also found no significant relationship between pain
intensity and depressive symptomatology in longitudinal, multivariate models (Hawker,
et al., 2011). In all but the study by Karoly et al. (2006), the lack of significant findings
might be attributable to the constellation of other variables included in the models. For
example, the multivariate model tested by Pence et al. (2008) included marital
satisfaction and reports of the spouse’s reactions to the patient’s pain—factors that could
potentially mediate or moderate the relationship between pain intensity and depressive
symptomatology. Multicollinearity may have been a factor in the pattern of findings
observed in Tan et al. (2005) and Hawker et al. (2010). More will be said about this issue
of potential multicollinearity issue later in this review.

A fair number of studies have reported inconsistent findings. For example, in one study of community-dwelling adults with hip or knee pain, Path A was not statistically significant in cross-sectional models, but became significant in longitudinal models (e.g., Hutchings et al., 2007). Significant associations have been observed in clinical samples between pain intensity/severity and depressive symptomatology/depression when constructs have been assessed using some scales, but not others (e.g., Creamer et al., 1999; Mystakidou et al., 2006; Teunissen, De Graeff, Voest, & de Haes, 2007). However, it is difficult to evaluate whether the variability observed across studies is associated with the use of particular measures, characteristics of the study samples, or the intersection of the two (i.e., the use of a specific measure within certain samples).

Finally, some prior studies have reported differences in the significance and/or strength of Path A by gender. However, studies have varied widely as to the domains measured for each construct. Large-scale studies of community-dwelling adults in Canada (Munce & Stewart, 2007) and in Europe (Onder et al., 2005) found that, although the relationship between pain intensity and reports of significant depressive symptomatology or depression was significant for both men and women, the relationship was stronger in women. In another community-dwelling sample, Bonnewyn et al. (2009) observed that the bivariate relationship between reports of persistent pain and scoring as depressed on the CIDI was significant only among females. A few studies using samples of cancer patients (e.g., Pud, 2011) and chronic pain patients (e.g., Haley et al., 1993) have reported similar findings. However, in the majority of these studies, it does not appear that the authors conducted any statistical tests (or appropriate tests) of the
differences observed in either bivariate or multivariate relationships.

A few studies have reported gender differences in the opposite direction. A study of chronic back pain in the community found that pain intensity was significantly related to major depression (as measured using the CIDI); however, a significant interaction effect involving gender was also found, such that this relationship was stronger among men. Moreover, this finding was robust, as it was little reduced in analyses controlling for other variables (Currie & Wang, 2004). Meanwhile, a study by Affleck et al. (1999) found no significant overall effect associated with daily ratings of joint pain intensity on negative mood the next day among rheumatoid and osteoarthritis patients; yet, comparative analyses revealed a significant effect for males, but not for females.

A somewhat larger body of literature was located in which no gender differences have been found in the strength or existence of the relationship between pain and depressive symptomatology or depression. Notably, all of these studies used samples comprised of chronic pain patients (e.g., Cano et al., 2000; Herr et al., 1993; Keogh et al., 2006). Sample sizes in these studies seemed adequate and most researchers also reported descriptive data to show that males and females did not tend to differ on other variables.

Overall, the bulk of the existing evidence attests to a modest, but significant relationship between pain intensity and depressive symptomatology. This relationship has been observed in diverse samples and appears relatively consistent, despite variation in selected measures.

Path B: The Relationship Between Pain and Activity Limitation

As discussed earlier in this chapter, numerous conceptual definitions have been developed around “activity limitation.” The present study was concerned primarily with
physical limitations—i.e., limitations in basic physical abilities such as climbing stairs, walking a certain distance, stooping/kneeling, lifting/reaching, etc.

Several sources of difficulty are encountered when attempting to summarize the empirical evidence pertaining to the relationship between pain and activity limitation (Path B). First, the evidence base is fairly limited—that is, proportionately more studies have examined the impact of having pain (i.e., pain presence) on activity limitation than have examined pain intensity. Second, when activity limitation is examined, the terminology used across studies is very inconsistent. Not only does the terminology vary across studies in different areas (e.g., chronic pain, osteoarthritis, gerontology), it often varies within area as well. Third, as was mentioned earlier in the review of terminology selected for the proposed conceptual model, numerous instruments have been developed to measure activity limitation. Because the names of these measures are often unclear, considerable effort is required to uncover the content of scales. Fourth, differences exist across studies in terms of the method by which some measures are obtained. For example, studies of aging and health—especially cohort studies—increasingly have moved toward assessing performance of physical abilities (e.g., timed chair stands, gait speed; see Fried et al., 2000), whereas studies of chronic pain patients and large population-based studies rely on self-report measures. In an attempt to limit its scope, this review concentrates primarily on the findings with respect to Path B in studies using self-report measures of activity limitation.

Some additional observations about existing measures are warranted. Very few studies within the chronic pain literature have used scales and/or items developed by Nagi (1979) or others that are consistent with the construct of physical limitations as
defined in the present study. Instead, pain researchers generally have either (a) used broad measures of health that have typically been used in medical research (e.g., Medical Outcomes Study SF-36), or (b) developed measures specifically for use with pain populations (e.g., Brief Pain Inventory [BPI]; Cleeland, 1989). Many measures in the latter category can be conceptualized as “pain-specific.” That is, most have item stems, or are prefaced by general instructions, that explicitly refer to the respondent’s pain (e.g., “[Rate the] extent to which pain has interfered with…”). Examples of such measures include scales of pain interference on the Brief Pain Inventory (BPI; Cleeland, 1989), the WHYMPI (Kerns et al., 1985), the PDI, and the Rolland Morris Disability Questionnaire (RMDQ; Rolland & Morris, 1983). Several other measures of activity limitation have been developed for specific pain conditions—e.g., the Headache Impact Test (HIT; Kosinski et al., 2003) and the Migraine Disability Assessment Questionnaire (MIDAS; Stewart, Lipton, Dowson, & Sawyer, 2001). A surprising number of studies have relied on single-item indicators of pain-specific activity limitation or pain interference. For example, some studies with chronic pain patients have asked respondents to indicate the extent to which they “cut down on activities due to [pain/pain condition]” (e.g., Hanley et al., 2006; Reid, Williams, & Gill, 2005). These single-item measures are open to interpretation—respondents may consider one or more of a variety of different types of activities (e.g., social, physical, vocational, personal care, etc.) in their responses.

Returning to the former type of measure—i.e., broad measures of health: Three measures appear to dominate published studies that have examined the association between pain and activity limitation. These include the Stanford Health Assessment Questionnaire (HAQ; Fries, Spitz, Kraines, & Holman, 1980) and the SF-36. Both of
these measures are extremely broad. The SF-36, for example, includes items that assess mental health functioning and symptomatology. However, both measures have a specific subscale or subset of items that closely approach the current study’s definition of physical limitations (i.e., the HAQ-Disability Index; the SF-36 Physical Functioning Scale). Some, though not all, measures developed for arthritis research (e.g., AIMS; the Western Ontario and McMaster Universities Osteoarthritis Index [WOMAC; Bellamy & Buchanan, 1986]) and cancer (e.g., the Functional Assessment of Cancer Therapy-General scale [FACT-G]; Cella et al., 1993) also have subscales that are more closely related to physical limitations. The SIP, too, has a physical disability dimension.

In community-based studies, researchers tend to use either broad measures like the SF-36. Within the gerontological literature, however, there is a distinct tradition associated with the use of multi-item measures of functional impairment or limitation. As mentioned earlier in this chapter, most of these scales are rarely limited to basic physical limitations (e.g., difficulty walking, reaching, kneeling, etc.). If physical limitations are included, a majority of studies combine such items with additional items measuring limitations in IADLs (e.g., shopping, leisure activities) and/or ADLs (e.g., self-care activities, such as bathing and dressing).

Given the variety of available measures being utilized and the diversity of samples reflected in published studies, the summary below is offered at a very “broad” level. Attention is directed toward studies that have used measures containing items that are most similar to basic physical limitations. However, this distinction is difficult to maintain, especially in studies of chronic pain where scales of pain interference tend to resemble Williamson’s Activity Restriction Scale—i.e., they tap multiple areas of
“functioning,” including social, occupational, recreational, and domestic activities.

Looking broadly across the various literatures: A majority of published studies document a positive relationship between pain intensity (or severity) and activity limitation. Although not all studies have reported estimates of effect size, among those that have done so, bivariate correlations have generally ranged in strength from moderate to strong. A few selected examples are presented below.

Several studies of married individuals have reported a strong bivariate relationship between pain intensity and activity limitation. Most of these studies have measured activity limitation specific to pain, using a scale of pain interference. In samples drawn from clinical settings, correlations of .52 in a sample of 127 married patients with chronic musculoskeletal pain (Burns et al., 1996) to .66 in a sample of 64 married patients with chronic headache (e.g., Pence et al., 2008). In a study in which 105 married persons with pain were drawn from community-dwelling couples recruited because of (at least) one spouse suffering from chronic musculoskeletal pain, Cano et al. (2006) reported bivariate correlations between pain severity and the SIP-physical disability scale of .48 and .61 for the MPI Pain interference scale. Correlations reported in non-chronic pain samples tend to be slightly lower. In a sample of 235 older community-dwelling persons with arthritis, a moderate correlation ($r = .34$) was reported between ratings of pain and a multi-item measure of disability that included IADLs and basic physical limitations (Tsai, 2005). Ohayon and Schatzberg (2010) reported a correlation of .42 between levels of musculoskeletal pain and physical limitations in a subsample of community-dwelling older adults who met criteria for major depression.

Substantial effect sizes have also been observed in a number of longitudinal
investigations—most of which have been conducted with community-based samples. For example, several large cohort studies document a moderate to strong positive relationship between knee and/or hip pain intensity and limitations in physical functioning. Studies include the Johnston County Osteoarthritis Study in the southern U.S. (e.g., Jordan et al., 1996, 1997), the MAK study in the midwest U.S. (e.g., Sharma et al., 2003), the Somerset and Avon Survey of Health in the U.K. (e.g., Ayis & Dieppe, 2009), and the KNEST study in the U.K. (e.g., Jinks et al., 2007). Evidence from these studies is especially compelling, not just because of the longitudinal nature of the data, but also because these studies restricted the measurement of activity limitation to the more purely physical items/subscales (identified earlier) on the SF-36, the WOMAC, or the HAQ. Investigations using samples drawn from HRS (e.g., Clark et al., 1997) and AHEAD (e.g., Clark et al., 1998) also provide evidence in support of Path B within community-dwelling aging populations.

In the majority of published reports, the relationship between pain intensity and activity limitation remains significant and sizeable after controlling for a number of covariates. Most prior studies have controlled for age and gender. A few investigations have controlled for other characteristics such as obesity (e.g., Jordan et al., 1996) or BMI (e.g., Ayis & Dieppe, 2009), and SES (e.g., Jinks et al., 2007). Furthermore, scholars in arthritis have often observed that physical limitations tend to be better explained by reports of pain than by “objective” evidence of disease severity, such as that shown on x-ray (e.g, Gettings, 2010; Sokka, Kankainen, & Hannonen, 2000).

Compared the amount of literature published with respect to Path A, considerably fewer studies have examined potential gender differences with respect to Path B. Using
data from over 2,700 older adults (age 70-79) in the Health ABC Study, Weiner et al. (2003) examined the relationship between low back pain severity (measured as a composite of intensity and frequency) and reports of functional difficulty (including basic physical limitations and some IADLs). Their analyses were quite detailed, as they examined this relationship separately by gender and also for each specific task. Overall, they found strong evidence that increased severity of low back pain was associated with increased difficulty across most tasks for both genders. However, they also observed some gender differences—for example, relationships were more consistent and appeared more linear among women; among men, increased pain was often tied to functional difficulty only when it reached the highest levels of severity or when the tasks was more strenuous (e.g., lifting, moving things). The findings of Weiner et al. were preserved after controlling for a number of relevant covariates. In contrast, a study by Hairi et al. (Hairi, Cumming, Blyth, & Naganathan, 2013) found that an observed gender difference in the relationship between pain severity and physical disability (measured as difficulty with several basic, IADL, and ADL tasks) was reduced as more covariates were added to multivariate models. In this analysis of “chronic pain” (pain every day for more than 3 of the past 6 months) among community-dwelling older adults (≥ 65), the initial bivariate relationship between pain severity and physical disability was stronger for men than for women. A pattern of gender differences reduced to nonsignificance in a multivariate context was also reported by Scudds and Robertson (2000), although in their study, the bivariate analyses suggested that women were more likely to report disability associated with pain (measured as difficulty with three or more basic physical, ADL, and IADL tasks).
Hirsh et al. (2006) reported bivariate correlations between chronic pain patients’ overall rating of pain severity (as measured by the MPQ) and physical disability (as measured by the PDI) of .44 for men and .48 for women. As might be expected a z-test comparing these two coefficients was not statistically significant. A few other studies have suggested that their findings support the existence of gender differences in Path B (e.g., Bingefors & Isacson, 2004; Sharma et al., 2003), but it is unclear from published reports whether such differences were tested statistically. Additionally, some authors appear to imply that gender differences in mean levels of pain intensity and/or physical limitations constitute a difference in Path B (e.g., Marcus et al., 2003).

Overall, a preponderance of literature supports a moderate to strong relationship between pain and activity limitation. Evidence specific to pain intensity and physical limitations is more sparse, but existing data also suggest that this relationship is of similar strength. This relationship has primarily been observed in clinical samples, but community-based cohort studies have also documented an association. Although the relationship may vary somewhat according to the specific measures used and the nature of the sample, the relationship appears to be relatively robust to the introduction of multivariate controls.

Path C: The Relationship Between Activity Limitation and Depressive Symptomatology

Theoretically, Path C can exist independent of any relationship to pain. That is, activity limitation could result from any number of possible sources, including chronic diseases that affect musculature, heart rate, or lung capacity, or even physical abnormalities (e.g., club foot). It is beyond the scope of the present study to conduct a
critical review of this more expansive literature. However, numerous studies attest to a
sizeable relationship between physical limitations (a.k.a., physical impairment, functional
limitations, mobility limitations, upper-body disability) and depressive symptomatology
or depression. This relationship was documented in early studies with older community-
dwelling adults by Berkman et al. (1986) and Blazer et al. (1991). Later studies focused
on specific segments of the population, such as those in midlife and older adulthood (e.g.,
Zeiss et al., 1996) and the oldest-old (e.g., Femia et al., 2001). Evidence supporting Path
C has also been found in studies using the HRS and AHEAD samples of community-
dwelling adults (e.g, Fonda & Herzog, 2001). Estimates of effect size available in
published studies suggest a small to moderate bivariate relationship (e.g., \( r = .20 \) to \( .49 \) in
Femia et al., 2001; \( r = .21 \) to \( .23 \) in Gayman et al., 2008; \( r = .26 \) in Yang, 2006).

When Path C is considered within the context of pain, the evidence base that
specifically attests to the relationship between physical limitations (as defined in the
proposed conceptual model) and depressive symptomatology is somewhat sparse. In
addition, many of the concerns raised earlier with respect studies related to Path B are
also applicable to the body of literature addressing Path C.

Again, however, looking broadly across the various literatures in which Path C
has been examined: The preponderance of evidence supports a positive relationship
between activity limitation and depressive symptomatology or depression. Again,
estimates of effect size are not always reported in the literature. However, published
bivariate correlations have covered a fairly broad range, from small (e.g., \( r = .20 \) to a few
in the strong range, e.g., \( r = .63 \)). On the whole, correlations have tended to be in the
low-moderate range.
Correlations for Path C in studies of married persons with pain have ranged from .44 in the study by Pence et al. (2008) to .59 in the study by Gauthier et al. (2008). Cano et al. (2006) reported a bivariate correlation between “psychosocial disability” on the SIP and the .63 on the SIP Physical disability scale, and .46 on the MPI Pain Interference scale. A study of 103 married RA patients reported a correlation of .32 (Manne & Zautra, 1989). One study of cancer patients (McMillan et al., 2008) reported a correlation the BPI pain interference scale and the CES-D of .56.

Reported correlations in studies using samples of persons with osteoarthritis have ranged from .33 for upper-body physical limitations and .44 for lower-body limitations with depressive symptomatology among primary care patients with osteoarthritis (Rosemann et al., 2007) to .50 among community-dwelling adults with arthritis (Tsai, 2005). Another study that examined reports of chronic pain among primary care patients observed a slightly stronger correlation ($r = .60$ in Karoly et al., 2006). Estimates of bivariate effect size in studies of community-dwelling persons with pain are less readily located. However, studies by Gayman et al. (2008) and Strine et al. (2004) both reported correlations between .20 and .30. Numerous studies have examined the relationship between activity limitation and the presence of significant depressive symptomatology or depression; most report a sizeable and relationship, both in cross-sectional (e.g., Reid et al., 2002) and longitudinal analyses (e.g., Arola, Nicholls, Mallen, & Thomas, 2010).

A handful of studies have not observed a significant relationship between activity limitation and depressive symptomatology or depression. No significant bivariate relationship was observed in studies of older adults with osteoarthritis (e.g., Dekker et al., 1992) and a study of older adults with persistent back pain (e.g., Weiner et al., 2004).
Other studies have reported inconsistent findings. For example, in one study of community-dwelling older adults in the UK reported a significant bivariate relationship between depression “caseness” on a diagnostic interview and a measure of activity limitation due to pain when it was used as a continuous variable, but did not find a significant association when the measure was dichotomized at the median (e.g., Livingston et al., 2000).

The few studies that have examined gender differences with respect to Path C have reported conflicting findings. For example, a study of chronic pain patients by Hirsh et al (2006) reported that the relationship between overall disability (as measured on the PDI) and a negative mood factor (a latent variable created from several measures of psychological distress, including the BDI) was significantly stronger among females than males. Two other studies with chronic pain patients by Keogh et al. (2006) and Bolton (2004) also reported that Path C was considerably larger in females than males. However, as noted by Hirsh et al. (2006), neither of these studies controlled for the effect of pain. Several other studies have reported not finding any evidence of gender differences in the activity limitation-depressive symptom relationship, including a study of chronic pain patients (Herr et al., 1993), community-based midlife and older adults (Zeiss et al., 1996), and a study using a national epidemiologic survey of substance use (Barry et al., 2012).

Most studies report that the relationship between activity limitation and depressive symptomatology or depression is robust to the introduction of multivariate controls. Only the study by Pence et al. (2008) reported that the effect was rendered non-significant with the introduction of covariates. It is notable however, that the controls in
this study of married pain patients included perceptions of the spouse’s response to the 
pain of the patient and a measure of marital satisfaction. For the most part, controls had 
little effect on the observed strength of Path C.

Summary of Evidence for Paths A, B, and C

In summary, available research supports the existence of relationships among all 
three of the key constructs in the proposed conceptual model—pain, activity limitation, 
and depressive symptomatology. In most studies, these relationships have ranged in size 
from small to moderate. Most of the limitations of existing research are related to the 
varied definitions of, and measures used to assess, key study constructs. A few examples 
are highlighted below.

In order to make use of existing data, some researchers have collapsed the 
response categories on measures of key study variables. For example, it is not unusual 
for measures of pain intensity to be reduced to a few categories for analysis. Although 
most of these studies report a significant relationship between (certain levels of) pain 
intensity and depressive symptomatology, it can be difficult to compare the resulting 
effect sizes with those derived from correlational analyses using a continuous measure of 
pain intensity. Moreover, studies vary considerably in how these categories have been 
created. To generate a dichotomy, some researchers combine responses indicating 
“moderate” or greater intensity (e.g., Covinsky et al., 2009; Herrick et al., 2004; Scudds 
& Ostbye, 2001), whereas others limit their consideration to “severe” (vs. lesser—i.e., 
“mild” or “moderate”) pain intensity (e.g., Emptage, Sturm, & Robinson, 2000; Reyes-
Gibby et al., 2007).

Still other researchers have developed cut-points on 10- and 11-point rating scales
to distinguish between “mild,” “moderate,” and “severe” pain (e.g., Duong et al., 2005; Serlin, Mendoza, Nakamura, Edwards, & Cleeland, 1995), or between “high” and “low” pain (e.g., Melzer, Gardiner, & Guralnik, 2005). Other researchers have created different categories of pain severity by combining pain intensity ratings with other available information. For instance, Weiner et al. (2003) examined the relationship between depressive symptomatology (as measured by the CES-D) and “level” of low back pain reported by participants in the Health ABC Cohort (all age 70-79 living in the community). Four pain levels were created by combining responses indicating the presence of low back pain with reports of usual pain intensity and frequency: None (no low back pain reported); Mild intensity pain of any frequency; Pain of at least moderate intensity experienced less than very often; and Pain of at least moderate intensity experienced at least very often. Although scores on the CES-D varied significantly across pain levels, and a linear trend test was statistically significant, it is challenging to assimilate these findings into the body of existing evidence. As has been noted by others (e.g., Paul et al., 2005; Zelman et al., 2003), the pain intensity categories that result from these approaches can vary considerably across diseases and conditions, as well as various study-specific sample inclusion and exclusion criteria.

Other issues related to measurement may account for some of the heterogeneity in effect sizes observed across studies. First, some differences in effect size appear to be related to specific measures. In studies involving pain intensity, correlations above .40 generally have been limited to those studies using either a visual analog scale (VAS) or a multi-item measure of pain intensity (e.g., the AIMS pain scale); this has been true of many studies of Paths A and B among patients with rheumatoid (e.g., Brown, 1990;
Waltz et al., 1998) or osteo-arthritis (e.g., Sokka et al., 2000). Second, larger effect sizes tend to be observed when one or both constructs are conceptualized as latent constructs or are measured by combining items from different indices. For example, “negative mood” (a factor comprised of items on the BDI, along with measures of anxiety and anger) was correlated with pain severity at .59 in male and .39 in female chronic pain patients (Hirsh et al., 2006). Bivariate correlations over .45 were reported in two RA studies (Brown et al., 1990; and Smedstad et al., 1995) in which a “composite” measure of pain intensity was correlated with depressive symptomatology. Smedstad et al.’s (1995) unusual approach combined respondents’ VAS pain intensity rating, their AIMS pain scale score, and their answers to several pain items from the Nottingham Health Profile. Third, differences in effect size may also be attributable to slight variations in the wording of measures. As noted above, differences have been reported in the extent to which ratings of “worst,” “least,” “average,” and “current” pain intensity are related to outcomes including depressive symptoms (e.g., McMillan et al., 2008; Rustoen et al., 2005).

Even among studies that utilize the same or a similar set of items, researchers may apply different operational definitions in their coding of the items. For example, Guccione et al. (1994) defined functional limitations as the need for human assistance with ADL, IADL, and physical limitations, whereas Lichtenstein et al. (1998) coded a similar set of items to indicate “any difficulty,” regardless of whether or not assistance was needed. A similar divergence can be seen in the treatment of depressive symptomatology. Most studies examining Paths A or C have used one of several established measures of depressive symptomatology (e.g., BDI, CES-D, GDS); however, the underlying construct has been conceptualized as (“likely” or “probable”) depression.
in roughly half of these studies. It is often difficult to incorporate the findings of these various studies into the body of existing evidence.

Finally, there are serious concerns related to construct contamination and overlap within the existing literature. This is most evident in studies that use subscales from the same measure to investigate Paths A, B, and/or C. For example, the Bodily Pain scale of the SF-36 scale conflates pain intensity and interference, and the Physical Health component score includes the BP scale. Consequently, the correlations between these scales and other measures of pain intensity and/or pain interference will necessarily be inflated. A more serious concern can be levied against the pain intensity and physical disability scales of the WOMAC. Numerous studies have reported these scales to be highly correlated—e.g., \( r > .83 \) in studies by Maly et al. (2006), and Sale (2008). Yet these scales were utilized by several other studies reported earlier in this review—none of which mentioned any potential problems with collinearity (e.g., Hawker et al., 2010; Hutchings et al., 2007). As was noted in the review of studies under Path A, multicollinearity may have been a contributing factor in the (non)-findings of Hawker et al. (2010). Many of the aforementioned concerns and issues regarding differences in the definitions and measurement of key study variables are highlighted in the review of mediation studies presented next. Special attention is also paid to the potential overlap of study constructs within these studies.

**Review of Studies Testing Mediation by Activity Limitation**

This section reviews prior studies that have investigated whether (or the extent to which) activity limitation mediates the relationship between pain and psychological distress. These studies constitute a relatively small body of research. Relevant studies
were identified through a systematic search of relevant databases (e.g., PsycInfo, Medline). The reference lists of identified publications were also reviewed in order to locate additional studies.

Several studies were retained in this review even though they focused on populations other than community-dwelling middle-aged and older adults (e.g., older adults in assisted living and congregate residences, cancer patients). This was justified because so few mediation studies have been conducted within the target population. Moreover, given the relative dearth of mediation research focused specifically on married couples (or persons) in later life, studies in which participants fell within the target age range were included, regardless of the marital status composition of the sample.

Consistent with earlier portions of this literature review, mediation studies involving chronic pain patients were included here. However, a handful of studies of chronic pain patients were excluded from this review because they focused solely on cognitive mediators (e.g., cognitive appraisals of control, pain beliefs, coping styles). Although cognitive mediators almost certainly play an important role in adjustment to pain, the emphasis of this study (and conceptual model) was on the role of activity limitation.

A handful of studies were cited more than one time in the review of evidence supporting Paths A, B, and C, but did not meet criteria for inclusion as a mediation study. Many of these studies provided evidence supporting two of the three requisite pathways, but did not evaluate (or provide adequate information to permit evaluation of) the third pathway. Often, the pain-activity limitation-depressive symptomatology mediation pathway was not of interest in these studies. A subset of studies reported significant bivariate correlations between pairs of study constructs, but either did not conduct
relevant multivariate analyses or did not report analysis results in a way that permitted an assessment of mediation (e.g., Bierman, 2011; Herr, Mobily & Smith, 1993; Wolfe & Hawley, 1993). Finally, selected studies tested models with a different causal ordering of constructs—for example, Hirsh et al. (2006) tested whether negative mood mediated the relationship between pain severity and physical disability.

There are a number of different guidelines and approaches to testing for and probing mediation effects (e.g., Baron & Kenny, 1986; MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002; Sobel, 1988). This study relied mainly on the approach outlined by Baron and Kenny (1986). According to Baron and Kenny, studies that provide support for mediation must do three things: (1) show that the focal independent variable is related to the dependent variable; (2) establish that the mediating variable is related to the focal independent variable; (3) establish that the mediating variable is related to the dependent or outcome variable of interest; and (4) show that when the mediating variable is added to a model predicting the dependent variable, the relationship between the focal independent variable and the dependent variable is either reduced (partial mediation) or eliminated (total mediation). In terms of the proposed conceptual model, these propositions correspond to: (1) Path A; (2) Path B; (3) Path C; and (4) the reduction in the strength of Path A once the mediating variable (and, by extension, the combination of Paths B and C) is added to a model predicting depressive symptoms.

Another major approach to testing for mediation (MacKinnon et al., 2002) argues that the first step outlined by Baron and Kenny (1986; above) may not be necessary for establishing that mediation exists. In some cases, the focal independent variable may not evidence a significant, direct relationship to the dependent variable, but it may still have
an indirect relationship to the dependent variable by virtue of its relationship with the mediating variable. Hence, MacKinnon et al. suggest that even if step one is not met, researchers should still proceed with the subsequent steps in testing for mediation. In recent years, Kenny has acknowledged that MacKinnon and colleagues’ arguments and approach have some merit (Kenny, 2008). Although the current study relied primarily on the Baron and Kenny approach to establishing mediation, the MacKinnon et al. approach was applied in reviewing some prior studies.

Again this body of literature mainly consists of studies of individuals with pain who have been sampled without regard to marital status. Studies that are exceptions to this characterization (when recognized as such) are clearly noted within the review; most often, such studies have reported data from only one member (the patient, generally) of a patient-spouse/caregiver dyad recruited to participate in studies related to family relationships in chronic pain or painful conditions (e.g., arthritis).

Priority was given to studies involving mediating constructs most similar to the conceptualization of activity limitation as outlined in the proposed conceptual model—i.e., basic physical limitations. However, the myriad definitions and labels applied to the mediating constructs investigated in prior studies made it necessary to incorporate studies that focused on “functional disability,” “activity restriction,” “(pain-related) disability,” “(pain-related) activity interference,” and other types of activity limitation. Thus, this review included some studies that have examined social, recreational, and IADL and ADL functioning, especially when these other areas were grouped with basic physical limitations into a composite measure.

In addition, because of variability in definitions and measures of depressive
symptomatology (as well as limited research focused exclusively on symptomatology), it was necessary to include studies that examined negative mood/affect and clinically-diagnosed (or diagnosable) depressive disorders, as well as some that examined depressive symptoms in conjunction with other symptoms of psychological distress. Although the majority of prior research has focused on pain intensity or severity, a few studies have defined and/or measured pain more broadly. Since most of these studies utilized composite measures that made at least tacit reference to the intensity or magnitude of the respondent’s pain, these studies were also included.

A total of 14 studies were retained for review. For convenience, these studies are referred to collectively—here, and throughout this document—as the prior “mediation studies.” Given the diverse collection of studies included in this review, careful attention is paid to the conceptualization and measurement of central constructs of interest, sample composition, and other aspects of study methodology (e.g., data analytic techniques). An overall summary (offered at the end of the review) considers the possible effects of such diversity on study findings.

*Gerontological and Chronic Pain Research: Separate, but Parallel Literatures*

Prior to reviewing this body of literature, one preliminary observation deserves attention: Historically, the bulk of research on pain has been produced by researchers studying chronic pain; yet, studies of chronic pain patients are notably under-represented within the literature that considers activity limitation as a mediating variable in the pain-depression relationship. Of the 14 studies reviewed here, only 5 have been conducted with chronic pain patients. There are three likely reasons for this.

First, studies in the chronic pain literature very often assume, a priori, that activity
limitation (broadly defined to include all manner of physical impairments and
disabilities) mediates the relationship between pain and psychological distress. This
assumption is so pervasive that many studies have not even tested the assumption
empirically, even if they had the ability to do so. For example, in a sample of over 100
male chronic pain patients, Goldberg, Kerns, & Rosenberg (1993) found that both pain
severity ratings and pain-specific activity limitation (measured as “pain interference”)
ratings were significant, independent predictors of depressive symptomatology—
explaining roughly 24% and 8% of the variance, respectively. However, these authors
did not include any intermediate models that would have explicitly tested the mediation
hypothesis. Although not a factor in the Goldberg et al. study, the ability to test
mediation hypotheses in chronic pain studies has likely been compromised by the
widespread use of extensive pain inventories. Many of these inventories include items
measuring several dimensions of pain and many different types of activity limitation.
Despite ample psychometric evidence that these inventories contain distinct pain
constructs and dimensions, too often, researchers simply sum or average the scores across
to create an overall measure of “pain” for use in multivariate analyses. This practice
seems to have hampered knowledge development regarding important mediation
pathways within the chronic pain literature.

Second, as is typical of many fields of study, the chronic pain literature seems to
have undergone distinct evolutions marked by periods of intense focus on specific topics.
For example, much of the early literature on chronic pain (e.g., up to the late 1980s)
tended toward descriptive studies aimed at creating profiles for use in treatment. An
individual with long-standing pain, who reported significant impairment in physical (and
other) functioning and a lot of depressive symptoms, was often labeled as having “chronic pain syndrome” (e.g., Pope, 1987). Hence, the three central constructs of interest in the present study were often treated as markers of a special group of pain patients. More recent areas of emphasis in chronic pain research include cognitions and coping (e.g., Flor, Behle, & Birbaumer, 1993; Jensen et al., 2003), neurobiological aspects of pain (e.g., Price & Staud, 2005), and factors influencing the transition from acute to chronic pain (e.g., Fransen et al., 2002; NIH, 2009).

Third and finally, there appear to be substantial disciplinary and institutional boundaries within the body of research addressing activity limitation as a mediator of the pain-depression relationship. In general, there is a lack of overlap between researchers who study aging and those who study pain. Although psychologists play a prominent role in both areas of research, the areas tend to be populated by different disciplinary subspecialists—psychologists in pain (and chronic pain) research tend to be clinical, medical, or experimental psychologists, while gerontological research has historically been dominated by social and developmental psychologists, and only within the past few decades a more substantial cadre of clinical psychologists. As data suggest that few patients over age 65 attend multidisciplinary pain clinics (e.g., Roy, 2000), there may be little perceived need for chronic pain researchers to consult the gerontology literature. It should be noted that the reverse also appears to be true—in studies of pain among older adults, relatively little attention has been paid to the chronic pain literature.

These factors appear to have resulted in two seemingly independent avenues of research related to activity limitation as a mediator of the relationship between pain and depressive symptomatology—one within the field of gerontology, and one within the
field of chronic pain. This lack of sharing and cross-germination of ideas between chronic pain and gerontological researchers will be considered again after reviewing the existing literature with respect to mediation in the pain-depression relationship.

**Organization and Presentation of Mediation Studies**

Table 1 provides an overview of the 14 studies included in this review. The table contains a description of the sample employed in each study, including information about age and gender composition (in as much detail as was provided in the original publication). The table also provides information about how the three key constructs were measured in each study. Studies are organized in the table (and in the subsequent review) in terms of their primary findings with respect to mediation:

1. Studies that showed evidence of *partial mediation*—i.e., the mediating construct explained part of the relationship between pain and depressive symptomatology (*n* = 5, Panel A);
2. Studies that demonstrated *total mediation*—i.e., the mediating construct completely (or nearly completely) explained the relationship between pain and depressive symptomatology (*n* = 4, Panel B); and
3. Studies that found *no mediation*—i.e., the mediating construct explained none (or very little) of the relationship between pain and depressive symptomatology (*n* = 5, Panel C).

Within each group (i.e., each panel in the table), studies are listed in alphabetical order, according to the first author’s last name.

This review begins with some general observations about this body of literature. Those observations are followed by a more detailed review of the three groups of studies.
Table 1

Prior Empirical Studies that Tested Mediation of the Relationship Between Pain and Psychological Distress by Activity Limitation (N = 14)

<table>
<thead>
<tr>
<th>Study</th>
<th>Sample Description</th>
<th>Psychological Distress Construct(s) &amp; Measure(s)</th>
<th>Pain Construct(s) &amp; Measure(s)</th>
<th>Activity Limitation Mediating Construct(s) &amp; Measure(s)</th>
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</thead>
<tbody>
<tr>
<td><strong>Panel A: Partial Mediation (n = 5)</strong></td>
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<tr>
<td>Arnstein et al. (1999)</td>
<td>126 chronic pain patients</td>
<td>Depressive symptomatology (CES-D)</td>
<td>Pain intensity (visual analog scale)</td>
<td>Pain-related disability (Pain Disability Index—interference with self-care, work, social, recreational, and other activities)</td>
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<tr>
<td></td>
<td>$M_{\text{age}} = 44$ ($SD = 12.5$)</td>
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<td></td>
<td>66% female</td>
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<tr>
<td>Goodland (2002, unpublished dissertation)</td>
<td>123 injured workers with chronic pain</td>
<td>Depressive symptomatology (BDI-II)</td>
<td>Pain severity (WHYMPI Pain Severity scale)</td>
<td>6 areas of disability (6 scales—ADL, Social, Recreational, Family, Physical concerns, &amp; Vocational)</td>
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<tr>
<td></td>
<td>$M_{\text{age}} = 48$ ($SD = 8.4$), $Range = 28 – 64$</td>
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<tr>
<td></td>
<td>29% female</td>
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<tr>
<td>Gureje, Simon, &amp; Von Korff (2001)</td>
<td>3,000 randomly selected primary care patients at 15 clinics in 14 countries</td>
<td>Presence of either depressive or anxiety disorder (ICD-10 diagnostic interview)</td>
<td>Persistent pain (pain for 6 months or more AND that required medication or doctor visit or interfered significantly with life)</td>
<td>Occupational role disability (items from Social Disability Schedule—impairment in role functioning, e.g., in work, usual activities)</td>
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<tr>
<td></td>
<td>$M_{\text{age}} \sim 42$ ($SD = 12.0$), $Range = 18 – 65$</td>
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<tr>
<td></td>
<td>65-70% female</td>
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<tr>
<td>Study</td>
<td>Sample Description</td>
<td>Psychological Distress Construct(s) &amp; Measure(s)</td>
<td>Pain Construct(s) &amp; Measure(s)</td>
<td>Activity Limitation Mediating Construct(s) &amp; Measure(s)</td>
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<tr>
<td>Panel A: Partial Mediation (cont.)</td>
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<tr>
<td>Williamson &amp; Schulz (1992a)</td>
<td>228 geriatric outpatients $M_{age} = 72$, all age 55 or over 69% female</td>
<td>Depressive symptomatology (CES-D)</td>
<td>Pain (composite index—general pain, pain in last week, general discomfort)</td>
<td>Activity restriction (Activity Restriction Scale—limitation/restriction in 9 areas, e.g., ADLs, IADLs, work, maintaining friends, hobbies, etc.)</td>
</tr>
<tr>
<td>Williamson &amp; Schulz (1995)</td>
<td>268 midlife &amp; older cancer outpatients $M_{age} = 65$, Range = 30 – 90, 49% age 65 or over 51% female</td>
<td>Depressive symptomatology (CES-D)</td>
<td>Pain (4-item composite—presence of pain in past month, severity in past month, current intensity, constancy)</td>
<td>Functional disability (adapted Activity Restriction Scale—limitation/restriction in 9 areas, e.g., ADLs, IADLs, work, maintaining friends, hobbies, etc.)</td>
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<tr>
<td>Panel B: Total Mediation (n = 4)</td>
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<tr>
<td>Cano et al. (2004)</td>
<td>110 married chronic pain patients (musculoskeletal back or neck pain only) $M_{age} = 46$ ($SD = 10.5$), $Range = 30 – 90$ 56% female</td>
<td>Depressive symptomatology (MASQ—General &amp; Anhedonic Depressive Symptom subcales) Depression (SCID for current depressive disorders)</td>
<td>Pain severity/intensity (WHYMPI Pain Severity scale)</td>
<td>Physical disability (SIP Physical Disability &amp; Functional Impairment subscale—impairment in ambulation, mobility, body care)</td>
</tr>
<tr>
<td>Study</td>
<td>Sample Description</td>
<td>Psychological Distress Construct(s) &amp; Measure(s)</td>
<td>Pain Construct(s) &amp; Measure(s)</td>
<td>Activity Limitation Mediating Construct(s) &amp; Measure(s)</td>
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<td><strong>Panel B: Total Mediation (cont.)</strong></td>
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</tbody>
</table>
| Rudy, Kerns, & Turk (1988)  | 100 chronic pain patients at VA pain clinic  
\(M_{age} = 51 \ (SD = 14.5)\)  
22% female | Depressive symptomatology (BDI & Depression Adjective Checklist)                                          | Pain intensity/severity (MPQ Pain Rating Index; WHYMPI Pain Severity scale; Average hourly pain intensity over 2 weeks) | Pain interference with life activities (WHYMPI Social/Recreational, Family/Domestic, & Work scales)  
Personal control (WHYMPI Life Control & Problem-Solving scales; Health Locus of Control scale) |
| Turk, Okifuji, & Scharff (1995) | 100 chronic pain patients referred to multidisciplinary pain center  
\(M_{age} = 57 \ (SD = 12.0)\)  
57% female | Depressive symptomatology (CES-D)                                                          | Pain severity (WHYMPI Pain Severity scale)                                                             | Pain interference with life activities (WHYMPI Social/Recreational, Family/Domestic, & Work scales)  
Perceived life control (WHYMPI Life Control scale) |
| Williamson (2000a)         | 95 female breast cancer out-patients, stage 1, 2, or 3  
\(M_{age} = 49 \ (SD = 9.3), \ Range = 26 – 75\)  
100% female                  | Depressive symptomatology (CES-D)                                                      | Pain severity (SF-36 body pain severity item)                                                          | Activity restriction (composite of modified SF-36 items—ADL, IADL, work, social, & physical functioning restriction/limitation) |
### Study Sample Description

<table>
<thead>
<tr>
<th>Study</th>
<th>Sample Description</th>
<th>Psychological Distress Construct(s) &amp; Measure(s)</th>
<th>Pain Construct(s) &amp; Measure(s)</th>
<th>Activity Limitation Mediating Construct(s) &amp; Measure(s)</th>
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<tbody>
<tr>
<td><strong>Panel C: No Mediation (<strong>n</strong> = 5)</strong></td>
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<tr>
<td>Bookwala, Harralson, &amp; Parmelee (2003)</td>
<td>367 older adults with osteoarthritis from rheumatology clinics, primary care practices, and the community $M_{age} = 68$ ($SD = 9.7$), all age 50 or over 64% female</td>
<td>Depressive symptomatology (CES-D)</td>
<td>Pain intensity (composite scale with items from AIMS-2 pain scale and PGC pain scale)</td>
<td>[Limitations in] Physical functioning (AIMS-2 functional subscales—limitations in mobility, self-care, household tasks, hand/arm/finger function) Social functioning (social &amp; leisure involvement)</td>
</tr>
<tr>
<td>Cohen- Mansfield &amp; Marx (1993)</td>
<td>408 residents in LTC facility $M_{age} = 85$, Range = 70 – 99% female = unk.</td>
<td>Depressed Affect (Depression Rating Scale, completed by staff nurse caregivers)</td>
<td>Pain intensity (verbal descriptor scale, rated by staff nurse caregivers)</td>
<td>ADL impairment (Rapid Disability Rating Scale-2, completed by staff nurse caregivers)</td>
</tr>
<tr>
<td>Geerlings et al. (2002)</td>
<td>652 community-dwelling older persons in The Netherlands $Range_{age} = 55 – 85$, 55% age 70 or over 58% female</td>
<td>Depressive symptomatology (CES-D) Depression ($\geq 16$ on CES-D)</td>
<td>Pain symptomatology (Nottingham Health Profile Pain subscale—pain levels &amp; symptoms across various activities &amp; positions, rated none, some/moderate, or many/high)</td>
<td>Physical disability (difficulty with each of 3 tasks—climbing stairs, cutting toenails, using public transportation)</td>
</tr>
<tr>
<td>Study</td>
<td>Sample Description</td>
<td>Psychological Distress Construct(s) &amp; Measure(s)</td>
<td>Pain Construct(s) &amp; Measure(s)</td>
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<tr>
<td>Panel C: No Mediation (cont.)</td>
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<tr>
<td>Kahana et al. (1997)</td>
<td>804 older adults in good health randomly selected from 3 retirement communities in FL (used data from Wave 2 only) (M_{age} = 80, \text{Range} = 71 – 98) 66% female</td>
<td>Depressive symptomatology (10-item CES-D) Negative affect (PANAS)</td>
<td>Pain (composite of intensity and frequency of pain during the past year)</td>
<td>Personal disability (OARS ADL &amp; IADL subscales) Social disability (lack of involvement in social activities, leisure, and hobbies)</td>
</tr>
<tr>
<td>Parmelee, Katz, &amp; Lawton (1991)</td>
<td>598 congregate apartment &amp; nursing home residents at Jewish CCRC (M_{age} = 84 (SD = 5.9), \text{Range} = 61 – 99) 70% female</td>
<td>Level of depression (DSM-IIIR symptom checklist—possible major, minor, or no depression) Depressive symptomatology (Geriatric Depression Scale)</td>
<td>Pain intensity (averaged across 6 items from MPQ)</td>
<td>Functional disability (ADL impairments, as rated by staff or via self-report) Physician-rated system-level disease impairments (CIRS)</td>
</tr>
</tbody>
</table>

Note. ADL = Activities of Daily Living; AIMS = Arthritis Impact Measurement Scales; CCRC = Continuing Care Retirement Center; BDI-II = Beck Depression Inventory, 2nd edition; CES-D = Center for Epidemiologic Studies-Depression Scale (refers to original, 20-item scale, unless otherwise specified); CIRS = Cumulative Illness Rating Scale; DSM-IIIR = Diagnostic and Statistical Manual of Mental Disorders, 3rd edition-revised; IADL = Instrumental Activities of Daily Living; ICD-10 = International Classification of Diseases, 10th revision; LTC = Long-term care; MASQ = Mood and Anxiety Symptom Questionnaire; MPQ = McGill Pain Questionnaire; OARS = Older Americans Resources and Services multidimensional functional assessment questionnaire; PANAS = Positive and Negative Affect Schedule; PGC = Philadelphia Geriatric Center; SCID = Structured Clinical Interview for the DSM-IV (4th edition); SF-36 = Short form (36-item) Health Survey; SIP = Sickness Impact Profile; WHYMPI = West Haven-Yale Multidimensional Pain Inventory; unk. = Unknown.

*Although not conceptualized as activity limitation, these constructs (personal/life or health control) were considered potential mediators and were included by the authors in tests of mediation.*
and their major findings with respect to the proposed mediation pathway. A final subsection offers an overall summary and discussion of the limitations and gaps within the existing literature.

**General Observations**

As shown in Table 1, studies of mediation have used widely varying samples. Some have sampled community-dwelling persons (e.g., Kahana et al., 1997), others have recruited patients from primary care clinics (e.g., Gureje, Simon, & Von Korff, 2001), others have examined patients with specific diseases like osteoarthritis (Bookwala, Harralson, & Parmelee, 2003) and cancer (e.g., Williamson, 2000a), and others have sampled patients from chronic pain clinics (e.g., Rudy, Kerns, & Turk, 1988) or nursing homes (e.g., Cohen-Mansfield & Marx, 1993). Across the 14 studies, participants have also represented a wide age range, including young adulthood, midlife, the young-old, the old-old, and the oldest old. The samples have also varied considerably in their gender composition. One study had a sample comprised of women only (Williamson’s 2000 study of breast cancer patients); Cohen-Mansfield and Marx presented no gender information about their nursing home sample, but it was likely comprised mostly of women. Across the remaining studies, the percentage of female participants ranged from 22% (Rudy, Kerns, & Turk, 1988) to 70% (Parmelee et al., 1991). Only the study by Cano and colleagues had a sample comprised solely of married persons (Cano, Gillis, Heinz, Geisser, & Foran, 2004). Among the remaining studies for which such information was provided, the percentage of respondents who were married or partnered ranged from 33% (Williamson & Schulz, 1992a) to 81% (Goodland, 2002).

Although most of the studies used self-report measures of all central constructs,
there are a few notable exceptions. In their study of nursing home patients, Cohen-
Mansfield and Marx (1993) had to (understandably) rely solely on staff caregiver reports.
A few studies used clinical evaluation data to measure one or more key constructs (e.g.,
Gureje et al., 2001, used a diagnostic interview to evaluate psychological distress) or to
supplement self-report measures of key constructs (e.g., Cano et al., 2004, also assessed
depressive disorders via structured clinical interview; Parmelee et al., 1991, used doctor-
rated illness impairments in addition to self- or staff-reported functional disability).

Although not shown in Table 1, some studies also examined other outcomes (in
addition to psychological distress). For example, Bookwala et al. (2003) included self-
rated health as an outcome variable, and Kahana et al. (1997) assessed several
dimensions of psychological well-being (e.g., life satisfaction, positive affect). This
review focuses on only those findings related psychological distress. Although some
variation exists in terms of conceptualizations of psychological distress, most prior
studies focused on depression, depressive symptomatology, or negative mood. Most
likely, this is because most existing scholarly work on the psychological consequences of
pain has focused on mood disorders and negative mood states, especially depression
and/or depressive symptomatology (Parmelee, 1997). In studying depression or
depressive symptoms, most studies have relied on commonly-used measures, such as the
CES-D, the Beck Depression Inventory, or a DSM- or ICD-based diagnostic interview.

The measurement of pain has also varied across studies. Most studies examined
pain intensity or severity, although assessment tools have varied considerably. Several
studies used a single-item to assess pain intensity (or severity). For example, Cohen-
Mansfield and Marx (1993) had nurse caregivers rate patients’ pain intensity on a simple
numerical rating scale, and Arnstein, Caudill, Mandle, Norris, & Beasley (1999) had chronic pain patients rate their current pain intensity using a visual analog rating scale. Several studies created a composite pain intensity score using multiple items. Some authors used pain intensity or severity items or scales from well-established health questionnaires such as the SF-36 (e.g., Williamson & Schulz, 1995) or the Nottingham Health Profile (Geerlings, Twisk, Beekman, Deeg, & Van Tilburg, 2002). Some authors used items or scales originally designed for use with chronic pain patient populations. For example, several studies used the WHYMPI pain severity scale (Cano et al., 2004; Goodland, 2002; Turk et al., 1995). Still other authors created composite indices of pain derived from various sources, including items developed for use with specific patient populations (e.g., Bookwala et al., 2003, combined pain items from an arthritis-specific measure with general pain items from another scale), or items adapted from those used in population studies of health (e.g., Williamson & Schulz, 1992a). It is notable that some multi-item scales and derived indices included items that measure other dimensions of pain along with pain intensity or severity, including pain frequency (e.g., Kahana et al., 1997) or the number of pain symptoms or sites (e.g., Geerlings et al., 2002).

Previous studies have varied most markedly in terms of the conceptualization and measurement of the mediating construct, activity limitation. Although most included at least some type of physical and/or functional difficulty or impairment, some conceptualized the construct much more broadly. Williamson and Schulz (1992) first defined the construct of “activity restriction” (operationalized as a mix of ADLs, IADLs, hobbies, and social activities), and proposed that this “activity restriction” mediates the relationship between pain and depressive symptoms. Other authors (including
Williamson herself, on occasion) have labeled the mediating construct “functional
disability/disabilities” or “functional limitation(s);” in such studies, the construct has
been operationalized mainly as ADL and/or IADL impairments (e.g., Cohen-Mansfield &
Marx, 1993; Williamson & Schulz, 1995). The range of conceptual and operational
definitions has also included occupational disability (e.g. Gureje, Simon, & Von Korff,
2001), body system-level impairment (e.g., Parmelee et al., 1991), and various physical
disabilities and difficulties related to mobility and personal care (e.g., Cano et al., 2004;
Geerlings et al., 2002). Some authors have even proposed multiple, parallel mediating
constructs. For example, Goodland (2002) identified and tested six different dimensions
of “disability” including ADL, recreational, physical symptoms, social, family, and
vocational disability that could potentially mediate the pain-depression relationship.
Arnstein et al. (1999) also measured different areas of disability (including occupational,
self-care, social activities, recreation, etc.), but they summed them into an overall score.
Both Kahana et al. (1997) and Bookwala et al. (2003) distinguished two categories of
mediators—those related to physical functioning difficulties, and those related to social
functioning difficulties.

An additional source of variation across studies is whether or not mediating constructs were measured specific to the context of pain (or even illness) or more
globally. In general, studies of pain patients or those with specific illnesses tend to use
measures that specifically link the activity limitation to the pain or illness (e.g., “To what
extent has your pain interfered with your functioning in…self-care activities?” in
Arnstein et al., 1999). In contrast, in studies utilizing non-clinical, community-based
samples, the wording tends to be more general (e.g., “How much difficulty do you have
climbing stairs?” in Geerlings et al., 2002). The choice of context- (i.e., pain-) specific versus general measures of activity limitation, of course, depends strongly on a priori knowledge or assumptions about the prevalence of pain (or a pain-producing illness) within the target population. In a chronic pain sample or a sample of arthritis patients, for example, one can expect that most respondents will have pain and will be able to respond to a query regarding the extent to which pain interferes with their usual activities. In community-based samples, more general questions about physical limitations may be more widely applicable to all respondents, including those with and without pain.

In the more detailed review below, attention is focused on study findings with respect to mediation of the pain-depressive symptomatology relationship. Unless stated otherwise, it can be assumed that these studies met all other “preconditions” for establishing mediation—i.e., that pain was related to depressive symptomatology (Path A) and activity limitation (Path B), that activity limitation (Path C) were related to depression. Findings that extend beyond the mediation pathway outlined in the proposed conceptual model—especially those resulting from any post-hoc analyses (e.g., testing whether mediation was affected by participant characteristics)—are discussed only as they relate to the limitations and gaps within the existing literature.

**Studies Showing Partial Mediation (n = 5)**

Five of the 14 studies in Table 1 found evidence of partial mediation. Since the study by Williamson and Schulz (1992a) is widely regarded as the seminal study (within gerontology), it is considered first. These authors proposed and tested one mediation pathway—“activity restriction” (i.e., the extent to which pain [or illness] restricts or limits routine daily activities or causes functional impairment or disability). In a sample
of geriatric outpatients, pain was moderately and positively related to both activity restriction and depressive symptoms. Activity restriction was moderately and positively correlated with depressive symptoms. In additional bivariate analyses, activity restriction was also significantly higher among those classified as “at risk” for depression (scoring ≥ 16 on the CES-D) than among the non-depressed. In a path analysis predicting continuous CES-D scores, activity restriction was found to partially mediate the relationship between pain and depressive symptomatology. That is, once activity restriction was added to the model, the effect of pain on depressive symptoms was significantly reduced (but not totally eliminated).

Evidence for partial mediation was also found both cross-sectionally and longitudinally by Williamson in a later sample of cancer outpatients (Williamson & Schulz, 1995). In predicting depressive symptomatology at baseline, these authors found that, when activity restriction was added to a model that included pain, activity restriction was significant and the effect of pain was reduced, but remained statistically significant. At eight months, increased pain predicted greater activity restriction, which in turn predicted increased depressive symptomatology. Williamson and her colleagues have used these studies to articulate and extend her proposed Activity Restriction Model of Depressed Affect (ARM; for example, see model outlined in Williamson, 2000a), reviewed earlier in this chapter.

The study by Arnstein et al. (1999) found evidence of partial mediation involving what they called “pain-related disability” (operationalized as interference from pain across several life domains) in a sample of chronic pain patients. Pain intensity had a strong, direct effect on depressive symptomatology, and pain-related disability was
related to both pain intensity and depressive symptoms. After adding pain-related
disability to a model predicting depressive symptomatology, the relationship between
pain intensity and depressive symptoms was weakened, but was still statistically
significant.

In another chronic pain sample, Goodland (2002) tested several mediating
constructs simultaneously. He found that “ADL disability” (operationalized broadly to
include limitations in ambulation, mobility, and body care) and “social disability”
(reduction in and problems with social functioning) both functioned as mediators in the
relationship between pain severity and depressive symptomatology. However, a
comparison of alternative models suggested that social disability fit equally well as either
a mediator of the pain-depressive symptom relationship or as an outcome of depressive
symptomatology. Other types of “disability,” including recreational, vocational, family
disruption, and physical health concerns, did not appear to play any significant role in
mediating the observed relationship between pain severity and depressive symptoms.
Because the different mediators were tested simultaneously, and because of the
conceptual confusion regarding social disability as mediator and/or outcome, this study is
regarded as providing evidence of at least partial mediation by activity limitation.

Finally, Gureje et al. (2001) examined the relationship between persistent pain
and psychological distress (defined as a diagnosis of either a depressive disorder or an
anxiety disorder) in a longitudinal study of adults in primary care practices around the
world. After adjusting for relevant covariates, the presence of persistent pain at baseline
was a significant and relatively strong predictor of the onset of a diagnosable depressive
or anxiety disorder. There was also evidence that “occupational role disability”
(measured as limitations or problems in work or usual activities) partially mediated this relationship: When added to the model, baseline occupational role disability was a strong predictor of psychological disorder onset, and the effect of baseline persistent pain, although still statistically significant, was considerably reduced. These authors concluded that disability, or as they further characterized it, "withdrawal from rewarding daily activities" (p. 199), served as the critical link between persistent pain and psychological disorders. Unfortunately, data were not presented in a way that permitted separation of results with respect to depressive and anxiety disorders. Although Gureje et al. (2001) studied pain presence or onset (vs. pain intensity), they required that the pain meet a strict definition of “persistent pain” which made reference to duration and—albeit somewhat indirectly—to severity (i.e., pain required medication or a doctor visit).

**Studies Showing Total Mediation (n = 4)**

Four studies in Table 1 provide evidence that activity limitation totally mediate the pain-depressive symptomatology relationship. Because the study by Rudy, Kerns, and Turk (1988) can be regarded as the hallmark study within the chronic pain literature, it is reviewed first. The authors examined pain interference with life activities and perceptions of personal control as mediators of the relationship between pain intensity and depressive symptomatology in sample of chronic pain patients. The results provided evidence consistent with total mediation. Despite a significant bivariate relationship between pain intensity and depressive symptoms, once the two mediators (both of which were significantly related to pain intensity and to depressive symptomatology) were entered into the model, this relationship was reduced to nonsignificance. Instead, pain interference and perceptions of control accounted for the bulk of the explained variance.
in depressive symptomatology. The authors also presented evidence that both pain interference and perceptions of control independently mediated the relationship between pain intensity and depressive symptom relationship.

The study by Cano et al. (2004) is the only known mediation study conducted with a sample comprised exclusively of married persons. Although the purpose of the study was to examine the contributions of marital satisfaction and interactions to the depressive symptomatology of patients with chronic musculoskeletal pain, study findings also provided support for the mediating role of physical disability (measured as limitations and impairments in ambulation, movement, and body care). In this largely middle-aged sample, greater pain severity was moderately associated with higher levels of depressive symptomatology and strongly associated with greater physical disability. Physical disability was also moderately associated with depressive symptomatology. When both pain severity and physical disability were entered jointly in multivariate analyses, however, only physical disability emerged as a significant predictor of depressive symptomatology. The same pattern of results was observed when Cano and colleagues repeated the analysis substituting a diagnosable depressive disorder as the outcome variable. These findings provided evidence consistent with total mediation.

A later study of chronic pain patients by Turk and colleagues (Turk, Okifuji, and Scharff, 1995) also found evidence of total mediation; however, the evidence was more consistent with the MacKinnon et al. (2002) approach to mediation than that of Baron and Kenny (1986). The initial (i.e., bivariate) relationship between pain severity and depressive symptoms was positive but weak and not statistically significant ($r = .17$; this criterion is relaxed under the MacKinnon approach). Using path analysis, Turk et al.
demonstrated that pain severity significantly influenced pain interference and perceived life control, which in turn affected depressive symptom scores. The observed relationship between pain severity and depressive symptomatology in the final path model was reduced \( (r = .07) \). Although pain interference and perceived life control were analyzed jointly as mediators, the authors did report that each was significantly correlated with both pain severity and depressive symptomatology. Thus (based on the MacKinnon et al. criteria), this study can be regarded as yielding evidence of total mediation.

Williamson’s (2000a) study of women with breast cancer yielded additional empirical support for her proposed activity restriction model of depressed affect. In her sample, activity restriction was found to completely mediate the relationship between pain severity and depressive symptomatology. It is notable that Williams utilized a different measure of “activity restriction” in this study.

*Studies Showing No Mediation (n = 5)*

The five studies in the last section of Table 1 found no evidence that activity limitation mediates the pain-depressive symptomatology relationship. In different samples of older adults in residential care settings, both Parmelee et al. (1991) and Cohen-Mansfield and Marx (1993) and found that pain intensity remained independently associated with depressed affect or depressive symptoms, even after controlling for measures of functional disability and health status. The study by Parmelee et al. (1991), regarded as the other *hallmark* study in gerontology, found that functional disability (self- or staff-reported ADL impairment) did not account for the relationship between pain intensity and depressive symptoms. Pain intensity was moderately positively related to scores on the Geriatric Depression Scale and to depressive symptoms levels derived from
a diagnostic checklist (none, mild/minor, possible major depression). Pain intensity was also positively related to functional disability and to physician ratings of illness (general impairment of system functioning). However, the relationship between pain and depressive symptomatology (and depression category) remained significant and of generally similar strength, even after controlling for functional disability. In the Cohen-Mansfield and Marx (1993) study, pain ratings were not even significantly related to ratings of ADL impairment (notably, both measures relied on staff reports).

Kahana and colleagues (1997) examined their proposed pain-disability cascade model (see Figure 3 earlier in this chapter) in a sample of community-dwelling older adults of advanced age. They found no evidence that either social disability or personal disability mediated the relationship between pain (a composite frequency/intensity measure) and depressive symptomatology or negative affect. Pain was weakly related to personal disability and moderately related to social disability. Pain maintained a moderate direct effect on negative affect and a weak direct effect on depressive symptomatology, even after controlling for both types of disability and relevant background variables. In fact, pain was the strongest predictor of both depressive symptoms and negative affect in their model.

The study by Bookwala, Harralson, and Parmelee (2003) was mentioned earlier because of their efforts to extend the Kahana et al. (1997) model and integrate it with Williamson’s ARM. Bookwala et al. tested their proposed model in a sample of community-dwelling “older adults” with osteoarthritis of the knee (technically, participants were in late-middle-age or older). There was no evidence that either physical or social functioning mediated the relationship between pain intensity and depressive
symptomatology. Although limitations in physical functioning were positively related to both pain intensity and depressive symptoms at a bivariate level, they were not significantly related to depressive symptoms in a multivariate model that also included relevant control variables, social functioning, and their other outcome (perceived global health). There was no evidence that social functioning mediated the pain-depression relationship, either. Rather, pain intensity maintained a significant direct relationship with depressive symptoms, even after controlling for physical and social functioning.

Finally, a longitudinal study of older adults in the Netherlands by Geerlings et al. (2002) found a strong relationship between pain symptoms/intensity assessed at a prior wave and both depressive symptomatology and “probable depression” (scoring above a widely-used clinical cutoff on the CES-D) assessed at later waves. Furthermore, these pain-depression relationships held, even after relevant demographic variables were controlled. However, neither physical disability at a prior wave, nor disability at the current wave, was found to mediate the pain-depression relationship. Physical disability (at either wave) was strongly related to depressive symptomatology and to probable depression, but, when added to models with pain, it did not significantly reduce the pain-depression relationship. The authors used data from a study designed to compare depressed and non-depressed individuals; as such, the study sample was stratified by baseline depressive symptomatology scores, with 50% of the sample scoring above the cutoff for probable depression. Concerned that the sample stratification might have somehow influenced the study’s results, the authors replicated their test of mediation within the non-depressed half of the sample; but, again, they found no evidence that physical disability mediated the pain-depressive symptomatology relationship.
Summary and Consideration of Limitations and Gaps

As a whole, this body of research appears to offer substantial empirical evidence that activity limitation at least partially mediates the relationship between pain intensity and depressive symptomatology. A majority (64%) of prior studies found evidence of at least partial mediation. However, support for partial mediation is subject to three important observations and qualifications:

1. An equal number of studies yielded no support for mediation ($n = 5$) as those that found only partial mediation ($n = 5$). This suggests that existing evidence is, on balance, more equivocal and that study results have been somewhat inconsistent.

2. Support for mediation may be limited to specific populations. Some of the larger, community-based studies of older persons (e.g., Bookwala et al., 2003; Geerlings et al., 2002; Kahana et al., 1997) have not found support for mediation, neither have studies of those living in residential care settings.

3. Partial mediation may exist only to the extent that activity limitation is:
   (a) defined to include a broad range of physical impairments, IADL (and perhaps ADL) disabilities, and difficulties in other areas of functioning, and/or (b) either defined to be pain-specific or measured in populations assumed to have high levels of pain.

Several potential limitations and gaps exist within the existing literature. These gaps and limitations may be an important source of some of the inconsistency observed across studies, and may help to illuminate some of the observations and qualifications offered above. Discussion of these limitations and gaps is organized around issues
related to study design and execution, those related to construct definition and
measurement, those related to additional explanatory variables, and other issues.

**Issues related to study design and execution.** Heterogeneity in study results
with respect to mediation could be due to differences in samples and/or target
populations. The diversity of samples across studies makes comparison of results
difficult. However, two patterns are detected. First, both studies conducted with cancer
patients (Williamson & Schulz, 1995; Williamson, 2000a) found evidence of at least
partial mediation. Second, all four of the studies conducted with samples of chronic pain
patients also found evidence of at least partial mediation. These patterns could be due to
demographic differences between these samples and those in other studies, differences in
the types of (and/or ranges of) pain experienced by these samples, variance in the pain-
depressive symptom relationship within these samples, or a variety of unknown factors.

Although study samples were diverse in terms of the different target populations
they intended to represent (chronic pain clinic patients, primary care outpatients, nursing
home residents, etc.), they were not very representative in terms of the demographic and
background characteristics of the population at large. Most studies had limited diversity
with respect to participants’ racial or ethnic background, socio-economic status, and
gender. Only Williamson and Schulz (1992a), Bookwala et al. (2003), and Gureje et al.
(2001, an international study) had significant numbers of non-White participants. Many
studies reported relatively high levels of education and income among their participants.

The inclusion or exclusion of individuals in the study with a diagnosable
psychiatric disorder has been offered by some as a factor that may have contributed to the
inconsistency in results related to mediation (e.g., Williamson & Schulz, 1992a). Some
studies specifically excluded persons with psychiatric disorders, including depressive syndromes, from their samples (e.g., Arnstein et al., 1999; Williamson & Schulz. 1992a). Meanwhile, others used a sample stratified on the basis of a suggested depressive disorder (Geerlings et al., 2002), or created subgroups based on levels of depressive symptomatology in order to test specific hypotheses (Parmelee et al., 1991). In fact, both of these latter studies specifically tested the mediation hypothesis in the sample as a whole and within the different depression-based subgroups; neither found any marked difference in the results (both found no mediation). However, both did observe an interesting pattern in the pain-depression relationship among subjects with “probable” (Geerlings et al.) or “possible” depression (Parmelee et al.): In these groups, the strength of the observed relationship between pain and depressive symptoms tended to be smaller or weaker (in Parmelee, this relationship even failed to reach statistical significance) than in the rest of the sample. Whether this pattern reflects something unique about the pain-depressive symptom relationship within persons with psychiatric disorders (especially those with a suggested depressive disorder), or whether it can be attributed to a possible restriction of range in depressive symptomatology levels within these groups, is unclear.

Some of the observed inconsistency in findings could be attributable to differences in the types or the nature of pain experienced by the different samples. Variation in the (presumed) underlying cause of or type of pain was limited in some studies by design—for example, to cancer patients (Williamson & Schulz, 1995) or to arthritis patients (Bookwala et al., 2003). Yet, most studies have had samples with a presumably broad mix of underlying disease conditions and different types of pain. Two very diverse samples—for example, Gureje et al.’s (2001) primary care patient sample
and Geerlings et al.’s (2002) older community dwelling adults in the Netherlands—yielded very different findings with respect to mediation. As was mentioned, both cancer patient studies and all four studies of chronic pain patients found evidence of at least partial mediation, including the one study limited to patients with chronic musculoskeletal pain (Cano et al., 2004). Yet, persons with musculoskeletal pain (presumably due mainly to arthritis) comprised over 50% of the Kahana et al. (1997) and 100% of the Bookwala et al. (2003) samples and both of these studies failed to find evidence of mediation. Whether these findings reflect differences in the types, duration, frequency, or the intensity of pain experienced by these samples is unclear. In addition, because some types of pain are associated with background characteristics such as gender and age, attributions regarding the potential source of such variation must be made with caution. Other patterns related to the different types of pain and/or underlying etiology within the samples are hard to detect, in part because detection would require that the studies had provided adequate descriptions of the sample in terms of members’ existing disease conditions, health-related characteristics, and types of pain—something not provided in all publications.

Inconsistency in study findings regarding mediation could be linked to samples with restricted range(s) on one or more of the central measures (pain, depression, activity limitation). This possibility is generally difficult to evaluate due to a lack of comparable measures across studies. Additionally, many of the studies neglected to present descriptive data on the level and observed range of scores on key measures.

Restriction in pain intensity ratings would most likely have the effect of reducing observed relationships between pain intensity and other variables in the study. Only two
studies reported relatively weak bivariate relationships involving pain (Cohen-Mansfield & Marx, 1993, and Turk et al., 1995). However, in both cases, the weak relationships involved only one other variable; in addition, restriction of range in pain intensity seems an unlikely explanation for the differences between the outcomes of these two particular studies. Heterogeneity of study results also does not seem to be the result of any restriction of range with respect to the outcome variable—viz., depressive symptomatology or depression. However, because of the diversity of the samples (e.g., clinical samples vs. community-based samples, geriatric samples vs. general adult samples), it is difficult to evaluate the adequacy of the observed range of depressive symptom scores across the entire set of studies.

Possible restriction of range on measures of activity limitation may have been a factor in some of the studies that did not find mediation. Some of these studies may have had samples in which activity limitation was restricted because of a generally low level of functioning—for example, the nursing home and congregate living samples of Parmelee et al. (1991) and Cohen-Mansfield & Marx (1993). Restriction of range could also have occurred if activity limitation was bounded within the upper range of functioning. Kahana et al. (1997) reported that in their sample, personal disability (i.e., ADL, IADL, physical limitations) had relatively weak relationships with pain, depressive symptoms, and negative affect. This may suggest a possible restriction of range in personal disability, even though participants were in the upper ranges of old age (71-98). That 82% of the sample rated their health as “excellent” lends further credence to this possibility. Bookwala et al. (2003) also raised this as possibility explanation for the lack of mediation in their sample. However, without appropriate normative data, it is difficult
to fully evaluate this possibility with respect to most of the studies in this review.

Although the majority of studies were cross-sectional, three used a longitudinal design (Geerlings et al., 2002; Gureje et al., 2001; and Williamson & Schulz, 1995). Of these three studies, the latter two showed evidence of partial mediation, and the former found no evidence of mediation. With so few longitudinal studies to consider, it is hard to detect any specific pattern related to this aspect of study design.

The majority of studies utilized self-report measures of most constructs. Given the lack of variability across studies in this regard, patterns with respect to the influence of this study feature on mediation results cannot be adequately assessed. Studies used a variety of data collection modes, including in person interviews, paper-and-pencil questionnaires administered on-site or via mail, staff-ratings and diagnoses, data abstracted from medical and/or psychological records, and various combinations of different modes. Data collection mode did not appear to influence whether or not a study found evidence of mediation. There is however, one general exception to this general characterization: The Cohen-Mansfield and Marx (1993) study used a markedly different methodology, relying on staff ratings for all three central constructs in the mediation model. Hence the extent to which this study tested the same conceptual mediation pathway as outlined in the proposed conceptual model and examined by other studies in this review could be debated. Despite this methodological difference, however, their findings (or lack thereof) closely resembled those of Parmelee et al. (1991).

Neither study sample size nor data analysis technique appears to account for inconsistencies in findings regarding mediation. Most studies had very adequate sample sizes; indeed, some (e.g., Gureje et al., 2001; Kahana et al., 1997) had extremely large
samples. All studies appear to have used appropriate statistical analysis methods—namely, ordinary least squares (OLS) regression, path analysis, and/or structural equation modeling (SEM). A few studies used other analytic techniques (e.g., Parmelee et al., 1991, used partial correlation analysis and ANCOVA), but these techniques seem generally appropriate given the study design, measures, and sample characteristics. Some studies had multiple mediating constructs that were tested simultaneously. In studies that relied on OLS or OLS-based path analysis (e.g., Parmelee et al., 1991), it was sometimes difficult to determine the independent contribution of each construct to the mediation process. Although most studies that used SEM successfully managed to avoid this problem (e.g., Kahana et al., 1997; Rudy et al., 1988), one study that tested several mediating constructs and alternative structural mediation models (Goodland, 2002) yielded results that were also hard to interpret clearly.

As a whole, this body of literature does suffer from some of the same pitfalls that hamper many studies that seek to test mediation hypotheses. First, some authors were not always clear in their language regarding mediation processes. This was especially true of those studies conducted in the early 1990s. For example, Parmelee et al. (1991) articulated an intention to test whether activity restriction (viz., functional disability) “moderates the pain-depression relationship” (p. P15); however, all subsequent discussion made it clear that the study tested a mediation hypothesis. The other seminal study in this area (Williamson & Schulz, 1992a) also used language that sometimes confused mediation and moderation (see p. P368 for example). Second, in most studies testing mediation hypotheses, it was not always clear how the authors evaluated the “significance” of an observed mediation effect. When total mediation is observed, the
strength and significance of the mediation effect is indisputable. However, with few exceptions (e.g., Rudy, Kerns, & Turk, 1998; Williamson, 2000a), study authors rarely reported the statistical test or evaluation criterion used to judge whether or not partial mediation had occurred. Such limitations are, of course, not exclusive to studies of mediation in the pain-depression relationship; many mediation studies fail to adequately detail the processes and criteria used to evaluate mediation effects.

**Issues related to construct definition and measurement.** Some of the studies included in this review appear to have been hampered by limitations with respect to the conceptualization and/or measurement of one or more central constructs (i.e., pain, depression, activity limitation). It is difficult to evaluate the extent to which differences in the conceptualization and/or measurement of pain may have influenced study findings. Prior mediation studies have focused almost exclusively on pain intensity (variously labeled severity), or on a mixed construct that often included intensity along with other dimensions such as pain frequency and site.

In an attempt to extend pain research beyond the population of chronic pain patients, some researchers have borrowed items (or scales) from multidimensional pain inventories developed for use with chronic pain populations. Despite evidence that these items and scales may represent different dimensions of pain, these researchers (like some of their colleagues in chronic pain research) have created a summary score intended to represent some overall “pain” or “pain intensity/severity” construct. Unfortunately, such summary scores include items that appear to measure constructs such as activity limitation or interference—making it difficult to accurately test mediating hypotheses.

To the extent that pain measures have overlapped conceptually with measures of
proposed mediating constructs in prior studies, findings regarding mediation may have been *either over- or under-stated*. Parmelee et al. (1991), for example, included an item from the MPQ measuring activity limitation (pain interference) in their measure of pain “severity.” The conceptual overlap between the pain interference item and items measuring ADL impairment in their study may have spuriously inflated the relationship between pain and functional disability and potentially distorted an underlying mediation effect. Although criticisms regarding possible overlap between definitions and measurement of pain and mediating constructs could be levied against other prior mediation studies (e.g., Geerlings et al., 2002; Gureje et al., 2001), there does not seem to be any discernible pattern related to whether or not mediation was found.

With the exception of the study by Gureje et al. (2001) that combined diagnoses of anxiety and depressive disorders, the studies did not appear to be hampered in relation to the conceptualization and measurement of psychological distress. There was no detectable pattern of findings related to the use of any specific measures. In addition, study results did not seem to vary in terms of whether studies relied on a continuous measure of symptomatology or a categorical measure indicating the presence of a disorder as the outcome. In fact, both studies that compared the two approaches reported that mediation results were unchanged (Cano et al., 2004; Geerlings et al., 2002).

As was previously observed, prior mediation studies have varied most noticeably in terms of the construct(s) proposed to mediate the pain-depressive symptom relationship. In fact, the mediating constructs were so variably defined and operationalized that it is difficult to compare results across studies. Some of the variability in study results, however, *may* be related to whether or not the mediator was
defined and measured in relation to pain. *All of the studies that found no evidence of mediation relied on more general (i.e., not pain-specific) measures of activity limitation.*

In contrast, both of the studies that relied solely on a pain-specific measure of activity limitation (e.g., Arnstein et al., 1999; Rudy et al., 1988) found evidence of at least partial mediation. But, alongside these general patterns, exceptions could also be found—for instance, general (not pain-specific) measures were used in studies that found evidence of partial (e.g., Williamson & Schulz, 1992a) and total (e.g., Williamson, 2000a) mediation.

It is important to recognize that in existing studies, measurement of activity limitation is confounded with the nature of the sample or target population—that is, in chronic pain populations, researchers tend to examine pain-specific activity limitation; in more general, community-based populations, researchers tend to examine IADL and/or ADL functioning or general physical limitations, irrespective of pain. Some of this confounding is reasonable and practical—it does not make much sense to assess pain-specific activity limitation in persons who do not have pain. Whether or not this confounding is always necessary is unclear. At issue here, however, is the fact that such confounding makes it difficult to determine if the variability in results is related more to construct conceptualization and measurement, or to sample composition. There are also exceptions to the observation that studies using pain-specific measures of activity limitation tended to find evidence of at least partial mediation. Take, for example, the study of chronic pain patients by Goodland (2002). Goodland studied several mediating constructs, some of which were assessed using pain-specific measures (e.g., vocational and social disability), whereas others were assessed independent of the respondent’s pain (e.g., ADL limitations, recreational disability). His results, however, did *not* follow the
general pattern identified above: ADL limitations did appear to function as mediators, evidence regarding social disability was mixed, and there was no evidence that any other types of disability functioned as mediators of the pain-depression relationship. Beyond whether activity limitation are conceptualized and measured as pain specific or not, it is possible that the specific activity limitation(s) that were measured contributed to some of the inconsistent results. The type of activity limitation that seemed to produce the most disparate results was ADL limitations. Findings from these mediation studies seemed to vary depending on: (a) whether or not ADLs were included in the measure of the mediating construct, and/or (b) the extent to which ADLs dominated the measure. So, for example, the nursing home/continuing care retirement community studies (Cohen-Mansfield & Marx, 1993; Parmelee et al., 1991) relied almost exclusively on indicators of ADL impairment, and found no evidence of mediation. In two of the other three studies that found no evidence of mediation (Bookwala et al., 2003; Kahana et al., 1997), ADL functioning was used as a primary indicator of activity limitation (i.e., physical disability); in addition, physical disability was conceptualized and measured separately from social limitations (or disability) in these studies. By contrast, among studies in which the activity limitation construct was broadly defined to include many different areas and dimensions of life (e.g., social, recreational, family, IADLs, and ADLs), there was a tendency to find at least partial mediation. This was especially true if these different dimensions were aggregated to create a global measure of activity impairment or interference. Exceptions to this general observation certainly exist, and some of the findings from specific studies are not easily explained using this paradigm (e.g., Goodland, 2002, results showing ADL mediation; Kahana et al., 1997
finding of no mediation for social disability). In addition, some of the more broadly-defined measures were also pain-specific measures, layering on yet another source of possible variability contributing to study results. However, these observations do raise some unanswered questions worthy of exploration in future research.

The notion that ADL-based measures of activity limitation may present problems in tests of pain-depression mediation is indirectly supported by the results of some additional analyses by Williamson and Schulz (1992a). In post-hoc analyses, these authors found that restrictions in certain activities (e.g., shopping, hobbies) seemed to have the strongest mediating roles in the relationship between pain and depressive symptoms. They also were able to replicate the mediation effect they observed with the Activity Restriction Scale by using just a subset of five IADL items (e.g., shopping, transportation, heavy housework, doing laundry, preparing meals) derived from another measure. In a later publication (but not in the original article), Williamson noted that they were unable to replicate the mediation results using a traditional ADL measure (Williamson, 2000b). These findings also lend support to Williamson’s contention (presented earlier in discussion of this author’s conceptual model) that it may be the earlier losses related to basic physical abilities that are of primary importance.

Issues of conceptualization and measurement of key constructs can be summarized thusly: To what extent have prior studies: (a) accurately conceptualized the central constructs under study, (b) created or selected measures that adequately (and accurately) represent those constructs (and all of their relevant dimensions); and (c) matched the measurements of those constructs to the distribution of those constructs in the population selected for study. In the group of studies reviewed here, studies have
been most troubled by difficulties with respect to the conceptualization and measurement of mediating constructs. To the extent that studies have relied solely on measures of IADLs and ADLs, they might have suffered from restricted sampling, or representation, of the activity limitation construct and/or a lack of fit between these measures and the theoretical range of the construct within their samples.

**Issues related to additional explanatory variables.** Across this set of studies, there was marked variability in the extent to which analyses controlled for the presence of other, potentially explanatory variables. Illustrative extremes in this regard include: (a) Williamson & Schulz (1992a), whose analysis controlled for nothing other than physician-rated physical illness (which was also being investigated as a potential mediator); (b) Geerlings et al. (2002), whose analysis adjusted only for age and gender; (c) Bookwala et al. (2003), who included several demographic variables (age, gender, race, education, marital status) and the number of co-existing health problems as control variables in their analysis; and (d) Gureje et al. (2001) who, in addition to including this same list of demographic and health condition variables, also included several other clinical characteristics (e.g., self-rated health, physician-rated health). Not surprisingly, studies with larger samples tended to include more covariates in the analyses. The types of covariates did vary across studies, but this may also have been a function of variability in the types of samples being studied. There was also substantial variability in whether or not (and the extent to which) study authors presented one or more rationales (e.g., based on theory, prior empirical evidence, evidence within the current dataset) for the selection and inclusion of control variables. Overall, however, there did not appear to be any systematic variance in study outcome related to the inclusion or exclusion of control
variables in the analyses.

A few study authors did conduct some additional, often post-hoc, analyses. The results of these analyses offer some potential insights regarding additional variables that may influence the mediating pathway between pain and depressive symptoms. While a number of factors have been suggested and explored in some studies, the two with the most relevance to the current study (and the most existing support) are gender and age.

It was previously observed that the gender composition of the samples in these studies varied considerably. However, among the 12 studies with information (or actual variation) on gender, there appeared to be no systematic association between the gender ratio of the sample and the outcome of the mediation analysis. Studies dominated by women or men seemed equally likely to appear in any of the categories that characterized mediation results. Two of the above studies (Parmelee et al., 1991; Geerlings et al., 2002) did examine possible gender differences in the (direct) relationship between pain and depressive symptomatology. However, neither of these studies (nor have any others) explored the possibility that gender may influence the indirect relationship between pain and depressive symptomatology—i.e., the mediating effect of activity limitation. This is somewhat surprising, given the existence of compelling evidence documenting gender differences in both pain and depressive symptomatology (reviewed earlier in this chapter). The findings of Parmelee et al. and Geerlings et al. are reviewed in a subsequent section on gender as a potential moderating variable.

Across the group of mediation studies, there does seem to be a detectable pattern related to the age of study participants. Studies that found evidence of at least partial mediation tended to have either: (a) Younger samples, on average (note: the mean age of
participants across the 5 partial mediation studies was approximately 55, and was approximately 51 across the 4 total mediation studies); or (b) samples that encompassed a broader age range, which included individuals of younger ages (e.g., age range 20-68 in Arnstein et al., 1999). In contrast, studies that found no evidence of mediation tended to have either: (a) Older samples, on average (note: the mean age across these 5 studies was approximately 77); or (b) samples that were comprised only of those beyond middle age (e.g., over age 50, 55, or 70). The only notable exception to this pattern is Williamson & Schulz’s (1992a) initial study of geriatric outpatients (all aged 55 or older, mean age = 72) that did find evidence of partial mediation. Several of the above studies did conduct analyses to examine potential age-related differences with respect to mediation. Some of these analyses appear to have been done in response to speculations by Williamson regarding the possible influence of age on study results (e.g., Williamson, 2000b; Williamson & Schulz, 1995).

One additional issue. One additional issue deserves brief mention. Earlier, it was observed that two parallel, but relatively independent, lines of research have evolved with respect to the role of activity limitation as a mediator of the relationship between pain and psychological distress. Two of the hallmark mediation studies (Parmelee et al., 1991; Williamson & Schulz, 1992a) were published in a prominent gerontology journal (Journal of Gerontology: Psychological Sciences), while the other (Rudy, Kerns, & Turk, 1988) appeared in the dominant journal in chronic pain, Pain. Within this set of mediation studies, there appears to be a “disciplinary-divide,” characterized by relatively little sharing and acknowledgement of ideas across the two groups of researchers.

Of the four chronic pain mediation studies published after 1992, only Arnstein et
al. (1999) acknowledged Williamson’s prior work as central in establishing the rationale for their study. Cano et al. (2004) did cite Williamson and Schulz (1992a), but only as one of several studies that found evidence of mediation by activity limitation. Note that the reverse was also true: Few of the gerontological studies identified the contributions of chronic pain researchers. For example, the study by Williamson and Schulz (1992a) did not cite the prior study by Rudy, Kerns, & Turk, 1988; and, while they did cite a few references from the chronic pain literature, they did not cite prior discussions regarding potential mediators of the pain depression relationship. The later publication by Bookwala et al. (2003) cited very few chronic pain studies, and made only passing reference to an earlier study by Gureje and colleagues. Although there are a few exceptions to this general characterization (e.g., Geerlings et al., 2002; Kahana et al., 1997), the majority of studies have done little beyond citing a selected study or two from the other field to either support their results or highlight contrasting findings. More central than a lack of citations and acknowledgements, however, is the apparent lack of critical dialogue between scholars in the two fields—something that seems to have hampered the furtherance of knowledge in this area of research.

**Gender as a Potential Moderator of the Relationship Between Pain and Depressive Symptomatology**

The proposed conceptual model (Figure 4) makes clear that any of the several identified background or health-related characteristics could potentially influence (i.e., moderate) the relationship between pain and depressive symptomatology. Within that context, a moderating variable could impact any or all of the following relationships:

1. The direct relationship between pain and depressive symptomatology (i.e., Path A);
(2) the indirect (i.e., mediated) relationship between pain and depressive symptomatology via (a) the relationship between pain and activity limitation (i.e., Path B), or (b) the relationship between activity limitation and depressive symptomatology (i.e., Path C), or even (c) the extent to which activity limitation mediates the relationship between pain and depressive symptomatology (i.e., the combined effect of Paths B and C on Path A). Any one or all of these paths could potentially differ by age, gender, race and/or ethnicity, SES, body weight, or any of a number of different disease conditions.

In order to restrict the scope of the current study, a decision was made to examine only one potential moderating variable—gender. Gender was selected for three primary reasons. First, as reviewed earlier, there is substantial empirical evidence suggesting differences by gender in the levels of several key constructs in the activity limitation model (viz., pain intensity and depressive symptomatology). Prior sections of this chapter have established that both pain intensity and depressive symptomatology vary considerably by gender, with women typically experiencing (or reporting) higher levels of both, as compared to men. This evidence has fueled speculation that the relationship between the two constructs may be different in women than in men (e.g., Campbell et al., 2003; Fishbain et al., 1986). Although differences in the mean levels of constructs by gender do not necessarily presage the existence of gender differences in the relationships between constructs, they do provide a reasonable justification for examining the potential influence of gender on such relationships. Second, gender is a manifest identifying characteristic within heterosexual married couples, and thus already occupied a central role in the current study, given the dyadic nature of the data. Third, there have been some limited attempts to examine how gender might influence the pain-depressive symptom...
relationship. In general, such attempts have occurred only in research on chronic pain or painful conditions (e.g., arthritis). Findings to date have been largely mixed.

An early study of adults (mainly younger adults) referred to a chronic pain clinic, Haley, Turner, and Romano (1985) reported that the pain-depressive symptomatology relationship was “stronger” for women than for men. Although levels of depressive symptoms levels were similar between male and female chronic pain patients, pain intensity/severity was significantly correlated with depressive symptomatology for women, but not for men. These findings were summarized in the text as: “In female patients, depression was related to self-reported pain severity…[F]or males, analyses revealed no significant relationship between pain report and depression.” (p. 341). These correlations were not compared statistically, and were calculated based on subsamples comprised of 27 males and 36 females. Yet, the abstract, stated: “For women, depression was closely related to pain report, whereas for men depression was more strongly related to impairment of activity.” (p. 337), despite a subsequent statement that “Pain report was related only minimally to activity for male and female patients.” (p. 337). Although the abstract implied that significant differences were observed in the relationship between pain and depressive symptomatology (and also, potentially, the indirect, or mediated relationship), this conclusion must be viewed as suspect.

Two of the mediation studies reviewed above—Parmelee, Katz, & Lawton (1991) and Geerlings et al. (2002)—also explored gender moderation in their samples. However, both groups of authors examined only potential gender moderation of the direct relationship between pain and depressive symptomatology. Parmelee, Katz, & Lawton (1991) specifically sought to replicate the findings of Haley et al. (1985) in their
study of nursing home and congregate living residents. However, they found that, even though older women reported greater pain intensity than older men, there was no evidence that gender moderated the pain intensity-depressive symptomatology relationship. In contrast, the mediation study by Geerlings et al. (2002) found that the longitudinal relationship between pain and depressive symptomatology was somewhat stronger in men than in women, even after controlling for the effects of age and physical disability. Men were more negatively affected by increased pain symptomatology than women, but only when pain symptoms reached high levels.

Other studies that have examined gender differences in the pain-depressive symptomatology relationship have also yielded inconsistent results. A recent study of 260 mostly middle-aged (35-55) patients referred to a hospital-based pain management clinic for rheumatic diseases in the UK found no evidence of gender differences (Keogh et al., 2006). Pain intensity ratings were positively (and relatively equally) related to depressive symptomatology scores among both women and men in this sample. A similarly large study of a diverse group of chronic pain patients by Edwards, Augustson, & Fillingim (2000) also failed to find a difference between men and women in terms of the relationship between pain and depressive symptomatology. In a sample of chronic pain patients who were married (though not to each other), Cano, Weisberg, et al. (2000) found no evidence of a gender difference in the relationship between pain severity and depressive symptomatology; the correlation was positive and of moderate strength in both male ($r = .39, p < .001, n = 77$) and female patients ($r = .38, p < .001, n = 88$).

No prior study has specifically examined the possible moderating effect of gender on the mediating pathway involving activity limitation. In reviewing their findings,
Geerlings et al. (2002) suggested that their analyses showed no differences in mediation by gender. However, these authors did not explicitly test for the presence of any significant mediation in the gender-specific models (despite an apparent reduction in the size of the coefficients for pain in both genders), much less a difference in the size of any potentially mediated effects. Details about the strength and direction of the relationship between activity limitation and depressive symptomatology in these gender-specific models were also missing. Thus, the conclusions of Geerlings et al. (2002) regarding a lack of gender difference in (no) mediation by activity limitation, may be suspect.

There may be some reason to expect that such a moderated-mediation effect might exist. One particularly compelling line of evidence comes from studies suggesting that women may experience greater activity limitation from pain than do men. Unruh’s (1996) comprehensive review of gender differences related to pain evaluated differences related to “disability due to pain.” Several categories of disability, also termed “impairment,” were considered. She observed that although several studies had shown higher rates of daily activity impairment due to pain in women than in men, similar rates of activity impairment had been found in a sizeable number of studies, and not all rate differences that had been observed were statistically significant. A similar pattern was observed among studies of that had examined the use of sick leave and work absenteeism due to pain. However, when work and usual activity impairment had been combined in a few studies (i.e., into a single, broad “disability” measure), there was a marked tendency for women to report more disability due to pain than men (e.g., Taylor & Curran, 1985; Lipton & Stewart, 1993, cited in Unruh, 1996). Based on her review, Unruh concluded that, “Women may also be at greater risk for pain-related disability than men…” (p. 123),
but that the variation in findings across studies suggested that other factors—either acting in addition to, or in interaction with, gender—play an important role in determining pain disability. One obvious “other” factor noted in her review was the tendency for women to also report greater pain intensity.

Studies completed after Unruh’s review have been mixed, but on the whole tend to favor gender differences in levels of pain–related disability, with females showing higher levels of pain-specific activity limitation than males. For example, one study of 168 married individuals with osteoarthritis knee pain found that women reported higher levels of disability due to osteoarthritis-related knee pain (e.g., impairment in ADLs, mobility, physical dexterity, ability to complete household tasks) than men (Keefe et al., 2000). In another study of older persons (age 70+) with pain, reports of pain-specific activity limitation (e.g., mobility, “normal tasks,” recreation) were higher for women than for men (Scudds & Ostbye, 2001). Together, these results tend to support Unruh’s observation that gender differences may be limited to studies using measures specific to impairment or limitation resulting from pain.

To date, relatively few studies have directly tested gender moderation hypotheses with respect to the pain-depressive symptom relationship. Existing studies have largely been limited to chronic pain patients and older persons with painful diseases. In addition, most studies have looked only gender differences in the relationship between pain intensity and depressive symptomatology. The Haley et al. (1985) and Geerlings et al. (2002) studies do provide some evidence that the strength of the relationship between pain and depressive symptomatology may vary by gender. However, findings have been inconsistent with respect to the nature of this relationship. Lack of a consistent pattern
was as noted in the review of studies under Paths A, B, and C.

No prior studies have examined possible gender differences in the mediation of the pain-depressive symptom relationship via activity limitation. Although there does appear to be some evidence suggesting that women may report greater pain-specific activity limitation than men (especially when such limitations are defined and measured broadly), these findings are limited to chronic pain patients and older persons with pain. The extent to which prior studies have controlled for other factors such as age and pain intensity is unclear. It is important that studies have also taken account of gender differences in the prevalence of activity limitation in the population of middle-aged and older adult, as numerous studies document that women display more physical limitations than men, irrespective of pain.

It is often unclear how to best test for the possibility that mediation processes may differ across different subgroups. There are also conceptual issues involved in interpreting potential differences across subgroups. In the case of gender differences, for example: Are observed effects actually due to differences in mediation processes between men and women, or could they be due to underlying differences in the prevalence and valence of key constructs (e.g., women are more likely to experience pain and pain of greater intensity, as well as depressive symptomatology). Or, perhaps apparent differences in prevalence reflect the influence of larger societal forces—e.g., women and men have similar experiences, but they are socialized to report their experiences in different ways). Although challenging, it is important to explore whether or not the proposed mediation pathway(s) in the pain-depressive symptom relationship are contingent upon some third variable (gender, for example). Additionally, since
existing research has largely focused on adults of younger ages, research is clearly needed on the link between pain and depression in aging men and women (Campbell et al., 2003). In conclusion, whether or not (and how) gender moderates the direct relationship between pain and depressive symptomatology or the indirect (i.e., mediated) relationship between the two remain important, unanswered empirical questions.

**Unique Features of This Study**

The present study sought to examine pain and its relationship to depressive symptomatology among community-dwelling middle-aged and older married couples. This study was designed to address several key limitations and gaps that were identified in the review of the existing literature concerning pain and psychological distress at the intra-individual level. These limitations and gaps are highlighted below in the discussion of four interrelated features that distinguished this study from much prior research.

First, this study addressed a gap in the knowledge base regarding pain among community-dwelling middle-aged and older adults. The bulk of existing research concerning pain and its relationship to depressive symptomatology has been conducted using samples of chronic pain patients. Research on the pain-depressive symptom relationship in community-based samples has been limited, and studies specific to those in middle-age and older adulthood sparse. This gap is especially pronounced with respect to those middle-aged and older adults who may experience pain that does not fit neatly into the precise categories used in much prior research—for instance, chronic pain (e.g., of minimum duration and/or frequency), disease-specific pain (e.g., osteoarthritis, cancer, rheumatoid arthritis), and/or pain in defined locations (e.g., lower back, knee, head/neck).

The sample for this study was derived from a large, representative survey of U.S.
households that included oversampling of racial and ethnic minority persons, as well as persons over age 70 (i.e., the Health and Retirement Study, HRS). Because HRS sampled participants without regard to their health or pain status, participants displayed considerable diversity in their pain experiences, as well as in their background and health-related characteristics. Although some scholars advocate the independent study of different pain conditions (e.g., musculoskeletal pain, TMJ, migraine, etc.), others tout the importance of large-scale, population-based studies. By including individuals with a wide array of pain conditions and experiences, such studies have the potential to reveal what pain-related processes and outcomes are similar across different types of pain (Dworkin et al., 1992; McWilliams, 2003).

Second, the present study was guided by a conceptual model that was developed by blending parts of disparate models from the fields of chronic pain and gerontology, and anchoring these constructs and ideas within a broader theoretical framework concerning health and the disablement process (e.g., Verbrugge & Jette, 1994). This study focused on two areas of the proposed model: (a) the background and health-related characteristics associated with pain, and (b) the relationship between pain and psychological distress. Although knowledge concerning the demographic and health factors associated with reports of pain among community-dwelling persons has accumulated steadily in recent years, much less is known about the factors that influence ratings of pain intensity. This gap is most pronounced for pain that does not merit the label of “chronic.” The current study aimed to fill this gap by investigating the relationships between several background and health-related characteristics and ratings of pain intensity among middle-aged and older adults in the community.
This study examined the relationship between pain and psychological distress and evaluated activity limitation as a possible mediator of this relationship. This review located just 14 prior empirical studies concerning this specific mediation pathway. As the majority of prior studies utilized clinical-derived samples, the present study sought to contribute to knowledge regarding this relationship as it occurs in community-dwelling adults in mid and later life. This study fell within the mainstream of existing research, in that it, too, examined one particular dimension of psychological distress—depressive symptomatology. And, like several prior activity limitation mediation studies, this study utilized a version of a commonly-used measure of depressive symptomatology (i.e., the CES-D). This investigation considered only pain intensity—the dimension of pain most commonly assessed in studies of pain generally, as well as in prior mediation studies. Recall, however, that the definition and measurement of this construct varied considerably across prior studies and sometimes overlapped with other dimensions or constructs (e.g., frequency, interference).

This study sought to extend previous research by testing a more narrowly-defined conceptualization of activity limitation—physical limitations. As the above review made clear, compared to limitations in ADLs, IADLs, and some more broad areas of function (e.g., work, family/social, leisure/recreation), physical limitations have received limited attention within research on pain and/or depressive symptomatology in general. Moreover, no prior research has tested physical limitations specifically as an independent mediator of the relationship between pain intensity and depressive symptomatology. By focusing on physical limitations, this study sought improved fit between available measures of activity limitation and the theoretical range of impairment that might be
observed within a sample of community-dwelling middle-aged and older adults. The limitations observed in prior mediation studies associated with an overemphasis on ADLs and IADLs (e.g., restricted range, inconsistent results) also provide an important rationale for focusing attention on basic physical limitations. A focus on more basic physical limitations was also regarded as more consistent with a conceptual interest in mediating mechanisms that might occur earlier in the disablement process, particularly among aging persons in the community (see Williamson and Schulz, 1992a; Verbrugge & Jette, 1994).

Like most prior research, the current study examined the relationship between pain intensity and depressive symptomatology using cross-sectional data. Conflicting findings with respect to both the direct and indirect (i.e., mediated) pathways involving these constructs suggests that existing research has not yet definitively answered basic questions regarding the relationships among pain, activity limitation, and depressive symptomatology within a cross-sectional context. Additionally, the proposed conceptual model becomes more complex as it is expanded to more fully incorporate the study’s dyadic design—that is, as it takes into consideration the possible *inter*-individual effects (as will be discussed in Chapter 3). This complexity made proposing a longitudinal study impractical. The current study sought to further expand existing knowledge by evaluating these relationships and pathways while controlling for many possible confounding variables, as depicted in proposed conceptual model.

Third, this study had a unique focus on married couples. While the body of literature concerning pain and the pain-depression relationship in community-dwelling middle-aged and older adults has been limited, research regarding these relationships within *married couples* in mid and later life has heretofore been non-existent. This
review located roughly a dozen studies with samples of married persons; these were mainly clinical samples, drawn from chronic pain clinics or recruited on the basis of a medical condition (e.g., arthritis). Just a handful featured data collected from couples recruited from the community on the basis of one partner having pain or a painful disease. Several of these studies contributed evidence to the review of predictors of pain or pain intensity and/or one or more component pathways in the proposed mediation model. Only one prior study (Cano et al., 2004) examined the activity limitation mediation hypothesis in a sample comprised of married persons (all of whom were chronic pain patients). Research on pain and depressive symptomatology in married persons who are not labeled “chronic pain patients” or selected on the basis of a diagnosed health condition is lacking. Moreover, as has been noted, prior studies have examined pain in individuals who were married; in no previous study have participants been married to each other.

In contrast to prior studies, this study utilized a fully dyadic design, making use of the data that HRS collected independently from each spouse within the married couples participating in the study. Thus, it was possible to examine pain reports within married couples—i.e., the pain reports of husband-wife pairs—and to use these identify a sample of couples in which both the husband and the wife reported pain (Study Aim 1). These data made it possible the examination of some unique research questions at both the intra-individual and inter-individual levels. More is said about former below, while the latter is discussed in the next chapter (Chapter 3).

Fourth, and finally, the present study extended prior research by systematically examining the potential influence of one intra-individual characteristic—gender—on
various aspects of the proposed conceptual model. Gender constituted an inherent part of
the design of this study, given that data were collected from husbands and wives within
married couples. It thus made sense to utilize this design feature to investigate gender
differences in constructs and relationships about which prior research has either been
unclear or insufficient (especially within the target population of middle-aged and older
community-dwelling adults). Below are two examples of how the design of this study
yielded a potentially valuable context in which gender differences could be examined.

Recall that, although a majority of studies has found that women are more likely
to report pain and to report greater pain intensity than men, conflicting findings have
continued to permeate the literature. A number of studies have found that factors like
widowhood and/or living alone (e.g., Bradbeer et al., 2003) can account for differences in
pain and pain severity that appear to be due to gender. The demography of the older
population can make it difficult to isolate the effects of gender from those of these other
variables. However, by limiting the sample to cohabiting spouse pairs, the current study
eliminated the specific threats to internal validity posed by variability in marital status
and living alone. As such, the current study was better positioned to examine gender
differences in the pain experience of aging, community-dwelling adults.

The broad literature base underlying the interrelationships among pain, activity
limitation, and depressive symptomatology is also replete with contradictory findings
regarding the presence and nature of gender differences. This review also suggested that
gender might be a factor contributing to variability in findings regarding. Some authors
have identified other possible causes for the discrepant findings regarding mediation,
including age, adoption of sick-role behaviors, and social support. Turk et al. (1995)
observed that the pattern of correlations among pain, activity limitation, and depressive symptoms seemed to differ between younger and older chronic pain patients in their sample, with the latter displaying a stronger relationship between pain and depressive symptomatology. Because younger patients were more often married and living with a spouse and older patients were more likely to be living alone, the authors speculated that the increased availability of social support provided by a spouse may have tempered the relationship between pain and depressive symptomatology among younger patients. To the extent that the presence of a spouse comprises a potential source of social support, then this possible confound was also controlled by the design of this study.

Understanding the impact of demographic factors, such as gender, age, and minority status, on the relationship between pain and depression has been identified as one of the most important directions for future research (Campbell et al., 2003; NIH, 2012). The present study sought to contribute to this endeavor by systematically evaluating gender as a potential moderator of the direct and/or indirect (i.e., mediated) relationship between pain intensity and depressive symptomatology in this sample. Having data from both spouses also provided a unique opportunity to investigate patterns and relationships within and across married couples. The importance of considering the relationships between pain and depressive symptomatology within the context of marriage is addressed in the next chapter (Chapter 3).

**Research Questions and Hypotheses**

**at the Intra-Individual Level**

The individual-level research questions and specific hypotheses concerning pain and its relationship to depressive symptomatology in middle-aged and older husbands
and wives (i.e., at the individual level) are presented below. Two general types of questions were posed: (a) Research questions (RQ) that are based on existing empirical evidence and/or that were strongly suggested by the theoretical and/or conceptual frameworks that underlie the proposed study; and (b) exploratory questions (EQ) that have little empirical support at present, but may be suggested by one or more principles embedded within existing theoretical frameworks, or by observations based on the review of the existing literature. A further distinction is that Research questions have (at least one) specific, directional hypothesis, whereas exploratory questions do not.

All questions are numbered consecutively (e.g., RQ1, RQ2, EQ3…and so on), following the order in which they are introduced. Hypotheses (H) are labeled using the number of the research question under which they are subsumed (e.g., H1, H2, H3, etc.); additional lower-case letters are used to distinguish multiple hypotheses falling under a particular research question (e.g., H1a, H1b, etc.). Some questions (both research and exploratory questions) are subsumed under a more broadly-framed research question; in these instances, sub-questions are labeled using the same scheme as hypotheses (e.g., RQ4a, RQ4b, RQ4c, etc.).

The main research (or exploratory) questions that guided the current study are presented in **boldface type**, and hypotheses are presented in *italics* to make them easy to identify. Sub-questions and hypotheses are indented under the particular research (or exploratory question) to which they relate. To help clarify the particular effect being discussed, occasional reference is made to one or more paths labeled in Figure 4 (e.g., “Path B”). In the presentation below, research questions (RQ) and exploratory questions (EQ) are generally phrased using broad, conceptual language (e.g., pain, activity
limitation), whereas sub-questions and hypotheses (H) typically refer to the specific constructs or indicators in the current study (e.g., pain intensity, physical limitations).

As mentioned earlier, a unique feature of this study was a focus on pain within married couples. This prompted the use of two conventions in the presentation of the research questions and hypotheses below. First, because respondents were married couples, role-specific labels (e.g., wives and husbands) were used in place of the nomenclature traditionally used to phrase questions regarding gender differences (i.e., women and men). Second, the focal sample for this study was comprised of midlife and older couples in which both partners reported pain. These couples were identified as “dual-pain” (DP) couples. All individual-level research questions were specific to the husbands and wives in these DP couples. However, in order to simplify the presentation of research questions and hypotheses, only RQ1 makes explicit mention of DP couples.

**RQ1. Are there gender differences in key study variables between husbands and wives in dual-pain (DP) couples in mid and later life?**

**H1a. Wives will report greater pain intensity than husbands.**

This hypothesis was consistent with a considerable body of evidence showing that women are more likely to report experiencing pain and to report higher pain intensity than men. These gender differences appear to be robust, having been demonstrated in laboratory-based research (see review by Unruh, 1996), as well as in large, population-based studies (e.g., Christmas et al., 2002; NCHS, 2006). Although findings in clinical samples have been slightly less consistent, similar gender differences have been documented in studies of chronic pain patients (e.g., Koegh et al., 2005) and in studies those with painful diseases (e.g., arthritis, Felson et al., 1995). Gender
differences in pain intensity have been examined less frequently among middle-aged and older, community-dwelling adults; although existing studies in this area have observed gender differences generally consistent with other studies (e.g., Scudds & Ostbye, 2001). Although the majority of studies conducted with samples comprised of married *individuals* have not found evidence of gender differences in pain intensity, these studies lack comparability with the present study along several key dimensions. Based on the bulk of findings from most existing research, it was expected that in DP couples, wives would report higher levels of pain intensity than husbands.

*H1b.* Wives will report greater depressive symptomatology than husbands.

*H1c.* Wives will report greater physical limitations than husbands.

As noted in the review, women, on average, tend to report higher levels of depressive symptomatology and greater physical limitations than do men. Such gender differences have been observed consistently in large, population-based studies documenting the epidemiology of depression/depressive symptomatology (e.g., Kessler et al., 2003) and physical limitations, independent of the existence of any pain (e.g., Femia et al., 2001). The above hypotheses were justified on the basis of these findings.

**RQ2. Beyond gender, what other background and health-related characteristics are related to reports of pain intensity?**

*H2.* Selected background variables (older age, minority racial status and/or ethnic heritage, lower SES) and health-related characteristics (more disease conditions, increased body weight) will be associated with greater pain intensity.
In addition to gender, a variety of other background and health-related characteristics have been associated with greater pain intensity. Although there has been some variability in findings, accumulated evidence from a variety of studies suggests that individuals of African American race (e.g., Avis et al., 2003; Green et al., 2003) and Hispanic ethnicity (e.g., Portenoy et al., 2004), and with lower SES (e.g., Creamer et al., 1999 for education; NCHS, 2006 for income), a greater number of disease conditions (e.g., Bennett et al., 2002), and higher body weight (e.g., Anderson et al., 2003; Hitt et al., 2007) can be expected to report greater pain intensity. Although findings with respect to age differences in pain intensity have been mixed, a number of studies conducted with large, community-based samples of middle-aged and older persons report a positive relationship between age and pain intensity (e.g., Jacobsson et al., 2003; Scudds & Ostbye, 2001; Reyes-Gibby et al., 2007). On the basis of sample similarity then, pain intensity ratings were expected to increase in conjunction with the age of respondents. Although the extent to which such findings might generalize to husbands and wives in married couples was unclear, it seemed reasonable to expect that these same factors would emerge as predictors of pain intensity in DP spouses.

EQ2. Are particular disease conditions more strongly associated with greater pain intensity?

Although reports of pain have been associated with many disease conditions, including arthritis, cancer, diabetes, heart disease, lung disease, and stroke, studies specific to pain intensity have been sparse. However, the available evidence suggests that some of these same conditions may be associated with greater pain intensity. Given the limited amount of prior research, this question was exploratory in nature.
RQ3. **Is there a relationship between pain intensity and depressive symptomatology?**

*H3. Greater pain intensity will be associated with greater depressive symptomatology.*

Research Question 3 corresponds to Path A in Figure 4. As noted in the review of the existing literature, a sizeable body of evidence links pain and depressive symptomatology. Considerable evidence also suggests a positive, roughly linear relationship between pain intensity and symptoms of depression. This relationship has been observed in numerous studies, including those of chronic pain patients (e.g., Geisser et al., 2000), primary care patients (Karoly et al., 2006), and community-dwelling middle-aged and older adults (e.g., Bierman, 2011; Reyes-Gibby et al., 2002; Ohayon et al., 2003). Moreover, this relationship has proved relatively robust.

RQ4. **Is the relationship between pain and depressive symptomatology mediated by activity limitation (as measured by physical limitations)?**

As discussed earlier in this chapter, empirical questions involving mediation were addressed using the series of steps outlined by Baron and Kenny (1986). The first step requires that the focal independent variable be directly related to the dependent variable—i.e., that Path A in the Figure 4 would be significant (note that this step is addressed by RQ3 above).

The next step requires that the focal independent variable (pain) be related to the potential mediating variable (activity limitation). In other words, Path B in Figure 4 must be significant. Finally, the potential mediating variable (activity limitation) must be shown to be related to the dependent variable (depressive symptomatology). Thus,
Path C in Figure 4 must be significant. Finally, mediation is demonstrated if the relationship between pain and depressive symptomatology is significantly reduced and/or eliminated once the relationship between activity limitation and depressive symptomatology is taken into account—i.e., Path A must be significantly reduced or eliminated once activity limitation (and Path C) is added to the model. These latter two steps were addressed with the following sub-questions and hypotheses:

RQ4a. Is there a relationship between pain intensity and physical limitations?

H4a. Greater pain intensity will be associated with greater physical limitations. In terms of Figure 4, Path B will be positive and significant.

As reviewed earlier in this chapter, there is ample evidence of a positive relationship between pain intensity and physical limitations. In addition, considerable evidence documents the relationship between pain intensity and the broader construct of activity limitation. A sizeable relationship between pain intensity and physical limitations has been reported in large population-based studies (e.g., Weiner et al., 2003). Studies of community dwelling adults in mid and later life (e.g., Tsai, 2005) and pain patients (e.g., Cano et al., 2006) have also found evidence in support of Path B.

RQ4b. Is there a relationship between physical limitations and depressive symptomatology?

H4b. Greater physical limitations will be associated with greater depressive symptomatology. In terms of Figure 4, Path C will be positive and significant.
An abundance of studies document a relationship between activity limitation and depressive symptomatology. Research specific to physical limitations has been more sparse, although the bulk of existing studies suggest an approximately linear relationship between pain and the two constructs, with more physical limitations associated with greater levels of depressive symptomatology. This relationship has been observed in studies conducted in large population-based studies of all adults (e.g., Clark et al., 1997, 1998; Cooper & Kohlman, 2002), as well as in studies limited to community-dwelling midlife and older adults (e.g., Yang & George, 2005; Gayman et al., 2008). Moreover, evidence of this relationship has also been found in a variety of clinical samples of persons with pain, including chronic pain patients (e.g., Hirsh et al., 2006; Koegh et al., 2006) and those suffering from rheumatoid arthritis (e.g., Manne & Zautra, 1989), osteoarthritis (e.g., Tsai et al., 2005), and cancer (e.g., McMillan et al., 2008). Finally, numerous studies document a relationship between the more general construct of activity limitation and depressive symptomatology among community-dwelling adults who report pain (e.g., Strine et al., 2004).

RQ4c. Do physical limitations mediate the relationship between pain intensity and depressive symptomatology?

H4c. The relationship between pain intensity and depressive symptomatology will be at least partially mediated by physical limitations. In terms of Figure 4, Path A will be reduced and/or nonsignificant.

As noted in the literature review, in the majority of prior mediation studies, activity limitation at least partially mediated the relationship between pain intensity and
depressive symptomatology. No prior study has examined a mediating construct restricted to physical limitations (i.e., limitations in basic physical abilities). However, physical limitations of a similar nature have been included in broader measures of activity limitation in several prior mediation studies (e.g., Bookwala et al., 2003; Cano et al., 2004; Williamson, 2000a). Moreover, a majority of these studies found evidence of at least partial mediation. Furthermore, as was suggested in the review of this literature, the studies that produced the most disparate results with respect to mediation were those in which the measure of activity limitation was comprised largely of items assessing ADL limitations (e.g., Parmelee et al., 1991) or those in which either the nature of the activity limitation measure (e.g., 3 items in Geerlings et al., 2002) or the nature of the sample (e.g., majority of sample in excellent health in Kahana et al., 1997) may have limited the observed range of limitations. Given that the bulk of all prior studies, and the majority of studies that included basic physical limitations in the measure of activity limitation, found evidence of at least partial mediation, physical limitations were expected to at least partially mediate the relationship between pain intensity and depressive symptomatology in the present study.

EQ5. Does gender moderate any of the relationships between pain intensity and depressive symptomatology? Specifically, do the direct or indirect (i.e., mediated) relationships between pain intensity and depressive symptomatology differ between husbands and wives?

Gender has been suggested as a factor that may help to explain inconsistent findings regarding the mediating role of activity limitation in the pain-depressive symptom relationship. Yet, it has received very limited treatment within the empirical
literature. As a potential moderator in the current context, gender could impact: (a) the direct relationship between pain intensity and depressive symptomatology (i.e., Path A); and/or (b) the indirect (i.e., mediated) relationship between pain intensity and depressive symptomatology via activity limitation (i.e., the combination of Paths B and C, and their effect on Path A). Accordingly, two exploratory research questions were developed:

EQ5a. Does the direct effect of pain intensity on depressive symptomatology differ between husbands and wives? In terms of Figure 4, does Path A for husbands differ from Path A for wives?

EQ5b. Does the indirect effect of pain intensity on depressive symptomatology differ by gender? Specifically, does the mediating effect of physical limitations differ between husbands and wives? In terms of Figure 4, does the change in Path A for husbands differ from the change in Path A for wives?

Recall that two of the activity limitation mediation studies reviewed in this chapter (Parmelee et al., 1991; Geerlings et al., 2002) examined possible gender differences in the direct relationship between pain and depressive symptomatology. Recall that Parmelee et al. found no evidence of a gender difference in the effect of pain on depressive symptoms. In contrast, Geerlings et al. found that the pain-depressive symptomatology relationship was, surprisingly, stronger in males than in females.

Other studies exploring gender differences in the direct relationship between pain and depressive symptomatology have also yielded mixed results. Recall that one study utilizing a sample of married chronic pain patients reported no significant difference between husbands and wives in the magnitude of the pain-depressive symptom
No known studies have explored the possibility that gender may influence the indirect relationship between pain and depressive symptomatology—i.e., the mediating effect of activity limitation. This is surprising, given abundant evidence documenting the existence of gender differences in pain, depressive symptoms, and activity limitation (reviewed earlier in this chapter). Such evidence provided a rationale for exploring whether a moderated-mediation effect related to gender might exist. Given a lack of prior research in this area, as well as a pattern of mixed findings from the few existing studies, these research questions were exploratory in nature.
# CHAPTER 3: PAIN AND PSYCHOLOGICAL DISTRESS AT THE INTER-INDIVIDUAL LEVEL IN MIDDLE-AGED AND OLDER MARRIED COUPLES

## TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Available Theoretical Models</td>
<td>3-1</td>
</tr>
<tr>
<td>Theoretical Foundations of This Study</td>
<td>3-4</td>
</tr>
<tr>
<td>Proposed Inter-Individual Conceptual Model Linking Pain and Psychological Distress in Married Couples</td>
<td>3-8</td>
</tr>
<tr>
<td>Literature Review: Pain in Middle-Aged and Older Married Couples</td>
<td>3-13</td>
</tr>
<tr>
<td>General Observations</td>
<td>3-13</td>
</tr>
<tr>
<td>Review of Findings Specific to the Proposed Conceptual Model</td>
<td>3-16</td>
</tr>
<tr>
<td>Path 1: Covariation of Pain</td>
<td>3-16</td>
</tr>
<tr>
<td>Path 2: Covariation of Depressive Symptoms</td>
<td>3-22</td>
</tr>
<tr>
<td>Path 3: Covariation of Activity Limitation in Married Couples</td>
<td>3-28</td>
</tr>
<tr>
<td>Path 4: Pain in One Spouse Affects Psychological Distress in His/Her Partner</td>
<td>3-31</td>
</tr>
<tr>
<td>Psychological Distress in Partners of Persons</td>
<td>3-31</td>
</tr>
<tr>
<td>With Pain</td>
<td>3-31</td>
</tr>
<tr>
<td>Evidence of a Relationship Between One Spouse’s Pain and His/Her Partner’s Psychological Distress</td>
<td>3-36</td>
</tr>
<tr>
<td>Path 5 and Mediation: Indirect Pathways Through Which Pain in One Spouse Might Affect Psychological Distress in His/Her Partner</td>
<td>3-41</td>
</tr>
<tr>
<td>Potential Moderators of the Cross-Spouse Effects of Pain in the Marital Dyad</td>
<td>3-49</td>
</tr>
<tr>
<td>Gender and the Cross-Spouse Effects of Pain on Psychological Distress</td>
<td>3-50</td>
</tr>
<tr>
<td>When Both Spouses Have Pain</td>
<td>3-55</td>
</tr>
</tbody>
</table>
Limitations of Prior Research and Identification of Gaps ........................................ 3-57
Unique Features of This Study .......................................................................................... 3-62
Research Questions and Hypotheses at the Inter-Individual Level ............................... 3-64
Preliminary Considerations ............................................................................................ 3-64
Review of Conventions Used in This Presentation...................................................... 3-64
Handling of Couple-level Background Characteristics ............................................ 3-65
Research Questions and Hypotheses ............................................................................ 3-67
CHAPTER 3: PAIN AND PSYCHOLOGICAL DISTRESS
AT THE INTER-INDIVIDUAL LEVEL
IN MIDDLE-AGED AND OLDER MARRIED COUPLES

The prior chapter provided necessary background information about pain and reviewed the conceptual and empirical bases underlying the relationship between pain and psychological distress at the *intra*-individual level. In contrast, this chapter considers pain and its relationship to psychological distress at the *inter*-individual level. The chapter begins by briefly reviewing the available theoretical models that exist to study pain within the marital context. Because few models exist, and available models were of limited relevance to the current study, it was necessary to draw on concepts developed within several broad theoretical frameworks that have been used to understand couples and families in general. A section labeled “Theoretical Foundations of this Study” introduces these frameworks and identifies relevant concepts. The conceptual model proposed in Chapter 2 is then expanded to depict the relationships between pain and psychological distress within the marital context—i.e., within the dyad comprised of middle-aged and/or older spouses. The empirical literature supporting this proposed conceptual model is then reviewed, and gaps in the knowledge base are identified. Finally, research questions and hypotheses concerning pain and its relationships to psychological distress at the *inter*-individual, or couple-level, are presented.

Available Theoretical Models

In general, there are few theoretical models that address the larger social context surrounding individuals with pain. Although the effects of pain on families have received considerable attention from researchers, much of the existing research is atheoretical.
Theoretical models that do acknowledge the importance of an individual’s social context tend to focus on the individual with pain; others in his/her social milieu are regarded mainly as sources of contingencies for the reinforcement of pain behavior. Moreover, such models have been developed by scholars working in clinical settings, and consequently tend to focus exclusively on the experiences of persons with chronic pain.

Early behavioral theories regarding pain in families (e.g., operant conditioning models, social learning theory) generally ignored the impact of pain on families and directed attention instead to the role of the family in developing and maintaining chronic pain behavior (Flor & Turk, 1985). For example, Fordyce’s (1976) operant conditioning model of chronic pain argued that pain behaviors of an individual are maintained and even enhanced by the significant others in his/her life (e.g., spouse, other family members, and even health care professionals). This reinforcement could be direct or indirect, and complex contingencies of reinforcement could operate within the relationship. Studies derived from this model tend to focus on the extent to which family reactions (especially those of the spouse) either reinforce or deter certain pain behaviors or other possible “secondary gains” of the family member with pain. The model has received support from a number of studies demonstrating a link between pain-specific interpersonal exchanges and interactions—especially solicitousness (or over-concern) of the spouse—and pain reports, pain behaviors, and levels of disability (e.g., Block et al. 1980; Flor, Kerns, & Turk, 1987; Romano, Jensen, Turner, Good, & Hops, 2000). However, researchers have challenged the operant model as being overly simplistic (e.g., Cano & Williams, 2010; Hadjistavropoulos, Craig, Fuchs-Lacelle, 2004).

Cognitive-behavioral transactional models (e.g., Kerns & Weiss, 1994; Turk &
Kerns, 1985) are more dominant within contemporary research on pain and the family. These models emphasize the role of cognitive appraisals and coping processes in both the pain patient and the family member. For example, studies derived from these models have shown that family member cognitions and appraisals (e.g., attributions about the patient’s pain) influence their reactions and responses, these reactions and responses influence the patient’s coping processes, which in turn influence the family member’s adaptation (e.g., Cano, Weisberg, & Gallagher, 2000; Johansen & Cano, 2007; Turk, Kerns, & Rosenberg, 1992; Weiss, 1996). Although cognitive-behavioral models have been increasingly applied to the study of marital relationships in the context of pain, these models require extensive measurement of family and marital interactions and functioning. Such measures were not available within the dataset utilized for this study; thus, these theories were of limited use in the current study.

Some systems models of family functioning and theories of family stress and adaptation have provided conceptual frameworks applicable to the study of illness in families—for example, McCubbin and Patterson’s (1983) family stress process model; Minuchin’s (1974) family systems model of psychosomatic and chronic illness; Olson et al.’s (1983) circumplex model of family functioning; and Patterson and Garwick’s (1994) family adjustment and adaptation model. Yet, these models have seen only limited application within studies of families and pain (e.g., Turk, Flor, & Rudy, 1987). As Kerns & Weiss (1994) noted, although family systems models of health and illness have been useful in guiding thoughts about the role of the family in chronic pain, none of these models has had extensive influence on the field. These authors speculated that this lack of influence could be due to either the complexity of these models and/or difficulties in
operationalizing key constructs.

**Theoretical Foundations of This Study**

Interdependence theory offers a general perspective from which to view the marital relationship, and provides a good starting point for consideration of the interrelationships and processes involving both spouses in marriages that may be affected by pain. Interdependence theory focuses on social situations and the ways in which those situations shape both intrapersonal and interpersonal processes (Kelley & Thibaut, 1978). “Interdependence” refers broadly to the ways in which, and extent to which, individuals influence one another within the context of social interactions. Within any social interaction, each person’s outcomes can be influenced by his/her own actions and behaviors (actor control), the actions or behaviors of the interaction partner (partner control), and the joint actions and behaviors of the social partners (joint control) (Rusbult & Van Lange, 2003). Relationships can thus be characterized in terms of their level of “interdependence,” depending on the extent to which each partner relies on or is influenced by the other—i.e., the extent to which the outcomes of each partner are influenced by partner control or joint control.

The original formulations of interdependence theory by social psychologists Thibaut and Kelley (1959; Kelley & Thibaut, 1978; Kelley, 1979) embodied a social exchange perspective and emphasized market-like forces (such as perceived rewards and costs), power dynamics, and conflicts that governed behavior in social groups. Later applications of interdependence theory to the study of close, personal relationships (e.g., Kelley, 1981; Kelley et al., 1983; Rusbult & Arriaga, 2000) placed greater emphasis on the core concept of “interdependence” and the role of behavioral and psychological
interdependence in the formation, maintenance, and dissolution of close relationships (Sabatelli & Shehan, 1993).

For Kelley et al. (1983), the essence of a relationship lies within the interaction between two people. Closeness is reflected in a high degree of interdependence. Kelley et al. (1983) suggest that the level of interdependence can be observed in four properties of the interaction between two persons: 1) the frequency of interaction; 2) the diversity of influence that each person has on the other (e.g., across a range of activities, behaviors, goals, and plans); 3) the strength with which each person responds to or is influenced by the other; and 4) the duration of time that these different properties have been part of the interaction pattern of the two persons.

Family relationships are inherently close relationships and characteristically exhibit high degrees of interdependence and mutual influence, as the well-being of each family member is closely tied to the behaviors and well-being of other family members. The marital dyad is the central unit of the family and is typically characterized by high degrees of interaction, physical intimacy, and psychological closeness (Kelley, 1981). Marital partners rely on each other to fulfill important needs, and the outcomes for each partner are likely to be influenced by the actions, behaviors, and dispositions of the spouse. Marriage, then, involves considerable interdependence between the partners (Kelley, 1981). This interdependence provides a basis for many important interpersonal processes and outcomes within the marital relationship, including the direction of attention and emotion to one’s partner and to the relationship in general, the expression of prosocial behavior (e.g., caring, sacrifice, accommodation), and a sense of commitment to the relationship (Rusbult & Van Lange, 2003). Interdependence theory provides a
general basis for expecting (a) the well-being of spouses to be interrelated, and (b) the well-being of each spouse to be influenced by factors that affect the well-being of the other spouse.

Other theoretical frameworks either extend or complement concepts drawn from interdependence theory or offer explanations for specific phenomena or interrelationships observed in marital dyads that may be affected by pain. A few of these frameworks are introduced briefly below, and are then applied briefly in discussion of the proposed conceptual model. Other frameworks that relate only to specific elements of the proposed model (or that offer alternative explanations for various effects), are discussed within the review of the existing literature.

Kelley’s conceptualization of closeness emphasizes behaviors and activities—readily observable characteristics of a relationship (Berscheid, Snyder, & Omoto, 2004). Other scholars working from different theoretical paradigms in cognitive and social psychology have offered other definitions of closeness that emphasize different aspects of the relationship. Yet, interestingly, the central notion of “interdependence” among relationship partners is often (explicitly or implicitly) preserved.

For example, Agnew, Van Lange, Rusbult, and Langston (1998) discuss the concept of “cognitive interdependence”—a mental state of “thinking close” that is characterized by a pluralistic, collective representation of oneself within a relationship. This tendency to cognitively incorporate one’s relationship partner into one’s sense of self is often reflected in a tendency to use plural pronouns (e.g., “we”) in relationship-relevant cognitions and speech (Agnew, Loving, Le, & Goodfriend, 2004). Cognitive interdependence is also closely related to Aron & Aron’s (1986) concept of “self-
expansion,” or the tendency of each person in a social relationship to include the other person (and his/her resources, perspectives, identities) in his/her sense of self.

Notions of emotional interdependence within social relationships can be found within social contextual, interpersonal, and interactional theories of emotion (e.g., Coyne, 1976; Hatfield, Cacioppo, & Rapson, 1992, Joiner & Katz, 1999). Broadly speaking, these theories argue that social contexts, and the interactions within them, play a critical role in the creation, transmission, and maintenance of emotional states. One concept central to many of these theories is “emotional contagion” (Hatfield, Cacioppo, & Rapson, 1992). Primitive emotional contagion is a relatively unconscious process defined in lay terms as the tendency to “catch others’ emotions,” or (more psychologically) to “converge emotionally.” Hatfield and colleagues argue that people are especially likely to experience emotional contagion in close relationships like marriage because the partners are invested in one another, direct attention to one another, and tend to see themselves in terms of their relatedness to the other. The concept of emotional contagion, along with general principles derived from social contextual, interpersonal, and interactional theories of emotion, provides a basis for expecting significant relationships between spouses in terms of (a) the emotional well-being of each spouse, and (b) the health of one spouse and the emotional well-being of the other.

Finally, theories that offer explanations for how romantic and marital relationships are formed also offer some additional guidance to researchers who study the marital dyad. One theory of mate selection drawn from population genetics—assortative mating—argues that the pre-existing similarities of partners leads them to select each other in reproductive pairings (Thiessen, 1999). Assortative mating, then, also provides a
basis for expecting greater than chance levels of association between marital partners in
terms of both health-related characteristics and levels of well-being.

Proposed Inter-Individual Conceptual Model Linking Pain and
Psychological Distress in Married Couples

Figure 5 extends the *intra*-individual model depicting the relationship between pain and psychological distress (proposed in Chapter 2) to the context of the marital dyad. The model has three unique features. First, it recognizes that the central constructs under investigation—namely pain, depressive symptomatology, and activity limitation—are likely to covary (i.e., be correlated) within married couples. This within-couple covariation is depicted using curved, dashed paths labeled 1 (covariation in pain), 2 (covariation in psychological distress), and 3 (covariation in activity limitation). It should be noted that other constructs in the model can also be expected to covary within couples (e.g., age, disease conditions); however, these relationships are not depicted in Figure 5 in order to focus attention on the central constructs in the current study.

“Covariation” is a central concept within interdependence theory. In most early theoretical publications, it is used to refer to “covariation of interests” and “covariation of outcomes” in relationships (Kelley & Thibaut, 1978; Kelley, 1979). In the proposed model, the notion of outcome covariation is invoked to help explain both Path 2 (covariation in psychological distress) and Path 3 (covariation in activity limitation), as both serve as endogenous constructs in at least part of the model. Interpersonal and interactional theories of emotion and emotional contagion may also help to explain why the depressive symptom levels of spouses are likely to covary (Path 2).
Figure 5. Proposed Conceptual Model Linking Pain and Psychological Distress in Married Couples

Note. Dashed lines represent inter-individual relationships. These relationships are labeled to indicate the following paths of interest: Covariation of spouses’ pain psychological distress and activity limitation. The remaining paths represent the cross-spouse effect of each spouse’s pain and activity limitation on his/her partner’s psychological distress, with the subscript indicating the source of the effect—e.g., Path 4 represents the effect of the husband’s pain on the wife’s psychological distress.
Path 1 (covariation in pain) is more difficult to explain by strictly drawing on the concept of covariation in “outcomes” or “interests” emphasized within historical presentations of interdependence theory. However as noted above, the general notion of interdependence has been extended by researchers to help explain the connectedness that often occurs between partners in their cognition and emotion. As was discussed in Chapter 1, the human experience of pain consists of more than just physical sensation and involves the interaction of sensation with cognitive, emotional, motivational, and behavioral factors (Yehuda & Carasso, 1997). Thus, covariation of pain between spouses (Path 1) may be explained (at least in part) using a more general interpretation of interdependence theory.

There also are some scholars who argue for the existence of pain contagion—that is, emotional contagion specific to the experience of pain (e.g., Morse & Mitcham, 1997). Indeed, early experiments by Lanzetta and colleagues on the biological substrates of empathy seem to offer some support for the notion that watching another’s physical pain can precipitate physiological reactions in the observer that seem to mimic those associated with the actual experience of pain (Vaughan & Lanzetta, 1980). Emotional contagion thus offers another potential explanation for covariation of pain in spouses.

Of course, assortative mating also can potentially explain covariation in all three constructs in the model; but, this explanation is extremely difficult to defend (or dispute) without knowledge of each spouse’s health status prior to marriage. Beyond assortative mating, the extent to which spouses share similar environments, habits, and lifestyles may also contribute to shared variation in both physical and mental health. For example, poor nutrition or an unhealthy diet might put both spouses at risk for certain disease
conditions that could lead to shared variation in pain (Path 1) and/or activity limitation (Path 3).

The second unique feature of this proposed model is that it identifies several inter-individual, or “cross-spouse,” effects involving pain. Cross-spouse effects can be said to occur when a given characteristic of one spouse affects a (different) characteristic of the other spouse. Note that “different” is presented in parentheses. Theoretically, all inter-individual relationships within the marital dyad could be considered cross-spouse effects. For example, the paths in Figure 5 that depict the covariation of key constructs between spouses (e.g., pain) could be described as cross-spouse effects. However, many researchers reserve this term for use in characterizing those inter-individual relationships that (a) have an implied causal direction, and (b) involve different substantive constructs. Thus, most researchers would regard the paths in Figure 5 that link one spouse’s pain to the other’s psychological distress (note: constructs differ between spouses) as cross-spouse effects. In this document, the term inter-individual is used most frequently—especially in this chapter and the next (Method, Chapter 4)—largely because it can refer to both covariation and implied directional effects between spouses. At the same time, however, the content of subsequent chapters is more integrated—i.e., referring both to relationships hypothesized to exist within individuals (e.g., Paths A, B, C) and between spouses (e.g., Paths 1 through 5). In order to more clearly distinguish between these intra-individual and inter-individual relationships, these prefixes are generally italicized and separated from the root term: For example, “intra-individual” and “inter-individual” relationships. Terminology such as covariation and cross-spouse effects is used with increased frequency in later chapters, as specific findings are reviewed and discussed.
Interdependence theory proposes that closeness exists in a relationship “…to the extent that one or both interacting persons affect the other’s well-being” (Rusbult, Kumashiro, Coolsen, & Kirchner, 2004, p. 157). According to interdependence theory then, cross-spouse effects are an expected part of the marital relationship. And, as previously mentioned, notions of cognitive and emotional interdependence (drawn from both interdependence theory and theories regarding the interpersonal nature of emotion) can also provide some basis for expecting cross-spouse effects.

Of specific interest in the current study were the cross-spouse effects of pain—that is, the effects of one spouse’s pain on the psychological distress level of his/her partner. Cross-spouse effects are represented in the model by the straight, dashed lines labeled with the numbers 4 and 5. These paths are also subscripted to identify the specific source of each effect (e.g., $W$ indicates that the effect emanates from the wife to the husband). Throughout this document, these paths are referenced either generally (e.g., “Path 4”) or specifically (e.g., “Path 4$W$”), depending on context.

The model proposes that the cross-spouse effect of pain can be direct and/or indirect. For example, greater pain intensity in the wife can contribute directly to increased psychological distress in the husband (Path 4$W$). The wife’s pain can also have an indirect (i.e., mediated) effect on her husband’s psychological distress through its effect on her ability to perform basic physical activities (Path 5$W$). These indirect effects expand Williamson’s activity restriction model (ARM) of depressed affect (outlined in Chapter 2) to the level of the couple. At a general level, this theoretical model argues that pain in one spouse has indirect effects on his/her partner through its restriction of the spouse’s ability to perform important activities.
A final unique feature of the proposed model is that it suggests that background characteristics of the couple (e.g., household composition, years of marriage), as well as of the individual spouses (e.g., gender, age), can influence many elements of the model. Characteristics that influence pain and psychological distress at the individual level—for example, the relationship between education and depressive symptoms—were discussed in Chapter 2. Note, however, that the model conceptualizes socioeconomic status (SES) as both an individual-level and a couple-level background characteristic. So, for example, household income was conceptualized as a background characteristic of the couple and was examined in relation to pain and depressive symptomatology in each spouse. More is said about the handling of background characteristics at the end of this chapter and in Chapter 4 (Method).

Of primary concern in the current chapter are those background characteristics—conceptualized at either the individual or the couple level—that may shape the joint experiences and interactions within the couple. Some of these characteristics may be more likely to influence the within-couple covariation of pain, physical limitations, and/or psychological distress. Other characteristics may be more likely to influence the cross-spouse effects of pain on psychological distress. Since very little empirical evidence or theoretical development exists in support of specific influences of these characteristics, research questions pertaining to these influences within the current study were necessarily limited and exploratory in nature.

**Literature Review: Pain in Middle-Aged and Older Married Couples**

*General Observations*

The current base of knowledge regarding pain in married couples stems from a
body of scholarly work that has focused broadly on pain and the family. Most of this literature has accrued over the past 25-30 years. A good deal of this literature is either conceptual in nature and/or advocacy focused (i.e., designed to bring attention to the impact of pain on the family). Most empirical studies have been either atheoretical or based largely on behavioral theories, and studies have been mainly cross-sectional. The majority of empirical studies have been done in the U.S., but prominent research teams work in Finland, the Netherlands, the United Kingdom, Australia, and Germany.

Some of the extant literature focuses on families in general and some on couples. Since most empirical studies have been done with couples, the term “couples” is used when summarizing the main findings from this line of research. Although samples sometimes include unmarried, cohabitating couples, most have been comprised of married couples. In summarizing the literature, “spouse” is used most often, although “partner” is also employed to reduce redundancy.

The empirical literature on couples and pain is dominated by studies using small samples, averaging 50-60 subjects (or couples). Relatively few studies have samples over 100 individuals (or dyads), and most of these studies have been conducted within the past 10 years. The small sample sizes are primarily a reflection of the most frequent target population for pain research: chronic pain patients and their spouses (or family members). Samples are almost exclusively drawn from patients attending specialty pain clinics. Of notable exception are several studies by Cano and colleagues (e.g. Cano, Johansen, & Franz, 2005; Leonard & Cano, 2006) that recruited “chronic pain” sufferers and their spouses from the community. Of those studies not focused on chronic pain patients, the majority have examined patients with pain stemming from rheumatoid
arthritides, osteoarthritis, or cancer. These studies tend to recruit subjects from outpatient centers (e.g., cancer treatment centers) or specialty clinics (e.g., rheumatology clinics). Thus, the bulk of knowledge about pain and couples is based on samples drawn from clinical populations.

Early studies of couples and chronic pain (in the 1970s-1980s) tended to have samples composed of male patients and female spouses, especially because chronic back pain and work-related disability research dominated the field. In addition, many early studies of pain and couples were conducted within clinical populations receiving services through the Veterans Administration (VA). Later studies have had more equal representation of male and female patients, although many studies using disease-specific populations (e.g., patients with osteoporosis or rheumatoid arthritis), have been dominated by samples comprised of female patients and male spouses.

This review found only a handful of empirical studies that explicitly identified the study target population as “later life,” “older,” or “elderly” couples. More research has been directed toward couples in which one spouse reports a disease or condition that is typically associated with aging (e.g., osteoarthritis). However, even studies that purport to examine couples in later life tend to have samples more heavily populated by those in middle-age and early older adulthood (the average age of samples is typically mid 60s).

More commonly, studies of couples and pain focus on “adults,” regardless of age. Many studies have subjects ranging in age from the early to mid-twenties to the mid-60s. Studies may have a few subjects who are in their 80s and 90s—but these are the exceptions, rather than the rule. The mean age of the subjects in most samples tends to range from the early 40s to the early 50s. Much of the existing research on couples and
pain is based on samples drawn from specialty pain clinics. As very few older adults attend pain clinics, the average age of patients upon admission to pain clinics is around 43 years of age (Roy, 1994). Thus, the majority of what is known about couples and pain is based on research with chronic pain patients who are in early middle age.

*Review of Findings Specific to the Proposed Conceptual Model*

This review of the literature is organized around the major pathways and relationships depicted in the proposed conceptual model (Figure 5). Findings from empirical studies of pain and couples (or families in general, if relevant) are summarized within each section below. Details about study sampling, design, measurement, and analyses are provided when they help to establish a context for specific findings, or to highlight limitations within the existing knowledge base. In instances where evidence from empirical studies specific to pain is limited, related findings from the gerontological literature or more general literature on married couples are presented.

**Path 1: Covariation of Pain**

One of the central tenets of the conceptual model presented in Figure 5 is that pain is likely to covary in married couples. The current study examined this covariation in two forms: (1) the co-occurrence of pain in married couples, and (2) the covariation between ratings of pain intensity in couples in which both partners suffer from pain.

Although the phenomenon of co-occurring pain in married couples has long been noted by clinicians, studies documenting its prevalence are relatively rare. An early study by Gentry, Shows, & Thomas (1974) requested information from a sample of low back pain patients ($N = 56$) about their family history of low back pain and other physical conditions. Twenty-three percent of patients reported that their spouses had experienced
low back pain that was also resistant to treatment. Although suggestive, these findings were limited to patient retrospective reports of their spouse’s pain problems.

Mohamed, Weisz, and Waring (1978) were the first to document empirically that the spouses of chronic pain patients tend to report problems with pain at levels exceeding those found in other populations. These authors compared a small group of chronic pain patients referred for treatment of depression with matched controls referred for treatment of depression but without any serious somatic complaints. Data were also collected from their spouses. More pain problems were evident in the spouses of the depressed-pain patients than in the spouses of the depressed-only patients. There was also marked similarity in reports of pain location (e.g., head, abdomen, and limb) between the depressed-pain patients and their spouses. However, the Mohammed et al. study was limited by an extremely small sample size (n = 13 per group).

A larger, more representative sample was studied by Sharp and Nicholas (2000). These authors found that 21% of the significant others of 321 chronic pain patients reported that they themselves also suffered from a chronic pain problem. Spouses of chronic pain patients have also been shown to report higher levels of pain than do spouses of persons suffering from other chronic physical illnesses. For example, Flor, Turk, & Scholz (1987) compared the number of physical symptoms reported by the spouses of chronic pain patients to those reported by spouses of diabetic patients. Although the overall number of physical symptoms reported by each group was similar, the number of pain complaints was significantly higher in the spouses of pain patients than in the diabetic patients’ spouses.

The co-occurrence of pain between marital partners may be especially likely
among couples in mid and later life, when pain prevalence rates are generally highest in the overall population. Studies documenting the phenomenon of co-occurring pain in older married couples are rare. In a large metropolitan area in the Midwest, Cano and colleagues (e.g., Cano, Johansen, & Franz, 2005; Leonard & Cano, 2006) recruited a sample of 139 community-dwelling couples in which one partner reported chronic musculoskeletal pain, and have since published several studies using various subsets of this sample. Although descriptive demographic data were not reported for the entire sample, most published accounts have indicated that the sample was comprised mainly of middle-aged couples—for example, the average age of the person with chronic musculoskeletal pain was 53.2 ($SD = 13.3; N = 108$; Pence, Cano, Thorn, & Ward, 2006); the mean age of the spouse was 53.6 ($SD = 13.6; N = 113$; Leonard & Cano, 2006).

In several publications, Cano and colleagues have reported that a sizable proportion of the spouses of persons with chronic musculoskeletal pain reported that they themselves also suffered from chronic pain. Depending on the specific subsample included in the publication, the percentage of spouses who also reported chronic pain has varied from a low of 26% ($N = 84$ couples over age 55; Cano et al., 2005) to a high of 49% ($N = 113$ couples; Leonard & Cano, 2006), with a majority of studies reporting percentages closer to the latter estimate (e.g., 46% of 104 couples in Cano, Miller, & Loree, 2009; 48% of 79 couples in Johansen & Cano, 2007).

Very few studies have examined pain reports among spouses of persons who may suffer from pain, but who have not been labeled “chronic pain patients” or “chronic pain sufferers.” One rare exception is a study by Jacobi, van den Berg, Boshuizen, Rupp, Dinant, and van den Bos (2003). These authors studied 134 patients with rheumatoid
arthritis and their spouses in Amsterdam. The patients were mainly women (84%), and most were in their 50s and 60s. Notably, almost half (47%) of spouses reported that they themselves suffered from moderate pain or discomfort. More recently, Martire, Schulz, Keefe, Rudy, & Starz (2007) published the results of a randomized clinical trial involving 242 osteoarthritis patients age 50 or older and their spouses. Based on data reported for each of the three study conditions, approximately 56% of spouses also had “osteoarthritis/chronic pain” (Martire et al., Table 1, p. 125).

Deriving estimates of the co-occurrence of pain from published studies involving married couples is made difficult by the limited amount of data and the imprecise manner in which such data are often reported. For example, in a study of 95 married persons admitted to an inpatient chronic pain clinic in the United Kingdom, Newton-John & Williams (2006) reported that 30% of spouses “also reported experiencing a chronic illness, including chronic pain” (p. 54). Such reports present a challenge to researchers interested in couples and pain. In other publications, pain co-occurrence is implied by data reported only in descriptive tables and is never addressed in the text. Such was the case in the Martire et al. (2007) study. A similar pattern was observed in a report of a study of 165 chronic pain patients and their spouses by Cano and colleagues. In Geisser, Cano, and Leonard (2005), summary statistics for duration of pain (in years) were presented in a table for both patients ($M = 6.6, SD = 6.1$) and spouses ($M = 7.3, SD = 8.0$). A brief statement in the Method section suggested that spouses were also asked about pain (presumably their own pain), but these data were never discussed.

Data regarding covariation of pain intensity between spouses is largely missing from the literature. This author is not aware of any published studies—using either
community samples or samples of chronic pain patients—that have examined the extent to which pain intensity levels are related in marital partners who both report pain. Several studies, including those by Cano and colleagues, appear to have collected some detailed data from each spouse regarding his/her own pain. However, details regarding the (non-patient) spouse’s pain are rarely reported. The study by Leonard and Cano (2006) is a notable exception: Summary data were presented for pain location, severity, and duration in a subset of spouses who also reported chronic pain. No estimates of covariation were computed. The authors did indicate that a significant mean difference existed between the pain severity ratings of married partners who both reported pain; however, this test served mainly to substantiate a decision made by the research team to select, within such couples, the partner with the “most severe or disabling pain” (p. 141) to serve as the target spouse (i.e., “person with pain”) for the study.

Although a few additional studies appear to have collected pain data from both spouses in married couples, it is often hard to determine the extent to which pain intensity levels, specifically, overlap. For example, a correlation of .40 was reported between the SF-36 bodily pain subscale scores of 43 patients with rheumatoid arthritis and their spouses (Walsh, Blanchard, Kremer, & Blanchard, 1999). No descriptive data were reported for these subscale scores, nor were any data presented regarding the number of spouses who also reported pain. The correlation is suggestive; however, as was noted in Chapter 2, the SF-36 bodily pain subscale actually measures a conglomeration of constructs (pain presence, intensity, and interference), making it hard to evaluate the level of covariation specific to spouses’ pain intensity ratings.

Earlier, this chapter reviewed several explanations that have been offered as
possible explanations for covariation observed between spouses on all types of constructs. As most explanations have not been studied in relation to pain, there is no definitive mechanism that explains the high prevalence of pain symptoms among the spouses of persons suffering from pain (Roy, 2001, Chapter 5).

Whatever the causal mechanism, it is important to recognize that pain is one of myriad aspects of physical health that have been found to covary among married partners. For example, a study by Hippsley-Cox, Coupland, Pringle, Crown, and Hammersley (2002) analyzed the medical records of 8,386 middle-aged and older married couples in 10 general health practices in England. They found that partners of people with specific diseases (e.g., peptic ulcer, asthma, hyperlipidemia, hypertension) were at increased risk for the disease themselves, even after adjusting for age, smoking status, obesity, and general practice clustering effects. A recent analysis of data from the initial wave of the Health and Retirement Study (HRS) by Wilson (2002) also found significant correlations between the health status indicators of married partners in the United States. These inter-spouse correlations ranged from .21 for a weighted index of chronic conditions to .26 for a self-reported measure of global health status. Although controlling for demographic variables and behavioral risk factors significantly reduced these correlations, the remaining correlations were still substantial.

This review of existing research makes clear that there is a dearth of studies documenting pain co-occurrence in spouses of persons who may suffer from pain, but who have not been labeled “chronic pain patients.” More specifically, there is a lack of studies investigating the co-occurrence of pain between community-dwelling, middle-aged and older marital partners. In addition, this author is not aware of any published
studies—using either community samples or samples of chronic pain patients—that go beyond just documenting that pain co-occurs to examining the extent to which pain intensity levels covary between marital partners who both report pain.

**Path 2: Covariation of Depressive Symptoms**

Like various aspects of physical health, psychological symptomatology has also been observed to covary in married couples. Depressive symptomatology in particular has been found to be associated in studies of marital dyads (Coyne & Smith, 1991; Galbaud duFort, Kovess, & Boivin, 1994; Kouros & Cummings, 2010; McLeod, 1993), including studies of older marital partners (e.g., Bookwala & Schulz, 1996; Goodman & Shippy, 2002). In studies of older couples, higher levels of depressive symptoms in one partner have been associated with higher levels of depressive symptoms in the other partner (e.g., Bookwala & Schulz, 1996; Eagles et al., 1987; Tower & Kasl, 1995). These findings have also been extended to more serious levels of depressive symptomatology, and even to a diagnosis of major depression. For example, Kivela, Luukinen, Viramo, & Koski (1998) found that both partners in 1 in 20 elderly couples (6%) in a large epidemiological study in Finland met criteria for a DSM-III diagnosis of depression. In their study of middle-aged and older married couples in England, Hippisley-Cox et al. (2002) found evidence of elevated risk of depression among wives whose husbands were depressed (OR = 2.08).

Substantial covariation in depressive symptoms has often observed between married partners when one spouse is suffering from a chronic illness. For example, in a study of 123 older adults with recent vision loss and their non-impaired spouses, Goodman and Shippy (2002) reported a correlation of .24 (p < .01) between the Center
for Epidemiologic Studies-Depression Scale (CES-D) scores of both partners. A similar level of covariation has also been found in aging couples in which one partner suffers from advanced renal disease \( (r = .23, p < .001, N = 315; \text{Pruchno, Wilson-Genderson, \& Cartwright, 2009b}) \). Spouse covariation in psychological distress has been fairly well-studied within the context of cancer. Numerous studies report small to moderate correlations between the depressive symptoms levels of cancer patients and their caregiving spouses—e.g., a significant correlation approaching moderate size \( (r = .27, p < .05) \) was reported in a study of 85 couples in which the husband was diagnosed with prostate cancer (Kim, Kashy, Wellisch, Spiller, Kaw, \& Smith, 2008). A meta-analysis over 40 studies of cancer patients and their partners reported an average effect size in the moderate range (Hagedoorn, Sanderman, Bolks, Tuinstra, \& Coyne, 2008).

Covariation of depressive symptom levels has also been observed in married couples sampled from the community at large. In an analysis of baseline data from over 5,000 middle-aged and older married couples from the initial HRS and AHEAD cohorts, Townsend et al. (2001) also found moderate correlations (ICC = .26 for AHEAD couples; .27 for HRS couples) between spouses’ levels of depressive symptomatology. As part of a longitudinal analysis of data from 1,700 couples in the original AHEAD study), Hoppmann, Gerstorf, and Hibbert (2011) reported a baseline correlation of .32 between husbands’ and wives’ depressive symptom levels. A study of 553 older Mexican American couples documented an even higher correlation \( (r = .38) \) between spouses’ depressive symptomatology levels (Peek, Stimpson, Townsend, \& Markides, 2006).

Substantial covariation has also been reported in a multivariate context. For instance, a recent analysis involving 2,000 couples from HRS 2004 reported a standardized path
coefficient of .19 between the general depressive symptom levels of husbands and wives, after controlling for a number of covariates (Ayotte, Yang, & Jones, 2010). Tower and Kasl (1995) also reported that a significant relationship between husbands’ and wives’ depressive symptomatology levels was observed after controlling for known, individual-level risk factors (e.g., age, illness, impaired functioning).

To what extent are depressive symptoms correlated within married couples in which one or both partners suffer from pain? As with most other areas of research on pain in couples, very little is known about those couples in which both partners suffer from pain. The majority of what is known comes from research on couples in which one partner suffers from pain—most often couples in which one partner is a chronic pain patient. Even within this line of research, many studies do not have dyadic data on depressive symptomatology—i.e., data from both spouses on equivalent measures of depressive symptoms. Studies more commonly focus on the depressive symptoms of either the pain patient (e.g., Turk, Kerns, & Rosenberg, 1992) or the spouse (e.g., Sharp & Nicholas, 2000). In studies that do measure depressive symptomatology in both spouses, the observed association between symptomatology levels in both spouses is often not reported. For example, a study by Romano, Jensen, et al. (2000) had both pain patients and their spouses complete the CES-D; although descriptive statistics were reported for patient and spouse CES-D scores, no correlation was reported.

A limited number of studies have reported the association between symptoms of depression in chronic pain patients and their spouses, with most finding correlations ranging between .30 and .35. The magnitude of correlations appears consistent across studies using two of the most commonly used measures of depressive symptoms in pain.
studies—the Symptom Checklist-90 (SCL-90) depression subscale (e.g., $r = .35$ in Taylor, Lorentzen, & Blank, 1990) and the Beck Depression Inventory (BDI; e.g., $r = .31$ in Schwartz, Slater, Birchler, & Atkinson, 1991).

A few studies have documented much stronger correlations. In a small sample of 44 chronic pain patients and their spouses, patient and spouse scores on the SCL-90 depression subscale were correlated at .62 (Shanfield, Heiman, Cope, & Jones, 1979). In other studies, correlations in the moderate range were not statistically significant. For example, a study by Kerns and Turk (1984) found a moderate, but not statistically significant, correlation ($r = .31$) between patient and spouse depressed mood ratings in a small sample of 30 male chronic pain patients and their spouses at the VA. The extent to which this lack of statistical significance could be due to a lack of power (due to the small sample size) or the use of a single-item rating of depressed mood is unclear.

Cano and colleagues did administer measures of depressive symptomatology to both spouses in their study of community-dwelling couples in which (at least) one partner reported chronic musculoskeletal pain. However, their publications have not always indicated the extent to which spouses’ reports overlap. Leonard and Cano (2006) reported that mean levels of general depressive symptomatology on the MASQ (Mood and Anxiety Symptom Questionnaire) did not differ between the persons with chronic musculoskeletal pain and their spouses in the subset of 113 couples in their analyses; however, no correlation estimate was provided. Moreover, reported estimates have varied across studies—e.g., $r = .16$, ns ($N = 79$ couples; Johansen & Cano, 2007); $r = .24$, $p < .05$ ($N = 108$ couples; Pence et al., 2006). It is difficult to evaluate whether differences in estimates are attributable to differences in the subset of couples under
study or to other factors, such as variability in the measure of depressive symptomatology used (e.g., some Cano studies have combined two MASQ depression subscales into a composite scale, but Pence et al., 2006, used only the MASQ subscale of “non-specific” depressive symptomatology). In addition, the estimates reported to date by Cano and colleagues have been for the entire sample—i.e., all couples in the study, of which only 26-49% are comprised of partners who both reported chronic pain.

Covariation of depressive symptomatology has also been observed among couples in which one partner suffers from a chronic condition involving pain. One such study examined depressive symptom levels of 101 older women with osteoarthritis and their caregiving husbands (Druley, Stephens, Martire, Ennis, & Wojno, 2003). Patient and caregiver CES-D scores at Time 2 (6-months) were significantly correlated at a moderate level ($r = .39$). However, the correlation of spouses’ baseline scores was relatively weak ($r = .13$) and not statistically significant. Interestingly, these authors found that the relationship between patient and caregiver depressive symptomatology was influenced by the patient’s pain behaviors: patients with higher baseline depressive symptomatology who also displayed more pain behaviors had caregivers with increased depressive symptomatology at Time 2. In their study of 43 (predominantly female) rheumatoid arthritis (RA) patients and their spouses, Walsh et al. (1999) reported moderate correlations between spouses’ scores on the CES-D ($r = .43$) and also the SF-36 Mental Health composite scale ($r = .31, p < .05$). A more recent longitudinal study involving 133 patients with RA (mainly female) and their spouses reported correlations between spouses’ scores on a 9-item CES-D of .23 at baseline and .29 at one year; cross-wave correlations were of similar strength (Lam, Lehman, Puterman, & DeLongis, 2009).
Some mixed results have also been reported. A study of 117 chronic low back pain patients and spouses did not find a significant correlation on the Depression subscale of the Minnesota Multiphasic Personality Inventory (MMPI; Ahern, Adams, & Follick, 1985). However, patient depressive symptomatology was a significant predictor of spouse depressive symptomatology in multivariate analyses controlling for patient scores on other MMPI scales (anxiety, psychasthenia). Another study of chronic pain patients and spouses (Romano et al., 1989) reported a significant correlation between spouse and patient CES-D scores in male-patient dyads ($r = .30$), but not in female-patient dyads ($r = .04$). However, depressive symptom levels among male spouses were fairly low and may also have constituted a more restricted range of scores than those observed among male pain patients and female spouses.

Taken together, the findings of Druley et al. (2003), Ahern et al. (1985), and Romano et al. (1989), do raise the possibility that the relationship between depressive symptomatology levels in couples affected by the pain of one partner may not always be consistent or straightforward. These studies also provide a rationale for exploring potential factors that might influence the relationship between depressive symptom levels among spouses in the presence of pain. However, there is not yet a consistent body of literature that would identify conclusively which specific factors should be studied.

In summary, prior research has amply documented significant covariation of depressive symptomatology levels in married couples. The strongest and most consistent empirical support comes from studies of community-dwelling adults who may or may not have been affected by pain (e.g., Galbaud et al., 1994; Townsend et al., 2001). However, covariation of spousal depressive symptoms has also been observed among couples in
chronic pain clinics (e.g., Taylor et al., 1990), and has, to some degree, also been
documented in couples sampled on the basis of one partner having a painful disease (e.g.,
Druley et al., 2003) or (at least) one partner having chronic musculoskeletal pain (e.g.,
Pence et al., 2006).

Path 3: Covariation of Activity Limitation in Married Couples

As outlined in Chapter 2, activity limitation was conceptualized in the current
study as physical limitations—specifically, limitations in basic physical abilities and
activities, such as walking, climbing stairs, stooping/kneeling/crouching, and reaching or
grasping objects. Very few studies have examined spousal covariation of physical
limitations. These data are available: Large panel studies like AHEAD and HRS
routinely collect physical limitation data from respondents. Numerous research teams
have published analyses using the data from couples who participated in these surveys.
To date, however, attention has been directed more toward outcomes like depressive
symptomatology (e.g., Tower & Kasl, 1995). Although some studies have used physical
limitation data from spouses, published reports do not include estimates of spouse
covariation on these measures (e.g., Siegel, Bradley, Gallo, & Kasl, 2004). Only an
unpublished study by Townsend colleagues reported an estimate of spousal covariation
on a measure that was specific to basic physical limitations. An intraclass correlation of
.28 was observed in a sample of 2,000 married White couples drawn from the first wave
of the HRS study (Townsend, Miller, & Min, 2001). Although these authors used only
items specific to lower-body functioning (e.g., stooping, climbing stairs, walking one
block), their study provides one of the only available estimates of covariation between
physical limitations of community-dwelling spouses.
Dozens of studies have been conducted with samples of couples selected on the basis of one spouse suffering from a painful disease or condition such as arthritis or cancer. Few of these studies assess physical limitations in both spouses. Similar to the pattern observed within research on pain, data collection is often concentrated on the partner who has the disease with minimal data collected from his/her spouse. A smaller subset of studies has focused on the (presumably) healthy spouses. Few studies collect comparable data from both partners, especially data specific to physical limitations. One exception is the study of 43 rheumatoid arthritis patients and their spouses by Walsh et al. (1999). Moderate correlations were observed between spouses’ scores on SF-36 scales of physical functioning ($r = .33, p < .05$) and role limitations due to problems with physical health ($r = .31, p < .05$). It is notable that the physical functioning scale of the SF-36 is comprised mainly of basic physical abilities (e.g., climbing stairs, walking a certain distance, stooping/crouching).

This author is not aware of any published studies that have examined the extent to which physical limitations covary between persons with chronic pain and their spouses, whether sampled from the community or from clinical settings. This observation holds, even if the definition of physical limitations is relaxed to accommodate the wide variety of limitations, functions, restrictions, and disabilities that have been conceptualized by some researchers as activity limitation (e.g., IADLs, ADLs, functional limitations/disability, physical limitations/disability; as discussed in Chapter 2). Cano and colleagues have frequently examined physical disability in their studies of “chronic pain couples” (i.e., couples in which one spouse reported chronic musculoskeletal pain). However, their publications to date (e.g., Cano et al., 2005; Geisser, Cano, & Leonard,
2005) have considered only the physical disabilities and/or limitations of the designated partner with pain (reports of which may have been collected from the person with pain or his/her spouse or both).

A larger number of studies have examined spousal covariation with respect to activity limitation, more broadly defined. Most prior research has been focused within community-residing couples. As an example, Peek et al. (2006) reported a correlation of .34 for a measure of “disability severity” between spouses in 500 Mexican American couples. In their study, disability severity was operationalized as a count of ADL and IADL difficulties. Correlations of slightly smaller size (< .25) have been reported on similar measures of functional difficulty between spouses in HRS (e.g., Wilson, 2002).

Not all prior studies have found evidence of significant spousal covariation when activity limitation has been more broadly defined. In their analysis of over 1,700 couples in the original AHEAD study, Hoppmann et al. (2011) reported a baseline correlation of only .08 (p > .05) between husbands’ and wives’ scores on a multi-item measure of functional limitations. The measure combined a handful of items assessing limitations in basic physical abilities (e.g., walking, climbing stairs, pushing/pulling objects) with an equal number of items related to IADL limitations (e.g., shopping, managing money, preparing hot meals). Another recent study utilized data from 400 couples collected as part of a large, longitudinal sample of community-dwelling adults in a diverse Florida county, with purposive over-sampling of individuals with a physical disability. Several different measures of activity limitation were collected from both spouses, including limitations in ADLs, IADLs, and basic physical abilities. Because of generally low rates of endorsement across most measures, dichotomous indicators were constructed to signal
the presence of different types of limitation/disability. Spousal concordance for limitation/disability was generally low, ranging from < 1% of couples in which both partners reported ADL or IADL limitations to 3.8% of couples in which both partners reported any activity limitation (across the whole set of measures, including physical abilities). The author did note, however, that levels of concordance were higher among older couples (Andress, 2010, unpublished dissertation). Findings from these recent studies suggest that estimates of spousal covariation activity limitation may be affected by the age and general health of the sample, as well the specific way in which activity limitation is measured.

In summary, there is limited evidence for the covariation of physical limitations between spouses. However, this lack of evidence mainly reflects a dearth of empirical study of this issue, rather than findings that fail to support the existence of such covariation. This assessment is bolstered by findings from several recent reviews of the empirical literature focused on the health of older couples (Hoppmann & Gerstorf, 2009; Meyler, Stimpson, & Peek, 2007; Walter & Lucszc, 2009). A majority of studies has focused on mental health. Definitions and measures of physical health have varied so widely across studies that few specific conclusions can be drawn from the existing evidence base (Meyler, Stimpson, & Peek, 2007).

**Path 4: Pain in One Spouse Affects Psychological Distress in His/Her Partner**

*Psychological Distress in Partners of Persons With Pain*

The existing evidence base suggests that pain, especially pain that persists or is chronic, generally has a negative impact on the family and its members (Otis, Cardella, & Kerns, 2004; Roy, 2001). Most empirical research to date has focused on the effect of
pain on spouses, probably because of their emotional proximity to, and high levels of interaction with, the person with pain. In general, studies of the impact of chronic pain on spouses have tended to focus on three broad effects: (a) marital dissatisfaction; (b) physical health; and (c) psychological distress, especially dysphoric or depressed mood (Turk, Flor, & Rudy, 1987). Available research suggests that many (though not all) spouses show some level of negative effects in all three areas—higher marital dissatisfaction, higher physical health problems, and elevated depressive symptoms and emotional distress (e.g., Flor, Turk, & Scholz, 1987; Romano, Turner, & Clancy, 1989; and Schwartz, Slater, & Birchler, 1996).

Studies have shown that the spouses of chronic pain patients tend to show elevated levels of psychiatric symptomatology in general (e.g., Ahern & Follick, 1985; Saarijarvi, Hyypa, Lehtinen, & Alanen, 1990; Saarijarvi, Rytokoski, & Karppi, 1990). For example, both patients’ and spouses’ scores on most SCL-90 subscales have been found to be significantly higher than the scores of normative non-patient samples (e.g., Shanfield et al., 1979; Taylor et al., 1990). However, neither patients nor spouses typically display levels of psychiatric symptomatology as elevated as those found in psychiatric patient comparison groups (e.g., Taylor et al., 1990).

In some studies, the level of psychological or emotional distress evidenced by the spouses of chronic pain patients exceeds the level of distress displayed by the spouses of persons suffering from chronic illnesses. For example, in a pilot study of chronic pain patients and their spouses, Subramanian (1991) found that spouses’ psychosocial distress levels (as measured by the Psychosocial Adjustment to Illness Scale) were significantly higher than those observed in the studies of the spouses of cancer patients.
Not all studies have found elevated rates of emotional distress in the spouses of persons affected by pain. For example, in a sample of 40 metastatic cancer patients and their spouses, Dar and colleagues (Dar, Beach, Barden, & Cleeland, 1992) reported that both patients and spouses evidenced low levels of mood disturbance (as measured by the Profile of Mood States [POMS]). A spouse’s reaction to pain in the partner may not always be manifested as florid psychological distress. Symptoms might be more subtle, such as chronic feelings of sadness (Roy, 2001, Chapter 5).

Interestingly, there have been no published studies documenting the rate of major depression among spouses of chronic pain patients (Schwartz & Edhe, 2000). However, numerous studies have examined depressive symptomatology among spouses. It is noteworthy that although some studies measure depressive symptomatology in spouses, publications do not always present descriptive data about spouses’ scores on these measures. For example, Turk et al. (1992) used the spouse’s CES-D score as a dependent variable in several regression models, yet provided no descriptive data.

In general, rates of depressive symptomatology in the spouses of chronic pain patients have been found to be higher than the rates of normative, non-patient populations (e.g., Taylor, Lorentzen, & Blank, 1990). In some studies, the spouses evidence even higher depressive symptom levels than do the chronic pain patients themselves. One study of chronic pain patients at the VA found that 28% of spouses scored in at least the mildly depressed range, whereas only 20% of patients scored at or above this range (Schwartz et al., 1991).

Most studies estimate that between 20% and 40% of spouses of persons with chronic pain experience significant depressive symptomatology. These estimates appear
fairly consistent, despite variability how depressive symptomatology is measured, the types of pain problems of patients, and the size of the samples. For example, in a sample of 117 predominantly male, chronic low back pain patients and their spouses, Ahern, Adams, and Follick (1985) found that 20% of spouses scored in the “distressed” range on the MMPI Depression Scale. Studies using the BDI have documented rates ranging from 23% in a sample comprised mainly of the male spouses of female chronic pain patients (Capitolo, 1998) to 26% and 28% among female spouses of VA pain patients (Flor, Turk, & Scholz, 1987, and Schwartz et al., 1991, respectively). Using the CES-D, Romano and colleagues found clinically significant depressive symptoms in 21% of spouses of chronic low back pain patients ($N = 83$, Romano et al., 1989) and 35% of spouses of chronic musculoskeletal pain patients ($N = 121$, Romano et al., 2000).

As a group, these studies also appear to yield fairly consistent estimates of the rate of depressive symptomatology, regardless of the specific gender composition of the samples under study. However, possible gender differences have not been well studied. Only Romano et al. (1989) specifically tested hypotheses related to gender; they found that, although female spouses scored an average of two points higher than male spouses on the CES-D, the difference was not statistically significant.

A study by Kerns & Turk (1984) reported a substantially higher estimate of depressive symptomatology: Over 50% of spouses reported “significant” levels of depressed mood, assessed as depressed mood over the past week rated on a visual analogue scale. The procedure for estimating “significant” depressed mood was not reported. The higher estimate reported in this study may be at least partially attributable to the unique measure used in the study, or to the relatively small sample ($N = 30$). In
addition, the spouse sample was almost entirely comprised of wives, and evidence shows that women tend to report more depressive symptoms in general than men (Blazer, 2002). It is also possible that distress levels in this sample were unusually high; in support of this assessment, over half of the male patients also reported significant depressed mood.

There are few published studies documenting levels of depressive symptomatology in the spouses of persons suffering from pain who are not labeled “chronic pain patients.” Several studies of couples in which one partner suffers from a painful condition such as arthritis, osteoporosis, or cancer have not reported information about the levels of emotional distress displayed by spouses or caregivers (e.g., Cremeans-Smith, Stephens, Franks, Martire, Druley, & Wojno, 2003; Roberto & Gold, 1997; Williamson, Robinson, & Melamed, 1997). Other studies have reported only limited descriptive data about spouses’ scores on measures of depressive symptomatology (e.g., Miaskowski et al., 1997).

More recent studies have reported on the psychological distress levels in caregivers to persons who (are presumed to) suffer significant pain, but who are not labeled “chronic pain patients.” Most of the caregivers were spouses. The study by Druley et al. (2003) examined the levels of depressive symptomatology displayed by the husbands of female osteoarthritis outpatients. In this longitudinal study, 7% of spouses at baseline, and 18% of spouses at 6 months had scores above a standard clinical cutoff on the CES-D. These rates are somewhat lower than those found in the studies of spouses of chronic pain patients. It is noteworthy that this spouse sample was comprised of all males. In contrast, Jacobi et al. (2003) found that 22% of the caregiving partners (84% were male) of patients with rheumatoid arthritis reported moderate or extreme problems
with “depression or anxiety” (measured as a mixed construct). Further research is needed to document the levels of depressive symptoms reported by the spouses of persons who suffer from pain, but who are not considered chronic pain patients.

This review has established that a sizable proportion of spouses/partners of patients with pain experience significant levels of psychological distress, including depressive symptomatology. The majority of this research has been conducted with samples drawn from specialty pain clinics, although some studies have been done with caregivers to patients recruited from disease-focused clinics (e.g., arthritis, cancer). This review failed to locate any published studies using community-based, non-clinical (or non chronic pain-identified) populations. Thus, the extent to which the spouses of community-dwelling, late middle-aged and older adults affected by pain display symptoms of depression is unknown. In addition, although a few studies in both the chronic pain literature and the literature focused on specific disease conditions are suggestive of possible gender differences, gender effects have not been well-studied.

**Evidence of a Relationship Between One Spouse’s Pain and His/Her Partner’s Psychological Distress**

One important issue neglected by many researchers who examine depressive symptomatology in the spouses of persons suffering from pain is: To what extent can spousal psychological distress be tied to the patient/partner’s pain? There is clearly an assumption within the chronic pain literature that spouse psychological distress is influenced by the patient’s experience of pain. And, published studies are replete with anecdotal evidence of a direct association between patient pain and spouse psychological distress. For example, 83% of spouses in one study attributed their psychological
symptoms directly to their partner’s chronic pain. These spouses also reported feelings of uncertainty and helplessness related to their partner’s pain (Rowat & Knafl, 1985). Similar reports are recorded in studies of spouses of patients dealing with pain related to cancer and other diseases. Spouses often see their psychological distress as related to the patient’s pain and commonly report feeling “frustrated” and “helpless” (Ferrell, Rhiner, et al., 1991). Cancer caregivers frequently report that the patient’s pain causes the patient and the family considerable distress and is a significant source of emotional burden or strain for them (e.g., Ferrell, Grant, Chan, Ahn, & Ferrell, 1995). Similarly, husbands whose wives suffer from pain symptoms associated with osteoporosis report that seeing their wives in pain is emotionally difficult for them (Roberto, Gold, & Yorgason, 2004).

However, the assumption that one partner’s pain is related to psychological distress in the other partner is not often tested empirically. In some cases, this oversight reflects researchers’ attention to other focal relationships: for example, the relationship between marital satisfaction and psychological distress in both pain patients and spouses (e.g., Saarijarvi, Rytokoski, & Karppi, 1990), the relationship between concordance in perceptions of one partner’s pain and perceptions of spousal support (e.g., Roberto & Gold, 1997), and congruence in ratings of patient pain and mood disturbance (e.g., Miaskowski, Zimmer et al., 1997). In congruence studies, however, it is generally the concordance (or, most often, lack of concordance or extent of disagreement) of spousal ratings that is the predictor of interest; most do not even examine the bivariate relationship between the pain intensity ratings themselves and spouse or patient outcomes. This oversight could also reflect restrictions imposed by the populations under study. Insofar as most studies have been restricted to chronic pain patients and their
spouses, comparisons of depressive symptomatology levels between spouses of persons with pain and without pain are not possible (although scholars have specifically identified a need for such comparative studies; see Turk et al., 1987, for a discussion of this issue).

A few studies have empirically tested the association between one partner’s pain intensity and the depressive symptomatology (or, more generally, emotional distress) of his/her spouse. Two chronic pain studies are often cited as providing evidence of a relationship between patient pain intensity and spouse depressive symptomatology. In their small sample of low back pain patients at the VA, Schwartz et al. (1991) found that average patient pain intensity levels were strongly related to spouse depressive symptomatology levels ($\beta = .36$), even after controlling for factors such as patient anger and hostility, patient’s level of physical difficulties, and the spouse’s level of marital satisfaction. Kerns and Turk (1984) also found moderate to strong correlations between patient pain intensity and spouse depressive symptomatology in a sample of VA patients and their spouses. Both studies had relatively small samples (i.e., 30 or fewer couples). Both samples also were comprised of male VA patients and their wives. In their study of 130 RA patients and spouses, Lam et al. (2009) reported small to moderate bivariate relationships between RA patient “disease activity” (a measure that predominantly represented pain intensity, but also assessed number of specific joints with pain) and spouse depressive symptomatology.

Several studies have not found a significant association between patient pain intensity and spouse depressive symptomatology. These include two studies of chronic pain patients (Capitolo, 1998; Flor, Turk, & Scholz, 1987), and two studies of older, community dwelling couples—the study of older wives with osteoarthritis by Druley et
al. (2003) and the study of couples in which one spouse suffered from chronic musculoskeletal pain by Leonard (2004). Both Flor et al. (1987) and Capitolo (1998) had small samples ($N = 60$ couples and $35$ couples, respectively); but, small sample size is an unlikely explanation, given that strong relationships were observed in the two VA studies cited above. The pain patients in the study by Flor et al. were male VA patients, while the Capitolo patients were predominantly female (86%); thus, gender does not appear to account for differences between the two groups of studies. However, Flor et al. (1987) did not report any bivariate results; the relationship between patient pain intensity and spouse depressive symptomatology was assessed only in a multivariate model that also included patient depressive symptoms, activity limitation, and coping, as well as spouse perceptions of control and marital satisfaction. In addition, as has been observed by several authors—within chronic pain patient samples, the range of patient pain intensity may not vary enough to permit an examination of the relationship between pain intensity and spousal psychological distress (e.g., Roy, 2001, Chapter 5; Snelling, 1994).

Variability in terms of whose report of the patient’s pain intensity is used may also help to explain some inconsistent findings. Most prior studies have used the patient’s report of his/her own pain intensity. Others have used the spouse’s report of the patient’s pain. For example, Capitolo (1998) found the spouse’s rating of patient pain severity was not a significant predictor of spouse scores on the BDI. The findings of Kerns and Turk (1984) also suggest that the relationship between patient pain and spouse depressive symptomatology may differ depending on whose report of pain intensity is used. Although patient and spouse ratings of the patient’s pain intensity were correlated ($r = .37, p < .05$), only the spouse’s pain intensity rating for the patient was significantly
related to the spouse’s depressed mood ($r = .48, p < .001$ vs. $r = .32, p > .05$ for patient’s own rating). The sample size was quite small, however ($N = 30$), and the lack of statistical significance for the relationship between the patient’s own pain rating and the spouse’s depressed mood may have been due to a lack of statistical power.

Some studies have used both the patient and spouse ratings of the patient’s pain intensity and found neither to be significantly related to depressive symptom levels in the spouse. For example, in Leonard and Cano’s (2006) sample of 113 couples in which one spouse suffered from chronic musculoskeletal pain, the bivariate correlations observed between the partner’s depressive symptomatology and the pain intensity of the patient as rated by the patient and by the spouse were both fairly small ($r = .17$ and .18, respectively). This finding was interesting in light of the strong level of agreement observed between the spouses’ ratings of patient pain intensity ($r = .49$). However, the authors did find evidence of a more complicated relationship between patient pain intensity and spouse depressive symptomatology—one involving marital satisfaction. Patient pain intensity was significantly related to spouse depressive symptomatology, but only among those spouses who reported high levels of marital satisfaction.

The lack of an observed relationship between one spouse’s pain intensity and the other’s depressive symptomatology in the Druley et al. (2003) study is less easily explained. In this study of older female osteoarthritis patients and their husbands, patient baseline pain intensity was not significantly correlated with either the baseline or 6-month follow-up depressive symptom levels of the husband. The authors did note that levels of depressive symptomatology reported by husbands were somewhat low.

There is also the possibility that gender differences might exist in the cross-spouse
relationship between pain intensity and spousal depressive symptom levels. Yet, without comparable data from studies with equal male-to-female ratios of patients and spouses, it is impossible to evaluate gender as a possible explanation for the lack of significant findings observed in some studies. It is also difficult to detect a specific pattern related to gender across the set of studies reviewed here. For example, although Druley et al. did not find a relationship between patient pain intensity and the depressive symptomatology of spouses in their all-female osteoarthritis sample, a significant relationship was observed in Lam et al. (2009)’s sample of (predominantly female) rheumatoid arthritis patients. The issue of possible gender differences is revisited later in this chapter.

**Path 5 and Mediation: Indirect Pathways Through Which Pain in One Spouse Might Affect Psychological Distress in His/Her Partner**

There are probably several mechanisms through which pain in one spouse may be related to the psychological distress of the other partner (e.g., pain may impact roles within the marital relationship, pain may diminish the ability of partners to provide emotional support, pain may reduce expressions of intimacy, etc.). Given that this study made use of an existing dataset constructed for other purposes, the ability to test a variety of mechanisms underlying any observed cross-spouse effects of pain was limited. However, study data did permit examination of one possible pathway through activity limitation (Path 5 in Figure 5). In general, Path 5 argues for the mediating role of the spouse’s activity limitation in the relationship between one spouse’s pain and his/her partner’s depressive symptomatology.

It is challenging to identify studies that have specifically examined activity limitation as a mediator of the relationship between one spouse’s pain and the other’s
depressive symptomatology. Part of this difficulty arises from the sheer variety of terms that researchers have used to refer to the difficulties in physical functioning experienced by persons with pain. As was discussed in Chapter 2, such difficulties have been labeled functional limitations, activity restrictions, functional impairments, physical functional difficulties, and all manner of “disabilities,” “difficulties,” and “interference” (e.g., ADL, IADL, mobility, lower body, and upper body disabilities). Even researchers focused on chronic pain in couples have been inconsistent in their use of terminology. Some of the terms used in chronic pain research are highly specific and not easily reconciled with similarly-named constructs in the gerontological or public health literatures—for example, within clinically- and vocationally-oriented publications, “disability” due to chronic pain most often refers to work-related disability.

It can also be challenging to interpret and compare the results of relevant studies. Most difficulties are tied to the use of measures that have been developed specifically for research and clinical work with chronic pain patients. In general, these measures are extremely detailed and generate volumes of data about the chronic pain patient’s condition and circumstances; most also have vast literatures attesting to their dimensionality, reliability, validity, and utility. However, in practice, researchers studying couples and pain (like many other pain researchers) tend make use of global summary scores—for instance, a patient’s total “pain score” on the McGill Pain Questionnaire, or a “composite score” from the West Haven-Yale Multidimensional Pain Inventory. One or more activity limitation items are typically embedded within these summary scores. This makes it difficult to isolate the effect of activity limitation. It can be equally challenging to assimilate the results of studies in which researchers utilize a
single activity limitation item (often derived from a multidimensional pain measure) that is described only in generic terms. In many studies, the underlying construct is labeled using the wording of the item, resulting in a measure of “pain impact” or “pain interference.” Without knowledge of the specific measure that was used, it can be difficult to determine whether this “impact” is limited to a specific area of functioning (e.g., physical, psychological, social, work-related) or if it represents an overall assessment of the effects of pain on one’s life.

Finally, as was mentioned earlier in this chapter—especially within the chronic pain literature, there has been a general lack of attention to spouses. Even studies focused specifically on couples demonstrate a lack of interest in understanding the mechanisms through which partners could be negatively affected by a spouse’s pain. More often, interest is in effects moving in the opposite direction (i.e., from the spouse’s behavior or reaction to pain in the partner to an outcome in the partner with pain). Lack of interest in attempting to elucidate potential intervening mechanisms may also represent a natural outgrowth of a perspective identified earlier—that the patient’s pain is assumed to influence levels of psychological distress in the spouse—the patient’s activity limitation may similarly be assumed to contribute to spousal psychological distress. As a consequence, the study of intervening mechanisms may be considered irrelevant.

There are two important exceptions to this apparent lack of interest in intervening mechanisms. First, there is a fairly well-developed body of literature that has focused on marital adjustment and marital satisfaction within couples affected by pain (e.g., Cano, Gillis, Heinz, Geisser, & Foran, 2004; Romano, Turner, & Jensen, 1997; Subramanian, 1991; Saarijarvi, Rytokoski, & Karppi, 1990; Turk et al., 1992). Within this literature,
some studies have explored how problems in marital functioning or low levels of marital satisfaction may help to explain the relationship between one partner’s pain and the psychological distress of the spouse (e.g., Leonard, 2004; also see review by Leonard, Cano, & Johansen, 2006). Second, there is growing interest in examining perceptions of pain within the marital dyad. Several studies have explored the extent to which spouses are congruent in their perception of pain-related constructs and how congruence (or incongruence) affects their emotional well-being (e.g., Cano et al., 2005; Cremeans-Smith et al., 2003; Riemsma et al., 2000; Roberto & Gold, 1997). Neither marital functioning nor perceptual congruence regarding pain was of interest in the present study.

This author identified only one published study that provided data regarding the role of activity limitation in mediating the relationship between one spouse’s pain and the depressive symptomatology of his/her partner. Flor, Turk, and Scholz (1987) presented a multivariate model that predicted the wife’s depressive symptomatology from several variables, including the husband’s pain intensity and the extent to which pain limited his activities. In this model, neither his pain intensity nor his pain-specific activity limitation was a significant predictor of the wife’s depressive symptomatology. The fact that the husband’s activity limitation was not significantly related to his wife’s depressive symptomatology in a model that also included his pain intensity suggests that his activity limitation did not function as a mediator in this study. However, it is impossible to draw a substantive conclusion regarding possible mediation from this study given that (a) the bivariate relationship between the husband’s pain intensity and his wife’s depressive symptomatology was never reported, and (b) the model contained other predictors, including the husband’s level of depressive symptomatology and pain coping behaviors,
as well as the wife’s perceptions of control and level of marital satisfaction. Most notably, the model included both the patient’s and his wife’s ratings of the extent to which his pain “interfered with” his usual activities.

In the absence of studies offering an explicit test of the extent to which activity limitation mediates the cross-spouse relationship between pain and depressive symptomatology, indirect support for mediation can be culled from studies that have examined one or more steps necessary to demonstrate mediation. The preceding section reviewed existing evidence regarding the relationship between one spouse’s pain and the depressive symptomatology of his/her partner (i.e., Path 4). Chapter 2 reviewed evidence regarding the *intra*-individual relationship between pain and activity limitation. Thus, the most relevant evidence in the current context is evidence of a relationship between the spouse’s activity limitation and his/her partner’s depressive symptomatology (irrespective of the spouse’s pain intensity or severity).

Within the literature specific to pain in married spouses, there exists modest support for the existence of a relationship between one partner’s activity limitation and his/her partner’s depressive symptomatology. The strongest support comes from the study of chronic back pain patients by Romano et al. (1989). These authors observed a relatively strong relationship \( r = .45, p < .01, N = 41 \) between the patient’s activity limitation (as measured by a modified version of the Sickness Impact Profile [SIP]) and the spouse’s depressive symptomatology (as measured by the CES-D). However, this relationship was only observed in male-patient dyads, and only for the spouse’s perception of the patient’s activity limitation. Sharp & Nicholas (2000) also found a sizable relationship \( r = .42 \) between the activity limitation of chronic pain patients and
the depressive symptomatology levels of their spouses. However, the study used only the *spouse’s* rating of the patient’s activity limitation; moreover, the questions specifically tapped the extent to which the patient’s pain was interfering with the spouse’s own life.

More recently, Lam et al. (2009) examined the relationship between the physical limitations of RA patients and the depressive symptomatology of their spouses. Significant bivariate correlations between these constructs were found both between and within the two-wave study; correlations were in the small to moderate range (.18 to .28). In their study, Lam et al. assesses physical limitations using a measure called the DASH (Disabilities of Arm, Shoulder, and Hand). Thus, their findings were specific to upper-body physical limitations. Clearly, these prior studies offer only qualified support for a cross-spouse relationship between activity limitation in a spouse with pain and the depressive symptom levels of his/her partner.

Several other studies have generally failed to find a significant relationship between activity limitation in the pain patient and depressive symptomatology in the partner. Most of these studies emanate from the chronic pain literature. Examples include a small study of male back pain patients at the VA (Schwartz et al., 1991) and a larger study of back pain patients of both genders (Ahern, Adams, & Follick, 1985). Both of these studies also used the SIP to measure activity limitation; however, unlike Romano and colleagues, the authors did not indicate whether the measure was adapted to be specific to the “impact” or limitations caused by pain. Another study using the SIP in a small sample of chronic pain patients did find a strong relationship between patient SIP scores and spouse psychological distress ($r = .51$); however, this relationship only existed for the SIP subscale measuring psychosocial dysfunction/difficulties, and not for the
subscale measuring physical dysfunction/difficulties (Subramanian, 1991).

Given the relative paucity of empirical studies evaluating the role of the spouse’s activity limitation in mediating the cross-spouse relationship of pain to depressive symptomatology, can supporting evidence be culled from studies outside of the literature on pain? Support here is mixed, as the findings seem to vary widely. Unfortunately, variation in findings does not seem to be related to any particular feature of the studies. The studies have varied widely in terms of the nature of the samples (e.g., couples drawn from disease-specific clinical settings vs. community-dwelling couples), the size of the samples (e.g., thousands of couples vs. less than a hundred), the measures of activity limitation (e.g., focused mainly on IADLs or ADLs vs. focused on basic physical abilities), and the measures of psychological distress (e.g., specific to depressive symptoms vs. more general symptoms of distress), along with whether or not other constructs (and which other constructs) were included in the assessment of a relationship between one spouse’s activity limitation and the other’s psychological distress. A few examples illustrate both the diversity of these studies and their findings.

In their study of over 5,000 couples in HRS waves 1 and 2, Siegel et al. (2004) found that higher levels of a spouse’s physical limitations (at baseline) were associated with worsened depressive symptomatology in the partner (at two-year follow-up) in multivariate models controlling for the respondent’s own health and other demographic variables. However, this relationship did not hold when other variables (including the spouse’s baseline depressive symptomatology and change in depressive symptomatology between waves) were added to the model; unexpectedly, an increase in the physical limitations in the spouse was associated with improved partner depressive
symptomatology at follow-up. A study of patients recovering from a heart attack and their caregivers (mainly spouses) did not find a significant relationship between the activity limitation of the patients and the psychological distress of the caregivers (Kahana, Young, Kercher, & Kaczynski, 1993).

Looking more broadly at the available literature on cross-spouse effects in middle-aged and older couples, there is considerable evidence that physical health problems or concerns in one spouse are related to the emotional well-being of the other spouse. Such evidence comes from a variety of different areas of research, but only selected examples from studies of community-dwelling adults are reviewed here. In a large, random sample of community-dwelling adults, Salokangas and Poutanen (1998) found that the poor health status of one’s spouse was a risk factor for increased depressive symptomatology, even after controlling for the effects of one’s own poor physical health, lower SES, negative life events, work problems, problems with alcohol, poor marital functioning, and other poor interpersonal relationships. In studies using data from middle-aged and older community-dwelling couples in California, Strawbridge and colleagues have documented a variety of cross-spouse effects, including the negative impact of one spouse’s sleep problems (Strawbridge, Shema, & Roberts, 2004), hearing loss (Wallhagen, Strawbridge, Shema, & Kaplan, 2004), and vision impairment (Strawbridge, Wallhagen, & Shema, 2007) on the other partner’s depressive symptomatology and general mental health. Additionally, cross-spouse effects in these studies have been observed even after adjusting for the partner’s own relevant predictors (e.g., the partner’s own sleep or sensory impairment).

In summary, there is lack of evidence regarding the cross-spouse mediation of
pain by the spouse’s activity limitation—especially when conceptualized as physical limitations. This lack of evidence generally reflects a lack of empirical study, rather than findings that discount the existence of such mediation. The rationale for examining activity limitation (specifically physical limitations) as a potential mediator of the cross-spouse pain-depressive symptomatology relationship is four-fold. First, as reviewed in Chapter 2, there is both theoretical and empirical support to suggest that activity limitation at least partially mediates the pain-depressive symptom relationship at the intra-individual level. Second, although the evidence is somewhat limited and some contradictory findings do exist, one spouse’s pain does appear to be related to the depressive symptomatology of his/her partner (Path 4); but this association has not been adequately explained by existing theory or empirical data. Third, as reviewed above, there is some (albeit limited and often qualified) empirical evidence that is suggestive of a relationship between one spouse’s activity limitation (broadly defined) and the other spouse’s psychological distress. Fourth, as just reviewed, there is solid empirical support that one spouse’s physical health concerns and problems are associated with depressive symptomatology in the other spouse. On the basis of these lines of evidence, it seemed reasonable to extend Williamson’s ARM (discussed in Chapter 2) to the level of the marital dyad, and to investigate activity limitation as a possible mediator in the relationship between one spouse’s pain and the other’s depressive symptomatology.

Potential Moderators of the Cross-Spouse Effects of Pain in the Marital Dyad

Interest in the possible moderation of cross-spouse relationships involving pain and depressive symptomatology within the marital dyad was justified primarily on the
basis of some of the inconsistent and/or unexpected findings reviewed above (e.g., Druley et al., 2003; Romano et al., 1989; Siegel et al., 2004). Additional justification can also be drawn from arguments presented in Chapter 2 to support the exploration of factors that might influence the *intra*-individual relationship between pain and depressive symptomatology. In the proposed conceptual model, several factors could potentially moderate the cross-spouse effects of pain. Such constructs could be located at either the individual or the couple level. Given the lack of prior research in this area, however, the current study limited consideration to one potential moderator at each level. As discussed below, existing evidence regarding moderation is limited to the direct cross-spouse effects of pain, and even that evidence is relatively sparse and largely suggestive. Hence, research questions around potential moderation effects are mainly exploratory in nature.

**Gender and the Cross-Spouse Effects of Pain on Psychological Distress**

As was the case with the *intra*-individual relationship between pain and psychological distress, there has been relatively little empirical research investigating possible gender differences in the cross-spouse effects of pain. There is some limited evidence that females may be more sensitive to, and more affected by, the pain and/or activity limitation experienced by their male partners. As noted earlier, Romano et al. (1989) found that the relationship between patient activity limitation and spouse depressive symptomatology was stronger in male-patient couples than in female-patient couples; in addition, depressive symptomatology levels were more highly correlated in male-patient couples than in female-patient couples. However, depressive symptomatology levels in the male spouses in that study were fairly low and may have constituted a restricted range. Additionally, not all studies that have explicitly examined
There appears to have been a resurgence of interest in possible gender differences in cross-spouse relationships involving pain within the last ten years. This interest seems to have prompted many researchers to move in one of two directions: (a) conducting comparative studies of male and female spouses of pain patients; or (b) limiting empirical investigations to patient-spouse pairs of a particular gender combination (e.g., female patients-male spouses). Chronic pain researchers have tended to adopt the former approach. Consistent with the behavioral theories that have historically dominated research with chronic pain populations, these comparative studies tend to focus largely on gender differences in spouse responses (e.g., negative or punishing reactions vs. solicitousness) to the patient’s pain (e.g., Fillingim, Doleys, Edwards, & Lowery, 2003; Smith, Keefe, Caldwell, Romano, & Baucom, 2004).

Researchers studying persons with couples in which one spouse has disease-specific pain and those studying community-dwelling couples in which one partner reports pain have generally adopted the latter approach—i.e., using samples with a specific gender constellation. This approach presumably reflects increased recognition of gender differences in pain (and in related outcomes) at the intra-individual level; it may also represent an attempt to remove gender variability as a source of potential differences in terms of the cross-spouse effects of pain. Examples of this line of research include studies of older women with osteoporosis and their husbands (e.g., Roberto, Gold, & Yorgason, 2004), several studies involving female osteoarthritis patients and their male
spouses (e.g., Cremeans-Smith et al., 2003; Druley et al., 2003), and studies of fibromyalgia patients and their husbands (e.g., Bigatti & Cronan, 2002). That a majority of these studies involve female patient-male spouse pairs suggests the possible influence of gender disparities in disease prevalence or perhaps increased attention to women’s health issues.

The relevant findings from studies representing both approaches have already been reviewed in this chapter. And, as noted, some findings are suggestive of possible gender differences (e.g., Druley et al., 2003; Romano et al., 1989). However, the existing evidence base is clearly weak.

Indirect support for possible gender differences in the cross-spouse effects of pain may exist within the broader body of pain research. Some research suggests that females may be more aware of, or attuned to, pain in others. For example, studies have found that females tend to recall or report a greater number of family pain models (i.e., family members who suffered pain problems) than do males (e.g., Koutantji, Pearce, & Oakley, 1998). Beaupre et al. (1997) found higher levels of agreement in the ratings of the intensity of an osteoarthritis patient’s pain among male-patient dyads than among female-patient dyads, suggesting that female spouses may be better at judging the intensity of their partner’s pain than males.

Additional indirect support also may be found within studies of gender differences in the context of marriage and other relationships, as well as theoretical efforts to increase understanding of what appear to be basic differences between men and women in the ways in which they view the world. Several leading feminist theorists have suggested that women are more relationship-oriented than men (e.g., Chodorow, 1978;
Gilligan, 1993). Relationships appear to be valued more by women and considered more central to their self-definitions (Oyserman & Markus, 1989). Women also tend to view other’s stressors as their own (Wethington, McLeod, & Kessler, 1987), and to feel increased responsibility for the well-being of others (Thompson & Walker, 1989). When considered within the context of marriage, some of these differences might explain why women appear more likely to assume the role of monitor and controller of the health-related behaviors of family members (Rook, 1995).

Studies of emotional expression and reactivity also suggest that women may be more reactive and susceptible to emotional contagion than are men (e.g., Doherty, Orimoto, Singelis, Hatfield, & Hebb, 1995). Within the context of marriage, several studies report that husbands’ negative emotions more reliably predict wives’ negative emotions than the converse (Larson & Almeida, 1999; Notarius & Johnson, 1982; Roberts & Krokoff, 1990). Studies have also found that wives are generally more emotionally reactive to marital disagreements than husbands (e.g., Almeida et al., 2002).

Findings from some studies suggest that wives may be more affected by, or “sensitive to,” the health status of their spouses than are husbands. In a study of 120 middle-aged and older couples, Quirouette and Gold (1992) found that the husband’s level of marital satisfaction, his well-being and positive orientation toward life, and his physical health significantly predicted the psychological well-being of the wife. However, the psychological well-being of the husband was most strongly associated with his own physical health, and variables related to his wife were not significant predictors of his psychological well-being. Simonsick (1993) found that the physical health status of the husband was significantly associated with the emotional well-being (specifically,
depressive symptom levels) of older married women. Moreover, the magnitude of the association was similar to the effect of the wife’s own physical health status.

Gender differences have also been observed in Wallhagen and colleagues’ studies of the effects of one spouse’s sensory impairment on the well-being of the other (Wallhagen et al., 2004; Strawbridge et al., 2007). In their studies, husbands’ sensory impairment seemed to have larger negative effects on the mood, negative affect, and psychological distress of wives than the converse. Some reviews of caregiving studies have observed that women tend to report greater caregiver burden and depressive symptomatology than do male caregivers (e.g., Pinquart & Sorensen, 2006).

Of course, support can also be amassed for a counter argument. Studies of the social networks of older persons suggest that older men tend to accord their wives a more prominent social role in their lives (e.g., as confidant or best friend) than wives do their husbands (e.g., Turner, 1994). Furthermore, evidence suggests that reliance on one’s spouse for social support is more closely tied to health outcomes for men than for women (Cutrona, 1996). Studies of marital satisfaction have also generally found older men to be more affected by their wives’ conflict resolution styles and strategies than the reverse (e.g., Kurdek, 1995). If these data are interpreted as suggesting that men are more dependent on their wives, especially in older married couples, then husbands might potentially be more negatively affected by health concerns of their wives.

The balance of available evidence, drawn from pain studies, studies of health and illness in the marital context, and emotional reactivity appears to favor expecting that women may be more strongly affected by their husbands’ pain intensity than the reverse. However, this evidence is weak and largely speculative. Regardless of expected direction
of effects, the examination of gender as a possible moderator of cross-spouse effects is important. Reflecting on the diverse and sometimes contradictory findings evidenced across a range of studies on older couples and relationship and health dynamics prompted Walker and Luszcz (2009) to call for increased study of the “differential impact of…spousal characteristics on husband and wife well-being” (p. 468). The study of potential gender differences in cross-spouse effects could ultimately help to advance current understanding about the mechanisms through which the cross-spouse effects of pain occur. As no prior study has investigated a possible gender differences in an indirect (i.e., mediated) cross-spouse relationship between the spouse’s pain and the partner’s depressive symptomatology involving the spouse’s activity limitation, there is no basis for expecting a specific direction or nature of effect.

**When Both Spouses Have Pain**

There are currently no published studies that have explored the relationships between each partner’s pain and each partner’s psychological distress in couples in which both partners experience pain. Outside of acknowledging that the situation presents a unique challenge to clinicians, very little has been written about marriages characterized by co-occurring pain in both partners. One of the most prolific authors on the topic of pain and family relations noted that, ”A very special category of family relations arises when both partners are victims of pain” (Roy, 1994, p. 78); yet he offers no further discussion of this point. Clearly, this area of research is ripe for further exploration.

Only two studies have examined whether the spouse’s own pain (broadly considered) might also affect his or her level of depressive symptomatology, above and beyond the pain of the designated “patient” or “partner with pain.” The limitations of the
studies by Leonard and Cano (2006) and Jacobi et al. (2003) have already been reviewed. Both, however, offer some stimulating ideas for consideration. In their study of 113 chronic musculoskeletal pain patients and their spouses, Leonard and Cano found an interaction effect involving the spouse’s own pain: Spouse catastrophizing about the patient’s pain was related to spouse depressive symptom levels only for those spouses who reported pain themselves. Although spouse catastrophizing was not of interest in the current study, the idea that co-occurring pain in spouses could fundamentally alter the nature of the relationship between pain and psychological distress is intriguing. In a study of caregivers to patients with painful rheumatoid arthritis, Jacobi et al. (2003) found that the caregiver’s own pain problems were strong predictors of subjective caregiver burden ($\beta = .72$). Although they did not explicitly evaluate any interaction effects, this finding also raises interesting questions about whether pain in both partners might dramatically alter perceptions and outcomes within the marital relationship.

Drawing on ideas from interdependence theory and interactional models of emotion, questions of multiplicative pain “exacerbation” or, conversely, “attenuation” effects on psychological distress in one or both partners were explored within the current study. Possible questions (phrased from the point of view of either a husband or wife) might include: Does my pain affect me more if my partner also has pain? Does my spouse’s pain affect me more if I also have pain? Are my spouse and I both at risk for higher levels of depressive symptoms if we both have high pain intensity? Alternatively, attenuation effects could occur, whereby the cross-spouse effect of pain intensity might be diminished. So, for example, could having or experiencing pain of one’s own prompt a spouse to respond in a more empathetic and supportive manner to his/her partner’s
pain? Is it the case that “a problem shared is a problem halved?” Might sharing the same experience enhance closeness within a couple? Or, perhaps serve as a distraction for one of the spouses to focus on the pain and needs of the other? Such questions are purely exploratory at this point, given the lack of prior study.

Limitations of Prior Research and Identification of Gaps

This review makes clear that the knowledge base regarding pain within the context of the marital dyad is based mainly on studies that have used small samples drawn from populations with specific pain problems in treatment at specialty pain clinics. Research focused on the pain experiences of community-dwelling couples not explicitly identified as “chronic pain patients” is limited.

The importance of examining pain in community-dwelling couples is made clear when one considers the myriad ways in which a community-dwelling sample might differ from a clinically-derived sample of chronic pain patients and spouses. Crook, Tunks, Rideout, & Browne (1986) compared a sample of adults from a community-based family medical group practice who reported experiencing persistent pain to a sample of patients attending a specialty pain clinic in a mid-sized Canadian city. The two groups were remarkably similar with respect to most demographic and background characteristics, history of pain complaints, body location of pain, and self-reported medical conditions. However, the pain clinic patients reported their pain as more severe and more constant, had greater levels of impairment in most areas of functioning, evidenced higher levels of health care utilization, and reported experiencing more psychosocial difficulties as a result of their pain than did the community practice patients. As Turk, Flor, & Rudy (1987) have observed, patients seeking treatment at specialty pain clinics are probably
those coping most poorly with pain. Thus, patients referred to chronic pain clinics are not likely to be representative of persons in the general population who suffer pain problems. There may then be problems extending the findings from studies of chronic pain patients and their spouses to community-dwelling married couples.

The existing literature on pain in couples is almost exclusively focused on the issues of middle-aged chronic pain patients and their spouses (Roy, 1995). Moreover, the average age in most samples suggests that these patients are mainly of younger middle age. Research on the experiences of pain in older couples affected by chronic pain is sparse. One author who has done a considerable amount of research on older pain patients reported that it took him one full year to recruit 9 older patients and their spouses into a pilot study investigating family relationships in the context of pain (Thomas & Roy, 1999). The delay was not due to low levels of consent, but rather to extremely low rates of enrollment of older patients at the pain clinic from which the sample was drawn. This is partly due to the fact that older patients tend to be “filtered out” of the referral process to specialty pain clinics (Walco & Harkins, 1999).

Samples of married couples recruited from pain clinics may differ in other important ways from community-dwelling married couples. For example, it is possible that couples recruited from pain clinic samples are more highly interdependent or connected than are the larger population of married couples living in the community. If the pain patient him/herself is experiencing considerable psychological distress, a higher level of interdependence within the couple may also mean that his/her spouse will be experiencing high levels of distress. The dynamics of interaction and levels of conflict in these couples may also differ significantly from couples not drawn from pain clinics.
These potential differences are not limited to samples derived from chronic pain clinics. It is possible that couples recruited from other medical settings (e.g., arthritis clinics) also present a somewhat biased picture of relationships involving pain and depressive symptomatology within the marital context. Even studies using samples of older couples drawn from the community can yield results influenced by selection bias. This would be likely if the couples were recruited on the basis of one or more partners experiencing significant pain or a medical condition associated with pain. And, of course, a convenience sample of couples recruited from the community could be markedly different from the general population of community-dwelling couples.

One major limitation of prior research on pain in married couples is that studies almost always overlook the possibility that pain could be experienced by both members of a marital dyad. Even within the literature that examines pain within the context of the marital relationship, a number of studies have relied solely on the reports of either the designated “patients” (e.g., Goldberg, Kerns, et al., 1993) or their spouses (e.g., Bigatti & Cronan, 2002). Thus, some studies have not even collected data from both spouses. Other studies have collected data from both spouses, but have focused on only one partner’s experiences with pain. In these studies, the designated “patients” completed extensive inventories about their pain experience and spouses completed similar inventories about the patient’s pain experiences (but not their own pain). In rare studies in which pain is measured in both partners, pain in the non-patient “spouse” is often just reported as a descriptive characteristic (e.g., Cano, 2005).

Of the few studies that have collected comparable information from each marital partner about his/her individual pain experiences and his/her own symptoms of
psychological distress, the majority fail to make use of all available data. So, for example, study authors may report the level of association of depressive symptom levels within these dyads, and they may even use information about one partner’s pain to predict that partner’s and/or the spouse’s depressive symptomatology. But most studies fall short of a truly dyadic analysis in that they tend to (a) focus on only one partner’s depressive symptomatology (often that of the “spouse”) and/or (b) fail to use information about each partner’s own pain to help predict or explain the pain-depressive symptom relationship in both partners. For example, Flor, Turk, & Scholz (1987) reported a significant correlation ($r = .43$) between the spouse’s own pain and his/her depressed mood; however, they did not include the spouse’s own pain as a predictor of his/her own depressed mood in multivariate models that included clinical and background characteristics about the pain patient. Although Leonard and Cano (2006) did incorporate the presence of pain in the spouse into the multivariate analysis of the spouse’s depressive symptomatology (along with the pain intensity level of the partner with pain), they stopped short of including the spouse’s level of pain intensity. Of course, this would have been difficult, given that less than half of the spouses in their study also reported pain; limiting the analysis to this subsample of couples would likely have resulted in too great a compromise in statistical power.

Given the established link between depression and pain, failure to consider the role of each spouse’s pain in contributing to his/her own psychological distress could result in biases in attribution and explanation. Potential misattribution could occur if a spouse’s own pain (and possible mediating variables) is not included in an analytic model predicting his/her own depressive symptomatology from the pain of his/her partner. To
the extent that the spouse’s own pain is significantly related to his/her own distress, the resulting model could mistakenly attribute the spouse’s psychological distress to the effects of the partner’s pain. Potential over-attribution is also possible. Depending on the true relationship between the partner’s pain and his/her spouse’s psychological distress, not accounting for the effects of the spouse’s own pain could result in over-attributing the spouse’s distress to the pain of the partner. Finally, to the extent that the pain levels of both partners’ levels help to explain the depressive symptomatology of each spouse, resulting models could be limited in terms of the amount of variability in psychological distress that they could explain.

Little prior research has examined potential intervening mechanisms in the relationship between one partner’s pain and the spouse’s psychological distress. Despite the availability of theories and some empirical evidence suggesting that activity limitation may play an important role in mediating the effect of pain on depressive symptomatology at the individual level (see Chapter 2), this potential mediation pathway for the cross-spouse effect of pain has been largely unexamined in studies of pain in married couples.

Research regarding potential moderators of any cross-spouse effects of pain on psychological distress has also been scarce. In fact, few studies have examined how any sociodemographic characteristics (including gender, race, ethnicity, SES, length of marriage, etc.) might influence the experience of pain within married couples. Contradictory findings have emerged from some empirical studies of the relationship between one spouse’s pain and the other’s depressive symptomatology—findings that have not supported the prevailing assumptions of most pain scholars. The existence of
such findings, along with suggestive findings from various literatures and theoretical perspectives regarding gender differences, suggests that gender differences in the cross-spouse effects of pain have been under-studied. Finally, as existing studies have generally overlooked the possible existence of pain in both spouses, there has been very limited consideration of potential synergistic effects involving the existence of pain in both partners, and no prior consideration of potential synergy between both spouses’ levels of pain intensity.

**Unique Features of This Study**

The current study sought to address many of the limitations and gaps identified above. The study made use of a large, community-based sample of middle-aged and older persons and their spouses. Data were drawn from an ongoing national survey, intended to represent the population of persons age 51 and older in the U.S. Spouses of married respondents were also interviewed. Information about pain was collected from all study respondents, and did not rely on the recruitment of chronic pain patients or persons who suffered from painful disease conditions. The study also collected data from both partners regarding their own psychological distress (specifically, depressive symptomatology), as well as other key constructs in the proposed conceptual model.

The couples drawn from this source were expected to be more diverse in terms of race, ethnicity, and socioeconomic status than are couples included in most prior studies, especially those drawn from specialty pain clinics. The community-dwelling sample was expected to more fully represent the broad continuum of the pain experience (e.g., frequency, intensity), as well as the kinds of pain and/or locations of pain that respondents experience. Older adults are underrepresented in samples drawn from
specialty pain clinics. In contrast, the current sample covered a more broad range of ages within the spectrum of middle and older adulthood, particularly when compared to studies of chronic pain patients. The diversity in age (across the spectrum of middle and older age) contrasts markedly with studies of chronic pain patients in which older adults are seriously underrepresented. The variability and diversity of a national, community-based sample can only enhance the generalizability of this study’s findings with respect to the relationships between pain and psychological distress in middle-aged and older married couples.

The present study focused on couples in which both spouses report pain, a population about which very little is known. The study design permitted comparison of these “Dual-Pain” (DP) couples to three other types of couples within the broader HRS sample in which neither spouse, or only one spouse, reported pain. This comparison helped to address Study Aim 1. More details about these efforts are presented in the Method chapter.

The dyadic design of this study further permitted an examination of several critical gaps in the existing knowledge base. One was the investigation of covariation between spouses in key study constructs such as pain intensity, depressive symptomatology, and physical limitations. The dyadic design also made it possible to investigate the potential cross-spouse effects of pain, and to do this in a way that overcame many of the shortcomings evident in existing research. Specifically, the present study examined the extent to which each spouse’s pain is related to the depressive symptomatology of his/her partner, controlling for the possible effects of each partner’s own experience of pain (along with other relevant predictors). The current study also
explored an indirect pathway involving the spouse’s activity limitation; here again, the dyadic design of the study enabled the investigation of this potential cross-spouse mediation for each spouse. Finally, the dyadic design also made it possible to examine the extent to which selected background characteristics might influence some of the relationships between pain and psychological distress within married couples.

**Research Questions and Hypotheses**

**at the Inter-Individual Level**

Research questions and hypotheses at the *inter-*individual level are presented below. These questions were built around the inter-individual conceptual model introduced earlier in this chapter. Where possible, reference is made to the pathways in the figure to help clarify the relationships that are being tested. Before presenting these questions and hypotheses, several preliminary considerations are reviewed.

**Preliminary Considerations**

**Review of Conventions Used in This Presentation**

As outlined in this chapter, one central aim of the current study (Study Aim 3) was to examine the direct and indirect pathways through which one spouse’s pain might affect the psychological distress of his/her partner. Because the present study also examined relationships between pain and depressive symptomatology at the *intra-*individual level, the specific pathway(s) being tested must be clearly identified. Toward this end, two conventions have already been adopted: (a) differentiating between intra-individual and inter-individual research questions, effects, and issues (hence, the separate presentations of material in Chapters 2 and the current chapter); and (b) using the terminology “cross-spouse” to indicate *inter-*individual effects.
It is often necessary to refer to these cross-spouse effects generally, as well as to specify the origin and direction of a particular effect. Consequently, several additional conventions have been adopted. Gender-neutral language is used in statements relating to a general cross-spouse relationship or effect. Grammatically, such statements typically position the “spouse” or “respondent” as the subject (or implied actor) and “his/her partner” as the object (or implied recipient)—e.g., “the relationship between each spouse’s pain and the depressive symptomatology of his/her partner.” When the origin and direction of a particular cross-spouse effect is relevant, gender-specific language is used—for example, “the effect of the husband’s pain intensity on the wife’s depressive symptoms”. To help clarify the particular effect being discussed, occasional reference is made to either the general path number in Figure 5 (e.g., “Path 3”) or the subscripted path number that identifies the origin of the effect (e.g., “Path 3H”—i.e., the effect of the husband’s pain intensity on the wife’s depressive symptomatology).

**Handling of Couple-level Background Characteristics**

Research questions involving relevant individual-level background characteristics (e.g., gender, race, age, etc.) were presented at the end of Chapter 2. Several additional background characteristics were added in the current chapter, when the proposed conceptual framework was extended to the level of the marital dyad: household socioeconomic status, household composition, and years of marriage.

Household socioeconomic status was regarded mainly as a covariate in the proposed study. However, Chapter 2 reviewed evidence suggesting that lower socioeconomic status (broadly defined) is associated with reports of pain (e.g., Taylor & Curran, 1985), and also increased pain intensity (e.g., Creamer et al., 1999). These
findings have also been documented in studies of middle-aged (e.g., Avis et al., 2003) and older persons (e.g., Reyes-Gibby et al., 2007). Numerous studies have also found lower SES to be positively associated with depressive symptomatology (e.g., Blazer et al., 1991; Choi & Bohman, 2007) and physical limitations (e.g., Zimmer & House, 2003). Thus, in the present study, household-level SES variables will be examined as predictors of pain intensity in DP spouses. It will also be important to control for the effects of SES when examining potential cross-spouse effects of pain on depressive symptomatology.

Even though there is no available evidence to suggest that household composition is related to any of the key constructs in the proposed conceptual model, it was controlled in testing most of the hypotheses involving cross-spouse effects of pain. The current sample consisted of middle-aged and older married couples, couples likely to have some variation in household size. Hence, it seemed reasonable to at least control for such heterogeneity among couples when attempting to answer research questions posed at the couple-level—something that studies of marital dyads and pain have rarely done.

Years of marriage constitutes another background variable that is often controlled in studies of married couples (e.g., Pienta, 2003; Townsend et al., 2001). There is little prior evidence to suggest that marital duration is related to any of the key constructs in this study. However, as with household composition, it is important to control for any differences in years of marriage that might exist between couples, especially when substantive cross-spouse effects are being tested.

Research questions and hypotheses at the inter-individual level are presented in the next section. Household SES was the only couple-level characteristic around which a hypothesis was developed—and this was limited to an expected negative relationship
with levels of pain intensity. The hypothesis regarding the relationship of SES to pain intensity (i.e., H2) was already introduced in Chapter 2; thus, no separate restatement of this hypothesis is offered here. The evaluation of household SES as a predictor of pain intensity, along with the inclusion of couple-level background variables as covariates in planned analyses, are discussed further in the Analysis Strategy in Chapter 4.

Research Questions and Hypotheses

The numbering of the research questions and hypotheses below is continued from Chapter 2. As before, research questions (RQ) and exploratory questions (EQ) generally use broader, more conceptual language (e.g., pain), whereas sub-questions and hypotheses (H) tend to reference specific constructs or indicators in the proposed study (e.g., pain intensity). Sub-questions and hypotheses are again indented under the particular research (or exploratory) question to which they relate. Main research (or exploratory) questions are in **boldface type** and hypotheses are presented in *italics* to make them easy to identify.

**RQ6. Are spouses’ (i.e., wives’ and husbands’) key study variables related?**

*H6a. Spouses’ reports of pain intensity will positively covary.*

In terms of the relationships highlighted in Figure 5, this hypothesis asserts that Path 1 will be significant both for the presence of pain and pain intensity in married couples. Prior research has found evidence of spousal concordance for the presence of pain. Concordance rates for pain problems have ranged between 21% and 49% across chronic pain patient samples drawn from both clinical settings (e.g., Sharp and Nicholas, 2000) and the community (e.g., Cano, 2005), as well as in a sample of middle-aged and older community-dwelling persons not identified as chronic pain sufferers in
the Netherlands (e.g., Jacobi et al., 2003). Co-occurrence of pain problems has received little study on a national level in the U.S. However, given that baseline prevalence rates for pain range from 66% to 86% among middle-aged and older community-dwelling adults (see Chapter 2), it seems likely that pain will co-occur with some frequency among late-life married couples in the U.S.

Although some studies (e.g., Cano, 2005) have focused on the extent to which spouses are congruent in their rating of one partner’s pain intensity, no prior research has examined the extent to which each spouse’s ratings of his/her own pain intensity are related. However, based on evidence that pain is likely to co-occur among married couples, along with studies showing covariation in other health characteristics (e.g., disease conditions in Hippisley-Cox et al., 2002), spouses’ pain intensity ratings were expected to covary positively as well.

\( H6b. \) Spouses’ depressive symptomatology will positively covary.

This hypothesis proposed that Path 2 in Figure 5 would be significant. Prior research has documented sizeable associations between depressive symptom levels in married partners in a variety of different samples of married couples, including community-dwelling couples in mid and later-life (e.g., Galbaud et al., 1994; Goodman and Shippy, 2002). Although less well-documented in other populations, this phenomenon has also been reported in studies of couples sampled from chronic pain clinics (e.g., Schwartz et al., 1991) and from other clinical settings (e.g., outpatient rheumatology clinics, e.g., Druley et al., 2003). As mentioned in the earlier review, many existing pain studies either lack comparable data for both spouses—largely because researchers tend to focus only on the patient or the spouse—or fail to report the
bivariate association between spouse depressive symptom levels. Nevertheless, based on findings from the existing literature on married couples and pain, and support culled from studies of married couples in general, significant positive covariation of depressive symptoms was expected within the marital dyads in this study. Moreover, as will be discussed in Chapter 4, this expected covariation provided a rationale for selecting statistical techniques that appropriately control for this covariation.

\( H6c. \) \( \text{Spouses’ reports of activity limitation (specifically, physical limitations) will positively covary.} \)

Stated in terms of Figure 5, \( H6c \) proposes that Path 3 will be significant. As noted in the prior literature review, empirical support for this hypothesis is limited within existing studies of pain in married couples. Based on some prior studies suggesting covariation in physical limitations (e.g., Townsend et al., 2001; Walsh et al., 1999), a significant positive covariation was expected to be observed. This hypothesis was also supported by interdependence theory, as well as a growing body of research documenting substantial covariation of other health constructs within married couples (e.g., count of disease conditions, Wilson, 2002; also review by Meyler et al. 2007).

\textbf{RQ7.} \textit{Is there a cross-spouse relationship between each spouse’s pain intensity and his/her partner’s depressive symptomatology?}

\( H7. \) \( \text{Each spouse’s pain intensity will be related to greater depressive symptomatology in his/her partner.} \)

Research Question 7 concerned the existence of a direct relationship between one spouse’s pain and the other’s psychological distress. This direct, cross-spouse effect is represented by Path 4 in Figure 5. Two cross-spouse effects can be specified: Path
4_H, representing the effect of the husband’s pain on the wife’s depressive symptomatology; and Path 4_W, representing the effect of the wife’s pain on the husband’s depressive symptomatology.

As reviewed in this chapter, some prior empirical evidence exists to expect that higher levels of pain intensity in one spouse will be associated with greater depressive symptomatology in his/her partner. Support for this hypothesis exists within previous studies of chronic pain patients and their spouses (e.g., Romano et al., 2000), research with couples in which one partner experience significant disease-related pain—especially rheumatoid arthritis (e.g., Lam et al., 2009), cancer (e.g., Hagedoorn, et al., 2008), and to a slightly lesser extent, osteoarthritis (e.g., Druley et al., 2003). Evidence supporting a relationship between one spouses’ pain intensity and the other’s depressive symptomatology is generally lacking in studies of community-residing midlife and older adults. Considerably less evidence exists supporting a direct relationship between a spouse’s pain intensity level and his/her partner’s depressive symptom levels. However, as noted earlier, this relationship has not often been empirically tested. In addition, prior studies using chronic pain patient samples to examine this relationship may have been hampered by inadequate variability with respect to pain intensity ratings. Findings from a few studies (e.g., Schwartz et al., 1991; Kerns & Turk, 1984), as well as from studies linking the presence of pain in one spouse with the depressive symptoms of his/her partner, provided adequate evidence to formulate this hypothesis.

**RQ8. Is the cross-spouse relationship between each spouse’s pain intensity and the depressive symptomatology level of his/her partner mediated by the spouse’s activity limitation (as measured by physical limitations)?**
Potential mediation of cross-spouse effects was investigated using a series of questions and sub-questions consistent with the steps outlined by Baron and Kenny (1984). These steps were introduced in Chapter 2, and will be discussed in greater detail in Chapter 4. Thus, they are reviewed only briefly here.

First, a significant relationship must be observed between one spouse’s pain and his/her partner’s depressive symptomatology. In terms of Figure 5, Path 4 must be significant. This step is addressed by RQ7 above. Next, there must be a significant relationship between the spouse’s own pain and his/her own activity limitation (i.e., Path B must be significant). As this step involved an *intra*-individual relationship, it was addressed by an individual-level research question presented in Chapter 2 (RQ4a).

Next, a significant relationship between one spouse’s activity limitation and his/her partner’s depressive symptomatology must be observed. In terms of Figure 5, Path 5 must be significant. Mediation is demonstrated if the relationship between the spouse’s pain and the depressive symptomatology of his/her partner (i.e., Path 4) is significantly reduced and/or eliminated, once the relationship between the spouse’s activity limitation and the partner’s depressive symptomatology is taken into account—i.e., if Path 4 is reduced once activity limitation (via Path 5) is added to the model.

**RQ8a.** Is there a relationship between each spouse’s physical limitations and the depressive symptomatology of his/her partner?

**H8a.** Greater physical limitations in each spouse will be related to greater depressive symptomatology in his/her partner.

**RQ8b.** Do the spouse’s physical limitations mediate the relationship between each spouse’s pain and his/her partner’s depressive
symptomatology?

H8b. *The relationship between each spouse’s pain intensity and his/her partner’s depressive symptomatology will be at least partially mediated by the spouse’s physical limitations.*

As noted in the review of the literature regarding Path 5, evidence in support of activity limitation functioning as a mediator of the relationship between one spouse’s pain and the depressive symptomatology in the other partner is notably absent from the literature on couples and pain. However, a four-fold rationale was offered to justify extending the activity restriction theory of depression (as discussed in Chapter 2) to the level of the marital dyad: (1) there is both theoretical support and associated empirical evidence (reviewed in Chapter 2) that activity limitation at least partially mediates the relationship between pain and depressive symptomatology at the *intra*-individual level; (2) there is some empirical evidence of a relationship between one spouse’s pain and depressive symptomatology in the other spouse (i.e., Path 4), and the process underlying this relationship is not well-explained by existing theory or empirical data; (3) there is some (albeit limited and often qualified) empirical evidence that supports a relationship between one spouse’s activity limitation (broadly defined) and the other spouse’s psychological distress; and (4) there is ample evidence in studies of community-based couples that the physical health concerns and problems of one spouse are associated with depressive symptomatology (and general psychological distress) in the other spouse. On the basis of evidence culled from studies in each of these areas, it seemed reasonable to investigate physical limitations as a possible mediator in the relationship between one spouse’s pain and the other’s depressive symptomatology.
RQ9. Does gender moderate any of the cross-spouse relationships between each spouse’s pain intensity and the depressive symptomatology level of his/her partner? Specifically, do any of the direct or indirect (i.e., mediated) relationships between each spouse’s pain intensity and his/her partner’s depressive symptomatology differ between husbands and wives?

H9. The direct relationship between the husband’s pain intensity and the wife’s depressive symptomatology will be stronger than the direct relationship between the wife’s pain intensity and the husband’s depressive symptomatology.

As suggested in the literature review presented earlier in this chapter, there is more compelling empirical evidence and theoretical justification for expecting that wives would be more “reactive” to or influenced by their husband’s experience with pain. In relation to the conceptual model presented in Figure 5, this hypothesis proposed that Path 4_H (the path from the husband’s pain to the wife’s depressive symptomatology) would be significantly stronger than Path 4_W (the path from the wife’s pain to the husband’s depressive symptomatology).

EQ9. Does gender moderate the indirect (i.e., mediated) cross-spouse relationship between each spouse’s pain intensity and his/her partner’s depressive symptomatology?

Gender has not previously been studied in relation to the mediation of any cross-spouse (i.e., inter-individual) effects of pain. However, given that (a) gender has been offered as a possible moderator of intra-individual pain-depressive symptom mediation processes (as reviewed in Chapter 2) and (b) gender differences have been observed in
some existing studies of the cross-spouse relationship involving pain and depressive symptoms (as noted in the review in this chapter), there seemed sufficient justification to explore whether gender differences might exist in the extent to which the cross-spouse relationship between pain intensity and depressive symptomatology is mediated by the spouse’s physical limitations. In terms of Figure 5, this exploratory question asked: Are Paths 5_H and 5_W significantly different?

In this context, gender moderation could be said to exist if the mediated cross-spouse effect was found to be stronger in one spouse than the other (e.g., if Path 5_H—the path from the husband’s activity limitation to the wife’s depressive symptoms—was stronger than Path 5_W), or if mediation was observed for one gender only (e.g., if activity limitation mediated the relationship between the husband’s pain intensity levels and the wife’s depressive symptoms, but not the relationship between the wife’s pain intensity and the husband’s depressive symptoms).

EQ10. **Do spouses’ levels of pain intensity interact to jointly affect the depressive symptomatology of either partner?**

Lack of prior empirical and theoretical work regarding couples in which both spouses experience pain makes posing an explicit hypothesis about how spouses’ pain intensity levels might interact difficult. Therefore, this was an exploratory question.
# CHAPTER 4: METHOD

## TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Data Source</td>
<td>4-1</td>
</tr>
<tr>
<td>HRS 1998 Survey Procedures</td>
<td>4-4</td>
</tr>
<tr>
<td>Rationale for Choosing HRS 1998</td>
<td>4-5</td>
</tr>
<tr>
<td>The RAND Version of HRS Data</td>
<td>4-6</td>
</tr>
<tr>
<td>Sample</td>
<td>4-7</td>
</tr>
<tr>
<td>Sample Selection Criteria</td>
<td>4-7</td>
</tr>
<tr>
<td>Derivation of the Study Sample</td>
<td>4-9</td>
</tr>
<tr>
<td>Measures</td>
<td>4-15</td>
</tr>
<tr>
<td>Outcome Variable</td>
<td>4-16</td>
</tr>
<tr>
<td>Psychological Distress (Depressive Symptomatology)</td>
<td>4-16</td>
</tr>
<tr>
<td>Key Explanatory Variables</td>
<td>4-24</td>
</tr>
<tr>
<td>Pain</td>
<td>4-24</td>
</tr>
<tr>
<td>Pain Intensity</td>
<td>4-26</td>
</tr>
<tr>
<td>Disease-Specific Pain</td>
<td>4-29</td>
</tr>
<tr>
<td>Activity Limitation (Mediating Variable)</td>
<td>4-30</td>
</tr>
<tr>
<td>Health-Related Characteristics</td>
<td>4-38</td>
</tr>
<tr>
<td>Disease Conditions</td>
<td>4-38</td>
</tr>
<tr>
<td>Body Weight</td>
<td>4-40</td>
</tr>
<tr>
<td>Background Characteristics</td>
<td>4-42</td>
</tr>
<tr>
<td>Individual-Level Background Characteristics</td>
<td>4-43</td>
</tr>
<tr>
<td>Gender</td>
<td>4-43</td>
</tr>
<tr>
<td>Age</td>
<td>4-44</td>
</tr>
</tbody>
</table>
### Race and Ethnicity

- 4-45

### Socioeconomic Status

- 4-46

### Couple-Level Background Characteristics

- 4-47

### Analysis Strategy

- 4-55

#### Preliminary Steps

- 4-56

- Data Cleaning and Item-level Data Transformation

- Examination of Missing Data

- Handling of Missing Data: Multiple Imputation

- Overview and Rationale

- Data Preparation

- Specifications in the MI Procedure

- Processing of Imputed Data

#### General Considerations and Procedures in the Analysis of Dyadic Data

- 4-77

#### Analytic Implications of Dyadic Data

- 4-77

#### General Procedures and Guidelines Used in the Multilevel Analyses

- 4-80

- Concessions Required in Multilevel Modeling of Dyadic Data

- Benefits of the Multilevel Modeling of Dyadic Data

- Model Specification Options

- Model Building and Assessment of Model Fit

- Data Management and the Use of MI Data in HLM6

#### Analyses Addressing Research Questions and Hypotheses

- 4-89
<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>General Considerations</td>
<td>4-89</td>
</tr>
<tr>
<td>Preliminary Analyses</td>
<td>4-91</td>
</tr>
<tr>
<td>Describing DP Couples and Comparisons With Other HRS Couples</td>
<td>4-91</td>
</tr>
<tr>
<td>Examination of Basic Bivariate Relationships Within DP couples</td>
<td>4-92</td>
</tr>
<tr>
<td>Substantive Analyses in the Focal Sample of Dual-Pain Couples</td>
<td>4-93</td>
</tr>
<tr>
<td>Analyses Used to Address Intra-Individual Research Questions</td>
<td>4-94</td>
</tr>
<tr>
<td>Analyses Used to Address Inter-Individual Research Questions</td>
<td>4-104</td>
</tr>
</tbody>
</table>
CHAPTER 4: METHOD

Data Source

Data for this study were drawn from the 1998 merged Health & Retirement Study (HRS) and the Study of Assets & Health Dynamics among the Oldest Old (AHEAD). HRS and AHEAD are longitudinal, biennial panel studies of nationally representative samples of community-dwelling men and women initially aged 51-61 (HRS) and 70 and older (AHEAD). The studies are funded by the National Institute on Aging (NIA; grant number NIA U01AG009740) and conducted by the University of Michigan Institute for Social Research (Juster & Suzman, 1995).

The original HRS (first wave of data collection in 1992) was designed to provide scientists and policy makers with information about retirement and disability patterns. Thus, HRS focused on a cohort of persons of pre-retirement age, and followed them into retirement. The AHEAD study (first wave of data collection in 1993) was originally funded as a supplement to HRS, and focused on cohorts in the post-retirement period of later life. AHEAD was designed to address research questions related to the interplay between economic, family, and social welfare program resources and age-related changes in health and well-being. Both surveys tap a broad range of content domains including socio-demographics, family structure and resources, physical and mental health status, labor force participation, and economic status and resources.

For Wave 1, HRS used a multi-stage area probability sample design to select household units that included at least one age-eligible member born between 1931 and 1941 (Juster & Suzman, 1995). AHEAD Wave 1 participants (those born ≤ 1923) were identified through (a) household unit data from the initial screening done for HRS, and
The initial HRS sample included 7,705 households (12,654 persons). Over 6,047 households (8,222 persons) comprised the initial sample for AHEAD (HRS, 2002). In order to obtain samples of racial and ethnic minority groups large enough to permit independent analyses, Black and Hispanic persons were oversampled in both HRS and AHEAD at a rate of approximately 2:1 relative to White persons. Florida residents were also systematically oversampled in an effort to give special attention to areas with high concentrations and large numbers of older adults (Juster & Suzman, 1995).

Initial (baseline) interviews were conducted in person (or by phone, for some AHEAD respondents). On average, interviews lasted one to one and one-half hours. A Spanish language interview was made available to Spanish-speaking persons. HRS and AHEAD study participants are re-interviewed by phone (or in person, if warranted) every two years. Both HRS and AHEAD permit proxy interviews (usually with a spouse or other family member, or knowledgeable other) if a study participant is unable to participate because of physical or cognitive impairments, or if he/she is unwilling to answer questions but grants permission for someone else to do so. Participants are followed from their entry into the survey until death. Exit interviews are conducted with a surviving spouse or other informant after a participant’s death (Juster & Suzman, 1995).

Response rates for HRS and AHEAD generally have been high. The baseline (Wave 1, 1992) response rate for HRS was 81.7% (computed based on the total number of eligible respondents identified through screening). The baseline (Wave 1, 1993) response rate for AHEAD was 80.4% (HRS, 2002). Re-interview rates with surviving respondents have also been high—ranging between 91.8 and 95.4% for all subsequent
waves, as of the 2000 survey (Rodgers, Ofstedal, & Herzog, 2003; HRS, 2007b).

A unique feature of HRS and AHEAD, and one crucial to this investigation, is the inclusion of the spouses of respondents. If a target individual was married or living with a partner, an interview was sought with that spouse/partner, regardless of his/her age. Interviews were also sought with any new spouses/partners reported by respondents. HRS and AHEAD have had a high rate of success in recruiting spouses/partners into the study. For example, Wave 1 interviews were obtained with both spouses/partners in 93% of those households containing an HRS age-eligible, married/partnered respondent (HRS, 2002). To date, there are no published data regarding the success rate in getting re-interviews with both members of couples who responded in the initial study waves.

Field efforts and data collection instruments for HRS and AHEAD were merged in 1998, and the combined HRS/AHEAD study from 1998 onward is now simply referred to (and will generally hereafter be referred to in this document) as HRS. Two new groups of participants were added to the study in 1998. The first group includes individuals born between 1942 and 1947 (age 51-56 in 1998) and their spouses. This group is referred to as the War Babies (WB) cohort because of the proximity of their births to World War II. Given the aging of the original HRS cohort that had taken place by 1998, the WB cohort essentially replenished HRS with persons in their early fifties.

The second group of participants recruited into the study is labeled the Children of the Depression Age (CODA) cohort and their spouses. These individuals, born between 1924 and 1930 (age 68-74 in 1998), were so named because they grew up mainly during the Great Depression. The CODA cohort thus helped to fill the age gap between the original HRS and AHEAD cohorts (National Institute on Aging, 2007).
members were identified through the initial screening of household units done for the original HRS sample. The CODA sample (similar to part of the original AHEAD sample) was drawn from the Medicare enrollment database.

*HRS 1998 Survey Procedures*

HRS 1998 data were collected between February 1998 and March 1999. The collection of baseline data from new study participants (e.g., WB and CODA cohorts) was done through in-home interviews (HRS, 2007a.). Individuals (and their spouses) in the HRS and AHEAD cohorts completed their re-interviews either by telephone or in face-to-face interviews, depending on their health status and/or age (more frail respondents and those age 80 and over and were typically interviewed in person). In addition, AHEAD respondents born between 1918 and 1920 were randomly assigned to a telephone or face-to-face interview in HRS 1998 as part of a multi-wave experiment to assess possible mode effects (HRS, 2007a). In HRS 1998, the overall response rates (including exit interviews with proxies) for the original panel of HRS and AHEAD respondents (and their spouses) who participated in one or more prior waves were 84.9% for HRS and 90.4% for AHEAD (HRS, 2007b).

It should be noted that some of the WB and CODA individuals were already included in HRS as spouses of persons included in HRS or AHEAD; thus, sampling was done in a way that ensured that the total WB and CODA samples represented the complete population of persons born in each birth cohort. The baseline response rates for the WB and CODA cohorts in HRS 1998 were slightly lower than those observed for the original HRS and AHEAD cohorts at baseline—69.9% for WB, 72.5% for CODA—but were still very respectable (HRS, 2007b). With the merger of the two studies into a
single data collection effort and the ongoing recruitment of new cohorts into the study every six years, HRS has become a broad source of data regarding the health and well-being of persons over age 50 in the United States.

**Rationale for Choosing HRS 1998**

The decision to use data from HRS 1998 (as opposed to earlier waves) was motivated by several important considerations. By combining data from HRS and AHEAD, researchers can address important questions across two large portions of the life-span—middle-age and older adulthood. However, researchers who attempt to utilize data from early waves of both HRS and AHEAD (prior to 1998) must create complicated coding algorithms or rely exclusively on latent variable measurement models in order to overcome a lack of comparability in many of the questions and response categories used in the two studies. When HRS and AHEAD merged operations in 1998, data collection procedures and instruments were updated in order to (a) address problems experienced in earlier waves of either study, and (b) reflect contemporary survey research and practice. Respondents in all cohorts now answer the same questions and use the same response categories. As a result of adding the two new cohort panels alongside the original (now older) HRS and AHEAD cohort panels, the age distribution of participants in HRS 1998 became more broad and complete. The aging HRS cohort and the newly recruited CODA cohort filled the roughly 10-year age gap that existed between the original HRS and AHEAD cohorts. In addition, by recruiting new cohorts into the study, HRS 1998 contained data from an expanded number of married couples.

Numerous characteristics made HRS an ideal data source for addressing the research questions posed in this study. However, HRS does have some significant
limitations. First, all data collected in the early waves of HRS are self-report survey data; as such, they are subject to all biases inherent in self-reports. For example, self-reports can be subject to both intentional and unintentional efforts to bias self-presentation in either a favorable or unfavorable way (Fowler, 1995). However, self-reports are advantageous in that they provide data concerning internal, subjective phenomena (like pain or depressive symptoms, for example) that may not be accessible via other approaches (e.g., observation). Unfortunately, HRS 1998 did not collect any anthropometric data, data from medical records, or data from physical performance tests with which to compare self-report data like physical limitations (although HRS has added some of these measures in recent years). Second, although HRS is a valuable source of secondary data, research questions and analyses are necessarily limited by the questions that were asked (or not asked) of respondents. Sometimes depth of questions in any particular content area was sacrificed in favor of increased breadth of HRS survey content. Finally, the extent to which the married couples in HRS are representative of the larger population of middle-aged and older married couples in the United States is unknown. However, the sampling methodology employed in HRS ensures some degree of representativeness of individuals (and, by extension, their households) in terms of age, race, ethnicity, geographic region, and household composition.

*The RAND Version of HRS Data*

The data for this study were drawn primarily from the RAND version of HRS data—specifically, the RAND HRS Datafile, Version I (RAND Corporation, 2009). The RAND HRS Datafile is a cleaned, user-friendly version of HRS public use datafiles that contains fewer variables from all available waves of data collection. The files were
developed by the RAND Center for the Study of Aging through funding from the NIA and the Social Security Administration. The current study benefitted greatly from the fact that RAND matched the data from spouses participating in HRS and merged household-level data into each spouse’s row of data in the file. RAND also resolved several (although not all) of the known problems with HRS public release datafiles.

As not all variables of interest in this study were included in the RAND HRS Datafile, selected data were also drawn from special datafiles prepared by RAND, called the RAND-enhanced HRS Fat Files, Version I (RAND Corporation, 2009). The development of these files was sponsored by NIA. These files are wave-specific HRS raw datafiles that also include variables created by RAND to facilitate data cleaning and the merging of data across study waves. The files are released to researchers only upon request. The specific variables drawn from the RAND-enhanced Fat file for HRS 1998 are identified within the measures section of this chapter.

Sample

Sample Selection Criteria

This study utilized data from a subset of married couples interviewed in the 1998 HRS survey. The sample included individuals (and their spouses) from the original HRS and AHEAD studies who were successfully re-interviewed in 1998, as well as individuals (and their spouses) who were recruited into the study in 1998. Thus, four identifiable cohorts were included in the 1998 survey: (a) the HRS cohort (age 57-67 in 1998), and their spouses; (b) the AHEAD cohort (age ≥ 75 in 1998), and their spouses; (c) the War Babies (WB) cohort (age 51-56 in 1998), and their spouses; and (d) the Children of the Depression Age (CODA) cohort (age 68-74 in 1998), and their spouses.
To be eligible for inclusion in the present study, *each spouse must have completed the 1998 HRS interview independently*. Couples for whom data were obtained for one or both spouses via a proxy interview were excluded. This decision was based on accumulated evidence suggesting that, although proxy reporting is generally accurate for factual data (e.g., date of birth, number of children), proxy responses to subjective questions (e.g., evaluation of one’s health or well-being) are considerably less reliable and valid than are self-report data (Aday, 2006; Fowler, 2002). Studies comparing proxy reports to self-reports for key constructs such as pain (e.g., Ferrell, 2001) and physical health or functioning (e.g., Magaziner, Zimmerman, Gruber-Baldini, Hebel, & Fox, 1988) have found that proxy reports tend to be biased, sometimes in unpredictable ways.

Eligible couples were required to be *(legally) married and residing together in the community at the time of the 1998 interview*. Recall that HRS attempted to recruit both the spouses (“married”) and partners (“living with a partner [as if married]”) of participants into the study. Although it is now possible for gay and lesbian couples to obtain marriage licenses in some states in the U.S., it was not possible in 1998. In addition, although some states recognize cohabitating heterosexual couples as participants in a “common-law” marriage, not all states recognize these relationships or accord them the same legal status accorded to couples who are legally married. Because of the myriad definitions of marriage employed across the U.S., as well as the lack of empirical research regarding pain and depressive symptomatology among partners in non-traditional family structures, this study excluded couples comprised of same-sex partners. Couples were also required to report themselves as married and living together at the time of the interview. Although HRS interview questions did not specify that
reported marriages be legally-recognized; the use of the “living with partner” response option and follow-up questions about marriages (e.g., specific month & year married, current marital status, if/how the marriage ended) suggest that the marriages recorded for participants in HRS are those that were recognized legally.

Although HRS required participants to be living in the community at the time of their initial entry into the study, HRS does attempt to follow participants into institutional settings. Thus, some of the original HRS and AHEAD survey respondents could have been living in an institutional setting at the time of their re-interviews in 1998. Because the present study sought to examine the experiences of pain in community-dwelling couples, couples in which one or both spouses were living in an institutional setting at the time of the 1998 interview were excluded. The exclusion of such couples was expected to have been (at least partially) accomplished during earlier steps (e.g., excluding couples in which one or both spouses required a proxy interview, requiring spouses to report being legally married and living together at the time of the interview. Interview location data were also examined to ensure that eligible couples were residing in the community.

The pattern of responses given by each husband and wife pair to an HRS 1998 survey question regarding experience of pain was used to identify couples who met the final criterion for inclusion in the present study. The focal sample for the present study was comprised of those married couples in which both spouses reported being “often troubled with pain.”

Derivation of the Study Sample

The aforementioned study selection criteria were operationalized using variables available in the RAND HRS dataset; most were derived variables that had been
constructed by researchers at RAND. The sample was then derived by applying these selection criteria to the 1998 HRS survey data contained in the RAND HRS Datafile, Version I (RAND Corporation, 2009). Figure 6 illustrates the specific steps followed in this process. At each step, information is also provided about the number of excluded cases, in addition to any relevant descriptive characteristics.

A total of 21,384 individuals had interview data recorded for the 1998 HRS survey. Roughly 76% of these interviews were longitudinal (i.e., conducted with original HRS or AHEAD respondents and their spouses of any age), while approximately 24% were new interviews (i.e., conducted with newly-recruited WB and CODA cohort members and their spouses of any age; HRS, 2003). Step 1 shows that interview data were available in 1998 for both spouses in 6,989 couples (13,978 individuals).

Because only couples in which neither spouse required a proxy interview were eligible, Step 2 shows that 1,359 couples were excluded because one (96%) or both (4%) spouses required a proxy interview in 1998. After proxy interviews were excluded, 5,628 couples (or 11,256 individuals) remained eligible for inclusion in the present study.

In order to assess whether couples were (legally) married and living together in the community at the time of the 1998 interview, the responses of each spouse to questions regarding current marital status and household living arrangements were compared. Two hundred sixteen couples were excluded because one or both spouses reported a marital status other than “married” (e.g., separated, divorced, living with a partner) and/or reported that they were not currently living in the same household as their spouse. A total of 5,414 couples remained eligible at the end of Step 3; all reported being legally married and living together at the time of the 1998 interview.
Figure 6. Sample Derivation Flowchart

RAND HRS Data for 1998 Survey
Version I Data Set
N = 21,384 individuals with interview data

**Excluded N = 7,406 individuals**
(mostly single, divorced, or widowed; some were part of a couple in which the other spouse did not participate in 1998 survey)

Part of a couple in which both spouses had 1998 interview data?

Couples with 1998 Interview Data
N = 6,989 couples
(13,978 individuals)

**Excluded N = 1,359 couples**
(proxy interview required for both spouses in n = 49, the male only in n = 1,304, and the female only in n = 274 couples)

Neither spouse required a proxy interview in 1998?

N = 5,630 couples
(11,260 individuals)

**Excluded N = 216 couples**
(one or both said not married and/or not living together at time of 1998 interview)

Both spouses reported being (legally) married and living together at time of 1998 interview?

N = 5,414
(10,828 individuals)

**Excluded N = 2 same-sex couples**

Filter out any remaining same-sex couples

continued on next page
N = 5,412 couples (10,824 individuals)

**STEP 5 (post-hoc filter)**
Excluded N = 15 couples (In each of these couples, 1 spouse was reportedly living in a nursing home)

N = 5,397 couples (10,794 individuals)

**STEP 6 (post-hoc filter)**
Excluded N = 9 couples (both spouses too young to be eligible for HRS study sample in 1998)

N = 5,388 couples (10,776 individuals)

**STEP 7 (post-hoc filter)**
Excluded N = 2 couples (lived outside US)

N = 5,386 couples (10,772 individuals)

N = 4,963 couples (one or both spouses not troubled by pain at time of 1998 interview)

**STEP 8**
Both spouses reported being "often troubled by pain"?

Y

N = 423 “Dual-Pain” couples (846 individuals)
Several post-hoc filters were applied in Steps 4-7. Use of the label “post-hoc” indicates that they were created after all but the final study selection criteria had been applied. The filters were needed to address several anomalous cases observed within the remaining set of 5,414 eligible couples. In Step 4, any remaining same-sex couples were excluded; 2 such couples were identified, reducing the remaining sample to 5,412 couples. Interview location data revealed 15 couples in which 1 spouse reported that he/she was living in a nursing home at the time of the 1998 interview. Step 5 filtered out these couples, leaving 5,397 married couples living together in the community at the time of the 1998 interview.

Preliminary analyses of the demographic and background characteristics of these couples revealed the need for two additional filters. First, analyses identified 9 couples in which neither spouse was old enough in 1998 to be “cohort-eligible” for inclusion in HRS (HRS, 2002). Once an individual participates in HRS, attempts are made to follow him/her longitudinally through various life events, including changes in marital status. With few exceptions, HRS attempts to recruit the new spouses/partners of study participants into the study, regardless of their age. These 9 “young” couples were comprised of former spouses of original HRS and AHEAD survey respondents and their new spouses—information verified by staff at RAND (RAND HRS Helpdesk, personal communication, March 19, 2010). These former spouses were quite young at the time of their initial inclusion in HRS/AHEAD (e.g., in their 30s or 40s); when they re-married, they married individuals of similar age. HRS is intended as a study of middle-aged and older adults. Younger individuals—initially recruited by HRS as the spouse of an age-eligible respondent—later become eligible for inclusion in HRS as members of specific
age-defined cohorts (e.g., War Babies, CODA). Such individuals are then folded into the HRS panel, along with other, newly-sampled, cohort-eligible participants. At the time of the 1998 interview, neither spouse in these 9 “young” couples was cohort-eligible for inclusion in HRS. Most would be considered members of the “Early Baby Boomer” (born 1948-1953) or an as-yet-unnamed cohort (born 1954 or later)—neither of which had been formally incorporated into HRS as of 1998. Since the focus of the present study was on middle-aged and older couples, these 9 couples were excluded in Step 6, reducing the number of eligible couples to 5,388.

Preliminary analyses related to location of residence revealed the need for the filter in Step 7. Two couples reported that, at the time of the 1998 HRS interview, they were living outside of the contiguous U.S. (i.e., the lower 48 states). HRS specifically samples only households in the contiguous U.S., but they do attempt to follow participants wherever they may move, including foreign countries (HRS Helpdesk, personal communication, June 2, 2010). Unfortunately, location data are masked in HRS, preventing further specification of the locations of residence of these couples (i.e., whether a U.S. territory or a foreign country). Given that these were the only couples reportedly living outside the contiguous U.S., and the effect of residence outside the contiguous U.S. on pain and its relationship to depressive symptoms is unknown, these couples were excluded from the sample. HRS staff concurred with this decision, especially since the present study was cross-sectional in nature (HRS Helpdesk, personal communication, June 2, 2010). At the end of Step 7, a total of 5,386 couples remained eligible for inclusion in the present study.

The final step (Step 8) involved consideration of the responses of both spouses
within each couple to the 1998 survey question about pain. *Both spouses reported being “often troubled with pain” in 423 couples; these dual-pain (DP) couples represented 7.9% of the 5,386 couples identified as initially eligible for inclusion in the present study. The response pattern of couples to this general HRS pain question is displayed in the Results chapter (Chapter 5), along with descriptive information about the focal sample of DP couples. Chapter 5 also provides information about the ways in which the 423 DP couples differed from those couples not included in the current study. Note that the Analysis Strategy section of the current chapter provides information regarding the amounts, patterns, and strategies used to address missing data in the present study.

**Measures**

This section reviews the measures used in this study. Measures are organized according to the conceptual model presented in Figure 5 (Chapter 3), moving first from consideration of the model’s outcome variable (depressive symptomatology), next to key explanatory variables in the model (pain and activity limitation), then to health-related characteristics (disease conditions and body weight), and finally to individual- and couple-level background characteristics (e.g., age, gender, household income). In addition to describing each of the measures, evidence from prior studies concerning the reliability and validity of each measure is also presented (if available). Reliability estimates obtained in the focal study sample of DP couples ($N = 423$) are presented for multi-item scales. Because of planned gender comparisons, reliability estimates (Cronbach’s alpha estimates of internal consistency) were calculated separately for husbands and wives. Note also that reliability estimates were averaged across multiply-imputed datasets (details about the multiple imputation process are presented later in this
chapter). Except for the couple-level background variables, all of the variables in the model were measured (or constructed) separately by HRS for each spouse. Appendix A contains additional details regarding each measure, including (where appropriate) the exact wording of HRS interview questions, the original response options, and information regarding any necessary recoding.

Outcome Variable

Psychological Distress (Depressive Symptomatology)

The psychological distress of each marital partner served as the outcome in this study. Psychological distress was assessed with an adapted version of a widely-used measure of depressive symptomatology—the Center for Epidemiologic Studies- Depression Scale (CES-D, Radloff, 1977). Beginning in 1998, HRS measured depressive symptomatology with an abbreviated version of the CES-D. This version of the CES-D contains 8 of the original 20 items: felt depressed, everything was an effort, restless sleep, happy, felt lonely, enjoyed life, felt sad, and could not get going. Each respondent was asked to indicate whether or not he/she experienced each of the eight symptoms “much of the time during the past week” using a simple yes (1) or no (0) response format. After reverse-coding the positively-worded items (happy, enjoyed life), a summary or total score can be created that ranges from zero to eight, with higher scores indicating greater depressive symptomatology.

This 8-item version of the CES-D, along with the yes/no response format and question stem, was taken from a 10-item version of the CES-D developed for use in the Epidemiological Studies of the Elderly (EPESE; Kohout, Berkman, Evans, & Cornoni-Huntley, 1993). Kohout et al. (1993) demonstrated that the psychometric properties of
the dichotomous 10-item CES-D were good and roughly comparable to those of the original 20-item measure. For example, the estimated internal consistency of the 10-item scale was .80, and scores on the 10-item scale correlated highly \( (r = .88, N = 2,339) \) with scores on the original 20-item scale that used 4 response categories. The 8-item CES-D used in HRS 1998 excludes two interpersonal items (“People were unfriendly,” and “I felt that people disliked me”) that were part of Kohout et al.’s 10-item scale. In empirical studies, these items tend to have some of the lowest item-total correlations and consistently yield low estimates of subscale internal consistency (e.g., Ensel, 1986).

Turvey, Wallace, and Herzog (1999) analyzed data from the 8-item CES-D in HRS 1998 for members of the AHEAD cohort only \( (N = 6133) \). These authors obtained an internal consistency estimate of .78, with item-total correlations ranging from .33 to .79. The criterion-validity of the 8-item CES-D was supported in an analysis showing that clinical “caseness” (based on six or more symptoms) on the 8-item CES-D was generally comparable to “caseness” based on a shortened version of the DSM-based Composite International Diagnostic Interview-Short Form (i.e., CIDI-SF). The analysis also suggested that the 8-item CES-D was more sensitive to symptoms that did not meet the duration and intensity levels required for a diagnosis of depression using the CIDI-SF, thus supporting its usefulness in detecting subclinical or subthreshold depressive symptomatology (as discussed in Chapter 2). The 8-item CES-D and the CIDI-SF performed similarly in terms of their associations with self-reports of medical illness, psychiatric diagnosis, and psychiatric treatment (Turvey et al., 1999).

Steffick (2000) created this 8-item CES-D in early waves of HRS and AHEAD. She calculated Cronbach’s alpha estimates of .81 and .83 for Waves 2 and 3 of HRS, and
.77 and .79 for Waves 1 and 2 of AHEAD. Gallo, Bradley, Siegel, and Kasl (2000) also created and analyzed an 8-item dichotomous CES-D measure in a sample of 3,116 respondents drawn from HRS Waves 1 and 2. They calculated Cronbach’s alpha coefficients (adjusted for short test length using the approach by Nunnally, 1967, p. 223), resulting in alpha estimates of .82 and .88, respectively.

There is some debate regarding the best fitting factor structure for the 8-item CES-D (indeed, even the factor structure of the 20-item version of the CES-D has been subject to debate). Steffick (2000) found evidence of a two-factor structure within the 8-item scale (i.e., depressed mood [5 items] and somatic complaints [3 items]), while Gallo and colleagues (2000) found support for a unidimensional model of depressive symptoms. Analyses by Turvey et al. (1999) also supported a two-factor solution (depressed mood and somatic complaints); however, they noted that the two factors were moderately correlated in the AHEAD study sample ($r = .43$).

The issue of whether or not to distinguish somatic complaints from mood-oriented depressive symptoms has also been a controversial conceptual issue, debated within both the gerontological literature and the literature on chronic pain. For example, some have suggested that the use of self-report measures of depressive symptoms that include somatic complaints may produce artificially elevated scores among older adults who experience increased incidence of somatic symptoms associated only with increased age, physical illness, and/or medication side-effects (e.g., Berry, Storandt, & Coyne, 1984; Steur, Bank, Olsen, & Jarvik, 1980). However, an analysis by Foelker and Shewchuck (1992) demonstrated that scores on the CES-D increase in response to increased depressive symptomatology among medically-ill persons, but don’t necessarily lead to
“false positives” attributable to symptoms unrelated to depression. A review by Devins and Orme (1988) suggested that the CES-D has less of an emphasis on somatic symptoms than do other self-report measures of depression, and that this feature makes the CES-D valuable for use in research with persons with medical conditions. A study by Hertzog, Van Alstine, Usala, Hultsch, and Dixon (1990) also found little evidence of a biasing influence of age on the endorsement of somatic symptoms measured by the CES-D. A subsequent study using the CES-D in a sample of frail older persons found that neither age nor health status appeared to bias scores on the somatic items; furthermore, participants’ total scores were not disproportionately influenced by their responses to the somatic items (Davidson, Feldman, & Crawford, 1994).

Together, these and other studies offer support for the validity of the CES-D, and have led many authors to recommend the use of the CES-D with older adults and persons with chronic health conditions. For example, Bradley and colleagues (Bradley, 1994; Bradley & McKendree-Smith, 2001) recommend the CES-D for use in studies of persons with chronic diseases that produce a lot of somatic disturbances (e.g., rheumatological disorders). More recently, Parmelee (2005) suggested that the CES-D may be a preferred measure for use with community-dwelling older adults, resulting in less missing data and closer correspondence to diagnosed depression than measures like the Geriatric Depression Scale.

Some chronic pain scholars have raised similar arguments about the biasing effect of including somatic complaints in self-report measures of depressive symptoms (e.g., Dohrenwend, Raphael, Marbach, & Gallagher, 1999; Taylor, Lovibond, Nicholas, Cayley, & Wilson, 2005). Empirical work by Geisser and colleagues examined both the
CES-D and another self-report measure—the Beck Depression Inventory (BDI)—in a sample of chronic pain patients (Geisser, Roth, & Robinson, 1997). They found that both the CES-D and the BDI were able to discriminate between patients with and without major depression (as measured by DSM-IV diagnostic interview) at a rate significantly better than chance. They examined the performance of the somatic items on both scales and observed that the depressed pain patients displayed significantly more somatic depressive symptoms than the non-depressed pain patients. Furthermore, the differences between the two pain patient groups on the somatic subscales were similar in magnitude to those observed on other affect-oriented or cognitive depressive symptom subscales.

Even more importantly, when Geisser et al. removed the somatic items from the calculation of the total scale scores for both the CES-D and the BDI, they found that the accuracy of the measures’ classification of patients as depressed or not depressed actually decreased slightly. The authors concluded that somatic items contained on these scales do, in fact, contribute to the identification of significant depressive symptomatology among persons with chronic pain. Geisser et al. did note that higher cutoff scores may be necessary when using the CES-D or the BDI in chronic pain populations to screen for major depression, in order to optimally balance the numbers of false positives and false negatives. In comparing the two measures, these authors found that the CES-D had better sensitivity and a better negative predictive value than the BDI when used as a screening tool for major depression in chronic pain patients. The CES-D is also the measure of depressive symptomatology most often recommended for use in studies of older persons and pain (Gibson, Katz, Corran, Farrell, & Helme, 1994).

Thus, evidence from both the gerontological and chronic pain literatures supports
the use of the CES-D measure with older adults and persons with pain problems. It further suggests that concerns about the biasing effects of the somatic items may be overstated. It is also important to note that, even though 3 items on the CES-D8 can comprise a symptom cluster that has been labeled “somatic,” two of these items tap symptoms that are not purely somatic in nature. For example, the items “felt like everything was an effort” and “could not get going” largely reflect the motivational aspect of depressive symptomatology—an aspect of depression that many other measures do not include (Shafer, 2006).

In the current study, the 8-item CES-D scale was assumed to comprise a single-factor model of depressive symptomatology. Several lines of evidence converge to support the decision to treat these items as a unidimensional construct and, therefore, to use a total depressive symptomatology score in all analyses. The magnitude of the correlation between the somatic and mood factors observed by Turvey et al. (1999) suggests the strong possibility that the two factors represent an over-arching, higher-order construct (i.e., depressive symptomatology). Gallo et al. (2000) demonstrated that a single-factor model can fit the 8-item CES-D data in HRS reasonably well. In addition, both Steffick (2000) and Turvey et al. (1999) provide evidence that a total “depressive symptomatology” score derived from these eight items yields good internal consistency and construct validity. Finally, it is useful to remember that the original CES-D was designed to measure a variety of indicators of a single construct—depressive symptomatology—and that even the scale’s author suggested that it was best used as a total score (Radloff, 1977). HRS also instructs users to calculate a CES-D8 “summary score ranging from zero to eight, created by summing the number of ‘yes’ answers across
the eight items (with the positive items reverse-scored)” (Steffick, 2000, p. 6).

A total score was thus constructed for the 8-item CES-D scale in the present study. The internal consistency (Cronbach’s alpha) of the measure was estimated at .75 among DP husbands, and .80 among DP wives. These estimates indicate respectable internal consistency for the measure (DeVellis, 1991), and are in line with estimates reported in prior studies (e.g., Steffick, 2000).

As will be shown in the Results chapter, the distribution of total CES-D8 depressive symptomatology scores was positively skewed in this sample, with most husbands and wives reporting a relatively low number of symptoms. Significant positive skewness of CES-D scores is commonly encountered in most community- and population-based studies. Yet, very few researchers attempt any distributional correction of scores prior to data analysis. Most publications documenting the reliability and validity of shortened, dichotomously-scored CES-D scales, including the 8-item CES-D (e.g., Steffick, 2000; Turvey et al., 1999), have been conducted using untransformed total scores. Derived variables representing total scores on the CES-D8 are readily available in most of the publicly-released HRS datasets, as well as in the RAND HRS dataset. In addition, numerous studies using untransformed total CES-D8 scores have been published in prominent journals, including some by leading investigators affiliated with HRS (e.g., Fonda, Wallace, & Herzog, 2001; Louh & Herzog, 2002; Turvey, Schultz, Arndt, Wallace, & Herzog, 2002).

Several researchers have drawn attention to the problematic distribution of CES-D8 scores in HRS. The solutions enacted by these researchers, however, have varied considerably. Some have opted to create a dichotomous variable indicating the presence
(or absence) of “significant” depressive symptoms (e.g., Choi & Kim, 2007; Keating, Norredam, Landrum, Huskamp, & Meara, 2005). There is disagreement, however, on the specific cut-point (e.g., 3, 4, or 6 symptoms) that should be used to create the dichotomous variable. There is also debate surrounding the utility of cutoff scores on the original CES-D (e.g., Radloff & Teri, 1986; Devins & Orme, 1985), as well as on other shortened and/or dichotomously-scored versions of the measure (e.g., Santor & Coyne, 1997; Tuunainen, Langer, Klauber, & Kripke, 2001). Dichotomous CES-D8 total scores also have the undesirable effect of limiting the variability in scores, which, in turn, can limit the ability to explain more subtle variability in depressive symptomatology.

In their work with HRS data, Gallo and colleagues readily acknowledged the pronounced skewness of CES-D8 total scores. In one study based on an ordinary least squares (OLS) regression analysis of the untransformed total CES-D8 scores, the authors reported the results of two alternate analyses—an OLS regression using log-transformed scores, and a Poisson regression: “In both cases, the results were not qualitatively different from the reported estimation results using least squares estimation” (Gallo et al., 2000, p. S135). In a subsequent study, the research group reported results using a log transformation of CES-D8 total scores, although they noted that the distribution of transformed scores was still skewed (Siegel, Bradley, Gallo, & Kasl, 2004). Sensitivity analyses revealed that various transformation and normalization strategies had little effect on their estimation results.

Given the debate surrounding this measure, a sensitivity analysis was incorporated into the present study. An alternative version of the measure was created by applying a natural log transformation (natural log) to respondents’ CES-D8 scores (after
adding a constant of 1 to move the lowest scores off of zero). A log transformation was selected over other possible transformations, in part because it could be readily incorporated into the multivariate analysis strategy. The log-transformed CES-D8 scores were then analyzed in parallel with the untransformed scores. Results from these sensitivity analyses are presented in the Results chapter.

Key Explanatory Variables

Pain

Pain was a central construct of interest in this study. In both research and clinical practice, an individual’s self-report is generally regarded as accurate, reliable, and sufficient evidence of the existence of pain and its intensity (Turk & Melzack, 2001a). Indeed, self-report has long been regarded as the “gold standard” for understanding the subjective experience of pain (Craig, 1992; Craig & Hadjistavropoulos, 2004). As previously mentioned, HRS has only one question asked of all respondents regarding pain. In HRS 1998, all respondents were asked, “Are you often troubled with pain?” Respondents could answer “yes” (1) or “no” (0). The exact origin of this particular item is unknown, although one of the HRS principal investigators has suggested that the item may have derived from some early, community-based epidemiological studies of older adults (i.e., the EPESE studies; Robert Wallace, personal communication, December 15, 2005). However, this particular item does not appear in early EPESE publications that focused on pain (e.g., Herr et al. 1994; Lavsky-Shulan, Wallace, Kohout, Lemke, Morris, & Smith, 1985).

An early study of community-dwelling adults in Canada by Crook, Rideout, and Browne (1984) did use this same question (“Are you often troubled with pain?”), but in
combination with a question about whether or not they had experienced noteworthy pain in the past 2 weeks. Respondents’ answers to the pair of questions was used to identify those with “persistent” pain (i.e., those who responded “yes” and “yes”) and those with “temporary” pain (i.e., those who responded “no” and “yes”). For a randomly-selected subsample of 123 respondents, the authors then compared the resulting classification of respondents’ pain complaints with blind, independent assessments made by two clinical nurse specialists. The level of agreement between the classifications was high (81%). All respondents whose self-reports categorized their pain as “persistent” were also categorized as such by the nurse assessors; errors most often resulted from the nurses’ over-assessment of the persistency of respondents’ pain (classifying more than half of the respondents whose self-reports indicated “temporary” pain as “persistent”).

In the larger Crook et al. (1984) study sample (N = 500), 31.6% of adults reported some pain (i.e., responded “yes” to either question). The majority of these respondents (57.0%) reported that they were both often troubled with pain and had experienced noteworthy pain in the past two weeks; 27.2% reported experiencing noteworthy pain in the past two weeks, but did not report being often troubled with pain; and only 15.8% reported being often troubled with pain, but did not report experiencing noteworthy pain in the preceding two weeks. Together, these data suggest that: a) persons who endorse being “often troubled” by pain seem to regard their pain as something that is serious and enduring; b) judgments about the persistence of one’s pain show a high degree of concordance with judgments by medical professionals; and c) if anything, people “do not appear to exaggerate the persistence of their pain” (p. 313), but instead appear to overemphasize the temporary nature of their pain.
To this author’s knowledge, there are no published reports regarding the validity of this single item when used by itself to examine pain experiences, especially among middle-aged and/or older persons. It is interesting to note, however, that this item—modified slightly to read, “Are you regularly troubled with pain?”—has been incorporated periodically into the British Household Panel Survey, but only for respondents over age 50 (British Household Panel Survey, 2013). Evidently, some community-based researchers studying mid and later life have confidence that this relatively simple item generates useful information.

Pain Intensity

Pain intensity—a quantitative estimate of the severity or magnitude of pain—is one of the most commonly and easily assessed dimensions of pain (Jensen & Karoly, 2001). Intensity is also regarded as the most salient dimension of pain (Cleeland, 1989; Turk & Melzack, 2001a). In HRS 1998, respondents who reported that they were often troubled with pain were asked a follow-up question, “How bad is the pain most of the time?” Response options were “mild” (1), “moderate” (2), or “severe” (3). This question stem and response format is often referred to in the literature as a Verbal Descriptor Scale (VDS) or a Verbal Rating Scale (VRS) for pain intensity, because it uses words or verbal descriptions to indicate level of intensity (Jensen & Karoly, 2001).

Since there is no definitive measure of pain intensity, it is difficult to establish the criterion validity of verbal descriptor scales of pain intensity. However, verbal descriptor scales have been shown to be related to other measures of pain intensity, including numerical rating scales, visual analogue scales, vertically-oriented pain “thermometers,” and scales depicting pain intensity levels via a series of line-drawn faces (e.g., Jensen,
Karoly, & Braer, 1986; Woodforde & Merskey, 1972). Strong correlations between VDSs and other scales have also been observed in samples of middle-aged and older adults (e.g., Herr, Spratt, Mobily, & Richardson, 2004; Taylor & Herr, 2003). Research has also shown that VDS scores are sensitive to changes in pain intensity as a result of intervention (e.g., Onhaus & Adler, 1975). VDSs are easy to comprehend and generally have good completion rates across a variety of different groups, resulting in relatively low levels of missing data (Jensen & Karoly, 2001). Some research also suggests that verbal descriptor scales are preferred by older adult respondents (Herr & Mobily, 1993; Herr et al., 2004; Taylor, Harris, Epps, & Herr, 2005).

The wording of the pain intensity item in HRS suggests that respondents should reflect on their average or usual level of pain over some (unspecified) period of time. This raises issues about the reliability and validity of such “average” or “most of the time” ratings of pain intensity. Specific studies of the reliability and validity of such reports among middle-aged and older community-dwelling older adults are lacking in the literature. The extent to which pain and its intensity are dynamic phenomena among those in mid and later life is unknown, and this creates problems in attempting to measure the reliability of pain intensity ratings. However, studies have been done with chronic pain patients and with community-dwelling adults of all ages with pain. Based on their research with chronic pain patients, Jensen, Turner, Turner, and Romano (1996) concluded that patients’ ratings of their “average” or “usual” pain are, for the most part, adequately valid—providing “adequate estimate[s] of average pain for most research purposes…” (p. 39). For example, they found that patients’ recall of their “usual” pain over the past two weeks was highly correlated ($r = .78$) with the average pain ratings
calculated from their hourly pain diaries. Similar results were observed in a 3-month
diary study of migraine patients (Stewart, Lipton, Kolodner, Liberman, & Sawyer, 1999).
A study of 40 community-dwelling adults with pain problems lasting 6 months or more
found that ratings of pain intensity were very consistent over a one-month interval
(average $r = .74$; Salovey, Sieber, Smith, Turk, Jobe, & Willis, 1992). Together, these
and other studies suggest that average or usual pain reports are generally accurate, stable,
and valid—at least among adults who are either chronic pain patients or could be
considered to have chronic pain.

There is ongoing debate within the chronic pain literature regarding the “optimal”
number of points or levels to use on pain intensity scales (e.g., Anderson, 2005; Jensen,
Turner, & Romano, 1994). Most chronic pain researchers advocate for the use of more
finely-graded pain intensity scales and even multiple-item scales (e.g., Jensen et al.,
1996; Von Korff, Jensen, & Karoly, 2000), mainly because of ceiling effects observed in
the pain intensity ratings of many chronic pain patient populations (e.g., Cleeland, 1989).
However, studies of community-dwelling persons not identified as chronic pain patients
tend to rely on scales with fewer levels of severity/magnitude. Three- and four-category
pain intensity measures have been used in epidemiologic studies of pain in the general
population, such as the Southern Pain Prevalence Study (Hitt, McMillen, Thornton-
Neaves, Koch, & Cosby, 2007), and also in national polls reported in the popular press
(e.g., ABC News, USA Today, & Stanford University Medical Center, 2005).

It is also not unusual for researchers in community-based studies using 7- or 10-
point pain intensity scales to collapse these into 3 or 4 categories for the analysis. For
example, Scudds and Robertson (2000) used a 7-point verbal descriptor scale (ranging
from no pain to the worst pain imaginable) in a community-based sample of older adults. However, the authors collapsed the measure into 3 categories of intensity for all analyses involving persons reporting any pain: slight/mild; moderate; and severe/extreme/worst imaginable. Presumably, this was done to yield a variable with adequate distribution of cases for analysis, as over one-half (51%) of persons responded at the scale midpoint--reporting their pain as moderate in intensity. Slightly more than one-quarter (28%) characterized their pain as slight or mild, and just over one-fifth (21%) rated their pain as severe, extreme, or the worst imaginable.

Pain intensity measures with three or four points have been shown to have good validity and research utility. Three- and four-point verbal descriptor scales have been shown to be positively and significantly related to other measures of pain intensity (e.g., Jensen, Karoly, & Braver, 1986; Paice & Cohen, 1997). The 3-category scale created by Scudds and Robertson (2000) worked well in their study of older, community-dwelling adults—showing evidence of good predictive validity with respect to reports of physical disability due to pain. A review by Max (2003) noted that four-point pain intensity scales (ranging from “none” or “no pain” to “severe”) are often adequate in clinical trials to gauge the relative effectiveness of different analgesics. Three- and four-point scales have also demonstrated sensitivity to the effects of other pharmacologic and psychological treatments known to affect pain intensity (e.g., Ohnhaus & Adler, 1975).

Disease-Specific Pain

HRS 1998 does include some limited information regarding the location and/or type of pain experienced by respondents. Questions about specific pain were asked of individuals who reported certain disease conditions (the disease conditions are described
in detail later in this chapter). Those with arthritis were asked whether they sometimes had pain, stiffness, and swelling in their joints. Individuals whose responses indicated that they had “active” heart disease—defined as having seen a doctor for the condition in the past two years, had a heart attack or experienced other heart symptoms in the past two years, or were currently taking or carrying heart medication—were asked whether they had experienced recent heart or chest pain. Note that these questions were asked of respondents with these disease conditions (for heart disease, only those with “active” indicators of the disease), regardless of their answer to the general pain question. Both arthritis and heart/chest pain were coded as either present (1) or absent (0).

**Activity Limitation (Mediating Variable)**

As noted in Chapter 2, there is considerable inconsistency in the terminology used by various scholars to refer to the limitations that individuals may experience in their attempts to complete basic physical tasks and everyday activities. The term “activity limitation” was used in the current study in order to avoid confusion and to maintain a basic level of similitude with existing conceptual models of the relationship between pain and depressive symptomatology (e.g., Williamson & Schulz, 1992a, 1995). Physical limitations were used as indicators of activity limitation in the present study. Physical limitations are difficulties that individuals have in completing basic physical tasks. As discussed in Chapter 2, available theory and empirical evidence suggest that these limitations occur relatively early in the disablement process (Verbrugge & Jette, 1994; Fried & Guralnik, 1997). The middle-aged and older spouses in HRS were far more likely to experience these basic limitations in physical functioning than the severe functional difficulties tapped by measures of ADL and IADL limitations.
HRS 1998 respondents were asked, “Because of a health problem do you have any difficulty with [list of activities].” The activities included: (1) walking several blocks; (2) walking one block (skipped if no trouble walking several blocks); (3) sitting for about two hours; (4) getting up from a chair after sitting for long periods; (5) climbing several flights of stairs without resting; (6) climbing one flight of stairs without resting (skipped if no trouble with several flights); (7) stooping, kneeling, or crouching; (8) reaching or extending your arms above shoulder level; (9) pulling or pushing large objects like a living room chair; (10) lifting or carrying weights over 10 pounds, like a heavy bag of groceries; and (11) picking up a dime from a table. Response options included yes, no, can’t do, don’t do, don’t know, and refused. Because the goal was to assess respondents’ usual level of functioning (or, conversely, limitations) and to avoid attributing transient difficulties (e.g., those resulting from a recent injury or illness) to more permanent decline, the instructions directed respondents to “exclude any difficulties that you expect to last less than three months.”

Some of these items display a hierarchical dependence on one another, and conditional coding of responses is used by HRS to generate final values. For example, if a respondent reported no difficulty with item 1 (walking several blocks), then item 2 (walking one block) was not administered and the respondent was automatically coded as having no difficulty on item 2. It should be noted that the question sequence in HRS included one additional item (inserted between items 1 and 2 above): difficulty “running or jogging about a mile.” The running/jogging item was skipped if a respondent reported difficulty walking several blocks. This item was not included in the RAND HRS Datafile, partly because it was not asked in early waves of AHEAD. The original item
was retrieved from the RAND HRS 1998 FAT File and examined in relation to the other items. As a result of the skip pattern embedded in the question sequence, this item was missing for more than 60% of respondents. In addition, among those who were asked, very few respondents indicated that they had no difficulty running or jogging a mile. Consequently, the “jog” item was excluded from the present study—a decision consistent with the approach taken by most other researchers using HRS/AHEAD data (e.g., Jenkins, 2004; Wallace & Herzog, 1995; Wray & Blaum, 2001), including RAND.

These 11 items measure a broad range of basic physical abilities, including those requiring adequate functioning of muscles and joints in the upper-body (e.g., fingers, arms, shoulders) and those in the lower body (e.g., hips, legs, feet; Nagi, 1971; Wallace & Herzog, 1995). The items also measure strength, stamina, and flexibility. Some of the items require gross motor control (e.g., walking), while others require fine motor control (e.g., picking up a dime). HRS items were patterned after—or, in some cases, taken directly from—items originally developed by Nagi (e.g., 1976) and a scale developed for use in gerontological research (Rosow & Breslau, 1966). Similar items have been used in other large-scale, community-based studies, including the EPESE, and the Framingham Heart Study. These items are generally consistent with the construct of “functional limitations,” as depicted in the models of physical health and disability developed by Nagi (e.g., Nagi, 1991) and Verbrugge and colleagues (e.g., Verbrugge & Jette, 1994). However, they have been given a variety of different labels in prior research, including “functional limitations,” “functional impairments,” “physical limitations,” “physical functional limitations,” “physical disabilities,” “physical difficulties,” and “mobility (and/or strength) difficulties.” In addition, the items can be coded by researchers to
represent either limitations/difficulties or abilities. Again, in an effort to avoid confusion and be consistent, these items will hereafter be referred to as physical limitations.

Some researchers choose to differentiate conceptually and operationally between items that measure lower-body limitations such as climbing stairs and those that measure upper-body limitations such as picking up a dime (e.g., Johnson & Wolinsky, 1993; Lawrence & Jette, 1996). There is, however, some debate about the placement of specific items within this framework. For example, Lichtenstein et al. (1998) and Simonsick et al. (2001) classified lifting (item 10) as an upper-body activity, while Wolinsky and colleagues (e.g., Johnson & Wolinsky, 1993; Wolinsky & Johnson, 1991) and Lawrence and Jette (1996) classified it as a lower-body activity. Others have reported cross-loadings of items from the upper- and lower-body scales, low internal consistency estimates, and/or poor model fit when items are divided into separate scales (e.g., Clark et al., 1997; Stump et al., 1997; Wolinsky & Johnson (1991). Stump et al. (1997) and Clark et al. (1998) have also noted that the performance of some activities (e.g., pull/push heavy objects, carry 10 lbs.) may require both upper- and lower-body mobility/dexterity and strength. Johnson and Wolinsky (1993) further suggest that lower-body limitations can reflect systemic impairment (e.g., poor circulation, low oxygen), not just problems with lower-body physiology, and can thus be influenced by many diseases (e.g., diabetes, lung disease), not just those that directly impact the lower extremities.

Given these issues, Long and Pavalko (2004a, 2004b) argue against creating separate measures of upper- and lower-body limitations. They instead suggest that a single, composite measure of physical limitations works just as well, if not better, in many instances. For example, when plotting the prevalence of physical limitations by
age, Long and Pavalko (2004b) found that patterns of difficulty for many of the activities typically categorized as lower-body limitations closely matched those typically categorized as upper-body limitations. When used to predict future disability in activities of daily living (ADL) and work-related disability, they found that a single, combined measure performed better (i.e., showed more consistent and stronger relationships) than did separate measures of upper- and lower-body limitations.

The major limitation of the work by Long and Pavalko is that their studies were based only on data from females drawn from the National Longitudinal Survey (NLS)—a sample of women in midlife and early late life (age 37-68). However, evidence supporting their position can also be amassed from several other empirical studies, along with some conceptual work by scholars studying physical health and aging. For example, Gallo, Bradley, Siegel, and Kasl (2000) created a summed “physical functioning” score derived from some of these same HRS items in addition to some ADL items. A series of exploratory factor analyses supported the creation of a single, total score, and the resulting measure exhibited acceptable internal consistency in their sample (alpha = .74). Klein et al. (2004) created a single, summed “mobility impairment” score that incorporated most of these HRS items (they excluded the “picking up dime from table” item, but included the “jog” item) and reported acceptable psychometric properties. Scholarly examinations of other brief measures of health that use items similar to those used in HRS also buttress this position—e.g., see Hays et al. (2007) for discussion of the psychometric qualities of, and empirical support for, a unidimensional measure of physical functioning derived from the SF-36.

Results of both exploratory and confirmatory factor analyses of the 11 HRS
physical limitation items have provided support for treating the items as representing a single, underlying construct (Fonda & Herzog, 2004; Johnson & Wolinsky, 1993). For example, an exploratory factor analysis by Fonda and Herzog (2004) found support for a single factor underlying the “mobility, strength, and fine motor skills” items across five waves of HRS and two early waves of AHEAD. Although they noted that one item (“picking up a dime”) did not always load consistently or adequately with the other items, these authors still included this item when creating a “mobility, strength, and fine motor skills” summary score. This was reportedly done for both theoretical reasons and to be consistent with prior studies.

Researchers using the physical limitation items in HRS can make different decisions in coding the six response options (e.g., “yes [have difficulty],” “can’t do,” “don’t do,” “no [no difficulty],” “don’t know,” and “refused”). These decisions generally involve the handling of the “can’t do” and “don’t do” responses. For example, some researchers might exclude from further analysis any individuals who say that they “don’t do” a particular activity, arguing that a “don’t do” response may indicate that the respondents do not have the opportunity to engage in the behavior (e.g., climb a flight of stairs) and should therefore be excluded from any analyses involving that item. However, when applied over the entire set of HRS items, this strategy could potentially result in considerable missing data. Moreover, an equally compelling argument could be made that individuals report that they “don’t do” these activities largely because they cannot do them, and therefore avoid doing them. In order to maximize the use of data provided by respondents and to capture those responses that suggest any difficulty with a physical task, the “can’t do,” “don’t do,” and “yes” responses was coded as 1 (any
difficulty) in the current study. The “no” category was coded as 0, indicating no difficulty. Only those with “don’t know” or “refused” responses were considered to have missing data on these items. Creating dichotomous indicators that code for the presence of “any” difficulty is a strategy used by many researchers who regularly work with the physical limitation items in HRS (e.g., Clark, Stump, Hui, & Wolinsky, 1998; Gallo et al., 2000; Klien et al., 2004) and also a strategy commonly used in other studies (e.g., Cronin-Stubbs et al., 2000; Wolinsky et al., 2005).

In the current study, respondents’ answers to these 11 dichotomously-coded items were summed to yield a total number of self-reported physical limitations, ranging from 0 to 11. Creation of a summed score was consistent with the work of Fonda and Herzog (2004), cited above. Estimates of internal consistency (Cronbach’s alpha) were .80 for DP husbands and .82 for DP wives, indicating good internal consistency for the measure in this sample (DeVellis, 1991). These estimates were also consistent with those obtained in prior studies. For example, Fonda and Herzog (2004) reported internal consistency estimates for the mobility, strength, and fine motor skills summary scale of .81 to .87 across five waves of HRS. Similar internal consistency estimates have been reported by Garman et al. (2003) and Tager, Swanson, and Satariano (1998). Numerous studies have helped to establish the construct validity of the physical limitation items in HRS, as well as the derived summary score (see review in Fonda & Herzog, 2004).

Evidence from studies using other datasets also supports creating a total count of physical limitations derived from dichotomously-scored items. For example, Long and Pavalko (2004a) compared different strategies for assessing and scoring self-reported physical limitations in the NLS. Their findings showed that a physical limitation scale
created by summing dichotomous indicators of any difficulty worked as well as, or better than, scales created using multiple, ordered categories (e.g., no, some, lots) of difficulty, both in terms of having acceptable psychometric properties and in replicating known relationships with other constructs. Together, these studies provide considerable support for using a total, summed score. Although the distribution of physical functioning in most middle-aged and older samples is typically somewhat skewed, most researchers tend to use untransformed total scores in analyses (e.g., Clark et al., 1998; Freedman & Martin, 2000; Wray & Blaum, 2001).

Multiple lines of evidence offer broad support for the use of self-reported physical limitations data. Self-reported limitations in physical activities have been shown to be consistently and substantially related to performance on standardized, objective tests of physical functioning (e.g., Guralnik et al., 1994; Hazuda, Dhanda, Owen, & Lichtenstein, 2005; Simonsick et al., 2001). There is also evidence that self-reported physical limitations (like those assessed in HRS) are especially sensitive to early declines in physical functioning and, for many middle-aged and older persons, signal the beginnings of the disablement process (e.g., Alexander et al., 2000; Femia, Zarit, & Johansson, 2001; Fried, Ettinger, Lind, Newman, & Gardin, 1994; Fonda & Herzog, 2004; Fried & Guralnik, 1997; Fried, Young, Rubin, & Bandeen-Roche, 2001). Self-reported physical limitations have also been consistently related to other indicators of health, including self-rated health (e.g., Femia et al., 2001), numerous disease conditions (see review by Fried & Guralnik, 1997), and total number of disease conditions (e.g., Wallace & Herzog, 1995; Wolinsky, Miller, Andresen, Malmstrom, & Miller, 2005).
Health-Related Characteristics

Disease Conditions

In HRS 1998, respondents were asked “Has a doctor ever told you that you have [list of 7 conditions]?” The list included arthritis, chronic lung disease (e.g., chronic bronchitis, emphysema), diabetes, cancer (excluding minor skin cancers), hypertension, heart disease, and stroke. For most conditions, the phrasing of the question included a lay description of the condition—for example, “diabetes or high blood sugar” and “high blood pressure or hypertension.” These seven medical conditions were selected for HRS because of their public health significance in terms of their prevalence among middle-aged and older adults, their predictable impact on functional outcomes, and their amenability to prevention and intervention efforts (Wallace & Herzog, 1995). Although follow-up questions were asked about the history, recent symptoms, and current treatments for some (though not all) of these conditions, such follow-up information was not used in the current study. Instead, the current study relied on indicators of the presence of each of these seven conditions and a summary index of the number of conditions. Although theoretically, this index could range from zero to seven, this measure was top-coded at four or more conditions because relatively few husbands and wives reported more than four conditions. Because this measure was regarded as an index, no internal consistency estimate was calculated in this sample.

Several studies offer support for both the reliability and validity of information on chronic disease conditions reported by older persons (Bush, Miller, & Golden, 1989; Kehoe, Qu, Leske, & Chylack, 1994; Kriegsman, Penninx, van Eijk, Boeke, & Deeg, 1996). However, some researchers have found that the extent of agreement
between self-report measures of chronic disease and more “objective” measures (e.g., medical record review, physician diagnosis) may vary depending on the specific disease condition being examined, as well as the characteristics of the respondents. For example, extent of agreement is generally better for well-defined diagnoses, such as diabetes (e.g., Lampe, Walker, Lennon, Whincup, & Ebrahim, 1999). Also respondents with higher education and/or higher SES tend to evidence higher levels of agreement (e.g., Colditz et al., 1986). Reliability or consistency of self-report also seems to vary somewhat by disease condition—with greater consistency observed in reports of well-defined diseases (e.g., cancer) and/or those that involve current treatment (e.g., diabetes, hypertension; e.g., Becket, Weinstein, Goldman, & Yu-Hsuan, 2000).

Asking individuals to report diseases or conditions diagnosed by a physician, (as is done in HRS) is likely to yield more “objective” and valid data than just asking individuals to report whether or not they currently have a disease or condition. In general, self-reports of physician-diagnosed conditions have demonstrated good criterion validity. For example, in a sample of disabled older women, Simpson, Boyd, Carlson, Griswold, Guralnik, and Fried (2004) found that self-report of physician diagnosis of several conditions (e.g., cancer, stroke, Parkinson’s, diabetes) evidenced high levels of agreement with classifications derived from medical record data.

There is also evidence of the validity and utility of counts or indices derived from totaling the number of disease conditions reported by respondents. Fisher, Faul, Weir, & Wallace (2005) compared the frequency distribution of an index created from the number of physician-diagnosed disease conditions reported by respondents in HRS 2002 to the distribution observed among respondents over age 55 in the 2002 National Health
Interview Survey (NHIS). The two distributions looked remarkably similar, supporting the validity of this measure. Although a more detailed index that also incorporates an assessment of the severity of the disease (e.g., Charlson Comorbidity Index; Charlson, Pompei, Ales, & MacKenzie, 1987) might be preferable, the HRS 1998 dataset does not have the depth of data for all conditions to support the creation of such an index.

**Body Weight**

Currently, the most commonly-used measure of body weight, especially in community-based research, is body mass index (NHLBI, 1998; WHO, 2000). Body mass index (BMI) is a measure of body weight relative to height, and as such is a commonly-used indicator of both excess body weight (or adiposity or excess body fat relative to lean muscle mass) and extreme leanness or low body weight. RAND converted the self-reported height and weight of HRS respondents to metric units. BMI was then calculated as weight divided by height squared (kilograms/meters$^2$)

Valid self-report of these data depends on respondents’ knowledge of their current weight and height and also their willingness to report accurate values. As has been pointed out by some researchers (e.g., Schoenborn et al., 2002), middle-aged and older adults may not have had a recent physical examination at which measures of height and weight were taken (and reported to them). In addition, weight can fluctuate frequently in adulthood (Schoenborn et al., 2002).

In general, validation studies have demonstrated that self-reported weight and measured weight tend to be highly correlated (e.g., Spearman $r = .96$ in Rimm et al., 1990), and self-reported height is also correlated with measured height (with $r_s$ generally between .80 and .85; Kuczmarski, Kuczmarski, & Najjar, 2001). Although studies
generally support the validity of self-reported weight and height, self-reports do appear to be subject to systematic bias. For example, obese people tend to underreport their weight and underweight persons tend to over-report their weight (Kuskowska-Wolk, Bergstrom, & Bostrom, 1992). In general, men tend to over-report their weight and women tend to underreport it (e.g., Kuczmarski et al., 2001). People have also been shown to over-report their height. There is evidence of some height shrinkage during middle and old age (e.g., Cline, Meredith, Boyer, & Burrows, 1989); and perhaps because of this shrinkage, older adults tend to over-report their height more frequently than do younger adults (Rowland, 1990). Errors in self-reported height have been shown to be directly related to age, and unreliability increases steadily with increased age (Kuczmarski et al., 2001; Rowland, 1990). Taken together, these findings suggest that the BMI estimates in the current sample were likely to be somewhat conservative estimates of actual BMI. Ultimately, this might be expected to yield underestimates of the relationships between BMI and other study variables. The general direction and trend of these relationships, however, was expected to be valid.

BMI has been found to be positively correlated with total body fat content (NHLBI, 1998). BMI is also regarded as the current, most practical and least expensive measure of excess weight/body fat. Other existing measures (e.g., dual-energy x-ray absorptiometry) are more resource-intensive, and have not been demonstrated to be more effective for research purposes than BMI (NHLBI, 1998). As was discussed in Chapter 2, higher body weight (especially levels above the normal range) has been found to be associated with pain reports and pain intensity levels, physical limitations, and a variety of other background characteristics.
BMI was used primarily as an interval-level variable in most analyses. However, BMI categories were also created for use in describing the sample. BMI values were grouped according to the four-category scheme outlined by the World Health Organization (WHO, 2000). Underweight (< 18.5), normal weight (18.5 to 24.9), overweight (25.0 to 29.9), and obese (≥ 30.0). These cutoff values were derived from empirical studies of the association between BMI and risks of morbidity and mortality. This classification scheme was designed to be applied to all adults, regardless of age, gender, race, and ethnicity (NHLBI, 1998).

**Background Characteristics**

The individual- and couple-level background characteristics listed below were included in the proposed conceptual model because studies have shown them to be related to one or more of the key constructs in the study (e.g., pain, activity limitation, and depressive symptomatology), as described in Chapters 2 and 3. Some characteristics are also included because they are known to be related to other background and health-related characteristics. For example, household income is related to the age, gender, education, and race/ethnicity of the head of the household; and, gender, age, and race/ethnicity are related to body weight. In addition, as was outlined at the end of Chapters 2 and 3, exploratory research questions at both the *intra-* and *inter-*individual levels have been developed around gender.

Most of the individual-level background characteristics included in this study were derived from the baseline interview conducted with respondents. For respondents (and their spouses) in the initial HRS and AHEAD cohorts, these data were collected in the 1992 HRS or 1993 AHEAD surveys. Data from the newly-recruited WB and CODA
cohort members (and their spouses) were obtained during the HRS 1998 survey. Most
couple-level background data (e.g., household income, others living in household) were
obtained at each wave—in this case, from the HRS 1998 interview. Although many
individual- and couple-level characteristics were included in the RAND HRS Datafile,
some data were culled from the RAND-enhanced FAT files for 1998.

Couple-level background data were obtained from the HRS respondent or (in
multiple-respondent households) the report of the household respondent designated by
HRS as most knowledgeable about certain topics. As these are self-report data, they
suffer from a few well-known sources of bias. For example, household income tends to
be underreported in survey research and is typically subject to a large number of refusals
(see review by Moore, Stinson, & Welniak, 2000). HRS and RAND have taken steps to
try to ensure the accuracy and validity of the self-reported financial data—including the
use of specific strategies for eliciting responses, as well as the imputation of missing data.
Where relevant, these HRS strategies and imputation procedures are discussed in more
detail. Other self-reported data—for example, socio-demographic information such as
age or duration of marriage—can be subject to errors in memory or recall.

**Individual-Level Background Characteristics**

**Gender**

The gender of participants was embedded in the design of this study—by
definition, each married couple selected for the study consisted of a wife and a husband.
The literature is replete with evidence suggesting that gender differences exist on several
key study variables (e.g., pain, physical limitations, and depressive symptomatology).
Thus, gender was an important control variable in many analyses. As noted in the prior
literature review (Chapters 2 and 3), existing research is also suggestive of potential
gender differences in both the *intra-*individual and *inter-*individual relationships among
key study variables—for example, cross-spouse relationships of one spouse’s pain on
his/her partner’s depressive symptomatology may vary by gender. Thus, this study
examined potential gender differences in key study variables, as well as in the
relationships among them.

*Age*

Respondent chronological age at the time of the HRS 1998 interview was
measured in years. It was calculated by subtracting the respondent’s date of birth from
the date that the interview was completed. Partial years were rounded to the nearest one-
half year (e.g., 51 and 2 months was rounded to 51 years; 51 and 3 months was rounded
to 51.5 years). Age served primarily as a control variable.

The design of HRS does make it possible for researchers to explore the interface
between chronological age and a related sociodemographic construct—birth cohort. By
virtue of its sampling of multiple birth cohorts, intentional cohort-replacement features,
and longitudinal design, it is theoretically possible to use HRS to study the effects of
aging, birth cohort, and also historical or period effects (for discussion, see Alwin &
Campbell, 2001). In the context of the current study, however, the possibility of
disentangling these different effects was severely limited. As the current study was
cross-sectional in nature, age was highly confounded with cohort. In addition, it is
impossible to examine period effects without the longitudinal data for each cohort.
Perhaps most importantly, the inclusion of the spouses of HRS respondents and the
sample-selection design that later allowed spouses to become members of the cohort-
replacement samples creates a unique problem when attempting to analyze cohort data from spouse pairs: Many couples included in HRS 1998 have spouses in two different birth cohorts, making it extremely difficult to assign each HRS dyad to any particular cohort. Although potential cohort effects are important to consider in future research, cohort was not examined within the current study.

Race and Ethnicity

In HRS, respondent ethnicity was assessed with a single yes/no question: “Do you consider yourself Hispanic or Latino?” Each respondent was then asked to identify his/her race using one of five categories: Black/African American, White/Caucasian, American Indian/Alaskan native, Asian/Pacific Islander, or Other. Since few husbands or wives identified their race as American Indian/Alaskan native, Asian/Pacific Islander, or Other, these participants were combined into a single group reflecting Other non-White race. Thus, respondents were initially classified into three categories: White/Caucasian, Black/African American, and Other non-White race.

Empirical evidence regarding racial and/or ethnic differences is generally mixed for key variables in this study (e.g., pain, physical limitations, and depressive symptomatology. For example, as was reviewed in Chapter 2, some studies have found minority race or ethnicity to be associated with higher levels of pain, while other studies have found either no differences or differences in unexpected directions. Potential racial and ethnic differences in the relationships among key constructs (e.g., between pain and depressive symptoms) have not been subjected to much empirical study. Because the empirical evidence regarding racial and/or ethnic differences is relatively limited, race and ethnicity primarily served as covariates in the current study.
Socioeconomic Status

**Years of education.** Socioeconomic status was measured using three indicators—education, income, and wealth. Of these three, only education was measured at the individual level; income and wealth are considered in a later section addressing couple-level background characteristics. Education was measured as years of completed education. Respondents were asked, “What is the highest grade of school or year of college you completed?” For specific educational levels, a series of follow-up questions was asked (e.g., “Did you get a college degree?”). Reports were merged by RAND into a single variable that reports the highest number of years of education that was completed by the respondent. Because of the relatively low population prevalence of most post-Bachelor’s degrees (e.g., Masters, Doctoral, Law degrees), the number of years of completed education was top-coded at 17, so that the highest category reflected 17 or more years of education.

As described in Chapter 2, studies have shown a relatively robust relationship between lower education and higher depressive symptomatology. Low SES has also been found to be a risk factor for pain and physical limitations; relationships with low education levels have also been found (see Chapter 2). Education is also known to be related to other indicators of socioeconomic status, such as income and wealth. Given these relationships, respondent education was examined (along with other indicators of SES and other background characteristics) as a potential risk factor for pain. It was also used as a covariate in multivariate analyses. Years of education was treated as an interval-like variable in all analyses.
Couple-Level Background Characteristics

Socioeconomic Status

**Household income.** Household income is an important indicator of economic well-being. HRS solicited household income data from the designated “household financial respondent” (i.e., the spouse who told HRS that he/she was most knowledgeable about the family’s finances). In HRS 1998, the household financial respondent was asked to provide information about all sources of income for the prior year for both him/herself and the spouse or partner. Specific sources of income were queried at length, including wage/salary income, bonuses/overtime pay, business and real estate income, interest and dividend income, pensions/annuities, social security (disability and retirement), unemployment income, worker’s compensation, income from government transfer programs (e.g., veterans benefits, food stamps), and all other household income (e.g., alimony, lump-sum payouts from insurance or pension, inheritance). In constructing the various income categories and developing specific questions, HRS researchers drew heavily from other, existing large-scale surveys (Moon & Juster, 1995). Total household income in HRS was calculated as the sum of all income sources, with the exception of lump-sum income (e.g., inheritances or gifts, lump-sum payouts), as such lump-sum amounts were assumed to be one-time occurrences that would temporarily bias income estimates upward and would most likely be captured in estimates of household wealth (to be discussed below; Moon & Juster, 1995).

If a respondent refused or reported not knowing the exact value of a particular form of income, interviewers used a series of unfolding (i.e., increasingly specific) questions to obtain at least an approximate dollar range. For example, if a respondent
was unable or unwilling to provide an exact amount, the interviewer would ask whether or not the amount is over some value (e.g., income over $5,000). Depending on the respondent’s answer, the interviewer would then follow-up with a sequence of probes that would attempt to position the amount within increasingly narrow ranges (or “brackets”). HRS investigators believe that this unfolding-bracketing strategy has resulted in considerably less missing data than might otherwise have been observed, namely because data for typically unwilling respondents are now represented by at least categorical value estimates (Moon & Juster, 1995). A similar unfolding-bracketing strategy was recently adopted by NHIS, and preliminary analyses found a marked reduction in non-response, as compared to prior NHIS waves (Pleis & Cohen, 2007). Additional information about the bracketing strategy used in HRS can be found in Appendix B.

Despite the use of this unfolding-bracketing strategy, sizeable amounts of missing data exist for the income questions in the original HRS datafiles. Imputation procedures have been developed by both HRS and RAND. The current study utilized the RAND-imputed income data, in part because these data were already incorporated into the RAND HRS dataset. Additionally, the RAND imputation procedures are more consistent and are accompanied by better documentation than imputations offered by HRS. Additional information about the RAND income imputations is also in Appendix B.

The validity of the imputed household income data for HRS has been established in a number of different studies. Moon and Juster (1995) for example, compared household income data from Wave 1 of HRS to data obtained from the 1992 Current Population Survey (CPS). HRS data were very consistent with CPS estimates in terms of
the amount and distribution of total household income, as well as many of the specific
income components. There are no specific publications documenting the validity of the
RAND income imputation data for HRS. However, the RAND HRS dataset was partially
funded by, and created with input from, the Social Security Administration (SSA), with
the intention that HRS data be linked to other national datafiles (e.g., earnings and
benefits records from SSA; Medicare claims data from the Centers for Medicare and
Medicaid Services) to serve as a resource to inform public policies regarding retirement,
health insurance, and economic well-being (RAND, 2009). Starting in 2006, RAND
began providing the income and asset imputations for the public-release datasets issued
by HRS (St. Clair et al., 2009). Together, these observations provide strong support for
the soundness and utility of the RAND income imputation data.

Household income is related to other background variables included in the current
study, including age, education, and race and/or ethnicity. As was reviewed in Chapter 2,
various indicators of SES (including income) have been found to be related to key
constructs in the current study, including pain intensity, physical limitations, and
depressive symptomatology. Household income was explored along with other
background characteristics and SES indicators as a predictor of pain, but otherwise was
used mainly as a control variable in multivariate models.

Given the nationally-representative nature of HRS, household income in the
present sample was, not surprisingly, seriously positively skewed. Most households were
concentrated toward the middle and lower end of the income range. Household income
was transformed using the natural log function, after adding a constant ($45, the lowest,
non-zero observed value) to each household’s value to move the lowest values off zero.
Household wealth. Another key indicator of economic well-being is household wealth, referred to in some studies as net worth. Household wealth is generally defined as household assets minus any debts (Moon & Juster, 1995). HRS has data for both household income and wealth. Although the two are positively correlated, some scholars argue that wealth may be an even more important indicator of economic well-being than ordinary income, in part because wealth offers access to higher quality education, health, and other services (Kocchar, 2004). Wealth also provides economic security, especially during market downturns and periods of financial crisis such as unemployment. Wealth can make funds for higher education available, and can also be passed down to help ensure the financial security of future generations. Wealth is regarded as an especially important indicator of economic well-being for older persons, as it provides long-term security in the form of retirement income and (for some) stable housing (e.g., Moon & Juster, 1995; Smith & Kington, 1997).

A vehicle and a home are the assets most likely to be owned by households in the United States (Kocchar, 2004). Other components of wealth include a variety of financial and other types of assets. Financial assets include interest-earning and non-interest bearing accounts (e.g., checking or savings accounts, money market accounts, certificates of deposit), stocks, bonds, mutual funds, and retirement accounts (e.g., IRA, 401k, Keogh accounts). Other assets include business equity and other real estate (i.e., in addition to the primary residence). In the U.S., wealth is related to household income and region of residence, as well as to the education, age, gender, and immigrant status of the head of household (Kocchar, 2004).

In HRS 1998, the designated household financial respondent was asked to provide
detailed information about the net value of all of the different types of assets listed above (e.g., home, vehicle, financial, other). He/she was also asked for information about all debts, including the balance of all mortgages, other home loans (e.g., home equity lines of credit), and other debts (e.g., credit card debt, medical debt, loans from relatives). As was the case with household income, if the respondent refused or reported not knowing the exact value of an asset (or debt), interviewers used a series of unfolding bracketed questions to obtain at least an approximate dollar range (see Appendix B). This strategy helped to reduce missing data and also allowed for improved ability to estimate actual assets (Moon & Juster, 1995). Total household wealth was calculated as the sum of all household wealth components, less all debts. RAND imputed missing household wealth data using procedures similar to those used to impute income (see Appendix B). The RAND imputed household wealth data were used in the current study.

Household wealth data from HRS have been compared to data from several other large-scale social surveys, including the Survey of Income and Program Participation (SIPP) and the Current Population Survey (CPS). These analyses suggest that HRS wealth data appear to have “substantially less bias” than the wealth data available from other studies (Juster & Suzman, 1995, p. S36; see Moon & Juster, 1995, for detailed results). As was the case with income, above, the validity of the RAND imputed wealth data can be inferred from knowing that the RAND HRS dataset was developed in consultation with the SSA and that the RAND income and asset imputations were selected for use in HRS public-release datasets, beginning in 2006.

Like income, wealth is often related to a number of sociodemographic variables, including age, education, race, and ethnicity. However, wealth has been shown to
provide some additional information regarding the economic well-being of household residents—beyond that provided by household income alone. For example, studies have repeatedly shown that racial and ethnic disparities in household wealth are even greater than those in household income. In 2002, although the median income of Hispanic and non-Hispanic Black households was two-thirds of that of non-Hispanic White households, the median wealth of these households was approximately 9% (Hispanic) and 7% (non-Hispanic Black) that of White households (Kocchar, 2004). Hispanic and non-Hispanic Black households were also two to three times more likely to have zero or negative wealth values than were non-Hispanic White households.

Although SES in general has been found to be related to key constructs in the current study, including pain, activity limitation, and depressive symptomatology, the empirical data regarding specific relationships involving household wealth are relatively scarce. A few studies have found lower household wealth to be related to higher depressive symptomatology levels among couples in early waves of HRS and AHEAD (e.g., Min, et al., 2005; Siegel et al., 2004; Townsend et al., 2001). Consequently, household wealth was used primarily as a control variable in the current study. Household wealth was also examined (along with other indicators of SES) as a predictor of pain and activity limitation.

Household wealth, like income, was seriously positively skewed in the current sample, with the majority of households reporting relatively low levels of wealth. A number of households reported negative or zero wealth—typically reflecting the possession of few assets, combined with high debt. An inverse hyperbolic sine (IHS) function was used to transform household wealth data for the current study. Like the log
transformation, the IHS transformation helps to normalize the distribution and reign in extreme outliers. The IHS transformation, however, preserves zeros and negative values (St. Clair et al., 2009, p. 28; Zhang, Fortney, Tilford, & Rost, 2000).

**Household Composition: Others Living in the Household**

As part of the baseline HRS interview, respondents were asked to enumerate all of the persons living in the household at the time of the interview. In married couple households, if both spouses participated in HRS, the designated “family respondent” (i.e., the household member with the most knowledge of the family) provided these data. In subsequent waves, household enumeration data were reviewed with respondents and updated, if necessary. HRS totaled the number of persons residing in the household at the time of the interview into a variable indicating household size.

In the present study, these data were recoded into a dichotomous indicator reflecting the presence of other persons living in the household (in addition to the married couple). This measure (referred to hereafter as “Others in household”) served primarily as a control variable in analyses of respondents’ psychological distress. In this context, it could be viewed as a proxy for a variety of household-related factors that could potentially influence psychological distress (either positively or negatively). For example, having others in the household could provide the couple with increased social support. It is also possible that the presence of others in the household could represent a source of stress for the couple, as may be the case when the couple is caring for younger children or (more likely in the current sample) aging parents or other relatives. Having others in the household could signal increased financial resources or, conversely, could indicate increased strain on existing financial resources. In the current study, having
others in the household was used primarily as a control variable.

*Years of Marriage*

HRS respondents were asked to provide information about their marital histories, including the date on which their current marriage began. RAND then calculated the length of marriage as the number of years (rounded to one decimal place) between the reported date of marriage and the date of the interview. As with most other HRS interview data, the beginning date of the current marriage was obtained independently from each spouse. Thus, a RAND-calculated value representing the duration of marriage was present for each spouse. Because length of marriage was conceptualized as a couple-level background variable in the present study, a single value was needed for each couple.

Several researchers have reported problems with missing data and inconsistencies when attempting to construct marital duration reports from HRS couples. In their analysis of marital history data from couples in Wave 1 of HRS, Holden and Kuo (1996) had to exclude 269 of 5,029 (5.3%) HRS couples due to missing data related to the duration of their current marriage. The authors also reported having to review and correct purportedly discrepant marital history data for several hundred households (e.g., removing marriages that appeared to be duplicated in the marital history list of respondents, re-ordering marriages that were listed out of chronological order).

Townsend and colleagues (e.g., Townsend, Miller, & Guo, 2001) reportedly encountered similar difficulties related to the marital duration data for couples in HRS and AHEAD (A. L. Townsend, personal communication, November 14, 2005). Although the challenges associated with these data have not always been noted in published reports, some authors have resorted to collapsing length of marriage data into broad categories—
for example, Pienta, Hayward, and Jenkins (2000) used categories of less than 10 years, 10-19 years, 20-29 years, and 30 years or more.

Initial exploration of these data confirmed that some discrepancies and modest amounts of missing data were present among the married couples in the RAND HRS 1998 Datafile. Husband and wife values for years of marriage were essentially equivalent (i.e., within 1 year) in 96% of couples. In 2.5% of couples, the difference ranged from 1.5 to 10 years. In 1.5% of couples, the difference in reported years of marriage exceeded 10 years. Given the difficulties reported by some scholars in attempting to reconcile discrepant length of marriage data, a decision was made to use the years of marriage derived from the wife’s report. Wives served as the family informant for the 1998 HRS interview in over 90% of these couples, providing support for this decision. Additionally, less than 1% of wives had missing values for the years of marriage variable (note: decisions regarding the handling missing data are addressed in the next section). This strategy has been used by others, including Townsend et al. (2001, A. L. Townsend, personal communication, November 14, 2005).

**Analysis Strategy**

This section describes the basic steps undertaken in the analysis of these secondary, dyadic data. It begins with a discussion of preliminary steps, including data cleaning and transformation as well as the examination and treatment of missing data. The next section reviews some of the general considerations and procedures involved in the analysis of dyadic data and introduces multilevel modeling as the primary multivariate statistical analysis technique. A final subsection outlines the specific analyses used to address the study’s research questions and hypotheses.
Preliminary Steps

Data Cleaning and Item-level Data Transformation

Staff members at HRS and RAND have done a significant amount of checking and cleaning of HRS data over the years. However, problems have been discovered, even in the RAND dataset which has undergone extensive processing. Consequently, it was important to carefully check and clean the data. The handful of variables drawn from the RAND-enhanced FAT files were also carefully examined and, if possible, compared with related data in the RAND dataset. No major discrepancies were found.

Prior to creating any multi-item scales or conducting any substantive analyses, frequency distributions and univariate descriptive statistics (i.e., measures of central tendency and variability) were examined for all variables to be included in the study. Household-level variables were examined at the couple level and individual-level variables were examined separately by gender. Measures of skewness and kurtosis were examined for continuous variables. Items with potentially problematic (i.e., non-normal) distributions, insufficient variance, or sizeable amounts of missing data (e.g., more than 10%) were investigated in more detail. Potential univariate outliers (i.e., values lying $\pm 2$ SDs or more from the average) were also identified and examined more closely. A handful of extreme values were noted for husband BMI, wife BMI, household income, and household wealth. However, a review of these cases suggested that the coded values were, in fact, plausible, and that any potential negative effects associated with these extreme values would be minimized, either through the use of appropriate data transformations or via the large sample size. A variety of transformations were used to address identified problems, including top- and/or bottom-coding skewed variables.
and/or those with sparse data, collapsing data into specific value ranges or a smaller number of categories, and conducting appropriate numeric transformations (e.g., log transformation). The methods used to transform the data for specific variables were reviewed in the Measures section of this chapter. Steps taken to examine and deal with missing data are discussed next.

**Examination of Missing Data**

Item-level non-response was assessed via frequency distributions and univariate descriptive statistics generated for each study variable as part of the data cleaning and checking process. Frequency counts and percentages were used to characterize the amount of missing data for each item. The few variables with sizeable percentages of missing data (i.e., over 5-6%) were examined in greater detail in order to rule out possible coding or computation errors.

One minor coding discrepancy was observed in the handling of two sets of physical limitation items—difficulty walking and difficulty climbing stairs. The HRS questionnaire makes use of skip patterns designed to reduce the length of the interview and to utilize information already provided by respondents. Within the set of 11 physical limitation items, 2 questions about less physically-demanding tasks are contingent on the respondent’s answers to prior items involving more physically-demanding tasks. For example, if a respondent indicates that he/she has no difficulty walking several blocks, the item assessing difficulty walking one block is skipped (with the reasonable assumption that the respondent would also have no difficulty walking one block). The RAND versions of these variables did correctly account for the major contingencies embedded in these items (e.g., respondents who have no difficulty walking several blocks...
should be coded as having no difficulty walking one block; only respondents who report difficulty walking several blocks are eligible to report difficulty walking one block). However, analyses revealed a pattern of missing data involving the more demanding items that could be remedied using these item contingencies. If a respondent was coded as missing for the difficulty walking several blocks item, but then indicated that he/she had difficulty walking one block, then the respondent must, necessarily, also have difficulty walking several blocks. This logical imputation strategy was used to replace 8% and 14% of the missing values on the difficulty climbing several flights of stairs and walking several blocks items, respectively.

The dyadic nature of the data made it possible to logically impute several missing values for years of marriage. Recall that a decision was made to use the wife’s report of marital duration (see p. 54, this chapter). For couples in which the wife’s value for years of marriage was missing (< 1%), the husband’s value was used instead, leaving missing data for only two couples.

After implementing these two logical imputation strategies, the IBM SPSS Missing Values module, Version 18 (IBM/SPSS, 2010) was used to more thoroughly examine the amount and patterns of missing data across all study variables. At present, there are no published guidelines regarding the analysis and handling of missing data that occur in a dyadic context. In fact, scholars have only recently started to address in earnest the complex issues related to missing data within multilevel or hierarchical data structures (e.g., van Buuren, 2010; Yucel, 2008). For example, scholars note that analyses need to take into account the clustering that exists within grouped or nested data (e.g., Graham, 2009; Yucel, 2008). The decision was made to examine and address
missing data at the couple level in the present study. This strategy preserved the non-independence of husbands’ and wives’ data, permitted gross examination of gender differences in missing data patterns, and allowed consideration of the extent to which data were missing for both spouses. Use of a couple-level imputation strategy has also been advocated by leading figures in dyadic analysis (J. Z. Smith & A. G. Sayer, personal communication, January 22, 2010).

As operationalized in the present study, the couple-level conceptual model presented in Figure 5 (Chapter 3) included a total of 76 variables. Most were single items used to create multi-item scales (e.g., CES-D8). Other variables were single-item indicators of various constructs (e.g., household income) or the component measures used to create other single-item indicators (e.g., height and weight used to create BMI). Each case (i.e., couple) was comprised of 36 variables for the husband, 36 variables for the wife, and 4 household- or couple-level variables (76 variables total).

In keeping with Study Aim 1, analyses were planned to compare the focal sample for the present study—the 423 couples in which both spouses reported pain, the dual-pain (DP) couples—to the 4,963 remaining couples who were initially eligible for inclusion in the present study. In order to ensure the soundness of these comparative analyses, strategies to explore and address missing data were executed within the entire sample of initially-eligible couples (N = 5,386). Patterns of missing data were also examined separately for the 423 couples in the focal sample; any apparent differences between the findings of the two analyses are discussed at the end of this section.

Of the 5,386 couples, 1,677 (31%) had at least 1 missing variable. Although the overall percentage of missing values was very small (< 1% of over 400,000 possible
values in the dataset), 90% of variables (68 out of 76) had at least one missing value. Among variables with missing data, amounts missing ranged from < 0.1% (missing for 8 or fewer cases; e.g., wife’s or husband’s report of most disease conditions, such as cancer or heart disease) to 8.8% (wife’s report of difficulty climbing several flights of stairs, missing for 472 cases). Variables with the highest rates of missingness included the wife’s (and, to a lesser extent, the husband’s) response to several physical limitations (e.g., difficulty climbing one or several flights of stairs, pushing, lifting, or stooping), the wife’s weight, and the wife’s and the husband’s reports of heart pain.

Each case with missing data had missing values for an average of 1.7 out of 76 variables. Of course, this average masks considerable diversity. The majority of couples with missing data (62%; 1042 couples) were missing on only 1 variable, and the missing variable was usually one with the highest rates of missingness overall. Less than 10 couples were missing 8 or more variables (roughly 10% of variables), and these couples were often missing values for several physical limitation items for both the wife and the husband or were missing values on most physical limitation items for the wife only. Twenty-two percent of couples were missing on 2 variables, 8% were missing on 3 variables, and 7% were missing between 4 and 7 variables. A variety of missing data patterns were displayed by these couples, but three trends were noticeable: (a) wives tended to have more missing values than did husbands; (b) roughly one-third of patterns involved missing values for both husband and wife variables; and (c) variables that tended to be missing together included various combinations of physical limitation items (e.g., climbing several flights & pushing; pushing & lifting; climbing, lifting, & pushing) or physical limitation item(s) combined with (wife’s) weight, disease-related pain,
disease conditions, or other miscellaneous items.

The amounts and patterns of missing data within the sample of DP couples were generally very similar to those observed in the larger sample. The overall percentage of missing data in the DP sample was also less than 1% (294 values missing out of over 32,000 possible values). Compared to the larger dataset, the DP dataset did have fewer variables with missing values (42%, 32 out of 76). However, missing data affected a slightly larger proportion of DP couples, with 38% ($n = 159$) of couples having at least one missing variable.

Each DP case with missing data had missing values for an average of 1.8 out of 76 variables. As in the larger sample, the physical limitation variables were among those with the highest rates of missingness—for example, 9% and 10% of cases were missing the wife’s report of difficulties climbing several flights of stairs and pushing/pulling large objects, respectively. Relatively high missingness was also observed for the husband’s physical limitations, including difficulties climbing several flights of stairs (7% of cases missing) and pushing/pulling large objects (4% of cases missing). Higher rates of missingness were also seen for the wife’s weight and the husband’s report of heart pain. As in the larger sample, physical limitations, wife weight, and husband heart pain featured prominently in the different patterns of missing data seen in DP couples.

The majority of the 159 DP couples with missing data were missing on only 1 (60%) or 2 (21%) variables; relatively few couples (3%) were missing 4 or more variables. The general trends observed in the missing data patterns of the larger sample were also seen in the DP sample, with two notable exceptions. First, missing data seemed equally likely to occur in DP husbands and in DP wives. Second, few missing values were observed for
the doctor-diagnosed disease conditions reported by DP husbands and wives.

The pattern analyses suggested that the missingness in these datasets did not conform to a missing completely at random (MCAR) mechanism (Rubin, 1976). For example, several items from the physical limitations scale had the highest rates of missingness (e.g., difficulties climbing several flights of steps, pushing/pulling large objects, lifting/carrying 10 lbs.), and missing values on these items often occurred together. For both spouses, missingness on most physical limitation items was associated with increased age, more years of marriage, lower income, less wealth, less education, and greater height and/or weight. Wives with greater pain intensity and those who did not provide a current weight were more likely to have missing data on physical limitations. Patterns of joint missing data involving the physical limitations of both the husband and the wife were also relatively common. Furthermore, wives’ missingness on difficulty climbing stairs was even more strongly associated with the (lower) education level of their husbands than with their own level of education.

Such construct specificity and patterning would not be observed if the missing data were truly MCAR. This was confirmed statistically, as Little’s MCAR chi-square test (Little, 1988) indicated that these missing data could not be assumed to be missing at random, $\chi^2_{MCAR}(162) = 339.04, p < .001$. Note that if these data did meet MCAR assumptions, a complete case analysis strategy (a.k.a., listwise deletion of missing data) could be used. However, adopting this strategy with these data would exclude mainly those cases in which one or more physical limitations values were missing. Given the central role of physical limitations in the proposed conceptual model, and the relationships among the physical limitations items and between those items and other
variables, a complete case analysis approach would risk introducing bias into the results of the study. Moreover, in the larger sample, missing data occurred disproportionately among wives. Because of the dyadic nature of these data, a listwise deletion strategy would require excluding all couples in which either spouse had missing data.

It is important to note here that the primary multivariate statistical technique used in the present study (multilevel modeling) has the capacity to generate robust estimates in the presence of relatively small amounts of missing data on the dependent variable only. The technique does not accommodate missing data on any of the predictor variables in a multivariate model (including covariates). Based on the analyses reported above, use of a listwise-deletion strategy in the present study would have reduced the sample size by roughly one third (i.e., 31% of all eligible couples, and 38% of DP couples would have been excluded). Analyzing only those cases with complete data would have been inefficient and potentially could have yielded results that were seriously biased.

**Handling of Missing Data: Multiple Imputation**

*Overview and Rationale*

In order to retain more couples for analysis and to make use of the abundance of information provided by both spouses in the majority of couples, all remaining missing data were handled using multiple imputation (MI). Generally speaking, MI generates multiple possible values for each missing value in a dataset. The procedures used to generate imputed values differ across software programs, but all use the observed data in some sort of iterative process. Multiple imputation is typically described as having three phases: An imputation phase, an analysis phase, and a pooling phase (Enders, 2010). The imputation phase (briefly alluded to above) ultimately generates multiple, “filled-in”
or complete copies of the dataset, each of which has a different set of imputed values for the (originally) missing data. In the analysis phase, each of these complete datasets is analyzed using planned statistical analyses. In the final phase, the results obtained using each dataset are combined to yield a single set of results—that is, a single set of parameter estimates (e.g., means, regression coefficients, etc.) and their accompanying standard errors (or variance estimates).

MI is held in high regard by most statistical experts and has fast become the recommended method for handling missing data (McKnight, McKnight, Sidani, & Figueredo, 2007). MI offers several advantages over other common methods of handling missing data, including single imputation (e.g., mean replacement) and maximum likelihood estimation. First, compared to other methods, MI is less sensitive to the specific mechanism underlying the missing data. Even though MI technically assumes that data are missing at random (MAR), experts believe that MI can produce satisfactory results even when data deviate slightly from MAR assumptions (McKnight et al., 2007).

Second, MI allows researchers to improve the imputation of incomplete values by drawing on information contained in other variables within the dataset. In MI, it is relatively easy to incorporate additional variables that are not part of the intended analysis. Experts believe that including such “auxiliary” variables in the imputation process can help make the MAR assumption more plausible and increase power (Collins, Shafer, & Kam, 2001). The inclusion of auxiliary variables also has been shown to reduce bias in estimated parameters (Enders, 2010) and improve the overall efficiency of the imputation process (Raghunathan & Siscovick, 1996).

Third, MI provides a mechanism for estimating the rate (or fraction) of missing
information. Unlike a simple count or percentage of missing data, the rate of missing information gauges the impact of missing data on the resulting statistical estimates and inferences. The rate of missing information thus functions as a measure of statistical uncertainty attributable to missing data (McKnight et al., 2007).

Fourth, in the pooling phase of MI, the final standard errors are adjusted to incorporate two sources of variation (within- and between-imputation variance). In this way, MI formally incorporates the uncertainty associated with missing data (Enders, 2010). This results in more conservative and accurate standard errors.

Fifth, because MI generates multiple complete datasets, MI affords researchers tremendous flexibility. In addition to being available for any needed supplemental analyses (e.g., reliability or sensitivity analyses), the imputed datasets are also available for future studies. The datasets will be especially useful if an abundance of auxiliary variables were included in the imputation model, partly because MI programs generally do not distinguish between independent and dependent variables during the imputation phase (Enders, 2010). Provided that the variables of analytic interest were included in the imputation model, the MI datasets can be used in subsequent studies.

Data Preparation

The imputation process was carried out in the larger sample of all couples (\(N = 5,386\)). A special couple-level dataset was constructed for the MI procedure, for the reasons previously articulated. The major decisions and tasks involved in creating this dataset are described below.

At a minimum, an imputation model should contain all of the variables that are part of the planned analysis. Thus, the couple-level MI dataset was initially comprised of
the 76 item-level variables represented in the proposed conceptual model. Enders (2010) recommended that researchers conduct MI at the item level in order to maximize the information available during the imputation process. Drawing on this logic, separate variables for height and weight were included in the imputation model, rather than BMI; BMI was then (re-)calculated after the imputation process. Individual items from the CES-D8 (the primary outcome measure) were also included. Including measures of the dependent variable in the imputation model was once a matter of debate. However, scholars now agree that omitting the dependent variable actually introduces bias in the MI model because doing so assumes that the dependent variable is completely unrelated to other variables in the model (Graham, 2009). Also in keeping with the recommendations of various authors (e.g., Enders, 2010; Graham, 2009), measures of household income and wealth were transformed (using the log and IHS transformations, respectively) prior to their inclusion in the MI dataset. This helps to minimize any potential problems associated with extreme non-normality.

In addition to these initial 76 variables, the MI dataset included a number of auxiliary variables—variables that are not part of the conceptual or analytical model being examined, but that are (a) available in the larger dataset, and (b) related to one or more variables in the conceptual model and/or associated with missingness on those variables (Allison, 2001; Graham, 2009). These variables essentially function as covariates in the statistical models used to estimate missing values in the dataset. In selecting auxiliary variables to include in the imputation model, Graham (2009) has cautioned researchers to balance the desire to include all potentially relevant auxiliary variables with the exponential increase in computational burden and complexity.
associated with having a large number of variables in the model. Graham himself attempts to limit the total number of variables in his imputation models to 100 or less. Because of the dyadic nature of these data and the couple-level structure of the imputation dataset (i.e., each individual-level auxiliary variable was represented twice in the dataset—once for husbands and once for wives), the upper limit for the imputation dataset in this study was set at 200 variables. This limit was also informed by the results of early imputation attempts using files with varying numbers of cases and variables.

Roughly 100 variables were identified as potential auxiliary variables. All were screened for problematic distributions, insufficient variance, univariate outliers, and sizeable amounts of missing data (e.g., more than 10%). Several variables were flagged during this screening process and various transformations were applied. Variables with extremely-skewed distributions and/or extreme outliers were top- (or bottom-) coded, as appropriate (e.g., number of alcoholic drinks consumed per day was top-coded at 12). Variables with other problematic distributions and/or insufficient variability were also recoded (e.g., number of doctor visits in the past two years was collapsed into six ordered categories; number of bed days in past month was dichotomized into any vs. none). The entire set of variables to be included in the imputation model was also examined carefully in order to avoid incorporating redundant information. In some cases, variables that contained overlapping information were combined into a new variable (e.g., three dichotomous disability-related items were combined into a single variable). In other instances, unique information contained in one variable was incorporated into another (e.g., information from a dichotomous alcohol use variable was incorporated into variables indicating frequency and amount of alcohol use—specifically, respondents who
reported that they did not drink were coded as zero on variables reflecting the number of drinking days per week and the number of drinks per day).

Ultimately, 91 auxiliary variables were added to the imputation model. Several individual- and couple-level background characteristics were included (e.g., birth cohort, born outside of the U.S., census division of primary residence). Other, more contemporaneous, variables were incorporated on the basis of known (or presumed) relationships with one or more variables in the proposed conceptual model—especially the measures of the key constructs of pain intensity, physical limitations, and depressive symptomatology. Examples of such variables include reported difficulties with activities of daily living (ADLs) and instrumental activities of daily living (IADLs), health status indicators (e.g., self-rated health, level of sensory functioning), health and lifestyle behaviors (e.g., smoking and alcohol consumption), and healthcare access and utilization (e.g., insurance coverage, number of doctor visits). Most variables were drawn from the RAND HRS Datafile, although a few (e.g., had a fall in the past two years, self-rated childhood health) were retrieved from the RAND-enhanced FAT file for HRS 1998.

One exploratory research question posed in the present study (EQ10, Chapter 3) concerned a possible interaction between the pain intensity levels of husbands and wives. It is now generally accepted that the product terms representing any interaction effects of interest should be included in the imputation model. If such terms are not included, the imputation proceeds under the assumption that such effects do not exist—i.e., the effects have a correlation of zero with the outcome variable (Graham, 2009). Consequently, a product term representing a joint husband-wife pain intensity effect was created and added to the imputation model. Additional interaction terms of interest for future studies
were also included, including four *intra*-individual effects (e.g., Respondent Pain Intensity X Respondent Physical Limitations), and two *inter*-individual effects (e.g., Respondent X Spouse Physical Limitations). In order to reduce potential collinearity between higher- and lower-order terms, all lower-order terms were mean centered by gender prior to creating the interaction terms. Centering for the imputation model was done using the sample of all couples ($N = 5,386$). Following MI, and prior to conducting multivariate analyses with the focal sample of DP couples ($N = 423$), these variables were re-centered (again by gender, if appropriate) and interaction terms were re-created using the newly-centered variables. Seven interaction terms and two component terms (used to create some of the interaction terms) were added to the imputation model.

*Specifications in the MI Procedure*

The final couple-level MI dataset contained 182 variables, 173 of which were actually used in the imputation model (several variables were included for identification or diagnostic purposes). Appendix C contains a list of all variables in the final imputation datafile. The list also contains information about the nature of each variable (i.e., model, auxiliary, or other), whether or not the variable was used in the imputation model, and any special notes regarding variable derivation, coding, or constraints applied during the MI process.

Multiple imputation was conducted in the SPSS Missing Values module, v18 (IBM/SPSS, 2010), and fully conditional specification (FCS) was used to impute the missing values. FCS has been described as the “semi-parametric and flexible cousin” of joint modeling (van Buuren, 2007, p. 237), which makes more assumptions about the nature of the data and ties the imputation of different missing data patterns to parametric
density functions that must be specified in advance. Most MI software programs that use joint modeling (e.g., NORM by Schafer, 1999) assume that the data conform to an underlying multivariate normal distribution—an assumption that is often violated in real-world datasets. Under such circumstances, MI using joint modeling may fail to generate a solution, can produce biased estimates for categorical variables, and can yield imputed data that violate logical consistencies and relationships between variables (van Buuren, 2007). In contrast, FCS is better able to adapt to, and incorporate unique features of, the data (e.g., zero and non-zero values, skip patterns).

FCS is often referred to as imputation by chained equations because imputations are generated by performing a series of multivariate regressions on a variable-by-variable basis, moving from variables with the least amount of missing data to those with the most missing data. One benefit of this approach is that each predictive model can be tailored to the type of variable being imputed (e.g. logistic regression for dichotomous variables). FCS is thus especially well-suited for use in datasets containing mixed—i.e., continuous and categorical—data (van Buuren, Brand, Groothuis-Oudshoorn, & Rubin, 2006). In FCS (as implemented by SPSS Missing Values), the models used to generate the imputations are based on the level of measurement of each variable. Linear regression is used to impute scale- and interval-level variables. Categorical variables (nominal and ordinal) are imputed using logistic and multinomial logistic regression. In order to guide model selection, SPSS requires that a measurement level be specified for each variable used in the procedure (e.g., scale, ordinal, nominal). Thus, prior to executing the MI, each variable was assigned an appropriate level of measurement in the SPSS datafile.

As noted, the imputation of missing data via FCS is carried out in a sequence,
starting with the variable with the fewest missing values. The values imputed for that variable are then used in the imputation of the next variable with missing values, and so on. In this way, the distribution of missing values for any given variable is conditioned on both the observed, and the previously imputed, data. Thus, an additional benefit of FCS is that the prediction of missing values for any target variable borrows strength from the data contained in other variables, as well as from previously imputed values of the target variable itself, and from previously- and newly-imputed values for other variables in the dataset (Raghunathan & Siscovick, 2001). Because the conditional models are built in a series, variables must be arranged in increasing order of missing values. Thus, variables in the MI dataset were appropriately re-ordered prior to executing the imputation procedure. To facilitate replication, cases in the dataset were sorted in ascending order based on the household identification number.

FCS is an iterative Markov Chain Monte Carlo (MCMC) procedure (IBM/SPSS, 2010, p. 19). The sequence of regression-based imputations across the dataset is repeated (i.e., iterated) a fixed number of times. Each iteration represents a “step” in the Markov chain, which is essentially a sequence of random values in which each value is only related to the value that immediately precedes it (McKnight et al., 2007). The overall MCMC procedure uses a Bayesian-like algorithm to simulate a distribution of plausible missing values from which random values are then drawn (McKnight et al., 2007). By specifying the number of iterations (i.e., steps to be taken in the Markov chain), the analyst attempts to ensure that the values generated during each imputation differ from one another. Van Buuren and colleagues (e.g., van Buuren et al., 2006; van Buuren & Groothuis-Oudshoorn, 2011) have argued that because the application of MCMC in FCS
deviates from strict MCMC theory, the number of iterations can be fixed to a small value—e.g., 5 or 10. When this number is reached, the imputed values from that iteration are saved, creating a completely filled-in (i.e., imputed) dataset. This process is then repeated until the desired number of imputed datasets have been created. The SPSS Missing Values manual advises that the analyst can increase the number of iterations if the algorithm does not converge (i.e., generate a statistically-acceptable solution). Ten iterations were specified for the MI procedure in this study and no convergence problems were observed. Analysts can also specify a “seed” (i.e., starting value) for random number generation during the procedure; this allows the MI results to be replicated if needed. The random number seed specified for the MI procedure was 521.

SPSS Missing Values allows analysts to impose several optional constraints on the imputation process. Analysts can specify the role of each variable in the MI process (e.g., use only as a predictor, use only as a target to be imputed) and can potentially generate a custom prediction model for each variable with missing data. Following the recommendations of a number of authorities (e.g., Enders, 2010; IBM/SPSS, 2010), no role constraints were specified for the MI procedure—all 173 variables were used in the MI process as both predictors and as outcomes (if they had any missing data). Thirty-one of the 173 variables had no missing data; hence, these variables were not imputed and functioned only as predictors during MI. Also in keeping with the recommendations of others (e.g., Enders, 2010), no request was made to round imputed values. Minimum and maximum values were specified for 11 interval-level (or interval-like) variables because early imputation attempts generated a handful of out-of-range values for these variables. Specified values were based on either the possible range of values for a measure (e.g., 1
to 5 for self-rated health) or the range of values observed in the sample (e.g., 1.37 to 2.11 for husband height, in meters). Although analysts can request that SPSS exclude variables with extremely large amounts of missing data (e.g., 50%), this constraint was not necessary—no variable included in the MI model had more than 11% missing values.

Analysts must specify the number of imputed datasets to be created from the FCS MI procedure. Historically, most missing data scholars recommended creating 3 to 5 MI datasets (e.g., Allison, 2001; Rubin, 1996; Schafer & Olsen, 1998). Simulation studies conducted with varying amounts of missing data later led some researchers to suggest that 10 imputations would be adequate for most situations (e.g., Acock, 2005). More recently, Graham and colleagues suggested that the number of imputations should be higher than previously recommended and should take into account the rate of missing information and the decrease in power associated with using fewer imputations (Graham, Olchowski, & Gilreath, 2007). For low rates of missing information (.10 or .30), they suggested a minimum of 20 imputations in order to keep the loss of power to 3% or less. If the analyst is willing to accept a slightly larger drop in power, their simulations suggested that imputations could range from 5 (for < 3% power drop with rate = .10) to 10 (for < 5% power drop with rate = .30). The loss of power was most deleterious when attempting to detect relatively small effects when the rate of missing information was closer to .5 or higher; although parameter estimates were not biased, measures of variability around those estimates were increased.

Several practical problems can arise when attempting to apply Graham et al.’s recommendations. First, the calculation of the rate of missing information is specific to each parameter being estimated. This means that numerous factors contribute to its
calculation, including the amount of missing data for each variable in the analysis, the extent to which missingness overlaps across variables, sample size, and the number of imputations requested (Enders, 2010). Second, the simulations by Graham et al. revealed that the calculation of the rate of missing information can itself be biased.

In the absence of firm guidelines, current recommendations for the number of MI datasets vary widely. SPSS Missing Values v18 sets the number of imputations to five by default (IBM/SPSS, 2010), although a note suggests that the number can be increased if the rate of missing information is large. The manual for Stata, which implements a similar approach to FCS (Yucel, 2011), suggests 5 to 20 imputations for low rates of missing information, and 50 or more imputations when the rate of missing data is relatively high (StataCorp, 2011, p. 11).

According to Enders (2010), the rate of missing information for any given parameter is typically lower than the overall proportion of missing data. This was true in the current study. In the initial sample of all couples \((N = 5,386)\), the overall proportion of missing data across the set of 76 variables in the conceptual model was .31. In contrast, the rate of missing information for the post-imputation estimated sample mean on the physical limitations scale (the scale comprised of items with the largest amounts of missing data) was reported by SPSS to be .06 for husbands and .03 for wives. Based on these estimates, the rate of missing information in the current dataset could be characterized as either very low (e.g., < 10% Enders, 2010, and Graham et al., 2007) or small-to-moderate (e.g., > 25%, Collins et al., 2001; > 30%, Graham et al., 2007).

Ultimately, eight imputed datasets were requested from the MI procedure in the current study. This number was regarded as a compromise between historical
recommendations and the suggestions emerging from more recent work (as reviewed above). Two other practical considerations motivated the selection of eight imputations. First, SPSS Missing Values offers a limited number of statistical procedures that can appropriately pool (i.e., aggregate) the results of analyses using multiply-imputed datasets. Thus, the number of imputations was limited in order to minimize the number of manual calculations required to pool the results of some analyses. Second, the multilevel software used for the multivariate analyses in this study could accommodate no more than 10 imputed datasets without the use of special programming (M. du Toit, personal communication, February 24, 2010). Even with only eight imputations requested, the MI procedure required access to additional memory space on the computer hard drive (SPSS workspace was set to 20 MB), and took six hours to finish.

**Processing of Imputed Data**

At the end of the MI procedure, SPSS saves the imputed data into a single, stacked dataset—i.e., each case has multiple rows of data, corresponding to the number of imputations plus the original data. As an example, the post-imputation dataset in this study was comprised of over 48,000 rows of data (5,386 couples * 8 imputations, plus 1 set of original data). All subsequent data transformations (e.g., creation of multiple-item scales) were done using this stacked dataset. SPSS uses a variable, Imputation_# (where # is either the imputation number or zero for the original data), to distinguish the different imputations.

The analysis phase of MI is relatively straightforward: All analyses are run on each imputed dataset. The Imputation_# variable is used by SPSS to generate results for each imputation. Thus, even though all data are contained in a single datafile, SPSS
analyzes the data from each imputation separately. The output from SPSS displays the results for each imputation and even includes the results based on the original dataset (with missing values) for comparison.

SPSS assists in the pooling phase of MI by combining the results from the analysis of each imputed dataset into a final set of estimates—for example, a single parameter estimate (e.g., mean, regression coefficient) and the corresponding standard error. SPSS follows a set of generally-accepted “rules” (i.e., algorithms)—originally developed by Rubin (1987)—to “pool” (i.e., combine, aggregate) the results from the analyses of the imputed datasets (IBM/SPSS, 2010). The pooled estimate for any given parameter is achieved by calculating the arithmetic average of the parameter estimates obtained for each of the imputations. Pooled standard errors are calculated using more complicated formulae that take into account both within- and between-imputation variability. Drawing on Bayesian principles, it is the incorporation of the between-imputation variability that helps to ensure that the final estimate reflects the uncertainty that is attributable to missing data (Graham, 2009). The pooled standard error is then used to construct appropriate confidence intervals and associated $p$-values. SPSS generates pooled values for most point estimates (e.g., mean values, beta weights, correlations, frequencies and cell counts) and other values used in hypothesis testing (e.g., degrees of freedom, test statistics). For most—but not all—statistical tests, SPSS also provides estimates of the impact of missing data (e.g., rate of missing information, relative efficiency of estimates). SPSS does not support pooling for all statistical procedures, nor does it calculate a pooled value for every estimate or quantity of interest (e.g., standard deviations, percentages, Cronbach’s alpha, $F$ values for tests of
homogeneity of variance). In those instances, results were pooled using Excel.

SPSS (v19; IBM/SPSS, 2011) was used to conduct all post-imputation univariate and bivariate analyses. However, because of the dyadic nature of these data, multivariate analyses were conducted using different statistical analysis software. The section below makes clear why such software was necessary; it also describes adjustments made to accommodate these dyadic data in other analyses (e.g., bivariate analyses). Next, the general procedures used to build and evaluate the multivariate analytic models are described, along with information about how multiply-imputed datasets were incorporated into the analytic process. A final subsection provides details concerning the univariate, bivariate, and multivariate analyses used to address the study’s research questions and hypotheses.

General Considerations and Procedures

in the Analysis of Dyadic Data

Analytic Implications of Dyadic Data

The analysis of data drawn from individuals who are part of nested social groups, such as families, classrooms, or neighborhoods, presents challenges for social science researchers. The use of traditional statistical methods, such as independent samples t tests and OLS regression, ignores the relatedness inherent in the data. This often results in models that violate the assumption of the independence of errors, and can potentially bias conclusions regarding the nature and significance of effects. Specifically, non-independence distorts estimates of error variance, thereby biasing standard errors, confidence intervals, degrees of freedom, and p values used to test statistical hypotheses (Kenny, Mannetti, Pierro, Livi, & Kashy, 2002).
Beyond the effects of non-independence on significance testing, however, lies an issue of more conceptual importance—that is, that the non-independence of the data is, itself, a rich source of information about the impact of the social context on individuals (and vice-a-versa). As noted by Kashy and Snyder (1995), “the non-independence in couples research should not be treated as a statistical nuisance, but rather as an interesting and important…variable to be studied” (p. 340). As made clear in Chapter 3, non-independence of the data for DP husbands and wives was clearly of interest in this study.

As previously discussed, the fact that individuals may experience pain within the context of marriage has been recognized for some time by scholars within the chronic pain field and, more recently, by some gerontological researchers. The bulk of existing research, however, has focused either on the impact of one spouse’s pain on his/her partner or the influence of the partner’s behavior on the spouse’s experience of pain. Researchers have seldom considered that both spouses in the couple may be experiencing pain—a circumstance especially likely to be encountered in research on adults in mid and later life. Furthermore, although many researchers have examined depressive symptomatology as an outcome related to one partner’s pain, few studies of pain in the context of marriage have controlled for (much less purposely examined) the non-independence of marital partners’ depressive symptomatology. And yet, the interdependence that exists among members of social units, particularly close units such as married couples, reflects the reality of the social world. Studies that more accurately model the social world are better able to bridge the gap between research and practice.

One of the challenges faced by prior research in this area has been the lack of well-developed statistical frameworks and techniques to study social units and the
individuals within them. There are currently two major analytical approaches that have been developed to deal with the statistical problem of non-independence and also provide opportunities to study social processes—structural equation modeling (SEM) and multilevel (or hierarchical) linear modeling (MLM). Both approaches permit researchers to ask questions at different levels of analysis; in the case of marital dyads, researchers can ask questions at the level of the individual marital partners, the dyad, or both (Kenny & La Voie, 1985). In theory, both MLM and SEM should yield comparable results.

Multilevel modeling was used in this study for all multivariate statistical analyses. MLM was preferred over SEM for two main reasons. First, the proposed conceptual model identified a large number of background variables (e.g., socio-demographic variables, disease conditions) to be incorporated into statistical analyses of the relationships between pain and depressives symptoms at the individual and/or couple level. It is notoriously difficult to incorporate a large number of covariates in SEM, and the difficulty and complexity of the resulting statistical model would be compounded in the current study by the need to have both individual-level and dyad-level covariates as well as the *intra-*individual and cross-spouse effects of interest. Second, many readily available SEM packages (e.g., AMOS by SPSS) do not currently have the capacity to model a large number of dichotomous variables—especially dichotomous indicators of latent constructs, which dominate the multi-item measures in the current study.

Although the current study primarily used multilevel modeling, other selected statistical techniques were also used. Some of the study’s more descriptive and basic comparative questions (e.g., Do wives report greater pain intensity than husbands? To what extent do the pain intensity levels of husbands and wives covary?) were addressed
using bivariate analyses and tests appropriate for non-independent or “paired” data (e.g., Gonzales & Griffin, 2001). These included intraclass correlation coefficients (ICCs) and non-parametric tests for differences in proportions or medians in matched pairs (e.g., McNemar chi-square test), depending on the specific variables under consideration.

Details regarding specific analyses, and their correspondence to the research questions and hypotheses presented at the end of Chapters 2 and 3, are presented later in this chapter. First, however, it is necessary to consider some of the general procedures and directives that guided the application of multilevel modeling to the data from the marital dyads in the current study.

**General Procedures and Guidelines Used in the Multilevel Analyses**

It might be expected that multilevel modeling would be used to address many of the inter-individual questions tied to the proposed conceptual model—for example, the direct and indirect effects of each spouse’s pain on his/her partner’s depressive symptomatology, as represented Paths $4_H/4_W$ and $5_H/5_W$ in Figure 5. However, even research questions posed at the intra-individual level (e.g., the relationship between each spouse’s pain and his/her own depressive symptomatology, as represented by Path $A$ in Figure 5) must take into account the non-independence of spouses’ responses on the dependent variable—depressive symptomatology. Multilevel modeling affords researchers the opportunity to address both types of questions.

Although a variety of software packages can now be used to conduct multilevel modeling, this study used HLM 6.08 for Windows (Raudenbush, Bryk, & Congdon, 2009). In order to distinguish the software program from the acronym commonly used to characterize the general linear approach to multilevel modeling (HLM, hierarchical linear
modeling), the software program is hereafter referred to as HLM6 (with a numerical suffix). The next section outlines several considerations that influenced how the multilevel analyses were structured, as well as how the results were interpreted.

**Concessions Required in Multilevel Modeling of Dyadic Data**

In research with dyads, limited pieces of information are available to help estimate unknown parameters in multilevel models. In longitudinal dyadic research, this “degrees of freedom” problem can be overcome by collecting at least 5 waves of data. In cross-sectional dyadic research, however, concessions must be made in order to statistically estimate different elements of any proposed conceptual model. When there is interest in examining both *intra*-individual (i.e., within person) and *inter*-individual (i.e., across dyad members) effects, authorities in dyadic analyses (e.g., Sayer & Klute, 2005; Kenny, Kashy, & Cook, 2006) assert that one needs to: (a) create, or design the study to collect, two (or more) “parallel” (i.e., equivalent) measures of the dependent variable; or (b) fix the error variance of the dependent variable, based on some known quantity derived from the observed data; or (c) do both. All of these options result in what is referred to as a “Multivariate Outcomes Model for Distinguishable Dyads” (Sayer, 2007) or an “Actor-Partner Interdependence Model (APIM) for Distinguishable Dyads” (Kashy & Kenny, 2000). An early version of this type of model was outlined by Barnett, Marshall, Raudenbush and Brennan (1993). These models are all specific to dyads in which members are able to be distinguished based on some meaningful characteristic or trait; hence the clause, “for Distinguishable Dyads” in several of the names.

Gender was regarded as the distinguishing characteristic within dyads in the current study. Since this study was cross-sectional, and there was interest in examining
both *intra*-individual and *inter*-individual (i.e., cross-spouse) effects, concessions were required in order to estimate all parameters of interest. In terms of the above options, option A was not feasible to implement with the CES-D8 because the number of items was so small, item content was too asymmetrical (e.g., five mood items, and three somatic items), and item variability was too disparate, to permit the construction of parallel measures. Consequently, option B was used in the current study—i.e., the error variance of the dependent variable was fixed in the analysis.

In the HLM6 program, error variance is fixed by providing the program with estimate(s) of the error variance of the dependent variable in the data being analyzed. For example, the error variance for depressive symptomatology scores in the current study was estimated from the reliability (i.e., internal consistency estimate) and observed variance of the depressive symptomatology scores. The specific formula is presented in Appendix D. Error variance estimates were calculated separately by gender (i.e., for husbands and wives). The HLM6 program uses the error variance estimates to create precision weights (equal to the reciprocal of the error variance[s]) that are then used to conduct a weighted analysis (Sayer, 2007). This error variance-fixing strategy has been used successfully in several prior multilevel studies with dyadic data (e.g., Cano et al., 2005; Goldberg & Sayer, 2006).

In general terms, the multivariate outcomes model for distinguishable dyads (as specified in HLM6) models a latent score on the dependent variable for each member of the dyad. The latent score is comprised of each dyad member’s “true score” on the dependent variable plus some amount of error. These latent scores then become the outcomes at the next level of analysis, in which predictor variables are added to the
model. Models that include predictors at this next level of analysis are called “conditional” models (e.g., Raudenbush, Bryk, Cheong, & Congdon, 2004, p. 87), because the outcomes (i.e., each dyad member’s dependent variable score) are conditioned on (i.e., a function of) the values of predictor variables. Equations are then modeled for each dyad member separately, but simultaneously, yielding estimates of different intercepts and different intra-individual and inter-individual effects (a.k.a., different “actor” and “partner” effects per Kashy & Kenny, 2000). Because of the separate, but simultaneous, estimation of each dyad member’s dependent variable score, this model is sometimes referred to as the “two-intercept model” (e.g., Sayer, 2007).

In the current study, the HLM6 model was used to estimate an intercept (i.e., a predicted depressive symptomatology score) for husbands and, simultaneously, an intercept for wives. The model yields estimates of different intra-individual (or “actor”) and inter-individual (or “partner”) effects, depending on which independent variables are included in a given model. Applying the language of Kashy & Kenny (2000) to the current study, the depressive symptomatology of husbands (here, the “actors”) was expected to be affected by their own pain intensity (an “actor effect”) and also by the pain intensity of their wives (a “partner effect”). Appendix E presents the general multilevel equation that formed the basis of the multivariate analyses in this study.

Benefits of the Multilevel Modeling of Dyadic Data

The dual intercept (or multivariate outcomes) model has a number of features that were of benefit to the current study. First, this analytic strategy allows researchers to incorporate both individual-level predictors (i.e., those that have distinct values for each dyad member) as well as dyad- or couple-level predictors (i.e., those that have the same
value for both members of the dyad). In the current study, the effects of couple-level variables, such as household income or years of marriage, were estimated alongside the effects of individual-level variables, such as a respondent’s own pain intensity and age. Second, because the model yields separate (but simultaneous) estimates of the effects of predictor variables on each dyad member’s outcome, it is possible to conduct formal tests of the differences in these effects (Barnett et al., 1993). Several research questions concerning gender differences were addressed via this mechanism in the current study.

A third beneficial feature of the two-intercept model is that it generates an estimate of the amount of shared variance (or intraclass covariation) in the dependent variable between the two dyad members. In the present study, for example, the model provided an estimate of the interdependence of husbands’ and wives’ depressive symptomatology scores. Fourth, the two-intercept model also provides separate estimates of the residual variances (i.e., the amount of unexplained variance) in the dependent variable for distinguishable dyad members (Kenny, Kashy, & Cook, 2006; Raudenbush, Brennan, & Barnett, 1995; Sayer & Klute, 2005). So for example, the model provided estimates of the residual variance in husbands’ and wives’ depressive symptomatology. Finally, this analytic strategy is very flexible, in that it permits the development of separate models using different predictor variables for each dyad member (Raudenbush et al., 1995). Although in the present study no gender-specific (i.e., husband- and wife-specific) models were hypothesized a priori, such models could be specified if desired.

**Model Specification Options**

Although HLM6 can be used to analyze a variety of different types of dependent variables, all outcomes in the present study were estimated using a standard, hierarchical
linear model (HLM). The primary outcome variable in the current study—depressive symptomatology (CES-D8)—was analyzed as an interval-level, continuous variable. As previously mentioned, a sensitivity analysis was also conducted by analyzing a log-transformed version of the CES-D8 in parallel with the original CES-D8. As part of the test of proposed mediation hypotheses, the measure of physical limitations (an interval level, continuous variable) also served as an outcome variable in some analyses.

Estimation theory permits statistical inferences to be made about population parameters based on sample data (Raudenbush & Bryk, 2002). The HLM6 program employs a variety of alternative estimation methods, depending on the nature of the dependent variable and the need to compare resulting models. In this study, maximum likelihood (ML) estimation methods were used for all analyses so that the resulting models could be statistically compared (this will be discussed in more detail shortly). In HLM6, ML estimation employs either expectation-maximization (EM) or Fisher scoring computational algorithms (Raudenbush et al., 2004).

The HLM6 program calculates both unadjusted (i.e., normal) and robust standard errors for all parameter estimates. Robust standard errors adjust for non-normally distributed data and sample size (Hox, 2002; Raudenbush & Bryk, 2002). Given the non-normality observed on some model variables even after transformation, robust standard errors were reported for all multivariate analyses. Robust and normal standard errors were also compared to help identify any potential problems with model estimation.

Model Building and Assessment of Model Fit

The first step in multilevel modeling is to construct a baseline model. The main purpose of such a model is to serve as a comparison for later models—i.e., to assess how
well subsequent models adding various predictor variables can explain variation in the
dependent variable, as compared to the baseline model. However, a baseline multilevel
model also generates other useful data. For example, as noted in Appendix E, the
baseline model in the current study provided useful information about the average
husband and wife CES-D8 scores, as well as the variability in their scores and the
covaration between scores. The baseline model in the current study consisted of only an
intercept (and residual) for each spouse.

Research questions and hypotheses were addressed using a series of “nested”
conditional models (i.e., models that contain predictor variables, on which dependent
variables are conditioned). Models were built sequentially, with specific predictor
variables added alone (or in select combinations) to test specific hypotheses. The first
conditional model was compared to the baseline model discussed above. At subsequent
steps, predictors were added to create additional conditional models. Thus, models were
nested in the sense that all subsequent models include the same set of predictors present
in the prior model, plus any newly added predictors. Nesting allows researchers to
compare the relative fit of sequential models. In the current study, the extent to which a
subsequent model provided improved fit to the observed data (compared to the prior
model, or to the baseline model) was evaluated using a likelihood ratio test provided by
HLM6 (Raudenbush et al., 2004) that makes use of the deviance statistic calculated for
each model (Raudenbush & Bryk, 2002). Nested model comparison results (along with
individual parameter estimates) were used to help evaluate whether a given variable (or
set of variables) contributed significantly to a model that better fit the data.

Model building was driven primarily by the order of the proposed research
questions and hypotheses. The specific details about each model are reviewed in detail in the next subsection (see “Analyses Addressing Research Questions and Hypotheses”). In general, most research questions and hypotheses were addressed by examining the significance, direction, and strength of the parameter estimates for key study variables. The statistical significance of these parameter estimates was determined by an approximate $t$-value (Raudenbush et al., 2004). Variance and covariance estimates generated by HLM6 were evaluated by chi-square tests (Raudenbush et al., 2004).

Although the parameter estimates for key study variables were of most interest in the current study, the predictive value of multivariate models was also examined. This was done by calculating a proportion reduction in error measure (PRE). Within MLM, a PRE is much like a pseudo-$R^2$ and can be interpreted as the proportion of variance in the dependent variable than is explained by a model, as compared to the total (explainable) variance. Given the dual-intercept approach used in modeling, PREs were calculated separately by gender for each of model.

For each multivariate model, assumptions related to multivariate non-normality, the presence of multivariate outliers, potential multicollinearity, and poor model fit were checked. Model deviance values, residual files, and selected scatterplots of residuals against predictor variables were examined for any evidence that the underlying assumptions were violated. The output of all multilevel analyses was also examined carefully for parameter estimates that were suspiciously high, had large standard errors, and/or demonstrated unusual shifts in size or sign within a series of nested models. Multicollinearity diagnostics were calculated and examined. Suspected problems were investigated in more depth and attempts were made to address them prior to finalizing
each set of multivariate models. In order to conserve space, results from assumption and model-checking analyses are reported only for the final (or nearly-final) *intra-* and *inter-* individual multivariate analyses. Specific models are identified shortly.

*Data Management and the Use of MI Data in HLM6*

HLM6 is capable of performing phases two (analysis) and three (pooling) of MI data: The software can analyze multiply-imputed datasets and then pool the results into a single set of parameter estimates and associated standard errors. In order to apply HLM to these data, it was necessary to dismantle the single, stacked SPSS dataset used for most univariate and bivariate analyses. Data from each of the eight imputed datasets were saved as separate datafiles and read into HLM6. Selecting a “multiple imputation” option during the model specification process instructs HLM6 to run the specified model in each MI dataset and to pool the results of these separate analyses.

As is standard in MI, HLM6 calculates the pooled estimate of any given parameter by averaging the estimates obtained from the analysis of each imputed dataset. HLM6 provides pooled estimates for each of the following parameters: Fixed effects (gammas, $\gamma$), reliabilities, parameter variances and correlations, chi-square tests of parameter variances, standard errors for the variance-covariance components, and multivariate hypothesis tests for fixed effects (Raudenbush, Bryk, Cheong, & Congdon, 2004, p. 181). The calculation of standard errors associated with the pooled fixed effects ($\gamma$) takes into account both within- and between-imputation variability for the estimates, and appropriately reflects the uncertainty resulting from imputation. Formulae developed by Rogers et al. (1992; as cited in Raudenbush et al., 2004) are used to pool the standard errors. Pooled standard errors are then used to construct confidence intervals and derive
associated *p*-values. Like SPSS, HLM6 does *not* pool every estimate or value of interest. Consequently, model deviance values were pooled in Excel by averaging the deviance values from all imputations, and tests of model deviance were conducted using these pooled estimates. PRE measures were also calculated in Excel using the (already pooled) random effects estimates generated by HLM6.

In most multivariate analyses, continuous predictor variables were centered around either their respective gender-specific means for individual-level predictors (e.g., age, education, physical limitations) or the grand mean for household-level predictors (e.g., income, length of marriage). Centering was done both to increase the interpretability of specific parameter estimates and to reduce the collinearity that can result from the inclusion of interaction terms in multivariate models. Although centering can be done automatically by HLM6, the analyst retained control over the process by centering variables prior to moving the data into HLM6. Recall that several variables had been centered previously within the larger sample of all eligible couples as part of the preparation for the imputation process. Because multivariate analyses were conducted only in the focal sample of dual-pain couples (*N* = 423), these variables were re-centered (by gender, if appropriate) within the DP couples sample. Interaction product terms were also re-created within the focal sample. Note that these tasks were conducted separately within each of the imputed datasets, prior to reading the datasets into HLM6.

*Analyses Addressing Research Questions and Hypotheses*

**General Considerations**

Prior to commencing any substantive analyses, univariate descriptive statistics were again examined for all study variables (across all 8 MI datasets). Descriptive data
were also examined separately for all subgroups of interest (i.e., by gender and by subgroups defined by the pain reports of both spouses). All multi-item scales and derived variables (e.g., calculated variables, such as BMI; alternate versions of variables and/or variables re-coded for analysis, such as log-transformed CES-D8 scores) were then computed. Finally, the reliability of all multi-item scales was examined. Internal consistency estimates (viz., Cronbach’s alpha) were calculated for each scale in all subgroups of interest (and across all MI datasets) using SPSS. Final estimates were obtained by averaging the results from the 8 MI datasets in Excel. Recall that the internal consistency estimates for the CES-D8 and the physical limitations scales reported in the Measures section were those obtained for the husbands and wives in DP couples. Comparable internal consistency estimates for these scales were observed among the husbands and wives in the other groups of couples (couples typology is reviewed shortly). For the CES-D8, Cronbach’s alpha ranged from .68 to .74 for husbands and .73 to .76 for wives. Internal consistency estimates obtained for the 11-item physical limitations scale ranged from .77 to .82 among husbands and .76 to .81 among wives. Thus, both scales evidenced acceptable internal consistency within the current study.

By convention, alpha (i.e., Type I error rate) was set to .05 for all bivariate and multivariate analyses. The power of all statistical tests was adequate (i.e., above .90), as the sample size for all analyses was at least 423 couples (the N for some analyses was over 5,000 couples). Given the large sample size, care was taken in interpreting results, as even relatively small effects were likely to be statistically significant. Thus, estimates of effect size were calculated and reported where possible.
Preliminary Analyses

Describing DP Couples and Comparisons With Other HRS Couples

This study sought to identify and describe a national sample of community-dwelling middle-aged and older married couples in which both spouses reported pain (part of Study Aim 1). An earlier section of this chapter, “Derivation of the Study Sample,” reviewed the steps taken to identify 5,386 heterosexual married couples living together in the community in which both spouses participated in the 1998 wave of HRS. A typology of couples was created by cross tabulating the responses of both spouses to a survey question regarding whether the respondent was “often troubled with pain.” The 423 couples in which both spouses reported pain (i.e., the dual-pain [DP] couples) comprised the focal sample for this study.

Initial analyses focused on describing the focal sample. Univariate frequencies, percentages, and relevant descriptive statistics (e.g., mean, standard deviation, median, range) were used to characterize the background characteristics of these DP couples, as well as the husbands and wives comprising them. Similar procedures were used to describe the health-related characteristics, pain, activity limitation, and levels of psychological distress of DP husbands and DP wives.

To further address Study Aim 1, subsequent analyses examined the extent to which these DP couples differed from the other HRS 1998 couples represented in the couple-pain typology—i.e., the 4,936 couples in which only one, or neither, spouse reported being troubled by pain (refer to Table 3, Chapter 5, for typology). Planned contrasts were used to compare the household-level characteristics (e.g., income) and couple-level background characteristics (e.g., length of marriage) of DP couples to those
of each of the other types of couples. Gender-specific planned contrasts compared the individual background characteristics of DP spouses (e.g., age, years of education) to the characteristics of the spouses in other couples. Similar gender-specific comparisons were conducted for the health-related characteristics, pain, activity limitation, and levels of psychological distress of husbands and wives versus their peers in other couples. Interval-level (or interval-like) measures were compared using independent groups $t$ tests. Categorical variables were compared using chi-square tests. Significant omnibus chi-square tests involving more than two groups were decomposed using $z$ tests for pairwise comparisons of interest (adjusted for multiple comparisons). No explicit a priori hypotheses were developed for these comparisons. Additional details regarding these analyses are incorporated into the presentation of findings in the Results chapter.

*Examination of Basic Bivariate Relationships Within DP couples*

Data from the focal sample of DP couples were also subjected to additional preliminary analyses. Simple bivariate analyses (e.g., cross-tabulations, scatterplots, correlations) were performed at several different levels within the DP sample—across all individuals, across couples, for husbands and wives separately, and also within couples (i.e., between husbands and wives). Depending on the nature of the specific variables under consideration, some pairs of variables were analyzed at all possible levels, while others were analyzed at only one or two levels. As an example, consider the relationships among several SES variables. A bivariate correlation was used to characterize the general relationship between education and household income for all individuals in the sample; this same relationship was also assessed separately for each gender (i.e., for husbands and for wives). However, the relationship between household wealth and
household income was assessed by correlating these two variables at the couple level. Finally, the relationship between the education levels of spouses was assessed by correlating two individual-level variables—husbands’ and wives’ education—across the sample of DP couples.

These preliminary analyses had two primary purposes. First, they were used to detect any potentially problematic bivariate relationships, including nonlinear relationships between two continuous variables, sparsely-populated cells in the joint distribution of two categorical variables, and potential collinearity between measures to be used in subsequent multivariate analyses. Second, these analyses provided a preliminary sense of the unadjusted relationships between key variables. Selected bivariate analyses were also used to answer specific research questions. Additional details concerning those analyses are contained within the subsection below.

**Substantive Analyses in the Focal Sample of Dual-Pain Couples**

This section reviews the analyses used to address each of the study’s research questions and hypotheses. Recall that research questions and hypotheses were proposed at both the *intra*-individual level (at the end of Chapter 2) and at the *inter*-individual or cross-spouse level (at the end of Chapter 3). As noted earlier, however, because of the inherent non-independence of the data from the husbands and wives, many of the *intra*-individual level research questions were addressed using a statistical approach that appropriately modeled the couple-level data (i.e., multilevel modeling). The two-intercept multilevel model allowed many of the research questions posed at the *intra-*individual level to be addressed separately, but simultaneously, for husbands and wives. *Inter*-individual questions were then addressed by building on those initial models.
Table 2 summarizes the substantive analyses conducted in the focal sample of DP couples. In both the table and the text below, analyses pertaining to the intra-individual level research questions are presented first, followed by those that addressed research questions at the inter-individual. Throughout both sections, repeated reference is made to Figure 5—the conceptual model used to guide the current study.

**Analyses Used to Address Intra-Individual Research Questions**

Research Questions 1 through 5 were introduced at the end of Chapter 2. These questions were designed to address Study Aim 2—to examine the demographic and health-related characteristics associated with reports of pain in DP couples, as well as the relationship between each spouse’s pain and his/her own psychological distress. This series of questions also addressed Study Aim 4—to determine whether, and the extent to which, any of these intra-individual relationships involving pain and psychological distress differed by gender.

Research Question 1 concerned the existence of possible gender differences in key study variables between husbands and wives in DP couples. Associated hypotheses asserted that DP wives would report greater pain intensity (Hypothesis 1a [H1a]), depressive symptomatology (H1b), and physical limitations (H1c) than would DP husbands. Frequencies and descriptive statistics were used to examine husbands’ and wives’ reports on these measures. Paired t tests (adjusted for heterogeneous variances, if necessary) were used to evaluate whether the mean scores of husbands and wives on these measures differed significantly. Because it was important to examine these differences within the context of other gender differences, supplemental analyses also compared husbands and wives on other characteristics. For these analyses, paired t tests
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<th>Research Questions &amp; Hypotheses</th>
<th>Analyses&lt;sup&gt;a&lt;/sup&gt;</th>
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<tr>
<td><strong>Intra-Individual Questions &amp; Hypotheses (from Chpt. 2):</strong></td>
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<tr>
<td>RQ1. Are there gender differences in key study variables between husbands and wives in dual-pain (DP) couples in mid and later life?</td>
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<td>H1a. <em>Wives will report greater pain intensity than husbands.</em></td>
<td><strong>DV = Pain Intensity:</strong> Frequencies &amp; descriptive statistics by gender (IV) with <em>Paired t test</em> of means.</td>
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<tr>
<td>H1b. <em>Wives will report greater depressive symptomatology than husbands.</em></td>
<td><strong>DV = Depressive Symptomatology:</strong> Frequencies &amp; descriptive statistics by gender (IV) with <em>Paired t test</em> of means.</td>
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<tr>
<td>H1c. <em>Wives will report greater physical limitations than husbands.</em></td>
<td><strong>DV = Physical Limitations:</strong> Frequencies &amp; descriptive statistics by gender (IV) with <em>Paired t test</em> of means.</td>
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<tr>
<td>RQ2. Beyond gender, what other background and health-related characteristics are related to reports of pain intensity?</td>
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<tr>
<td>H2. Selected background variables (older age, minority racial status, lower SES) and health-related characteristics (more disease conditions, increased body weight) will be associated with greater pain intensity.</td>
<td><strong>DV = Pain Intensity:</strong> Series of two nested HLM models: (1) background variables; and (2) <em>adds</em> health-related characteristics.</td>
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<tr>
<td>EQ2. Are particular disease conditions more strongly associated with greater pain intensity?</td>
<td><strong>DV = Pain Intensity:</strong> Same as above, except with the set of disease conditions substituted for number of disease conditions in Model 2.</td>
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<td>Research Questions &amp; Hypotheses</td>
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<td><strong>RQ3.</strong> Is there a relationship between pain intensity and depressive symptomatology?</td>
<td><strong>DV = Depressive Symptomatology:</strong> Series of three nested HLM models: (1) background variables; (2) health-related characteristics; and (3) adds pain intensity.</td>
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<td><em>H3.</em> Greater pain intensity will be associated with greater depressive symptomatology. In Figure 5: Path A will be positive and significant.</td>
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<td><strong>RQ4.</strong> Is the relationship between pain intensity and depressive symptomatology mediated by activity limitation (as measured by physical limitations)?</td>
<td><strong>DV = Physical Limitations:</strong> Series of three nested HLM models: (1) background variables; (2) health-related characteristics; and (3) adds pain intensity.</td>
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<td><strong>RQ4a.</strong> Is there a relationship between pain intensity and physical limitations?</td>
<td><strong>DV = Depressive Symptomatology:</strong> Series of four nested HLM models: (1) background variables; (2) health-related characteristics; (3) pain intensity; and (4) adds physical limitations.</td>
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<tr>
<td><em>H4a.</em> Greater pain intensity will be associated with greater physical limitations. In Figure 5: Path B will be positive and significant.</td>
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<td><strong>RQ4b.</strong> Is there a relationship between physical limitations and depressive symptomatology?</td>
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<tr>
<td><em>H4b.</em> Greater physical limitations will be associated with greater depressive symptomatology. In Figure 5: Path C will be positive and significant.</td>
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<td><strong>RQ4c.</strong> Do physical limitations mediate the relationship between pain intensity and depressive symptomatology?</td>
<td>Examine change in the coefficient for pain intensity from <strong>HLM</strong> Model 3 (under <strong>H3</strong>) to Model 4 (under <strong>H4b</strong>) above.</td>
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<td><strong>H4c.</strong> The relationship between pain intensity and depressive symptomatology will be at least partially mediated by physical limitations. In Figure 5: Path A will be reduced and/or nonsignificant.</td>
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<td><strong>EQ5.</strong> Does gender moderate any of the relationships between pain intensity and depressive symptomatology? Specifically, do the direct or indirect (i.e., mediated) relationships between pain intensity and depressive symptomatology differ between husbands and wives?</td>
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<td><strong>EQ5a.</strong> Does the direct effect of pain intensity on depressive symptomatology differ between husbands and wives? In Figure 5: Does Path A for husbands differ from Path A for wives?</td>
<td>Compare the coefficient for pain intensity for husbands to that of wives in <strong>HLM</strong> Model 3 (under <strong>H3</strong>) above. If warranted, test for statistical significance by constraining coefficients to be equal.</td>
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<tr>
<td><strong>EQ5b.</strong> Does the indirect effect of pain intensity on depressive symptomatology differ by gender? Specifically, does the mediating effect of physical limitations differ between husbands and wives? In Figure 5: Is the change in Path A for husbands different from the change for wives?</td>
<td>Compare the extent of change in the coefficient for pain intensity between <strong>HLM</strong> Models 3 (under <strong>H3</strong>) and 4 (under <strong>H4b</strong>) above—essentially, the result of <strong>H4c</strong>—for husbands to that of wives. If warranted, impose equality constraints on selected coefficients to test for statistical significance.</td>
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### Research Questions & Hypotheses

#### Inter-Individual Questions & Hypotheses (from Chpt. 3):

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<th>Question</th>
<th>Hypothesis</th>
<th>Analyses</th>
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<tr>
<td><strong>RQ6.</strong> Are DP spouses’ (i.e., wives’ and husbands’) key study variables related?</td>
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<tr>
<td><strong>H6a.</strong> Spouses’ reports of pain intensity will positively covary.</td>
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<td><strong>DV = Pain Intensity:</strong> Calculation of an <em>Intraclass Correlation Coefficient (ICC)</em> that adjusts for mean differences by gender.</td>
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<td>In Figure 5: <em>Path 1 will be positive and significant.</em></td>
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<tr>
<td><strong>H6b.</strong> Spouses’ depressive symptomatology will positively covary.</td>
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<td><strong>DV = Depressive Symptomatology:</strong> Calculation of an <em>ICC</em> that adjusts for mean differences by gender.</td>
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<td>In Figure 5: <em>Path 2 will be positive and significant.</em></td>
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<tr>
<td><strong>H6c.</strong> Spouses’ physical limitations will positively covary.</td>
<td></td>
<td><strong>DV = Physical Limitations:</strong> Calculation of an <em>ICC</em> that adjusts for mean differences by gender.</td>
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<td>In Figure 5: <em>Path 3 will be positive and significant.</em></td>
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<td><strong>RQ7.</strong> Is there a cross-spouse relationship between each spouse’s pain intensity and his/her partner’s depressive symptomatology? In other words, does pain intensity in one spouse affect the level of depressive symptomatology in the partner?</td>
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<td><strong>H7.</strong> Each spouse’s pain intensity will be related to greater depressive symptomatology in his/her partner. In Figure 5: <em>Paths 4_W and 4_H will be positive and significant.</em></td>
<td></td>
<td><strong>DV = Depressive Symptomatology:</strong> Series of nested <em>HLM</em> models: Building on Model 4 under <em>H4b</em> above (which includes: background variables; the respondent’s own health-related characteristics, pain intensity, and physical limitations); (5) <em>adds</em> the spouse’s health-related characteristics; and (6) <em>adds</em> the spouse’s pain intensity.</td>
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<td>Research Questions &amp; Hypotheses</td>
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<td><strong>RQ8.</strong> Is the cross-spouse relationship between each spouse’s pain intensity and the depressive symptomatology level of his/her partner mediated by the spouse’s activity limitation (as measured by physical limitations)?</td>
<td><strong>DV = Depressive Symptomatology:</strong> Series of nested <strong>HLM</strong> models: Building on Model 6 under <strong>H7</strong> above (which includes: background variables; the respondent’s own health-related characteristics, pain intensity, and physical limitations; the spouse’s health-related characteristics and pain intensity); <strong>(7) adds</strong> the spouse’s physical limitations.</td>
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<tr>
<td><strong>RQ8a.</strong> Is there a relationship between each spouse’s physical limitations and the depressive symptomatology of his/her partner?</td>
<td><strong>H8a. Greater physical limitations in each spouse will be related to greater depressive symptomatology in his/her partner. In Figure 5: Paths 5&lt;sub&gt;W&lt;/sub&gt; and 5&lt;sub&gt;H&lt;/sub&gt; will be positive and significant.</strong></td>
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<tr>
<td><strong>RQ8b.</strong> Do the spouse’s physical limitations mediate the relationship between each spouse’s pain intensity and his/her partner’s depressive symptomatology?</td>
<td><strong>H8b. The relationship between each spouse’s pain intensity and his/her partner’s depressive symptomatology will be at least partially mediated by the spouse’s physical limitations. In Figure 5: Paths 4&lt;sub&gt;W&lt;/sub&gt; and 4&lt;sub&gt;H&lt;/sub&gt; will be reduced and/or nonsignificant.</strong></td>
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<td><strong>Examine change in the coefficient for each spouse’s pain intensity from HLM Model 6 (under H7) to Model 7 (under H8a) above.</strong></td>
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<td>Research Questions &amp; Hypotheses</td>
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<td><strong>RQ9.</strong> Does gender moderate any of the cross-spouse relationships between each spouse’s pain intensity and the depressive symptomatology level of his/her partner? Specifically, do any of the direct or indirect (i.e., mediated) relationships between each spouse’s pain intensity and his/her partner’s depressive symptomatology differ between husbands and wives?</td>
<td>Are the changes in Paths $4_w$ (wife to husband) and $4_h$ (husband to wife) different?</td>
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<tr>
<td><strong>H9.</strong> The direct relationship between the husband’s pain intensity and the wife’s depressive symptomatology will be stronger than the direct relationship between the wife’s pain intensity and the husband’s depressive symptomatology. In Figure 5: Path $4_H$ will be stronger than path $4_w$.</td>
<td>Compare the coefficient for the spouse’s pain intensity for husbands to that of wives in HLM Model 6 (under $H7$) above. If warranted, test for statistical significance by constraining coefficients to be equal.</td>
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<tr>
<td><strong>EQ9.</strong> Does gender moderate the indirect (i.e., mediated) cross-spouse relationship between each spouse’s pain intensity and his/her partner’s depressive symptomatology? Specifically, does the mediating effect of the spouse’s physical limitations differ between husbands and wives?</td>
<td>Compare the extent of change in the coefficient for the spouse’s pain intensity between HLM Models 6 (under $H7$) and 7 (under $H8a$) above—essentially, the result of $H8b$—for husbands to that of wives. If warranted, impose equality constraints on selected coefficients to test for statistical significance.</td>
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<tr>
<td><strong>EQ10.</strong> Do spouses’ levels of pain intensity interact to jointly affect the depressive symptomatology of either partner?</td>
<td><strong>DV</strong> = Depressive Symptomatology: Series of nested HLM models: Building on Model 7 (under $H8a$) above, (8) adds a Respondent X Spouse Pain Intensity interaction term.</td>
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**Note.** RQ = research question; EQ = exploratory question; H = hypothesis (presented in *italics*); DV = dependent variable; IV = independent variable; HLM = hierarchical linear model in HLM6. Referenced paths are in the conceptual model presented in Figure 5 (in Chapter 3). Some words underlined for emphasis.  
<sup>a</sup>Statistical tests and models indicated in **boldface** type.
were used to examine differences on continuous (or quasi-continuous) measures (e.g., age, years of education). In terms of categorical variables, differences in frequency counts/percentages between spouses were tested using specialized chi-square tests for paired data: The McNemar $\chi^2$ was used for dichotomous variables (e.g., presence of specific disease conditions), and the McNemar-Bowker $\chi^2$ was used for multinomial variables (e.g., BMI categories).

Research Question 2 asked: Besides gender, what other background and health-related characteristics were related to the pain intensity reported by DP husbands and wives? A directional hypothesis was offered for several socio-demographic and health characteristics (e.g., age, minority race, count of disease conditions) that had been shown in prior studies to be related to the pain intensity. Dual-intercept hierarchical linear models (described previously) were constructed in HLM6 to examine these predictors in a multivariate context. Predictors were examined separately, but simultaneously, for husbands and for wives. In order to isolate their relative contributions, background and health-related variables were entered in separate steps, creating nested models. To address Exploratory Question 2 (i.e., which disease conditions were more strongly associated with reports of greater pain intensity?), dummy variables representing the seven disease conditions (e.g., arthritis, heart disease) were substituted in place of the number of disease conditions in “Alternate” Model 2.

Research Questions 3 and 4 concerned the relationship between pain and psychological distress at the intra-individual level. Research Question 3 (RQ3) focused on the direct effect of pain intensity on depressive symptomatology. Based on prior research, it was expected that greater pain intensity would be related to greater depressive
symptomatology in both DP husbands and wives (Hypothesis 3, H3). Drawing on the activity limitation theory of depressed affect (Williamson & Schulz, 1992a), Research Question 4 (RQ4) asked whether the relationship between an individual’s pain intensity and depressive symptomatology was mediated by his/her own activity limitation (specifically, physical limitations). Thus, RQ4 focused on the possible indirect (i.e., mediated) effect of pain intensity on depressive symptomatology. Together, RQ3 and RQ4 embodied Paths A, B, and C in the proposed conceptual model (see Figure 5). The underlying hypotheses comprised the steps necessary to evaluate this proposed mediation effect (based on Baron & Kenny, 1984, as outlined in Chapter 2). When applied to the current study, these steps dictated that mediation would be established only if:

- pain intensity was related to depressive symptomatology (H3, Path A);
- pain intensity was related to physical limitations (H4a, Path B);
- physical limitations were related to depressive symptomatology (H4b, Path C); and
- when physical limitations were added to a model in which pain predicted depressive symptomatology, the strength of the relationship between pain intensity and depressive symptomatology was reduced or eliminated (H4c).

The above hypotheses were addressed using dual-intercept HLM models. RQ3 and H3 were evaluated in a series of two nested HLM models with CES-D8 scores (i.e., depressive symptomatology) as the dependent variable. The first nested model (Model 1) in the series included all relevant individual- and couple-level background characteristics (e.g., age, race, education, household income and wealth). Health-related characteristics (body weight, number of disease conditions) were added next in Model 2. The
respondent’s pain intensity was entered in Model 3. Thus, H3 (Path A) was evaluated only after controlling for relevant background and health-related characteristics.

Hypothesis 4a was addressed using a separate series of nested HLM models. In these models, the measure of physical limitations served as the outcome variable. The model-building strategy remained the same, however: After background variables and health-related characteristics were entered into Models 1 and 2, respectively, pain intensity was entered as a predictor in Model 3. Thus, H4a (Path B) was also evaluated after controlling for relevant covariates.

Hypotheses 4b and 4c were addressed by building upon the series of nested HLM models constructed to address RQ3. After accounting for the variance in depressive symptomatology associated with background variables (Model 1), health characteristics (Model 2), and pain intensity (Model 3), physical limitations were entered in the final intra-individual HLM model (Model 4). The significance of the parameter estimate for physical limitations provided a test of H4b (Path C). To address H4c, the change in the parameter estimates for pain intensity from Model 3 to Model 4 was examined. Support for mediation was obtained if a significant change was observed in the size of the estimate. Details about the formal significance test used for this comparison are presented in the Results chapter. Note that H4b and H4c were also evaluated after controlling for relevant covariates.

Exploratory Question 5 considered whether gender moderated any of the direct or indirect relationships between pain intensity and depressive symptomatology. Gender moderation was addressed by comparing the parameter estimates obtained for husbands and wives in the final HLM models for RQ3 and RQ4c. Possible gender differences in
the direct effect of pain intensity on depressive symptomatology were examined by comparing the coefficient for pain intensity (i.e., Path A) for husbands to that of wives in Model 4. Possible gender differences in the indirect (i.e., mediated) effect of pain intensity were assessed by comparing the extent of change observed in the pain intensity coefficient between Models 4 and 5 (basically, comparing the results of H4c above) for husbands and wives. If warranted, equality constraints were imposed on the coefficients to formally test whether a specific effect differed by gender (e.g., if the effect of pain intensity on depressive symptoms was equivalent in husbands and wives). Since the constrained model was essentially nested within the unconstrained model (Model 5), the difference between coefficients was evaluated statistically by comparing model deviance values. Additional information regarding equality constraints and associated statistical tests is provided within the Results chapter.

Analyses Used to Address Inter-Individual Research Questions

Research Questions 6 through 10 were introduced at the end of Chapter 3. These questions were designed to address Study Aim 3—to investigate the covariation of DP spouses’ reports of pain, activity limitation, and psychological distress, as well as the relationship between each spouse’s pain and the psychological distress of his/her partner. This series of questions also addressed Study Aim 4—to determine whether, and the extent to which, these inter-individual relationships varied by gender.

Research Question 6 (RQ6) concerned the extent to which DP husbands’ and wives’ reports (or scores) on key study variables were related. Accompanying hypotheses asserted that husbands’ and wives’ pain intensity (H6a), depressive symptomatology (H6b), and activity limitation (specifically, physical limitations, H6c)
would be positively related. These hypotheses corresponded to Paths 1, 2, and 3 in the conceptual model contained in Figure 5. Intraclass correlation coefficients (ICC’s) were used to estimate the covariation of spouses’ responses. ICC’s in the current study were calculated using methods that adjusted for the variability in these constructs attributable to gender, as such variability can potentially bias estimates of shared variation (e.g., Gonzales & Griffin, 2001). These adjustments are described in greater detail alongside the ICC’s data in the Results chapter.

The steps delineated in Research Questions 7 and 8 essentially mimicked those outlined above for RQ3 and 4, except that the direct and indirect effects of pain intensity on depressive symptomatology were inter-individual (or cross-spouse) effects, rather than intra-individual effects. Together, RQ7 and RQ8 examined whether the relationship between one spouse’s pain intensity and the depressive symptomatology of his/her partner was mediated by the spouse’s activity limitation (specifically, physical limitations). These effects corresponded to Path 4 and Path 5, respectively, in Figure 5.

The HLM models developed to test RQ7 and RQ8 (and associated sub-questions and hypotheses) differed from those developed for RQ3 and RQ4 above in three important ways. First, the potential effects of an individual’s (or respondent’s) own focal predictor variables (e.g., pain intensity, physical limitations) were controlled prior to introducing the parallel predictor belonging to his/her spouse. Second, because prior research has amply demonstrated the negative effects of one spouse’s poor health on the psychological well-being of the other (see Chapter 3), any cross-spouse effects associated with the spouse’s disease conditions and body weight were controlled prior to examining the cross-spouse effects of pain intensity and/or physical limitations on the depressive
symptomatology of the partner. Finally, the relationship between the respondent’s own pain intensity and his/her own physical limitations represented an \textit{intra}-individual relationship (i.e., Path B); as this relationship was assessed via the analyses under RQ4a, no additional modeling was necessary. Aside from these differences, the key features of these multilevel models were unchanged. Most notably, all effects were examined separately, but simultaneously, for husbands and for wives.

Because of the complexity of the multilevel models developed to examine cross-spouse effects, a sample model is presented in Appendix F. Specifically, Appendix F contains the HLM6 multilevel model (and underlying equations) that were used to test RQ7—Is there a cross-spouse relationship between each spouse’s pain and the depressive symptomatology of his/her partner? The equations in Appendix F show the basic parameterization of a two-intercept model that includes cross-spouse, or \textit{inter}-individual, effects. For each nested model in the series—i.e., Models 5, 6, and 7—equations are developed showing the variables (or variable sets) being added to the model. The accompanying text in Appendix F also links the parameters in Model 7 to the cross-spouse paths depicted in Figure 5 (i.e., Path 4\textsubscript{W} and Path 4\textsubscript{H}). Recall from Chapter 3 (and Figure 5) that the letter subscripts used for these cross-spouse effects or paths indicate the origin of the effect; for example, Path 4\textsubscript{H} represents the effect of the \textit{husband’s} pain intensity on the wife’s depressive symptomatology.

Research Question 9 considered whether any of these cross-spouse effects might differ by gender. Gender moderation was addressed by comparing several parameter coefficients for husbands and wives in the HLM models developed for RQ7 and RQ8. These comparisons evaluated whether any of the direct relationships (RQ7, Paths 4\textsubscript{H} and...
4_w) or mediated relationships (RQ8, Paths 5_H and 5_W) between one spouse’s pain and the depressive symptomatology of his/her partner differed between husbands and wives in terms of their significance and/or strength. A directional hypothesis was offered for the direct effect only—specifically, the effect of the wife’s pain intensity on the depressive symptomatology of the husband was expected to be stronger than the effect of the husband’s pain intensity on the depressive symptomatology of the wife (H9). The examination of possible gender differences in the mediation of any cross-spouse effects of pain intensity mediation was exploratory. If warranted, gender differences were tested statistically by imposing equality constraints on specific coefficients. Additional details regarding such tests are provided in the Results chapter.

Exploratory Question 10 (EQ10) concerned a potential joint effect of both spouses’ pain intensity on the depressive symptomatology of either spouse. As noted in Chapter 3, because this joint effect could be potentially exacerbating or protective in nature, no directional hypothesis was developed. In order to address EQ10, a product term was created using each spouse’s level of pain intensity (i.e., Husband Pain Intensity X Wife Pain Intensity). Note that pain intensity levels were mean-centered by gender prior to creating the product term. The product term was then added as a final step in a fully-specified HLM model (Model 8). Procedures recommended by Aiken and West (1991) were used to decompose any significant interaction effects.

After addressing all research questions and hypotheses, a final multivariate HLM model (Model 9), was constructed to predict the depressive symptomatology of DP husbands and wives. In composing this final model, attempts were made to balance principles of parsimony (e.g., trimming the model of nonsignificant terms), parallelism
(e.g., maintaining a conceptually similar model for husbands and wives, unless compelling theoretical, logical, and empirical evidence supported doing otherwise), and pragmatism (e.g., building a model that had good fit to the observed data). Whereas prior models attempted to isolate the effect of specific predictors, this last model was used to illustrate how DP husbands and wives with certain characteristics (i.e., based on significant predictors) might be at risk for increased depressive symptomatology.
CHAPTER 5: RESULTS

TABLE OF CONTENTS

| Description of Dual-Pain Couples and Comparisons with Other Couples | 5-2 |
| Couple-Level Characteristics and Comparisons | 5-4 |
| Individual-Level Characteristics and Comparisons | 5-10 |
| Husbands in Dual-Pain Couples and Selected Comparisons | 5-13 |
| Background Characteristics | 5-13 |
| Health-Related Characteristics | 5-15 |
| Pain Intensity | 5-19 |
| Physical Limitations | 5-21 |
| Psychological Distress | 5-23 |
| Wives in Dual-Pain Couples and Selected Comparisons | 5-24 |
| Background Characteristics | 5-24 |
| Health-Related Characteristics | 5-26 |
| Pain Intensity | 5-29 |
| Physical Limitations | 5-31 |
| Psychological Distress | 5-32 |
| Overall Summary | 5-33 |
| Differences and Similarities Between Spouses in Dual-Pain Couples | 5-38 |
| Differences Between Dual-Pain Husbands and Wives | 5-39 |
| Similarities Between Dual-Pain Husbands and Wives (Paths 1, 2, and 3) | 5-44 |
| Implications for Subsequent Analyses | 5-51 |
| Use of Multilevel Modeling | 5-51 |
Emphasis on Gender ................................................................. 5-53

*Intra*-individual Relationships .................................................. 5-54

Overview of Bivariate Relationships ........................................... 5-54

Predictors of Pain Intensity ......................................................... 5-59

Bivariate Results ........................................................................ 5-60

Multivariate Results ................................................................. 5-63

Pain in Relation to Psychological Distress at the Intra-Individual Level .............................................................. 5-73

Relationship Between Pain Intensity and Depressive Symptomatology (Path A) .......................................................... 5-73

Bivariate Results ........................................................................ 5-73

Multivariate Results ................................................................. 5-74

Relationship Between Pain Intensity and Physical Limitations (Path B) ........................................................................ 5-82

Bivariate Results ........................................................................ 5-82

Multivariate Results ................................................................. 5-83

Relationship Between Physical Limitations and Depressive Symptomatology (Path C) and Test of Mediation ................................. 5-89

Bivariate Results ........................................................................ 5-89

Multivariate Results ................................................................. 5-89

*Inter*-Individual (Cross-Spouse) Relationships ............................ 5-107

Overview of Cross-Spouse Bivariate Relationships ....................... 5-107

Pain in Relation to Psychological Distress at the Inter-Individual Level ........................................................................ 5-110

Relationship Between Each Spouse’s Pain Intensity and His/Her Partner’s Depressive Symptomatology (Path 4) ......................... 5-112

Bivariate Results ........................................................................ 5-112
CHAPTER 5: RESULTS

This chapter is divided into four sections. The first section reviews the steps taken to identify the couples who served as the focal sample for the present study. Next, these couples are described in terms of their background characteristics, and descriptive data are also presented separately for husbands and wives. Information is also provided regarding how, and to what extent, these couples differed from other couples in HRS 1998 (Study Aim 1). Section two considers the similarities and differences between husbands and wives in the focal sample. The husband-wife comparative analyses addressed several research questions and hypotheses that were outlined in Chapter 3, including Paths 1, 2, and 3 in the proposed conceptual model (part of Study Aim 3).

Section three presents the results of intra-individual (i.e., within-person) analyses with regard to (a) predictors of pain intensity, and (b) the relationship between pain and depressive symptomatology. Specific findings concerned Paths A, B, and C in the proposed conceptual model, and the hypothesized mediating role of activity limitation (Study Aim 2). Results pertaining to the analysis of inter-individual (or cross-spouse) relationships between pain and depressive symptomatology are presented in section four. These findings addressed Paths 4 and 5, as well as the hypothesized role of the spouse’s activity limitation in mediating the cross-spouse pain-depressive symptomatology relationship. This section also reviews the findings of a model that evaluated the joint effects of both spouses’ pain intensity ratings (also part of Study Aim 3). A final, trimmed model is presented at the end of section four in order to illustrate the additive effects of significant predictors. Within each of these sections, the results of analyses with respect to gender differences are reviewed (Study Aim 4).
Description of Dual-Pain Couples and Comparisons with Other Couples

Table 3 shows the pattern of responses to the HRS 1998 pain item for all 5,386 couples who were initially eligible for inclusion in the present study. The column and row totals summarize the individual responses of wives and husbands, respectively. Over one quarter of wives (28.4%) responded affirmatively to the question, “Are you often troubled with pain?” A slightly smaller, but sizeable percentage of husbands (22.4%) reported that they were often troubled with pain. A McNemar test for correlated proportions was used to compare the pain reports of wives and husbands. This test was statistically significant, $\chi^2_M(1) = 54.77, p < .001$, indicating that pain was more prevalent among wives than among husbands in this sample. The difference between the prevalence of pain among wives and husbands expressed as a percentage was 6%, with a 95% confidence interval ranging from 4.4 to 7.6%.

The four bordered cells in the center of Table 3 present the distribution of couples across the four categories that capture the joint responses of each husband and wife. The characteristic defined by the joint responses of each husband and wife may, for the sake of convenience, be referred to later in this document as “couple pain status.” The two-letter abbreviation enclosed in a circle in each cell will be used to refer to the different groups or types of couples. Although dual-pain couples have been mentioned briefly in prior chapters, they are re-introduced here as data in each cell of the table are reviewed.

Over half of all couples ($n = 3,070$) were comprised of husbands and wives who both reported not being troubled with pain. Hereafter, these couples will be referred to as “No Pain” (or NP) couples. Among couples in which one only spouse reported being
Table 3

*Pattern of Husband and Wife Responses to HRS 1998 Pain Item within Married Couples Initially Eligible for Inclusion in the Present Study (N = 5,386 couples)*

“Are you often troubled with pain?”

<table>
<thead>
<tr>
<th>Wife Response</th>
<th>No</th>
<th>Yes</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Husband</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>3,070</td>
<td>1,108</td>
<td>4,178</td>
</tr>
<tr>
<td>(57.0%)</td>
<td>(20.5%)</td>
<td></td>
<td>(77.6%)</td>
</tr>
<tr>
<td><strong>Yes</strong></td>
<td>785</td>
<td>423</td>
<td>1,208</td>
</tr>
<tr>
<td>(14.6%)</td>
<td>(7.9%)</td>
<td></td>
<td>(22.4%)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>3,855</td>
<td>1,531</td>
<td>5,386</td>
</tr>
<tr>
<td>(71.6%)</td>
<td>(28.4%)</td>
<td></td>
<td>(100.0%)</td>
</tr>
</tbody>
</table>

*Note.* Percentages were calculated relative to the total sample (N = 5,386). The symbol in the lower right corner of each cell indicates type of couple: NP = Neither spouse has pain, WP = Wife has pain, Husband does not, HP = Husband has pain, Wife does not, DP = Both spouses have pain (aka, “Dual-Pain” couples).
troubled with pain, those in which only the wife reported pain outnumbered those in which only the husband reported pain nearly three to two. Couples in which the wife had pain, but the husband did not will be referred to as “Wife Pain” (or WP) couples, while “Husband Pain” (or HP) couples will be used to denote couples with the reverse pattern. Couples in which both spouses reported being troubled with pain accounted for less than 10% of all couples who were initially eligible for the present study. However, they still constitute a sizeable sample. These “dual-pain” (or DP) couples, depicted in the bottom-right cell of Table 3, comprised the focal sample for the present study.

Couple-Level Characteristics and Comparisons

Table 4 contains summary statistics describing the couple-level background characteristics of the dual-pain (DP) couples (see column 2). Considering that the DP couples comprised a relatively small portion of the total number of HRS couples, initial analyses examined the extent to which they differed from the other types of couples. Given the lack of prior research on community-dwelling couples and pain, no a priori hypotheses were formulated. The middle columns of Table 4 contain comparable descriptive data for each of the other types of couples—No Pain (NP), Husband Pain (HP), and Wife Pain (WP) couples. The final column summarizes the results of planned contrasts that compared DP couples to each of the other three groups. Differences among the other 3 groups were not relevant to the present study, thus they were not tested.

DP couples are described below. Results of the planned contrasts with other couples are integrated into this description. Given the large sample sizes in each of the groups, relatively small differences were likely to be statistically significant. Consequently, exact \( p \)-values are reported for most statistical tests and effect size...
Table 4. Characteristics of Dual-Pain Couples and Comparisons with Each Other Group

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Years of marriageb</td>
<td>34.92 (15.78)</td>
<td>35.13 (14.00)</td>
<td>34.55 (15.42)</td>
<td>35.05 (15.12)</td>
<td>none</td>
</tr>
<tr>
<td>HH income (log)c</td>
<td>10.29 (0.96)</td>
<td>10.75 (0.92)</td>
<td>10.61 (0.92)</td>
<td>10.58 (0.87)</td>
<td>DP &lt; NP, HP, WP</td>
</tr>
<tr>
<td>HH wealth (ihs)d</td>
<td>11.16 (4.81)</td>
<td>12.57 (2.99)</td>
<td>12.18 (3.43)</td>
<td>12.06 (3.59)</td>
<td>DP &lt; NP, HP, WP</td>
</tr>
<tr>
<td>Others present in HH</td>
<td>127 (30.02%)</td>
<td>916 (29.84%)</td>
<td>237 (30.19%)</td>
<td>311 (28.07%)</td>
<td>none</td>
</tr>
</tbody>
</table>

Note. Cells contain M (SD) or n (%). For variables with missing data, descriptive statistics and the results of planned contrasts were pooled across the 8 multiply-imputed data sets. Only one variable in this table (Years of marriage) had any missing data. HH = Household.

aStatistically significant results of planned contrasts comparing DP couples to each of the other groups are presented in this column.
bThe groups displayed unequal variances, F_{Levene}(3, 5382) = 6.50, p < .001. Consequently, t tests that adjusted for unequal variances were used to conduct the a priori contrasts of group means.
cHH income is in log-transformed units. The mean (SD) untransformed HH income values for the groups were as follows: $43,446 ($59,784) for DP, $68,696 ($102,244) for NP, $56,968 ($55,454) for HP, and $55,569 ($65,166) for WP couples. Untransformed median HH income values were $30,532 (DP), $47,874 (NP), $41,725 (HP), and $40,000 (WP).
dHH wealth is in inverse hyperbolic sine units (ihs)—a transformation that preserves negative values. The mean (SD) untransformed HH wealth values for the groups were as follows: $311,193 ($1,175,450) for DP, $450,227 ($1,703,294) for NP, $322,629 ($454,807) for HP, and $340,838 ($728,340) for WP couples. Untransformed median HH wealth values were: $120,000 (DP), $220,000 (NP), $164,000 (HP), and $155,350 (WP). Because the groups displayed unequal variances, F_{Levene}(3, 5382) = 21.49, p < .001, t tests that adjusted for unequal variances were used to conduct the planned contrasts of group means.
measures and/or confidence intervals are presented as appropriate. Any adjustments made to the analysis plan laid out in Chapter 4 (Method) are also reviewed. As most variables had some missing data, descriptive statistics and the results of group comparisons were pooled (i.e., aggregated; see Chapter 4 for details) across the eight multiply-imputed (MI) datasets. Thus, all comparative analyses were based on the total number of couples initially eligible for the present study (i.e., 5,386 couples).

On average, DP couples had been married 35 years. However, considerable variability was also observed: A handful of couples had been married for 1 year or less, and a similar number had been married for 65 years or more. The majority of DP couples (60%) had been married between 20 and 50 years. Although DP couples displayed slightly more variability, and NP couples displayed slightly less, the relative shape of the distribution of years of marriage was similar across the four groups of couples. The average length of marriage ranged from approximately 34.5 years in HP couples to just over 35 years in NP couples. The minor discrepancy in group variances yielded a significant Levene test (see footnote “b” for Table 4), so planned contrasts involving DP couples were done using t tests that adjusted for unequal variances. None of these pairwise comparisons was statistically significant (t values ranged from -0.14 to 0.40, with corresponding p-values from .89 to .69). Therefore, the average length of marriage for DP couples was not appreciably different from that of any other group.

Not surprisingly, neither household income nor household wealth was normally distributed in any group. This non-normality can be most directly observed by comparing the standard deviations of the untransformed values (presented in footnotes c and d in Table 4) to the respective group means. In all instances, the standard deviations
exceeded the mean household income values (the same was also true for household wealth). Although the log transformation helped to normalize the distributions of household income, values were still mildly to moderately non-normal in several of the groups (see criteria in Curran, West, & Finch, 1996). Skewness values ranged from -1.25 to -2.06 and kurtosis values ranged from 9.15 to 14.39. Application of the inverse hyperbolic sine transformation (ihs) to household wealth values yielded similar results: Even the ihs-transformed distributions of wealth were moderately to severely non-normal in several groups (e.g., skewness values ranged from -3.38 to -5.56; kurtosis values ranged from 11.74 to 37.50).

Given the non-normality of these transformed distributions, nonparametric tests of group differences were considered. However, they were rejected for two primary reasons: (1) parametric tests of group differences in central tendency (e.g., F or t tests) have been shown to be relatively robust to even substantial violations of normality (Pett, 1997); and (2) when sample sizes are sufficiently large—for example, more than 20 or 30 cases per group (Jekel & Katz, 2001; Warner, 2008)—the Central Limit Theorem ensures that the “sampling distributions of means are normally distributed regardless of the distributions of variables” (Tabachnick & Fidell, 2001, p. 72). Additionally, parametric tests of group mean differences can be adjusted for unequal variances between groups—something that is not possible in nonparametric tests. In summary, the large group sizes in the present study suggested that parametric tests of group mean differences could be appropriately applied (with necessary adjustments) to the transformed values of household income and wealth.

The Levene test was not statistically significant for group comparisons of
(logged) household income, $F_{\text{Levene}}(3, 5382) = 0.65, p = .580$. Consequently, $t$ tests assuming equal variances were used to evaluate a priori contrasts of interest. Dual-Pain (DP) couples reported significantly lower average (logged) household income than did No Pain (NP) couples, $t(5382) = -9.73, p < .001$. The average (logged) household income of DP couples was also significantly lower than that of Husband Pain (HP) couples, $t(5382) = -5.74, p < .001$, as well as that of Wife Pain (WP) couples, $t(5382) = -5.53, p < .001$. Effect size estimates for these differences were calculated in terms of Cohen’s $d$, which measures the magnitude of the mean difference in pooled standard deviation units. Cohen’s $d$ values were 0.51 for DP versus NP, 0.35 for DP versus HP, and 0.32 for DP versus WP couples. According to Cohen’s (1988) guidelines for interpretation, the difference between the DP and NP couples was moderate, with the average (logged) household income of DP couples roughly one half of a standard deviation lower than that of NP couples. The mean differences between DP couples and the HP and WP dyads were relatively weak. Although differences in logged units can be hard to compare, inspection of the untransformed household income values of the groups (see footnote in Table 4) makes clear that DP couples had mean and median incomes that were substantially lower (i.e., at least $10,000 less) than those observed in each of the other groups. The largest differences were observed between DP and NP couples.

In untransformed dollar amounts, DP couples had an average household income of just over $43,400 per year, and a median income of roughly $30,500. Roughly 10% of DP couples reported an annual household income greater than $77,900. In contrast, 25% reported income below $18,900. The poverty threshold for two-adult households in 1998 was $10,634 for all households, and $9,862 for “elderly” households (i.e., at least one
householder age 65 or older; Almanac of Policy Issues, 2004). Using the more conservative elderly household threshold, 6% of DP households could be considered poor, and an additional 6% “near poor” (i.e., income below 150% of threshold).

As noted in footnote d for Table 4, the groups had significantly different variances in household wealth (ihs-transformed), with DP couples displaying the most variability, NP couples displaying the least variability, and HP and WP couples displaying levels of variability closer to that of the NP couples. Due to the differences in the dispersion of household values, a priori comparisons of interest were conducted using t tests that adjusted for unequal variances. DP couples reported household wealth values that were, on average, 1.41 ihs units lower than NP couples, $t(468.11) = -5.86, p < .001$. The mean differences between the household wealth of DP couples and those of HP and WP couples were somewhat smaller (1.02 and 0.90 ihs units, respectively), but still statistically significant: $t(658.06) = -3.86, p < .001$ for DP vs. HP; $t(610.08) = -3.48, p < .001$ for DP vs. WP. Considering that differences in ihs units are difficult to evaluate, the untransformed household wealth values are presented in footnote e of Table 4. Looking at these values, it is apparent that both the mean and median wealth of DP couples were substantially lower (i.e., at least $100,000 less) than the wealth reported by couples in each of the other groups. Differences between group means were statistically different, yet effect size estimates were relatively small (Cohen’s $d$ values ranged from 0.20 for DP vs. WP couples to 0.30 for DP vs. NP couples).

The untransformed median household wealth of DP couples was $120,000. The higher mean value (roughly $311,000) reflects the presence of a “heavy tail” at the upper end of the distribution. Considering that HRS calculates household wealth from the value
of all assets (e.g., primary residence, savings, other personal property) minus any household debts (e.g., mortgages, loans, outstanding medical or credit card debt), the median wealth reported by DP couples seems modest. Two additional observations are compelling. First, 121 DP couples (28.6%) reported total net assets under $44,500—the estimated annual cost of a private pay nursing home stay at the time of the 1998 HRS interview (Stewart, Grabowski, & Lakdawalla, 2009). Second, 6% of DP couples had wealth values at or below zero dollars.

Most DP couples (70%) were living in couple-only households at the time of the 1998 HRS interview. This proportion varied little among the groups. The overall chi-square test comparing these proportions across groups was not statistically significant, \( \chi^2(3) = 1.49, p = .685 \). DP couples living with others in the household reported between 1 and 9 other household residents. Half of these couples \((n = 64)\) had only one other person living in the household. Just over one third \((n = 44 \text{ couples})\) shared the household with two to three other persons. The remaining 15% of couples \((n = 19)\) shared the household with four or more other persons.

**Individual-Level Characteristics and Comparisons**

Table 5 summarizes the individual-level characteristics of DP husbands (in column 2) and wives (in column 3). As before, the middle columns of the table contain comparable data for the spouses in each of the three other types of couples. The final two columns summarize the results of planned contrasts comparing DP spouses to spouses in the other groups. Comparisons involving individual-level characteristics were gender-specific and restricted to the appropriate spouse in selected other groups. Husbands in DP couples were compared to husbands in NP couples and HP couples (not to husbands
### Table 5. Characteristics of Husbands and Wives in Dual-Pain Couples and Selected Comparisons to the Relevant Spouse in Other Groups

<table>
<thead>
<tr>
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</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Husband</td>
<td>Wife</td>
<td>Husband</td>
<td>Wife</td>
<td>Husband</td>
</tr>
<tr>
<td><strong>Background Characteristics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (yrs.)</td>
<td>66.56 (10.06)</td>
<td>62.93 (10.59)</td>
<td>65.32 (9.77)</td>
<td>61.66 (10.32)</td>
<td>65.78 (9.52)</td>
</tr>
<tr>
<td><strong>Race</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White/Caucasian</td>
<td>368 (87.00%)</td>
<td>369 (87.23%)</td>
<td>2,720 (88.92%)</td>
<td>700 (89.17%)</td>
<td>978 (88.27%)</td>
</tr>
<tr>
<td>Black/Afr. American</td>
<td>38 (8.98%)</td>
<td>40 (9.46%)</td>
<td>267 (8.03%)</td>
<td>60 (7.64%)</td>
<td>97 (8.75%)</td>
</tr>
<tr>
<td>Other</td>
<td>17 (4.02%)</td>
<td>14 (3.31%)</td>
<td>83 (2.70%)</td>
<td>25 (3.19%)</td>
<td>33 (2.98%)</td>
</tr>
<tr>
<td>Hispanic ethnicity</td>
<td>23 (5.44%)</td>
<td>26 (6.15%)</td>
<td>202 (6.58%)</td>
<td>48 (6.11%)</td>
<td>85 (7.67%)</td>
</tr>
<tr>
<td>Yrs. of education (0 - 17’)</td>
<td>11.44 (3.38)</td>
<td>11.70 (2.79)</td>
<td>12.83 (3.27)</td>
<td>12.71 (2.81)</td>
<td>12.09 (3.35)</td>
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<td><strong>Health-related Characteristics</strong></td>
<td></td>
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<tr>
<td>No. of disease conditions (0 - 4’)</td>
<td>2.12 (1.24)</td>
<td>1.93 (1.17)</td>
<td>1.26 (1.11)</td>
<td>1.08 (1.02)</td>
<td>1.86 (1.16)</td>
</tr>
<tr>
<td>Specific conditions (1 = yes)</td>
<td></td>
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</tr>
<tr>
<td>Arthritis</td>
<td>308 (72.81%)</td>
<td>307 (72.58%)</td>
<td>1,036 (33.75%)</td>
<td>1,180 (38.44%)</td>
<td>524 (66.75%)</td>
</tr>
<tr>
<td>Cancer</td>
<td>53 (12.53%)</td>
<td>63 (14.89%)</td>
<td>302 (9.84%)</td>
<td>280 (9.12%)</td>
<td>92 (11.72%)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>85 (20.09%)</td>
<td>72 (17.02%)</td>
<td>415 (13.52%)</td>
<td>220 (7.17%)</td>
<td>130 (16.56%)</td>
</tr>
<tr>
<td>Heart disease</td>
<td>159 (37.59%)</td>
<td>112 (26.48%)</td>
<td>604 (19.67%)</td>
<td>313 (10.20%)</td>
<td>257 (32.74%)</td>
</tr>
<tr>
<td>High blood pressure</td>
<td>207 (48.94%)</td>
<td>210 (49.65%)</td>
<td>1,231 (40.10%)</td>
<td>1,127 (36.71%)</td>
<td>361 (45.99%)</td>
</tr>
<tr>
<td>Lung disease</td>
<td>60 (14.18%)</td>
<td>50 (11.82%)</td>
<td>144 (4.69%)</td>
<td>117 (3.81%)</td>
<td>83 (10.57%)</td>
</tr>
<tr>
<td>Stroke</td>
<td>42 (9.93%)</td>
<td>25 (5.91%)</td>
<td>159 (5.18%)</td>
<td>92 (3.00%)</td>
<td>53 (6.75%)</td>
</tr>
<tr>
<td>Body weight (BMI)b</td>
<td>27.93 (5.24)</td>
<td>28.51 (6.46)</td>
<td>27.25 (4.14)</td>
<td>26.41 (5.18)</td>
<td>27.84 (4.70)</td>
</tr>
<tr>
<td>Body weight (BMI, 3 categories)b</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Underweight or Normal (≤ 24.4)</td>
<td>111 (26.24%)</td>
<td>126 (29.79%)</td>
<td>796 (25.93%)</td>
<td>1,251 (40.75%)</td>
<td>180 (22.93%)</td>
</tr>
<tr>
<td>Overweight (24.5 - 29.4)</td>
<td>171 (40.43%)</td>
<td>135 (31.91%)</td>
<td>1,496 (48.73%)</td>
<td>1,096 (35.70%)</td>
<td>357 (45.48%)</td>
</tr>
<tr>
<td>Obese (≥ 29.5)</td>
<td>141 (33.33%)</td>
<td>162 (38.30%)</td>
<td>778 (25.34%)</td>
<td>723 (23.55%)</td>
<td>248 (31.59%)</td>
</tr>
</tbody>
</table>

*a* Significant contrasts are reported for all characteristics.
Table 5 (cont.)

<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td></td>
<td>Husband</td>
<td>Wife</td>
<td>Husband</td>
<td>Wife</td>
<td>Husband</td>
</tr>
<tr>
<td><strong>Pain</strong></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Pain intensity (0 - 2)(d)</td>
<td>0.84 (0.68)</td>
<td>1.00 (0.65)</td>
<td>0.75 (0.65)</td>
<td>0.87 (0.65)</td>
<td>DP &gt; HP</td>
</tr>
<tr>
<td>Pain intensity (3 categories)(d)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mild</td>
<td>139 (32.86%)</td>
<td>89 (21.04%)</td>
<td>288 (36.69%)</td>
<td>315 (28.43%)</td>
<td>none</td>
</tr>
<tr>
<td>Moderate</td>
<td>214 (50.59%)</td>
<td>243 (57.45%)</td>
<td>407 (51.85%)</td>
<td>619 (55.87%)</td>
<td>none</td>
</tr>
<tr>
<td>Severe</td>
<td>70 (16.55%)</td>
<td>91 (21.51%)</td>
<td>90 (11.46%)</td>
<td>174 (15.70%)</td>
<td>DP &gt; HP</td>
</tr>
<tr>
<td>Arthritis pain (1 = yes)(e)</td>
<td>279 (90.58%)</td>
<td>289 (94.14%)</td>
<td>766 (73.94%)</td>
<td>910 (77.12%)</td>
<td>DP &gt; NP</td>
</tr>
<tr>
<td>Heart/Chest pain (1 = yes)(f)</td>
<td>75 (51.02%)</td>
<td>67 (67.68%)</td>
<td>148 (26.89%)</td>
<td>92 (33.38%)</td>
<td>113 (46.69%)</td>
</tr>
<tr>
<td><strong>Activity Limitation</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical limitations (0 - 11)</td>
<td>4.43 (2.94)</td>
<td>5.48 (3.21)</td>
<td>1.21 (1.85)</td>
<td>1.49 (1.98)</td>
<td>3.78 (2.91)</td>
</tr>
<tr>
<td><strong>Psychological Distress</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CES-D8 Depr. Sympt. (0 - 8)</td>
<td>2.21 (2.07)</td>
<td>2.61 (2.34)</td>
<td>0.85 (1.35)</td>
<td>1.11 (1.59)</td>
<td>1.82 (1.91)</td>
</tr>
</tbody>
</table>

**Note.** Cells contain \(M (SD)\) or \(n \%(\). Except for a few variables with no missing data (Age for husbands and wives; Race, Hispanic ethnicity, Yrs. of education, and Pain-specific activity limitation for wives only), cell values and the results of planned contrasts were pooled across the 8 multiply-imputed data sets. BMI = Body Mass Index (weight as kg\(^2\)/height as m\(^2\)). CES-D8 = 8-item version of the Center for Epidemiologic Studies-Depression Scale.

*Columns contain statistically significant results of planned contrasts. Husbands in DP couples were compared to husbands in NP and HP couples; Wives in DP couples were compared to wives in NP and WP couples. Univariate ANOVAs with planned contrasts (i.e., \(t\) tests) were used to test specific mean differences on continuous (or quasi-continuous) variables and chi-square tests were used to test differences in frequency counts/percentages on categorical variables.

\(^a\)Both continuous and categorical versions of BMI are presented here.

\(^b\)Underweight BMI (< 18.5) was combined with Normal weight BMI (18.5 - 24.4) because of low frequency counts.

\(^c\)During the imputation process, pain intensity was treated as quasi-interval variable (0 - 3, no pain to severe) and was mean-centered by gender in the total sample. Following imputation, pain intensity was returned to its original metric—Mild (0), Moderate (1), and Severe (2)—and is thus relevant only for those respondents with pain. Both a quasi-continuous and a categorical version of pain intensity are presented here. For the 6 husbands and 1 wife with missing data on pain intensity, the imputed pain intensity value in each MI data set was assigned to the nearest integer value. These individuals were then assigned to the pain intensity category that was closest to the average pain intensity value calculated for the total sample.

\(^d\)Question was asked of all those with arthritis, regardless of their answer to the general pain question (i.e., “Are you often troubled with pain?”). Percentages are based on those respondents who reported arthritis within each group (refer to data reported for Arthritis under “Specific disease conditions” above).

\(^e\)Question was asked of all those with recent heart-related symptoms, doctor visit, or medication use (i.e., “active” heart disease), regardless of their answer to the general pain question. Percentages are based on those respondents classified as having “active” heart disease within each group. \(N\) for husbands: 147 (DP), 550 (NP), 242 (HP), and 266 (WP). \(N\) for wives: 99 (DP), 276 (NP), 76 (HP), and 200 (WP).
in WP couples). Similarly, wives in DP couples were compared to wives in NP and WP couples. Due to the lack of prior research in this area, no a priori hypotheses were formulated or tested. Differences among the husbands (or wives) in each of the other 3 groups were not of interest, so they were not tested. Given the large sample sizes, effect size estimates were also calculated.

Results from the comparison of individual-level characteristics are organized (in text and in Table 5) by gender and, within gender, according to the order in which the constructs appear in the proposed conceptual model (Figure 5). Alternative formats were examined for a few characteristics; for instance, body weight (measured as BMI) data were examined as continuous scale values and as standard BMI categories. Any differences observed in the results are reported below. Adjustments made to the analysis plan are also noted. The review of comparisons by gender is followed by a summary highlighting the main characteristics of DP couples and the ways in which they differed from the other couples identified in the HRS 1998 sample.

**Husbands in Dual-Pain Couples and Selected Comparisons**

*Background Characteristics*

Husbands in DP couples ranged from 41 to 94 years of age. The majority of DP husbands (70%) were between the ages of 56 and 78. DP husbands had slightly more variability in age as compared to husbands in the other three groups, and this difference was statistically significant, $F_{\text{Levene}}(3, 5382) = 8.63, p < 0.001$. Thus, $t$ tests that adjusted for unequal variances were used to compare the average age of DP husbands to the average ages of NP and HP husbands. DP husbands were, on average, 1.5 years older than husbands in NP couples—a difference that was statistically significant,
The mean difference in age between DP and HP husbands, though smaller (1.2 years), was also statistically significant, \( t(843) = 2.07, p = .039 \). Cohen’s \( d \) effect size estimates (0.15 and 0.12, respectively) suggested that these mean differences were relatively small.

Only 13% of DP husbands were non-White. The majority of non-White husbands identified as Black/African American, and smaller numbers reported themselves to be of Asian, Indian, or some other race. The racial breakdown of husbands was similar across all groups. For example, between 8% and 9% of husbands in each group were of African American descent. An omnibus chi-square test confirmed that the racial composition of husbands did not differ significantly by type of couple, \( \chi^2(6) = 2.56, p = .860 \).

Just over 5% of DP husbands reported themselves to be of Hispanic ethnic heritage. This percentage varied minimally across groups, with men of Hispanic ethnicity comprising no more than 8% of each group of husbands. An omnibus chi-square test showed that the proportion of husbands reporting Hispanic ethnicity did not vary significantly across groups, \( \chi^2(3) = 3.01, p = .391 \).

On average, husbands in DP couples had completed just over 11 years of education. Considerable variability was observed, with these men reporting anywhere from 1 to 17 or more years. Although 12 years (i.e., completion of high school) was the most frequently-reported value among DP husbands, it was reported by just 30% of the group. Roughly one third (32%) of DP husbands reported completing more than 12 years of education. The remaining husbands (38%) had completed less than a high school education. Notably, a sizeable percentage of all DP husbands (12%) reported less than 8 years of formal education. Although husbands in the other groups displayed similar
levels of variability in educational attainment, their average level of education appeared slightly higher than that of DP husbands. This observation was supported statistically. A nonsignificant Levene test, $F_{\text{Levene}}(3, 5382) = 0.65, p = .581$, validated the homogeneity of variance assumption. A priori contrasts determined that the mean difference of 1.39 years between the NP and DP husbands was significant, $t(5382) = -8.08, p < .001$, as was the mean difference of 0.65 years between the HP and DP husbands, $t(5382) = -3.25, p = .001$. The effect size of the educational difference between NP and DP husbands bordered on moderate (Cohen’s $d= 0.42$, a difference of almost one half of a standard deviation unit), while the effect size for the difference between HP and DP husbands was fairly weak (Cohen’s $d = 0.20$).

Health-Related Characteristics

Husbands in DP couples reported having been diagnosed with between 0 and 6 of the seven doctor-diagnosed disease conditions assessed in the HRS interview. Recall that the total count of disease conditions was top-coded at 4 because of low numbers of both husbands and wives reporting 5 or more conditions. As a group, DP husbands were fairly dispersed over the entire (revised) range, with roughly 9% reporting zero, 26% reporting one, 26% reporting two, 22% reporting three, and 17% reporting four or more disease conditions. As a group, DP husbands averaged slightly more than two disease conditions (see column 2, Table 5). The variability observed in the disease condition counts of those husbands reporting pain (i.e., DP husbands and HP husbands) was noticeably higher than those of husbands who did not (i.e., husbands in NP and WP couples), $F_{\text{Levene}}(3, 5382) = 4.86, p = .002$. Consequently, planned comparisons utilized separate variance $t$ tests. DP husbands reported an average of almost 1 additional disease condition than their NP peers.
(mean difference = 0.86). This difference was statistically significant, \( t(519.26) = 13.48, p < .001 \). The effect size as measured by Cohen’s \( d \) was 0.70—a moderate-sized effect. Although DP husbands also reported significantly more disease conditions than husbands in HP couples, the mean difference between groups was small (0.26) and the effect size was fairly weak, \( t(816.39) = 3.46, p = .001, d = 0.21 \).

Table 5 presents frequency counts and percentages for each of the seven doctor-diagnosed disease conditions. Among husbands in DP couples, arthritis was most common, with almost three quarters of husbands reporting having been diagnosed with some form of this ailment (e.g., rheumatoid arthritis, osteoarthritis). Sizeable numbers of DP husbands reported having high blood pressure, heart disease, or diabetes. Fewer than one in seven reported being diagnosed with lung disease or any type of cancer (other than minor skin cancer). Less than 1 in 10 DP husbands reported having had a stroke.

A series of chi-square tests examined differences in the proportion of husbands reporting each disease condition across the different types of couples. All of these omnibus chi-square tests were statistically significant. In order to maintain consistency between the strategies used in parametric and nonparametric statistical testing, post-hoc decomposition of significant omnibus chi-square tests was restricted to two specific comparisons of interest: (1) comparing DP husbands to NP husbands, and (2) comparing DP husbands to HP husbands. As detailed in Chapter 3, the decomposition strategy involved testing the pairwise differences in proportions of interest using a \( z \) test (Jaccard & Becker, 2002, Appendix 15.1, pp. 156-157). A step-down testing procedure (based on Holm, 1979) ensured that the Type I error rate did not exceed .05 across the set of post-hoc tests for a given variable (i.e., a family-wise alpha of .05 was maintained). The
second-to-last column in Table 5 lists the significant pairwise differences of interest.

Of the seven disease conditions, arthritis was the condition for which the most sizeable differences were observed between DP husbands and those in the other two groups. The overall effect size as estimated by Cramer’s $V$ was .29, suggesting that group type was moderately related to reports of arthritis, explaining roughly 9% ($V^2$) of the variance. Notably, the proportion of DP husbands reporting a diagnosis of arthritis was more than twice that of husbands in the NP group and reflected an absolute difference of roughly 39%. The difference in arthritis reports between husbands in DP couples and those in HP couples was much smaller (an absolute difference of .06, or 6%), but was also statistically significant. Despite a statistically significant omnibus chi-square test for reports of cancer by type of group, decomposition revealed that the differences in reports of cancer between DP husbands and those of NP and HP husbands were not statistically significant. The difference most likely involved husbands in the WP group, who had the smallest proportion of husbands with cancer.

Decomposition revealed that the proportion of DP husbands reporting each of the remaining disease conditions was significantly higher than the proportion of NP husbands reporting these conditions. A relatively large difference was observed for lung disease, heart disease, and stroke, with roughly two to three times the proportion of DP husbands reporting these diseases as their NP counterparts. The differences observed between DP and NP husbands for high blood pressure and diabetes were modestly smaller (proportions of DP husbands were 1.2 and 1.5 times greater than those of NP husbands). Although a slightly higher proportion of DP husbands than HP husbands reported each of these 5 remaining conditions, decomposition procedures found none of these differences
to be statistically significant.

Body weight was the other health characteristic examined. As previously noted, body weight data are presented in two ways in Table 5. DP husbands had an average body mass index (BMI) of 27.93, with an average deviation of 5.24 points. DP husbands appeared to have greater variability in BMI than the husbands in most other groups. A significant Levene test confirmed the existence of discrepant variances, necessitating that planned contrasts utilize t tests that adjust for unequal variances, $F_{\text{Levene}}(3, 5382) = 12.20$, $p < .001$. The mean difference of 0.69 index points between the BMIs of DP husbands and NP husbands was statistically significant, $t(497.45) = 2.59$, $p = .010$. However, a calculated Cohen’s $d$ of 0.13 suggested that this difference was fairly small in magnitude. The difference between the average BMI values of DP and HP husbands was very small (.10 index points) and not statistically significant, $t(788.47) = .32$, $p = .750$.

In part because BMI values are difficult to interpret in the absence of objective standards, individuals were grouped into categories of body weight defined by the World Health Organization using ranges of BMI values (e.g., NIH/NHLBI, 1998). These BMI ranges are neither gender- nor age-specific—these are simply categories defined in reference to what authorities identified as a “healthy” or “normal” weight in 1998. Given the small number of DP husbands and wives with BMI values in the “underweight” range (i.e., $\leq 18.5$), this category was combined with the Normal weight category.

Two fifths of DP husbands had BMI values that classified them as overweight, one third were classified as obese, and just over one quarter of husbands were classified as normal or underweight. This distribution is in stark contrast to the distribution observed among NP husbands, which resembles a more symmetrical, bell-shaped curve.
with roughly equal proportions classified as underweight/normal or obese (.26 and .25, respectively). The distribution of HP husbands across the three weight categories was also top-heavy (like that of DP husbands), but less severely so. An omnibus chi-square test for categories of husbands’ body weight was statistically significant, \( \chi^2(6) = 25.26, p < .001 \). Decomposition revealed that DP husbands were both significantly less likely than their NP peers to have BMIs in the overweight range and significantly more likely than their NP peers to have BMIs in the obese range. These differences were statistically significant at \( p < .05 \), using the step-down procedure previously described. The slight differences in the proportions of DP and HP husbands in each BMI category were not statistically significant.

**Pain Intensity**

Comparison of husbands’ pain intensity ratings was necessarily restricted to husbands in DP and HP couples. Recall that respondents rated their pain intensity on a three-point scale—mild, moderate, or severe. Differences in pain intensity ratings were examined in two ways. First, pain intensity was treated as a continuous, quasi-interval level variable, ranging from 0 (mild) to 2 (severe). The average rating of DP husbands was .84 (between mild and moderate). A Levene test of the pain intensity ratings of DP and HP husbands indicated that both groups displayed similar levels of variability, \( F_{\text{Levene}}(1, 1206) = < 0.01, p = .971 \). A \( t \) test assuming equal variances revealed that DP husbands did rate their pain intensity slightly higher than husbands in the HP group, \( t(1206) = 2.25, p = .025 \). However, the mean difference between groups (.09 scale points) was very small (Cohen’s \( d = 0.14 \)).

The difference in pain intensity ratings between DP and HP husbands was clearly
evident when ratings of pain intensity were treated as a nominal level variable. As shown in Table 5, just over half of DP husbands rated their usual pain intensity as moderate, roughly one third rated their pain as mild, and less than one in five reported severe pain. A chi-square test indicated that pain intensity ratings of DP husbands were significantly different from those of HP husbands, \( \chi^2(2) = 6.51, p = .039 \). Note that this was consistent with the \( t \) test result above. The decomposition of this chi-square provided a valuable insight: proportionally more husbands in DP couples rated their pain as “severe” than did husbands in HP couples (.17 vs. .11).

Recall that respondents reporting certain health conditions were asked about pain associated with these conditions, regardless of their response to the general pain question (i.e., “often troubled with pain”). Fortunately, adequate numbers of husbands in each group of couples reported these disease conditions. It was thus possible to compare reports of disease-specific pain (among husbands with these conditions) across the three groups of husbands. Data for two specific types of pain—arthritis pain and heart/chest pain—are presented under the Pain heading in Table 5.

The most striking trend evident in the disease-specific pain data is the sheer frequency with which husbands who responded “No” to the general pain question admitted to pain on disease-specific follow-up questions. For example, almost three quarters of husbands with arthritis in Neither Pain couples (766 of 1,036) and in Wife-only Pain couples (291 of 394) groups reported pain as a symptom of their arthritis. The proportions of husbands with active heart disease in NP and WP couples who reported heart/chest pain were considerably smaller; however, the frequency counts were still sizeable (see Table 5).
A secondary, yet noteworthy, observation concerning these disease-specific pain data is a sizeable difference in the levels of endorsement of disease-specific pain between husbands with general pain (i.e., DP and HP husbands) and those who did not report general pain (i.e., NP and WP husbands). This difference was most marked in reports of heart/chest pain: Between one quarter and one third of NP and WP husbands with active heart disease reported heart/chest pain, while roughly half of HP and DP husbands with active heart disease reported heart/chest pain. Likewise, compared to roughly 74% of NP and WP husbands who reported pain as a symptom of arthritis, over 90% of husbands in HP and DP couples reported experiencing arthritis-related pain. Formal chi-square tests confirmed that statistically significant differences existed between the groups for both arthritis pain ($\chi^2 = 106.47, df = 3, p < .001$) and heart/chest pain ($\chi^2 = 47.41, df = 3, p < .001$). Associated effect sizes—calculated as Cramer’s $V$s—were .22 and .20, respectively, suggesting that type of couple explained approximately 4% of the variance in husbands’ reports of arthritis and heart/chest pain. Decomposition procedures that focused on the differences involving DP husbands offered additional insight. For instance, DP husbands with arthritis were significantly more likely to report arthritis-related pain than NP husbands with arthritis; DP husbands reported arthritis pain at 1.23 times the rate of NP husbands. Even more strikingly, DP husbands with active heart disease reported heart/chest pain at 1.89 times the rate of their NP peers. Decomposition procedures revealed that DP husbands were not significantly different from HP husbands in the rate with which they reported either type of disease-specific pain.

**Physical Limitations**

Activity limitation was operationalized as physical limitations in the present
study. DP husbands displayed the full range of possible scores on the physical limitations scale, from 0 to 11. On average, they reported just shy of 4.5 physical limitations, with the majority of husbands (81%) reporting 1 to 8 limitations. Only 8.3% of DP husbands \((n = 35)\) reported zero physical limitations. At the other end of the distribution, 11% of DP husbands \((n = 45)\) had nine or more physical limitations. Just over one fifth of the sample reported one or two, one third reported three to five, and roughly one quarter reported six to eight limitations. Over 70% of DP husbands reported difficulty stooping, getting up from a chair, and climbing several flights of stairs. Difficulty picking up a dime was reported by fewer than 15% of DP husbands.

Husbands in each of the other groups (NP, HP, and WP) also displayed the full range of possible physical limitations scores (i.e., 0 – 11). However, visual inspection of the data in Table 5 suggests that the distribution of DP husbands’ scores differed from those in most other groups in terms of central tendency and dispersion (with the likely exception of the HP husbands, who also displayed considerable variability). The Levene test confirmed the existence of discrepant variances, \(F_{\text{Levene}}(3, 5382) = 192.73, p < .001\). Consequently, \(t\) tests that adjusted for heterogeneous group variances were used to test the significance of the two mean differences of interest—between DP and NP husbands, and between DP and HP husbands. The average score of DP husbands was 3.21 points higher than that of NP husbands, and the estimated standard error of this difference was very small \((SE = .15)\), resulting in \(t(469.32) = 21.83, p < .001\). The calculated effect size confirmed that this was a very strong effect (Cohen’s \(d = 1.13\)). The average physical limitations score of DP husbands was also significantly higher than that of HP husbands, although the mean difference was just over one half of a scale point, \(t(853.36) = 3.64,\)
Cohen’s $d$ was estimated at 0.22, indicating a fairly weak effect.

**Psychological Distress**

On the CES-D8, the scores of DP husbands covered the entire possible range of scores, from 0 (no symptoms) to 8 (all symptoms). As a group, their scores were slightly positively skewed ($skewness = 1.02$), with progressively fewer husbands reporting more than a handful of depressive symptoms. DP husbands’ median score (2.00) was also somewhat lower than their average (2.21). However, this level of skewness was within normal limits (Curran et al., 1996). Additionally, adequate numbers of DP husbands populated the upper part of the distribution: roughly 26% reported three to five depressive symptoms, and just fewer than 10% reported 6 symptoms or more. The three items endorsed with the greatest frequency (over 38%) were: sleep was restless, felt that everything was an effort, and could not get going. One quarter of husbands endorsed the item, “I felt depressed.” Fewer than 15% of husbands reported that they did (not) enjoy life or that they felt lonely.

Looking across the last row of Table 5, the variability observed in the DP husbands’ CES-D8 scores appears greater than the variability in the scores of husbands in most of the other groups (again, with the possible exception of the HP husbands). Even more striking differences were evident from comparing the histograms of CES-D8 scores across the groups. First, the modal score for DP husbands was 1 symptom—endorsed by one quarter of husbands. In all other groups, the modal score was zero—for instance, nearly half (49%) of NP husbands reported no depressive symptoms. The distributions of scores within the other groups were more markedly skewed, having relatively fewer husbands in the upper reaches of the scale. A preliminary Levene test confirmed
substantial heterogeneity of variances across the groups, $F_{\text{Levene}}(3, 5382) = 99.68, p < .001$; thus, planned comparisons between husbands in the DP and those in the NP and HP groups utilized separate variance $t$ tests. DP husbands on average had CES-D8 scores that were 1.35 points higher than NP husbands, a difference that was statistically significant, $t(472.69) = 13.04, p < .001$. DP husbands also scored significantly higher than their HP peers, although the mean difference (0.38) was much smaller, $t(804.69) = 3.14, p = .002$. Cohen’s $d$ for the DP-NP mean difference was 0.68, indicating a moderate-sized effect; for the DP-HP mean difference, Cohen’s $d$ was .19, indicating a fairly weak effect.

**Wives in Dual-Pain Couples and Selected Comparisons**

*Background Characteristics*

Wives in DP couples were, on average, 3.5 years younger than their husbands, with a mean age of 63. Although DP wives ranged in age from 35 to 90, the majority (70%) were between 53 and 75 years old. As implied by the standard deviations in Table 5, DP wives displayed greater variability in age as compared to wives in the other groups (especially NP and WP wives). A Levene test confirmed that this difference was statistically significant, $F_{\text{Levene}}(3, 5382) = 3.35, p = .018$. For this reason, separate variances $t$ tests were used to conduct the planned comparisons between the mean ages of DP wives and their NP and WP counterparts. On average, DP wives were 1.3 years older than NP wives and this difference was statistically significant, $t(527.08) = 2.47, p = .014$. The Cohen’s $d$ effect size estimate was .13, indicating that this mean difference was of small magnitude. Although DP wives were slightly older than their WP peers, this difference (.78 years) was not statistically significant, $t(724.50) = 2.07, p = .190$.  

5-24
DP wives tended to be of the same race as their husbands, with the majority (87%) identifying as White/Caucasian. The racial composition of wives was similar across all groups of couples. For example, wives reporting a race in the “Other” category made up less than 3.5% of all groups. An omnibus chi-square test confirmed that the racial composition of wives in the DP group did not differ substantially from that observed in the other groups, $\chi^2(6) = 2.39, p = .880$. Also like their husbands, relatively few DP wives (6%) reported themselves as Hispanic. As can be seen in Table 5, this percentage varied little across the groups. An omnibus chi-square test confirmed that the proportion of wives reporting Hispanic ethnicity did not vary significantly by type of couple, $\chi^2(3) = 2.08, p = .556$.

On average, wives in DP couples had completed just under 12 years of education. Most DP wives (80%) reported completing between 8 and 14 years of schooling, but reports ranged from 0 to more than 17 years. Twelve years (i.e., completion of high school) was the most frequently-reported level of education, accounting for roughly 40% of the sample. Twenty-eight percent reported completing education beyond high school. The remaining wives (32%) reported less than a high school education, with roughly 7% reporting less than 8 years of schooling. The data in Table 5 suggest that DP wives had somewhat lower levels of education than wives in the other groups. The Levene test indicated the need to use $t$ tests that adjusted for unequal variances, $F_{\text{Levene}}(3, 5382) = 3.17, p = .023$. The mean difference of 1.01 years of education between DP and NP wives was statistically significant, $t(546.92) = -7.01, p < .001$. Although smaller, the mean difference of 0.43 years between the DP and WP wives was also significant, $t(818.07) = -2.64, p = .009$. The effect size of the educational difference between DP and
NP wives was relatively modest (Cohen’s $d = 0.36$). The effect size for the difference between DP and HP wives was even smaller ($d = 0.15$).

**Health-Related Characteristics**

Wives in DP couples reported having been diagnosed with 0 to all 7 of the seven doctor-diagnosed disease conditions assessed in HRS. DP wives’ distribution on the count of disease conditions was markedly positively skewed, with a steady decrease observed in the number of wives reporting more than 2 disease conditions. A sizeable percentage of DP wives (roughly 12%) reported no doctor-diagnosed disease conditions. Similar numbers of wives reported one, two, and three disease conditions (27%, 28%, and 23%, respectively). About 10% of wives reported four or more disease conditions. DP wives reported an average of just under two disease conditions. As previously noted, this measure was ultimately top-coded at four or more conditions. The variability in disease condition counts of DP and WP wives was noticeably higher than those of NP and HP wives; this was confirmed by a significant Levene test, $F_{\text{Levene}}(3, 5382) = 19.51, p < .001$. Consequently, separate variance $t$ tests were used to compare the mean disease condition count of DP wives to those of NP and WP wives. On average, WP wives reported almost 1 additional disease condition than their NP peers (mean difference = 0.85). This difference was statistically significant with a moderate to strong effect size, $t(514.07) = 14.28, p < .001$, Cohen’s $d = 0.74$. Although DP wives also reported significantly more disease conditions than wives in WP couples, the mean difference between these groups was small (0.23) and the effect size was fairly weak, $t(735.35) = 3.49, p = .001, d = 0.20$.

Column 3 of Table 5 presents the frequency counts and percentages of DP wives.
reporting each of the seven doctor-diagnosed conditions. Arthritis was the most commonly-reported condition (73%), followed by high blood pressure (49%). Just over one quarter of DP wives reported heart disease. Between one tenth and one fifth of DP wives reported having been diagnosed with diabetes, cancer, or lung disease. Stroke was reported by less than six percent of DP wives.

A series of chi-square tests compared the proportions of wives reporting each disease condition across the different types of couples. All of these omnibus chi-square tests were statistically significant. As before, the post-hoc decomposition of significant chi-square tests for wives was restricted to two comparisons (DP vs. NP, DP vs. WP) and utilized z tests of pairwise differences in proportion with a step-down testing procedure to maintain an overall alpha of .05. Statistically significant pairwise differences for each condition are listed in the last column in Table 5.

As was the case for husbands, the largest absolute difference in disease proportions for wives involved the condition of arthritis. An overall effect size was estimated by Cramer’s $V$ as .30, suggesting that group type was moderately related to, and explained roughly 9% of the variance in, reports of doctor-diagnosed arthritis. The proportion of DP wives reporting a diagnosis of arthritis was 1.9 times greater that of wives in the NP group, but reflected an absolute difference of 34% (very similar to the difference in arthritis reports observed between DP and NP husbands). For wives, however, this was the only statistically significant difference involving arthritis reports, as similar proportions WP wives and DP wives reported having arthritis.

Decomposition results revealed that the proportion of DP wives reporting each of the six other disease conditions was significantly higher than the proportion of NP wives
reporting them. A relatively large difference was observed for diabetes, heart disease, and lung disease, with DP wives reporting these conditions between 2.4 and 3.0 times the rates of NP wives. The differences observed between DP and NP wives with respect to reports of high blood pressure, cancer, and stroke were a bit smaller, with the proportion of DP wives reporting these conditions ranging from 1.2 to 2.0 times the proportion of NP wives. Decomposition procedures revealed that only three of these pairwise differences were statistically significant. Heart disease, lung disease, and cancer were reported by a greater proportion of DP wives than WP wives. DP wives reported these conditions between 1.3 and 1.5 times the rate of WP wives.

DP wives had an average BMI of 28.51, with an average deviation of 6.46 points on the index. Looking across the row in Table 5, it appears that DP wives displayed greater variability in BMI than did wives in the other groups. The Levene test confirmed the need to conduct planned contrasts using $t$ tests that adjusted for unequal variances, $F_{\text{Levene}}(3, 5382) = 24.15, p < .001$. The average BMI value of DP wives was over two index points higher than that of NP wives, but the calculated effect size was small, $t(500.37) = 6.38, p < .001$, Cohen’s $d = 0.33$. The average BMI value of DP wives was less than 1 index point higher than WP wives and was not statistically significant, $t(715.13) = 1.93, p = .055$.

In Table 5, wives’ BMI data are also grouped into the 1998 WHO body weight categories (WHO, 2000). The largest percentage of DP wives (almost 40%) had BMI values within the obese range. The remaining DP wives were evenly split between the underweight/normal weight and overweight categories. Much like what was observed for their husbands, the distribution of DP wives across these 3 BMI categories differed
markedly from the distribution of wives in NP couples, in which the underweight/normal weight and overweight categories were more heavily populated (with 41% and 36%, respectively). The distribution of WP wives was of an entirely different shape—more symmetrical and generally more flattened (i.e., platykurtic), with similar numbers of women in both the under/normal weight and obese categories and slightly more (about 37%) in the overweight category. The omnibus chi-square test for wives’ categorical body weight data was statistically significant, $\chi^2(6) = 81.97, p < .001$. Decomposition revealed that, compared to wives in NP couples, DP wives were both significantly less likely to have BMIs in the underweight/normal range and significantly more likely to have BMIs in the obese range. The proportion of DP wives in the obese range was also significantly greater than the proportion of WP wives in the obese range. These differences were all statistically significant at $p < .05$, using the step-down procedure previously described.

**Pain Intensity**

As a group, wives in DP couples characterized their pain intensity as moderate. Roughly equal numbers of DP wives (approximately 1 in 5) rated their pain as mild or severe. The pain intensity ratings of DP wives were compared to those of WP wives. When pain intensity was treated as a quasi-interval level measure, the Levene test evaluating the homogeneity of variance assumption was statistically significant, $F_{\text{Levene}}(1, 1529) = 7.28, p = .007$. A separate variances $t$ test revealed that DP wives reported higher pain intensity than did WP wives, $t(762.51) = 3.54, p < .001$. The calculated effect size indicated this was a fairly small difference (Cohen’s $d = 0.18$).

A more informative comparison resulted from treating wives’ pain intensity
ratings as nominal-level data. A chi-square test indicated that pain intensity ratings of the two groups were significantly different, $\chi^2(2) = 12.43$, $p = .002$. Although the result of this test was consistent with that of the $t$ test reported above, the $z$ tests done as part of the chi-square decomposition process helped to more clearly illuminate the differences between the ratings of the two groups. The distribution of WP wives’ pain intensity ratings was considerably less symmetrical than the distribution of DP wives’ pain intensity ratings. Proportionately more WP wives rated their pain as mild and fewer rated their pain as severe in comparison to the DP wives.

Table 5 also presents wives’ reports of the two types of disease-specific pain—arthritis pain and heart/chest pain. Recall that these pain questions were asked of all HRS participants, regardless of their responses to the general pain question. The two trends observed in the husbands’ disease-specific pain data were also evident in the wives’ data. First, disease-specific pain was reported by a surprising number of wives who did not report pain on the general HRS pain question (i.e., wives in the NP and HP couples). For instance, at least three quarters of wives with arthritis in NP and HP couples reported experiencing arthritis-related pain, even though they answered “no” to the general pain question (i.e., “Are you often troubled with pain?”). Second, there was a sizeable gap in the proportions of wives reporting disease-specific pain between those with general pain (i.e., DP and WP wives) and those without general pain (i.e., NP and HP wives). For example, in contrast to 77% of NP and 83% of HP wives, over 94% of DP and WP wives with arthritis reported experiencing arthritis-related pain.

A chi-square test comparing reports of arthritis pain across the different groups of wives was statistically significant, $\chi^2(3) = 137.36$, $p < .001$. Again, $z$ tests were used to
decompose this chi-square, specifically comparing the DP wives to the NP and WP wives. Only one pairwise comparison was statistically significant: Among wives with arthritis, wives in DP couples were significantly more likely (roughly 22% more likely) to report arthritis-related pain than were wives in NP couples.

Footnote “f” in Table 5 provides the number of wives within each group who were classified as having “active” heart disease at the time of the 1998 HRS interview. Just over two thirds of DP wives with active heart disease reported experiencing recent heart or chest pain. In contrast, heart or chest pain was reported by only about one third of wives with active heart disease in both the NP and HP groups. The omnibus chi-square test was statistically significant, $\chi^2(3) = 36.83$, $p < .001$. Decomposition confirmed that the proportion of DP wives reporting heart/chest pain was significantly greater than the proportion of wives in both NP (roughly 2.0 times greater) and WP (roughly 1.5 times greater) couples.

*Physical Limitations*

On average, DP wives reported just over five physical limitations. They also displayed considerable variability, with some reporting as few as 0 and others as many as 11 limitations, and a median of 6 limitations. Two dozen DP wives (roughly 6%) reported zero physical limitations, while three times as many ($n = 84, 20\%$) reported nine or more. Approximately 15% of DP wives reported one or two, 28% reported three to five, and 31% reported six to eight limitations. Much like their husbands, DP wives reported the most difficulty getting up from a chair, stooping, and climbing several flights of stairs (each over 70%). Less than 15% reported having difficulty picking up a dime.

Comparing the physical limitations data across the four groups, it would appear
that DP wives’ scores varied considerably more than the scores of other wives. A Levene test confirmed that the variances of the groups were discrepant, $F_{Levene}(3, 5382) = 212.73, p < .001$. Planned contrasts of group means thus utilized $t$ tests that adjusted for unequal variances. These tests revealed that, much like their husbands, DP wives had significantly more physical limitations than both NP and WP wives, $t(471.40) = 25.76, p < .001$ and $t(738.59) = 5.33, p < .001$, respectively. DP wives reported almost four more limitations than their NP peers—a very strong effect, as indicated by a Cohen’s $d$ of 1.34. The mean difference between DP and WP wives was just under than 1 limitation—a weak effect, as indicated by a Cohen’s $d$ of 0.31.

**Psychological Distress**

DP wives reported an average of about 2.5 depressive symptoms on the CES-D8. As suggested by the relatively large standard deviation, DP wives displayed substantial variability on this measure. As a group, their scores covered the entire range—from 0 to all 8 symptoms. DP wives’ scores were slightly positively skewed ($skewness = 0.79$), although their scores were less skewed that the scores of their husbands and were still within normal limits (Curran et al., 1996). Roughly 20% of wives reported no depressive symptoms, 38% reported one or two symptoms, 25% reported from three to five symptoms, and 17% reported six or more symptoms. Restless sleep, everything felt like an effort, and could not get going were the most frequently endorsed symptoms, each reported by more than 40% of DP wives. Nearly one third (30%) of wives reported that they felt depressed. Less than 20% of DP wives indicated that they did (not) enjoy life.

Similar to the pattern observed among husbands, wives with pain (i.e., DP and WP wives) tended to have greater variability in their CES-D8 scores than did wives.
without pain (i.e., the NP and HP wives). NP wives had the least variability and the
greatest skewness in CES-D8 scores (skewness = 1.9), with almost half (49%) of NP
wives reporting no depressive symptoms at all. A statistically significant Levene test
corroborated the observation of unequal variances, $F_{\text{Levene}}(3, 5382) = 107.14, p < .001$.
Consequently, group mean comparisons used separate variances $t$ tests. DP wives scored,
on average, 1.50 points higher on the CES-D8 than did NP wives. This difference was
statistically significant and of moderate size, $t(477.15) = 12.77, p < .001$, Cohen’s $d = 0.66$. DP wives also scored a bit higher on the CES-D8 than their WP peers. Although
this mean difference of .47 points was statistically significant, the effect was relatively
small, $t(693.01) = 3.60, p < .001, d = 0.21$.

**Overall Summary**

The focal sample for this study was the 423 married couples in which both
spouses reported pain on the 1998 HRS survey. Most Dual-Pain (DP) couples were
comprised of middle-aged or older spouses who had been married for several decades. A
majority lived in couple-only households. Their average household income was just over
$43,400, with a median of roughly $30,500. The median wealth reported in DP
households was roughly $120,000. DP couples reported markedly lower household
income and slightly less wealth than each of the 3 other groups of couples in HRS
1998—Neither Pain (NP), Husband-only Pain (HP), and Wife-only Pain (WP). In terms
of financial well-being, DP couples were most different from (i.e., considerably less well-
off than) NP couples. DP couples were not appreciably different from other couples in
terms of length of marriage or the presence of other household residents.

The average DP husband was in his mid-60s and his wife was somewhat younger,
generally in her early 60s. Most DP couples were of the same race and ethnicity, identifying as White and non-Hispanic. On average, both husbands and wives reported completing slightly less than a high school education.

The individual characteristics of DP husbands were compared to two selected groups of husbands—those from NP couples and those from HP couples. Similarly, DP wives were compared specifically to wives from NP couples and those from Wife Pain (WP) couples. The background characteristics of DP spouses differed somewhat from those of their (respective) peers. On average, both DP husbands and wives were over one year older than husbands and wives in NP couples. DP husbands were also somewhat older than HP husbands. Spouses in DP couples also had lower levels of educational than the spouses to which they were compared. The average NP husband and wife had completed roughly 1.5 years more education than the average DP husband and wife. The educational gap between DP and HP husbands and between DP and WP wives was smaller—generally just over one-half years.

In terms of health characteristics, both DP husbands and wives reported approximately 2 (of 7) doctor-diagnosed disease conditions. Arthritis was most common—reported by just over 70% of DP spouses. High blood pressure and heart disease were also fairly prevalent, and were reported by 25% to 50% of husbands and wives. DP spouses tended to be overweight, with average BMI values of roughly 28 for husbands and 28.5 for wives. Notably, one third of husbands and almost 40% of wives had a BMI value in the obese range.

Husbands and wives in DP couples were generally less healthy than those in comparison couples. The average DP husband and wife each reported one additional
disease condition than their NP peers, and the effect sizes for these differences were strong. With only one exception (cancer for husbands), they were also significantly more likely than NP spouses to report each of the seven disease conditions. Reports of arthritis, heart disease, and lung disease (also stroke for husbands, and diabetes for wives) were generally two to three times higher in DP spouses compared to NP husbands and wives. DP husbands and wives also reported slightly more disease conditions than their respective HP or WP peers. In addition, the proportions of DP wives reporting cancer, heart disease, and lung disease were also marginally higher than in WP wives. Both DP spouses tended to be heavier than the spouses in NP couples. DP husbands were less likely than NP husbands to be classified as overweight and more likely to be classified as obese. Compared to their NP counterparts, DP wives were less likely to be in the underweight/normal weight category and more likely to be in the obese category. DP wives were also somewhat more likely than WP wives to be classified as obese. Overall, health-related differences between DP and NP spouses were more pronounced than those between DP spouses and their respective HP or WP peers.

On average, DP wives rated their pain intensity as moderate. The average rating for DP husbands ranged between mild and moderate, but was closer to moderate. The pain intensity ratings of DP spouses could only be compared to those of their same-gendered peers in HP and WP couples. Although both husbands and wives in DP couples rated their pain intensity higher than their respective peers, effect sizes for these differences were very modest.

Over 90% of DP husbands and wives with arthritis reported experiencing arthritis-related pain. More than 50% of husbands and close to 70% of wives with active
heart disease reported experiencing recent heart or chest pain. These two types of
disease-specific pain were also reported by sizeable percentages of husbands and wives in
other couples. However, DP spouses reported arthritis pain at rates roughly 1.2 times
higher than those of NP spouses. Heart/chest pain reports were even more discrepant—
the proportions of DP spouses reporting such pain were approximately 2.0 times greater
than those of spouses in NP couples. Additionally, reports of heart/chest pain in DP
wives were also more common than in WP wives.

Both husbands and wives in DP couples reported, on average, a moderate number
of physical limitations. Out of 11 possible limitations, the average DP husband reported
almost 4.5, and the average DP wife reported roughly 5.5. Both genders also displayed
considerable variability on this measure. The largest differences between DP and NP
spouses were observed on the measure of physical limitations. DP husbands averaged
roughly three additional physical limitations than NP husbands. DP wives averaged
almost four limitations more than their NP counterparts. Effect sizes for these differences
were very large. DP husbands and wives also reported close to one additional physical
limitation than their HP or WP counterparts, but these effect sizes were fairly modest.

The average DP husband reported relatively modest levels of depressive
symptomatology, endorsing just over two of eight possible symptoms on the CES-D8.
The average DP wife endorsed closer to three symptoms. However, considerable
variability was observed in both genders. DP husbands and wives reported significantly
higher depressive symptomatology than their NP peers. DP husbands reported an
average of 1.4 symptoms more than NP husbands, and DP wives reported 1.5 symptoms
more than NP wives. Calculated effect sizes were fairly large. DP husbands and wives
also reported slightly more depressive symptoms than their comparison spouses in HP or WP couples, but these differences were relatively small (e.g., less than .5 symptoms).

Despite that fact that dual-pain couples represented only 8% of the total number of community-dwelling married couples who completed non-proxy interviews in the 1998 wave of HRS, these data suggest that they were roughly similar to the larger set of couples in terms of several key background and demographic characteristics. The racial composition and ethnic heritage of DP couples mirror those of the other couples. DP couples had also been married a comparable number of years and had similar living arrangements (in terms of other household residents). DP husbands and wives were a little older than other husbands and wives, especially those in NP couples, but these age differences were fairly modest in size. Most importantly, DP couples fell well within the target age range for the HRS study and can be characterized appropriately as middle-aged and older married couples.

DP couples did differ in some important ways from other couples in the 1998 HRS. In terms of background characteristics, DP couples differed most notably from other couples on socioeconomic status. On average, they had considerably less income and slightly less accumulated wealth than other couples. Additionally, both DP husbands and wives had somewhat lower levels of education than those in other couples. These differences were most pronounced in comparison to NP couples, and the largest discrepancies were observed on household income and husbands’ level of education.

The spouses in DP couples differed most notably from those in NP couples. DP husbands and wives were less healthy (both in number of diseases, and prevalence of specific diseases), more overweight and/or obese (especially wives), and more likely to
report disease-related pain. DP spouses also reported substantially more physical limitations and greater depressive symptomatology than their NP counterparts.

DP husbands and wives also differed in some ways from their same-gendered peers with pain (i.e., in HP or WP couples). In general, they reported worse health, more disease-specific pain, more physical limitations, and greater depressive symptomatology than their comparison spouses. DP spouses also rated their pain intensity a bit higher than did their peers. As previously noted, these differences tended to be less consistent (e.g., observed only for one gender) and of smaller magnitude than the differences involving NP spouses.

**Differences and Similarities Between Spouses in Dual-Pain Couples**

The section below presents the results of analyses comparing the spouses within the 423 DP couples—specifically, the similarities and differences between DP husbands and wives. In this and the sections that follow, results are generally presented in order of the proposed research questions and hypotheses (see Table 2, Chapter 4). Exceptions occur when a set of analyses addressed multiple questions or hypotheses that were not numbered consecutively. To help orient the reader, research questions and hypotheses are identified in **boldface** type (e.g., Research Question 2, RQ3, H2c).

Questions regarding the similarities and differences within DP couples were addressed using two sets of analyses. The first group of analyses addressed Research Question 1 (RQ1), which considered the existence of gender differences with respect to variables in the proposed conceptual model (part of Study Aim 4). A second set of analyses examined the extent to which DP husbands and wives were similar in terms of
key study variables (Research Question 6, RQ6; part of Study Aim 3). Note that these initial research questions (and associated analyses) were limited to the descriptive characteristics of individual DP husbands and wives—e.g., demographics, pain intensity ratings, levels of depressive symptomatology. More complex questions (and analyses) concerning the relationships between and among these variables are addressed in a later section of this chapter.

Findings regarding the differences and similarities between DP spouses are reviewed separately below. A final subsection considers the proximal implications of these findings for subsequent analyses. Because relatively small differences and similarities were likely to be statistically significant in a sample of this size, exact \( p \)-values are reported for most statistical results. In addition, effect size measures and/or confidence intervals are presented as appropriate.

Differences Between Dual-Pain Husbands and Wives

Table 6 summarizes the results from a series of bivariate analyses comparing DP husbands and wives on key study variables and other characteristics. Many of the descriptive data presented earlier (i.e., Table 5) are repeated in columns 2 and 3 of Table 6 for convenience. The results for key study constructs are presented in Panel A (pain, depressive symptomatology, and activity limitation), following the order in which associated hypotheses were developed under RQ1. Comparisons involving background and health-related characteristics (for which no a priori hypotheses were developed) are presented in Panel B.

Statistical procedures appropriate for paired data were used to evaluate husband-wife differences. Paired \( t \) tests were used to test mean differences on interval and
### Table 6. Husbands and Wives in Dual-Pain Couples Compared on Key Study Variables & Other Characteristics

<table>
<thead>
<tr>
<th>Variables</th>
<th>Dual-Pain Couples (N = 423)</th>
<th>Statistical Test</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Husbands</td>
<td>Wives</td>
<td></td>
</tr>
<tr>
<td><strong>Panel A: Key Study Variables</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Pain</strong></td>
<td></td>
<td></td>
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<tr>
<td>Pain intensity (0 - 2)</td>
<td>0.84 (0.61)</td>
<td>1.00 (0.65)</td>
<td>t (422) = -3.83</td>
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<tr>
<td><strong>Psychological Distress</strong></td>
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<tr>
<td>CES-D8 Depressive Sympt. (0 - 8)</td>
<td>2.21 (2.07)</td>
<td>2.61 (2.34)</td>
<td>t (422) = -3.13</td>
</tr>
<tr>
<td><strong>Activity Limitation</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical Limitations (0 - 11)</td>
<td>4.43 (2.94)</td>
<td>5.48 (3.21)</td>
<td>t (422) = -6.09</td>
</tr>
<tr>
<td><strong>Panel B: Other Characteristics</strong></td>
<td></td>
<td></td>
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<tr>
<td><strong>Health-related Characteristics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of disease conditions (0 - 4')</td>
<td>2.12 (1.24)</td>
<td>1.93 (1.17)</td>
<td>t (422) = 2.40</td>
</tr>
<tr>
<td>Specific conditions (1 = yes)</td>
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<tr>
<td>Arthritis</td>
<td>308 (72.81%)</td>
<td>307 (72.58%)</td>
<td>$\chi_M^2 (1) = 0.01$</td>
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<td>Cancer</td>
<td>53 (12.53%)</td>
<td>63 (14.89%)</td>
<td>$\chi_M^2 (1) = 0.84$</td>
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<tr>
<td>Diabetes</td>
<td>85 (20.09%)</td>
<td>72 (17.02%)</td>
<td>$\chi_M^2 (1) = 1.35$</td>
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<td>Heart disease</td>
<td>159 (37.59%)</td>
<td>112 (26.48%)</td>
<td>$\chi_M^2 (1) = 13.52$</td>
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<td>High blood pressure</td>
<td>207 (48.94%)</td>
<td>210 (49.65%)</td>
<td>$\chi_M^2 (1) = 0.03$</td>
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<td>Lung disease</td>
<td>60 (14.18%)</td>
<td>50 (11.82%)</td>
<td>$\chi_M^2 (1) = 1.14$</td>
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<td>Stroke</td>
<td>42 (9.93%)</td>
<td>25 (5.91%)</td>
<td>$\chi_M^2 (1) = 4.74$</td>
</tr>
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### Table 6 (cont.)

<table>
<thead>
<tr>
<th>Variables</th>
<th>Dual-Pain Couples (N = 423)</th>
<th>Statistical Test</th>
<th>(p)</th>
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<tbody>
<tr>
<td></td>
<td>Husbands  Wives</td>
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</tr>
<tr>
<td><strong>Panel B (cont’d)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body weight (BMI)(^b)</td>
<td>27.93 (5.24) 28.51 (6.46)</td>
<td>(t) (422) = -1.62</td>
<td>.106</td>
</tr>
<tr>
<td>Body weight (BMI, 3 categories)(^b)</td>
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<td>(\chi^2) (_{M-B}) (3) = 7.09</td>
<td>.069</td>
</tr>
<tr>
<td>Underweight or Normal ((&lt; 24.4))(^c)</td>
<td>111 (26.24%) 126 (29.79%)</td>
<td>(\chi^2) (_{M-B}) (3) = 7.09</td>
<td>.069</td>
</tr>
<tr>
<td>Overweight (24.5 - 29.4)</td>
<td>171 (40.43%) 135 (31.91%)</td>
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<td></td>
</tr>
<tr>
<td>Obese ((\geq 29.5))</td>
<td>141 (33.33%) 162 (38.30%)</td>
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<tr>
<td><strong>Background Characteristics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (yrs.)</td>
<td>66.56 (10.06) 62.93 (10.59)</td>
<td>(t) (422) = 12.50</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Non-white race (1 = yes)(^d)</td>
<td>55 (13.00%) 54 (12.77%)</td>
<td>(\chi^2) (_M) (1) = 0.03</td>
<td>.866</td>
</tr>
<tr>
<td>Hispanic ethnicity (1 = yes)</td>
<td>23 (5.44%) 26 (6.15%)</td>
<td></td>
<td>.508</td>
</tr>
<tr>
<td>Yrs. of education (0 - 17(^*))</td>
<td>11.44 (3.38) 11.70 (2.79)</td>
<td>(t) (422) = -1.79</td>
<td>.073</td>
</tr>
</tbody>
</table>

**Note.** Cells contain \(M (SD)\) or \(n\) (%). Except for a few variables for which neither spouse had any missing data (Arthritis pain, Age, Hispanic ethnicity, Arthritis, Diabetes, Lung disease, and Stroke), cell values and statistical test results were pooled across the 8 multiply-imputed data sets. BMI = Body Mass Index (weight as kg\(^2\)/height as m\(^2\)). CES-D8 = 8-item version of the Center for Epidemiologic Studies-Depression Scale. Statistically significant (i.e., \(p \leq .05\)) results are indicated by \(p\) -values in boldface type.

\(^a\)Paired \(t\) tests were used to test mean differences on continuous (or quasi-continuous) variables. To test differences in frequency counts/percentages for categorical variables, the McNemar-Bowker test (\(\chi^2\) \(_{M-B}\)) was used for multinomial and the McNemar test (\(\chi^2\) \(_M\)) was used for dichotomous variables.

\(^b\)Both a continuous and a categorical version of BMI are presented here.

\(^c\)Underweight BMI (\(< 18.5\)) was combined with Normal weight BMI (18.5 - 24.4) because of low frequency counts.

\(^d\)Non-white race includes individuals who reported themselves to be African American/Black or of other race.

\(^*\)Because of small observed counts in several cells, an exact test based on the cumulative binomial distribution was used, rather than calculating a chi-square value.
quasi-interval variables. Differences in proportions were examined using the McNemar chi-square test ($\chi^2_M$) for dichotomous variables, and the McNemar-Bowker chi-square test ($\chi^2_{M-B}$) for multi-category variables. Statistical test results are presented in the last two columns. Significant differences ($p < .05$) are indicated in boldface type. As before, statistical results for variables with missing data were pooled across the eight MI datasets. Thus, these analyses were based on data for all 423 DP couples.

Empirical support was found for all three hypotheses under RQ1. Test results and significance levels are reported in the final columns of Panel A in Table 6. The pain intensity ratings of wives were significantly higher than those of husbands, providing support for Hypothesis 1a. Recall that the average DP wife reported “moderate” pain, whereas the average husband reported between “mild” and “moderate” pain. This mean difference was relatively small, with wives’ average pain intensity rating roughly a quarter of a (pooled) standard deviation higher than husbands’ (Cohen’s $d = 0.26$). Additional analyses that treated pain intensity as a categorical variable are presented in the next section on spouse similarities.

Consistent with Hypothesis 1b, DP wives displayed significantly greater depressive symptomatology than did husbands. The calculated effect size (Cohen’s $d = 0.18$), however, suggested that this was a weak effect. Support for Hypothesis 1c was also found, with DP wives reporting, on average, 1 additional physical limitation than their husbands. Although the effect size of the husband-wife difference in physical limitations was the largest among the key study variables (Cohen’s $d = 0.34$), it was still a relatively small effect.

In order to gain a more thorough understanding of the gender differences in DP
couples, additional tests compared the health-related and background characteristics of husbands and wives. These corresponding descriptive data and test results are presented in Panel B of Table 6. DP husbands reported slightly more disease conditions than DP wives; yet the calculated effect size was fairly small (0.14). When disease conditions were examined individually, only two comparisons were statistically significant. The percentage of husbands reporting heart disease was 11.0% higher than wives, with a 95% CI ranging from 5.1% to 16.7%. Husbands were also more likely to report having had a stroke, although the 95% CI for the 4.0% difference between husbands and wives ranged from 0.2% to 7.4%. With the exception of exploratory analyses related to predictors of pain intensity, subsequent analyses made use of the number of disease conditions, rather than the individual conditions themselves.

In terms of body weight, the average BMI of DP wives was somewhat higher than that of their husbands. Notably though, the mean difference was just 0.6 index points, and was not statistically significant. Table 6 also shows how DP husbands and wives were distributed across the three categories of body weight. There was a discernible trend for proportionally more husbands to be overweight than wives (40% vs. 32%); however the omnibus McNemar-Bowker test was not statistically significant. Note that the continuous measure of body weight (BMI) was used in all subsequent analyses.

As shown in Table 6, DP husbands and wives were markedly similar on most background characteristics, including years of education, race, and ethnic heritage. Note that three categories of race (White, Black/African American, and Other) had been used in comparing the 4 types of couples (i.e., in Table 5). However, the number of DP husbands and wives reporting Other race was deemed too small to yield reliable estimates
in analyses limited to DP couples. Supplemental analyses (not shown) revealed that DP spouses of other race were most similar in terms of demographic and background characteristics to those who identified as Black/African American. Consequently, Black and Other race were combined into a single category of non-White race.

DP husbands and wives differed significantly on only one background characteristic—age. As might be expected within cohorts of middle-aged and older couples participating in HRS 1998, husbands were a bit older than wives. The average age difference was 3.6 years, which seems sizeable until viewed in relation to the overall variability in the sample. Cohen’s d was 0.35, indicating a relatively small effect.

**Similarities Between Dual-Pain Husbands and Wives (Paths 1, 2, and 3)**

As emphasized in Chapter 3, similarity or correspondence is important to consider within hierarchical or nested data (e.g., data from married couples). Intraclass correlations (ICCs) were used to examine the extent to which DP husbands and wives were similar in terms of observed study variables. In contrast to t tests and chi-square tests that examine differences, ICCs focus on shared variability within nested or paired data. ICCs are commonly estimated using traditional Pearson correlations. However, when applied to dyadic data, this approach has been shown to yield biased results if the two groups display differences in central tendency (Gonzales & Griffin, 1999). In order to reduce the potential for bias, experts generally recommend that ICCs for dyadic data be estimated using partial correlations, wherein the correlation coefficient is adjusted for group mean differences (e.g., Gonzales & Griffin, 1999; Kenny, Kashy, & Cook, 2006). Following these recommendations, ICCs in the present study were calculated as partial
correlation coefficients—partia ling out differences attributable to gender. For the sake of clarity and convenience, these partial correlation coefficients are hereafter referred to as adjusted ICCs or labeled with a subscript signaling this adjustment (e.g., ICC_adj).

Table 7 presents the adjusted ICCs calculated to address Research Question 6—i.e., whether DP husbands and wives exhibited significant covariation on measures of key study constructs (part of Study Aim 3). Results are again organized in two panels: Panel A for key study variables, and Panel B for health-related and background characteristics. Adjusted ICCs were calculated based on the full sample size of 423, as correlations involving variables with any missing data in the original dataset were pooled across the eight MI datasets. Unlike most other correlation coefficients (which, for interpretation purposes, must first be squared), ICCs can be interpreted directly as proportion reduction in variance measures (e.g., Gonzales & Griffin, 1999). For instance, an ICC of .40 between husbands’ and wives’ scores on a test suggests that 40% of the variance in one spouse’s test score could be explained by the score of the other spouse.

A statistically significant, but relatively small, positive adjusted intraclass correlation was observed for the pain intensity ratings of husbands and wives. This ICC_adj signals the presence of some similarity in the pain intensity reported by DP spouses, thereby providing support for Hypothesis 6a and Path 1. Specifically, 10% of the variability in spouses’ pain intensity ratings was shared.

Additional steps were taken to investigate the patterns of pain intensity ratings within these couples. This investigation was motivated by several related issues. First, and foremost, pain was undoubtedly the most central construct in the present study and therefore worthy of detailed consideration. Second, because DP couples were identified
Table 7. Intraclass Correlations between Husbands and Wives in Dual-Pain Couples (N = 423)

<table>
<thead>
<tr>
<th>Model Variables</th>
<th>Adjusted Intraclass Correlations (ICC)(^a)</th>
<th>(p)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Panel A: Key Study Variables</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain intensity (0 - 2)</td>
<td>.10</td>
<td>.034</td>
</tr>
<tr>
<td>CES-D8 (0 - 8)</td>
<td>.28</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Physical limitations (0 - 11)</td>
<td>.32</td>
<td>&lt; .001</td>
</tr>
<tr>
<td><strong>Panel B: Health-Related &amp; Background Characteristics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of disease conditions (0 - 4(^+))</td>
<td>.17</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Body weight (BMI)</td>
<td>.22</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Age (yrs.)</td>
<td>.83</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Yrs. of education (0 - 17(^+))</td>
<td>.54</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Non-white race(^b,c)</td>
<td>.82</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Hispanic ethnicity(^b)</td>
<td>.81</td>
<td>&lt; .001</td>
</tr>
</tbody>
</table>

Note. CES-D8 = 8-item Center for Epidemiologic Studies-Depression Scale. BMI = Body mass index.

\(^a\)Calculated as partial correlation coefficients in a pairwise dataset, controlling for mean differences by gender. Statistical significance tested via z-test (Gonzales & Griffin, 1999); significant results (\(p < .05\)) are indicated by \(p\)-values in boldface type. For variables with missing data for either spouse, ICCs were pooled across the 8 multiply-imputed data sets.

\(^b\)Dichotomous (1 = yes).

\(^c\)Non-white race includes individuals who reported themselves to be Black/African American or of other race.
on the basis of each spouse reporting problems with pain, it seemed prudent to examine their responses to the only additional information collected about this defining characteristic. Third, as outlined in the analysis plan (Chapter 3), pain intensity was treated in multivariate analyses as a quasi-interval level variable; analyses will generally underestimate the degree of similarity that exists between two variables that only approximate interval-level measurement properties. Fourth, it was expected that a more detailed examination of couple-level patterns in pain intensity ratings would provide additional insights regarding other similarities and differences observed in these couples. Consider that the paired $t$ test (in Table 6) indicated that, on average, wives’ pain intensity ratings were significantly higher those of husbands. At the same time, the ICC$_{adj}$ (.10 from Table 7) suggested the presence of significant similarity in spouses’ pain intensity ratings. Neither result provides information about the specific nature of the observed similarity in spouses’ ratings. Furthermore, beyond characterizing and quantifying the mean difference between husband and wife ratings (0.16), neither result conveys information regarding the existence of specific dyadic “discrepancies” or dissimilarities in the pain intensity ratings of couples.

A cross-classification table of husband and wife pain intensity ratings is presented as Table 8. The cells comprising the left-to-right diagonal of the table showed that, in roughly 45% of DP couples ($n = 189$), the pain intensity ratings of husbands and wives were exactly the same. This level of consistency was appreciably greater than implied by the ICC$_{adj}$ of .10. As might be expected, the greatest consistency was observed among couples in which both husband and wife reported “moderate” pain intensity. Thus, the data in Table 8 provided additional support for the hypothesis that DP husbands’ and
Table 8. Cross-Classification Table of Pain Intensity Ratings of Husbands and Wives in Dual-Pain Couples (N = 423)

<table>
<thead>
<tr>
<th>Husband Pain Intensity</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
<th>Marginal Totals (for husbands)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>33 (7.80%)</td>
<td>80 (18.91%)</td>
<td>26 (6.15%)</td>
<td>139 (32.86%)</td>
</tr>
<tr>
<td>Moderate</td>
<td>43 (10.17%)</td>
<td>131 (30.97%)</td>
<td>40 (9.46%)</td>
<td>214 (50.59%)</td>
</tr>
<tr>
<td>Severe</td>
<td>13 (3.07%)</td>
<td>32 (7.57%)</td>
<td>25 (5.91%)</td>
<td>70 (16.55%)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Marginal Totals (for wives)</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
<th>Overall Total (for all couples)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>89 (21.04%)</td>
<td>243 (57.45%)</td>
<td>91 (21.51%)</td>
<td>423 (100%)</td>
</tr>
</tbody>
</table>

Note. Cells contain frequency counts and percentages pooled across the 8 multiply-imputed data sets.
wives’ pain intensity ratings would positively covary (H6a).

Differences in the pain intensity ratings of spouses were also evident in Table 8 (i.e., in cells other than those along the left-to-right diagonal). Husbands and wives provided pain intensity ratings that were dissimilar in 234 DP couples (55%). A McNemar-Bowker chi-square test was statistically significant, indicating an overall difference between husband and wife pain intensity ratings in DP couples, $\chi^2_{M-B}(3) = 16.135, p = .001$. Only a small proportion of the 234 couples with dissimilar ratings (roughly 17%, $n = 39$) displayed the most extreme type of dissimilarity (i.e., with one spouse reporting “severe” pain, and the other “mild” pain). Among dissimilar couples, it was far more common for the wife’s pain intensity to be higher than her husband’s pain intensity; in fact, this ratio was almost 2:1 (146 W > H couples vs. 88 H > W couples). A specialized chi-square test for ordered-categorical data (a McNemar chi-square test of overall bias), revealed that the wife > husband pain intensity rating pattern was statistically more prevalent than the reverse, $\chi^2_M(1) = 14.38, p < .001$. Thus, Table 8 provided additional evidence to support H1a—that DP wives would report greater pain intensity than DP husbands. In summary, Table 8 provided evidence of both similarities and differences in the pain intensity ratings of DP husbands and wives. More importantly, these supplemental data helped to illuminate the specific nature of observed similarities and differences in ways that tests of mean difference and ICC could not.

Returning to Table 7, a moderately-sized, positive ICC$_{adj}$ was observed between husbands’ and wives’ CES-D8 depressive symptomatology scores. Roughly 28% of the variance in one spouse’s CES-D8 score could be explained by the CES-D8 score of the other spouse. This finding was consistent with Hypothesis 6b—that significant
covariation would be observed in spouses’ depressive symptomatology levels (Path 2). A slightly stronger, positive ICC_{adj} was calculated between the scores of husbands and wives on the physical limitations scale. Approximately 32% of the variance in one spouse’s physical limitations score could be explained by the physical limitations score of the other spouse. **Hypothesis 6c**—which predicted that the physical limitations of DP spouses would positively covary (Path 3)—was also supported by these data. These adjusted ICCs represent sizeable amounts of shared variability between husbands and wives, especially when considered in light of observed gender differences on health and background characteristics. Thus, evidence for Paths 2 and 3 in Figure 5 was strong.

Additional adjusted ICCs were computed to examine the similarity of DP husbands and wives in terms of health-related and background characteristics. These findings are presented in Panel B. Relatively modest, yet statistically significant, positive adjusted ICCs were observed for the two health-related characteristics included in the conceptual model. Even though husbands reported significantly more disease conditions than wives (as reported in Table 6), statistically significant spousal similarity was observed on this measure. Roughly 15% of the variance in the number of disease conditions reported by husbands can be explained by the number of disease conditions reported by their wives, and vice-versa. Even greater homogeneity was observed in the body weight (BMI) of spouses. After controlling for systematic variance related to gender, almost one quarter of the individual variance in BMI in the sample could be explained by knowing the couple to which an individual belonged. Very strong, positive adjusted ICCs were also observed between various demographic variables of spouses, including age, education, non-White race, and Hispanic ethnicity (all ICC_{adj} > .50).
Implications for Subsequent Analyses

The findings from husband-wife comparative analyses confirmed the existence of both similarities and differences between spouses. Most importantly, this duality was observed on the primary outcome variable—depressive symptomatology (CES-D8)—as well as on the other key study variables (pain intensity, physical limitations). Such duality is noteworthy from both an empirical and a theoretical perspective, and had specific implications for subsequent analyses. First, DP husband-wife similarity on key constructs provided additional justification for using multilevel modeling to analyze these data. Second, the differences observed between spouses reinforced the planned emphasis on gender in the current study. Both issues are addressed in more detail below.

Use of Multilevel Modeling

The adjusted ICCs offer strong support for the use of multilevel modeling to analyze the CES-D8 scores (i.e., the primary outcome) of DP husbands and wives, as well as their physical limitation scores (as the proposed mediator, it is an intermediate outcome). Husbands’ and wives’ scores on these measures were clearly interrelated, violating a critical assumption of the traditional linear model—indeed, non-independence of observations. Consequently, statistical techniques (e.g., multilevel modeling) that take account of such non-independence are warranted in modeling these outcomes.

Whether or not the analysis of DP husbands’ and wives’ pain intensity ratings (i.e., the primary independent variable, but also an outcome in some analyses) necessitates the use of multilevel modeling is potentially debatable. There is disagreement among scholars as to the necessity of using multilevel modeling for outcomes that are only weakly correlated within cluster or group, especially at the low
levels observed here (e.g., ICCs ≤.10). Some contend that it is not until ICCs reach more sizeable levels (e.g., > .20) that researchers should become worried about generating seriously biased results using traditional, single-level models (see Browne & Rabash, 2004; and Lee, 2000, for discussion). However, others point to simulation studies and personal experiences that illustrate how ignoring even small amounts of non-independence within the data can lead to significant bias (e.g., Kenny et al., 2006; Musca et al., 2011). Raudenbush and Bryk (2002, p. 258), and more recently Roberts (2007), reminded researchers that it is erroneous to conclude from the presence of a small, or near-zero ICC, that non-independence does not exist.

ICCs are most often calculated at a strictly bivariate level (i.e., no other variables are controlled or partialled-out). Roberts noted, however, that “the degree of observational dependence actually is determined by the nature of the covariates/predictors chosen to be included in the model” (2007, p. 3). He provided two empirical examples in which the initial ICCs were very tiny (e.g., less than 2.00 E-7). In one case, non-independence was masked by the violation of distributional assumptions; in the other, it was apparent only after a specific predictor was added to the model.

In theory, if the ICC for an outcome is truly very small, the results of traditional and multilevel modeling approaches should yield similar results. If one were to apply multilevel modeling in the absence of a sizeable ICC, it is possible that adjustments made to the degrees of freedom and to the calculation of standard errors might result in some minor bias in decisions regarding statistical significance. However, such biases are likely to be conservative in nature—i.e., to be overly-protective in guarding against Type I errors (Roberts, Monaco, Stovall, & Foster, 2010). Thus, from a practical standpoint, the
use of multilevel modeling for all multivariate analyses in the present study (even those of data with small ICC estimates) can be regarded as a conservative analytic strategy.

One additional note regarding other estimates of spousal covariation observed in these data: Sizeable adjusted ICCs were observed on some demographic and background variables (e.g., age, education). In most linear modeling applications, predictor variables correlated at levels above .80 would fuel realistic concerns about multicollinearity (e.g., Tabachnick & Fidell, 2001). However, because the background characteristics of each spouse were used only as predictors of his/her own outcome in the present study, these high ICCs were not regarded as problematic. Diagnostics were also conducted to ensure that multicollinearity was not a problem in multivariate models.

**Emphasis on Gender**

Married (heterosexual) couples are mentioned often as a classic example of “distinguishable dyads;” that is, dyads in which members can be distinguished on the basis of some characteristic—in this case, gender. However, Kenny and colleagues (2006) have argued that distinguishing characteristics should also be “meaningful.” Accordingly, Chapters 2 and 3 reviewed existing evidence of gender differences in pain, activity limitation, and depressive symptomatology. Relevant theoretical or empirical explanations for such differences were also reviewed briefly. And, as just presented, analyses revealed some significant differences between DP husbands and wives in terms of key study variables including depressive symptomatology, physical limitations, and pain intensity. These different sources of data attest to the “meaningfulness” of gender within the present context. Moreover, such data provide additional justification for exploring gender differences in DP couples with respect to the presence and magnitude of
the relationships between key study variables. As noted in Chapter 4, dual-intercept multilevel models permit the systematic investigation of gender differences. By using such models, DP husbands’ and wives’ data could be analyzed separately, but simultaneously, in order to examine whether the hypothesized relationships among pain intensity, physical limitation, and depressive symptomatology differed by gender.

**Intra-individual Relationships**

This section presents the results of analyses at the *intra*-individual level in DP husbands and DP wives. Prior to reviewing results pertaining to specific research questions and hypotheses, consideration is given to the bivariate correlations calculated separately by gender. A general overview of these bivariate relationships is presented in the first section below. The next section presents the substantive findings related to **Research Question 2**—i.e., predictors of pain intensity. A final section discusses the results of analyses addressing the hypothesized intra-individual relationships among pain intensity, physical limitation, and depressive symptomatology (i.e., **Research Questions 3 and 4**). Findings with regard to gender differences (**Exploratory Question 5**) are reviewed throughout.

**Overview of Bivariate Relationships**

Bivariate (Pearson) correlation coefficients were calculated at the *intra*-individual level for each pair of variables to be included in the proposed multivariate models. For variables with missing data in the original dataset, correlation coefficients were pooled across the eight MI datasets. Thus, all correlations were based on \( N = 423 \). Correlation matrices are presented separately—husbands in Table 9, wives in Table 10. The large sample size resulted in even relatively weak correlations (e.g., \( r = .10 \)) being statistically
Table 9. Correlations Among Model Variables for Husbands in Dual-Pain Couples (N = 423)

<table>
<thead>
<tr>
<th>Model Variables</th>
<th>Pain intensity</th>
<th>Physical limitations</th>
<th>No. disease conditions</th>
<th>Body weight (BMI)</th>
<th>Age</th>
<th>Non-white race&lt;sup&gt;a, b&lt;/sup&gt;</th>
<th>Hispanic&lt;sup&gt;c&lt;/sup&gt;</th>
<th>Yrs. education</th>
<th>(CPL) HH income (log)</th>
<th>(CPL) HH wealth (ihs)</th>
<th>(CPL) Others in HH&lt;sup&gt;d&lt;/sup&gt;</th>
<th>(CPL) Yrs. married</th>
</tr>
</thead>
<tbody>
<tr>
<td>CES-D8 (0 - 8)</td>
<td>.18</td>
<td>.38</td>
<td>.16</td>
<td>-.08</td>
<td>-.02</td>
<td>.10</td>
<td>.08</td>
<td>-.22</td>
<td>-.26</td>
<td>-.19</td>
<td>.13</td>
<td>-.03</td>
</tr>
<tr>
<td>Pain intensity (0 - 2)</td>
<td></td>
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<tr>
<td>Physical limitations (0 - 11)</td>
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<tr>
<td>No. disease conditions (0 - 4&lt;sup&gt;c&lt;/sup&gt;)</td>
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<tr>
<td>Body weight (BMI)</td>
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<td>Age (yrs.)</td>
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<tr>
<td>Non-white race&lt;sup&gt;a, b&lt;/sup&gt;</td>
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<tr>
<td>Hispanic&lt;sup&gt;c&lt;/sup&gt;</td>
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<tr>
<td>Yrs. education (0 - 17&lt;sup&gt;c&lt;/sup&gt;)</td>
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<tr>
<td>(CPL) HH income (log)</td>
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<tr>
<td>(CPL) HH wealth (ihs)</td>
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</tr>
<tr>
<td>(CPL) Others in HH&lt;sup&gt;d&lt;/sup&gt;</td>
<td></td>
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<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. Correlation coefficients were pooled across the 8 multiply-imputed data sets. Correlations in shaded cells are statistically significant at $p < .05$; those in bold are of at least moderate strength ($r > .30$). CPL = Couple-Level variable. CES-D8 = Center for Epidemiologic Studies-Depression Scale. BMI = Body mass index. HH = Household. ihs = Inverse hyperbolic sine transformation.

<sup>a</sup>Dichotomous (1 = yes).

<sup>b</sup>Non-white race includes individuals who reported themselves to be African American/Black or of other race.
Table 10. Correlations Among Model Variables for Wives in Dual-Pain Couples (N = 423)

<table>
<thead>
<tr>
<th>Model Variables</th>
<th>Pain intensity</th>
<th>Physical limitations</th>
<th>No. disease conditions</th>
<th>Age</th>
<th>Non-white race&lt;sup&gt;a,b&lt;/sup&gt;</th>
<th>Hispanic&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Yrs. education</th>
<th>(CPL) HH income (log)</th>
<th>(CPL) HH wealth (ihs)</th>
<th>(CPL) Others in HH&lt;sup&gt;a&lt;/sup&gt;</th>
<th>(CPL) Yrs. married</th>
</tr>
</thead>
<tbody>
<tr>
<td>CES-D8 (0 - 8)</td>
<td>.30</td>
<td>.36</td>
<td>.15</td>
<td>.04</td>
<td>-.04</td>
<td>.10</td>
<td>.15</td>
<td>-.27</td>
<td>-.24</td>
<td>-.20</td>
<td>.08</td>
</tr>
<tr>
<td>Pain intensity (0 - 2)</td>
<td>.35</td>
<td>.26</td>
<td>.07</td>
<td>.07</td>
<td>.04</td>
<td>.06</td>
<td>-.12</td>
<td>-.09</td>
<td>-.13</td>
<td>&lt;.01</td>
<td>.08</td>
</tr>
<tr>
<td>Physical limitations (0 - 11)</td>
<td>.39</td>
<td>.18</td>
<td>.11</td>
<td>.11</td>
<td>.00</td>
<td>-.26</td>
<td>-.32</td>
<td>-.19</td>
<td>&lt;.01</td>
<td>.01</td>
<td></td>
</tr>
<tr>
<td>No. disease conditions (0 - 4&lt;sup&gt;a&lt;/sup&gt;)</td>
<td>.15</td>
<td>.23</td>
<td>.05</td>
<td>-.03</td>
<td>-.20</td>
<td>-.13</td>
<td>-.15</td>
<td>-.06</td>
<td>.09</td>
<td></td>
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</tr>
<tr>
<td>Body weight (BMI)</td>
<td></td>
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<tr>
<td>Age (yrs.)</td>
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</tr>
<tr>
<td>Non-white race&lt;sup&gt;a,b&lt;/sup&gt;</td>
<td></td>
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</tr>
<tr>
<td>Hispanic&lt;sup&gt;a&lt;/sup&gt;</td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>Yrs. education (0 - 17&lt;sup&gt;a&lt;/sup&gt;)</td>
<td></td>
<td></td>
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<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>(CPL) HH income (log)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(CPL) HH wealth (ihs)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(CPL) Others in HH&lt;sup&gt;a&lt;/sup&gt;</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

Note. Correlation coefficients were pooled across the 8 multiply-imputed data sets. Correlations in shaded cells are statistically significant at $p \leq .01$; those in **bold** are of at least moderate strength ($r \geq .30$). CPL = Couple-Level variable. CES-D8 = Center for Epidemiologic Studies-Depression Scale. BMI = Body mass index. HH = Household. ihs = Inverse hyperbolic sine transformation.

<sup>a</sup>Dichotomous (1 = yes).

<sup>b</sup>Non-white race includes individuals who reported themselves to be African American/Black or of other race.
significant at $p \leq .05$. For this reason, correlations of at least moderate strength (i.e., $r \geq .30$) are presented in boldface type. Note that each matrix also contains the couple-level background characteristics (e.g., household income). These correlation matrices are reviewed here in general terms only. Several specific bivariate correlations are discussed in more detail in subsequent sections, prior to the presentation of findings from multivariate analyses.

The correlations presented in Tables 9 and 10 provided adequate empirical justification for proceeding with the planned multivariate analyses. Both matrices contained a large number of statistically significant bivariate correlations (shaded cells). A sizeable number of these were of at least moderate strength (boldface text), especially those involving pairs of key study variables. Moreover, these correlations were in the expected direction—i.e., they were consistent with hypothesized relationships.

For the most part, individual and couple-level indicators of SES (years of education, household income, household wealth) evidenced consistent—albeit relatively small—associations with key study variables. Most of these relationships were in the expected directions (e.g., higher education was associated with lower depressive symptomatology). SES indicators were also related to each other, as well as to other background measures. Relatively few statistically significant relationships were observed between other individual- and couple-level background characteristics (age, non-White race, Hispanic ethnicity, others in household, and years married) and key study variables. Instead, most background variables were selectively associated with health-related characteristics and/or each other, and most of these relationships were modest in size. One notable exception was the strong relationship between age and years of marriage.
Of the two health-related characteristics, number of disease conditions demonstrated more sizeable and consistent relationships with key study variables and background characteristics than body weight (measured as BMI).

Several additional observations deserve special comment. First, a general comparison of Tables 9 and 10 reveals slightly more statistically significant correlations in DP wives’ variables than in husbands. In fact, most observed correlations were marginally larger for wives than for husbands. This pattern is most evident in the correlations among key study variables. This issue is addressed in more detail later in this chapter.

Second, the strong correlation between age and years of marriage ($r = .55$ in husbands, $r = .60$ in wives) raised concerns regarding possible collinearity in multivariate analyses. Although years of marriage had small, but statistically significant relationships with some other background and health-related variables (e.g., others living in the household, body weight), it was not significantly related to pain intensity, physical limitations, or depressive symptomatology in either DP husbands or wives. Hence, years of marriage was excluded from all subsequent analyses.

Third, race is represented in Tables 9 and 10 as non-White vs. White. Race is regarded as a background characteristic in the present study, and a variable to be controlled in multivariate analyses. In preliminary bivariate analyses (data not shown), most relationships between race and other variables were not appreciably different for Blacks and those of other minority race. For instance, both Black husbands and those of other minority race had significantly fewer years of education, as compared to white husbands ($r = -.16$ and -.13, both $p < .01$). When more sizeable differences were
observed, they were generally differences in the strength of relationships, rather than direction. For example, in husbands, both Black and other minority race were negatively related to household income \( (r = -.10 \text{ and } -.22, \text{ respectively}) \). In several instances, combining Black/African American and other minority race into a single category yielded correlations that were a bit stronger than had been observed for either race separately. For instance, the correlation in Table 10 between non-White race and age among wives was \( r = -.13 \) \( (p = .007) \); this was slightly larger than the correlations between age and both Black race \( (r = -.11, p = .029) \) and other minority race \( (r = -.07, p = .151) \). Given the relatively small number of DP spouses who were Black (9\% of husbands and wives) or other minority race (4\% of husbands, 3\% of wives), race was collapsed into a dichotomous variable indicating non-White race.

Fourth, although Hispanic ethnicity was significantly related to a few other background variables (e.g., years of education, household income), ethnicity demonstrated few statistically significant relationships with key study variables. In fact, Hispanic ethnicity was only weakly related to CES-D8 among wives \( (r = .15, p = .002) \). The lack of a sizeable number of significant bivariate relationships involving Hispanic ethnicity, along with the small numbers of DP husbands \( (n = 23, 5.4\%) \) and wives of Hispanic descent \( (n = 26, 6.2\%) \), provided additional justification for excluding Hispanic ethnicity as a variable in further analyses.

**Predictors of Pain Intensity**

**Research Question 2** sought to identify the background and health-related characteristics related to the pain intensity ratings of DP husbands and wives. Specifically, **Hypothesis 2** proposed that older age, minority race, a greater number of
disease conditions, and higher body weight would be associated with greater pain intensity. **Exploratory Question 2 (EQ2)** asked if particular disease conditions were more strongly associated with greater pain intensity. Findings from the bivariate analyses are reviewed first. Multivariate results are then presented. In order to address EQ2, the seven individual disease conditions were substituted for the count of disease conditions in an alternative multivariate model. A final subsection briefly summarizes all findings and ties them back to the proposed research questions and hypotheses.

**Bivariate Results**

Relatively few individual- or couple-level background characteristics were significantly related to pain intensity in DP husbands (Table 9) or wives (Table 10). However, there were two exceptions. First, greater household wealth was weakly associated with lower pain intensity levels in both husbands and wives. Second, greater education was related to lower pain intensity levels, but only in wives.

In terms of health-related characteristics, BMI was not significantly associated with the pain intensity ratings of either husbands or wives. Number of disease conditions was significantly related to greater pain intensity in both DP husbands and wives. In order to provide a context for the observed relationships between pain intensity and number of disease conditions, the distribution of disease conditions in DP spouses was explored in greater detail.

Table 11 shows the distribution of number of disease conditions reported by DP husbands (in columns 2 and 3) and DP wives (in columns 5 and 6). A majority of husbands (64.7%) and wives (61.4%) reported two or more of the seven disease conditions queried in HRS. Over one third of wives and almost 40% of husbands
Table 11. Disease Condition Counts & Top 3 Patterns for Husbands and Wives in Dual-Pain Couples (N = 423)

<table>
<thead>
<tr>
<th>Number of Conditions</th>
<th>HUSBANDS</th>
<th></th>
<th>WIVES</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>(%)</td>
<td>Number of Patterns</td>
<td>n</td>
</tr>
<tr>
<td>0 (none)</td>
<td>40</td>
<td>(9.5%)</td>
<td>1</td>
<td>49</td>
</tr>
<tr>
<td>1</td>
<td>109</td>
<td>(25.8%)</td>
<td>7</td>
<td>114</td>
</tr>
<tr>
<td>2</td>
<td>109</td>
<td>(25.8%)</td>
<td>12</td>
<td>120</td>
</tr>
<tr>
<td>3</td>
<td>92</td>
<td>(21.7%)</td>
<td>21</td>
<td>96</td>
</tr>
<tr>
<td>4 or more</td>
<td>73</td>
<td>(17.3%)</td>
<td>24</td>
<td>44</td>
</tr>
<tr>
<td>Total</td>
<td>423</td>
<td>(100.0%)</td>
<td>65</td>
<td>423</td>
</tr>
</tbody>
</table>

Top 3 Patterns

- (1) Arth only (17.0%)
- (2) Arth + HiBP (10.9%)
- (3) No Conditions (9.5%)

Note: Disease count data were pooled across the 8 multiply-imputed data sets. The 7 disease conditions queried in HRS included: Arthritis (Arth), High blood pressure (HiBP), Heart disease, Lung disease, Diabetes, Cancer, and Stroke.
indicated 3 or more conditions. A greater proportion of husbands than wives reported four or more conditions (17.3% vs. 10.4%). These distributional data provided clear evidence of multi-morbidity among both DP husbands and DP wives.

Given that the majority of spouses reported two or more disease conditions, supplemental analyses explored the patterns of disease co-occurrence. As suggested by the data in columns 4 and 7 of Table 11, a variety of different disease combinations were observed. For instance, among those with four or more disease conditions, husbands displayed 24 and wives displayed 20 different combinations of the 7 specific disease conditions queried by HRS. In total, husbands displayed 65 different combinations and wives displayed 61 different combinations of the 7 conditions. Both spouses displayed roughly half of all possible disease condition combinations ($2^7 = 128$). The bottom row of Table 11 lists the three most prevalent patterns observed in husbands and wives. Interestingly, although prevalence rates differed to some degree by gender (as shown in Table 5), the three most common patterns (arthritis only, arthritis and high blood pressure, and no conditions) were the same for both spouses. Together these three patterns accounted for over one third of DP husbands ($n = 158$, 37.4%) and over forty percent of DP wives ($n = 188$, 44.4%).

Additional correlation analyses (not shown) examined the relationships between pain intensity and each of the seven specific disease conditions. Four of the seven disease conditions were significantly related to pain intensity in DP husbands. Higher pain intensity was observed among husbands reporting lung disease, heart disease, arthritis, and/or high blood pressure. Although statistically significant, these correlations were relatively weak. Three conditions—cancer, diabetes, and stroke—were not
significantly related to pain intensity in husbands.

Much like their husbands, DP wives with heart disease, arthritis, lung disease, and/or high blood pressure reported greater pain intensity. Unlike in husbands, however, higher pain intensity in wives was also associated with diabetes. Neither stroke nor cancer was significantly related to wives’ pain intensity.

**Multivariate Results**

Table 12 displays the results of multilevel analyses in which the pain intensity ratings of DP spouses were regressed on individual- and couple-level background and health-related characteristics. In recognition of the nested nature of these dyadic data, dual-intercept hierarchical linear models were used to examine the pain intensity of husbands and wives separately, but simultaneously. Recall that the estimated reliability of the pain intensity measure in husbands and wives was used to fix the initial error variances in these models (see Chapter 4 and Appendix C). Model results for husbands are presented in Panel A; those for wives are presented in Panel B.

Model 0 served as a baseline, estimating the dual intercepts—one for husbands, one for wives—and their associated variances. As per the analysis plan, predictors were added to this baseline model in blocks in order to examine the relative strength of each set of predictors. Individual- and couple-level background characteristics were entered in Model 1. Health-related characteristics were entered in Model 2. In Alternate Model 2, the seven individual disease conditions were substituted in place of the number of disease conditions. With the exception of the number of disease conditions, all individual-level continuous predictors were mean-centered by gender, and all couple-level predictors were centered across the entire sample of 423 couples. A proportion reduction in error
Table 12. Intra-Individual Predictors of Pain Intensity in Dual-Pain Husbands and Wives (N = 423)

<table>
<thead>
<tr>
<th>Panel A: Husbands (N = 423)</th>
<th>Model 0</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Alt. Model 2</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Fixed Effects</strong> [unstand. coefficient (SE)]:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>0.84 (.03)</td>
<td>0.87 (.04)</td>
<td>0.60 (.07)</td>
<td>0.56 (.07)</td>
</tr>
<tr>
<td><strong>Background characteristics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>-0.01 (&lt;.01)</td>
<td>-0.01 (&lt;.01)</td>
<td>-0.01 (&lt;.01)</td>
<td></td>
</tr>
<tr>
<td>Non-white race</td>
<td>-0.01 (.11)</td>
<td>0.01 (.11)</td>
<td>0.04 (.11)</td>
<td></td>
</tr>
<tr>
<td>Yrs. education</td>
<td>-0.01 (.01)</td>
<td>-0.01 (.01)</td>
<td>-0.01 (.01)</td>
<td></td>
</tr>
<tr>
<td>HH income (log)</td>
<td>-0.05 (.04)</td>
<td>-0.03 (.04)</td>
<td>-0.04 (.04)</td>
<td></td>
</tr>
<tr>
<td>HH wealth (ihs)</td>
<td>-0.02 (.01)</td>
<td>-0.01 (.01)</td>
<td>-0.01 (.01)</td>
<td></td>
</tr>
<tr>
<td>Others in HH</td>
<td>-0.10 (.08)</td>
<td>-0.09 (.08)</td>
<td>-0.07 (.07)</td>
<td></td>
</tr>
<tr>
<td><strong>Health-related characteristics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| No. disease conditions (0 – 4+)
  Specific conditions (1 = yes) | 0.12 (.03)    |                  |                  |              |
| Arthritis                   | 0.19 (.07)       |                  |                  |              |
| Cancer                      | 0.04 (.10)       |                  |                  |              |
| Lung disease                | 0.35 (.10)       |                  |                  |              |
| Diabetes                    | -0.04 (.08)      |                  |                  |              |
| Heart disease               | 0.16 (.07)       |                  |                  |              |
| High blood pressure         | 0.12 (.07)       |                  |                  |              |
| Stroke                      | -0.08 (.11)      |                  |                  |              |
| Body weight (BMI)           | -0.01 (.01)      | -0.01 (.01)      |                  |              |
| **Random Effect** [variance (SD)]: | 0.37 (.61)       | 0.36 (.60)       | 0.34 (.58)       | 0.32 (.57)   |

PRE$^a$ | .02 | .08 | .12 |
## Panel B: Wives ($N = 423$)

### Fixed Effects  [unstand. coefficient (SE):]

<p>| | | | | |</p>
<table>
<thead>
<tr>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td><strong>Intercept</strong></td>
<td>1.00</td>
<td>0.97</td>
<td>0.76</td>
<td>0.75</td>
</tr>
<tr>
<td><strong>Background characteristics</strong></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>0.04</td>
<td>0.01</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>Non-white race</td>
<td>-0.01</td>
<td>-0.02</td>
<td>-0.02</td>
<td>-0.02</td>
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<tr>
<td>Yrs. of education</td>
<td>-0.02</td>
<td>-0.01</td>
<td>-0.01</td>
<td>-0.01</td>
</tr>
<tr>
<td>HH income (log)</td>
<td>-0.02</td>
<td>-0.01</td>
<td>-0.01</td>
<td>-0.01</td>
</tr>
<tr>
<td>HH wealth (ihs)</td>
<td>-0.01</td>
<td>-0.01</td>
<td>-0.01</td>
<td>-0.01</td>
</tr>
<tr>
<td>Others in HH</td>
<td>-0.02</td>
<td>-0.01</td>
<td>-0.01</td>
<td>-0.02</td>
</tr>
<tr>
<td><strong>Health-related characteristics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. disease conditions ($0 – 4^*$)</td>
<td>0.13</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Specific conditions ($1 = yes$)</td>
<td></td>
<td>0.16</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arthritis</td>
<td></td>
<td></td>
<td></td>
<td>0.16</td>
</tr>
<tr>
<td>Cancer</td>
<td></td>
<td></td>
<td></td>
<td>-0.01</td>
</tr>
<tr>
<td>Lung disease</td>
<td></td>
<td></td>
<td></td>
<td>0.21</td>
</tr>
<tr>
<td>Diabetes</td>
<td></td>
<td></td>
<td></td>
<td>0.16</td>
</tr>
<tr>
<td>Heart disease</td>
<td></td>
<td></td>
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<td>0.13</td>
</tr>
<tr>
<td>High blood pressure</td>
<td></td>
<td></td>
<td></td>
<td>0.09</td>
</tr>
<tr>
<td>Stroke</td>
<td></td>
<td></td>
<td></td>
<td>0.06</td>
</tr>
<tr>
<td>Body weight (BMI)</td>
<td></td>
<td></td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td><strong>Random Effect [variance (SD):]</strong></td>
<td>0.35</td>
<td>0.33</td>
<td>0.31</td>
<td>0.31</td>
</tr>
<tr>
<td>PRE$^a$</td>
<td>.03</td>
<td>.10</td>
<td>.11</td>
<td></td>
</tr>
<tr>
<td><strong>Model Deviance$^b$</strong></td>
<td>3755.12</td>
<td>3736.20</td>
<td>3693.61</td>
<td>3675.16</td>
</tr>
</tbody>
</table>
Note. HH = Household; log = Log-transformation; ihs = Inverse hyperbolic sine transformation. Pain intensity was rated on a 3-point scale from 0 (mild) to 2 (severe). Effects for husbands and wives were estimated separately, but simultaneously, via a dual-intercept hierarchical linear model. With the exception of the number of disease conditions, all individual-level continuous variables were mean-centered by gender and all couple-level continuous variables were centered across the 423 couples. To permit estimation, initial error variances were fixed by gender (details in text and Appendix C). Model 0 estimated the dual intercepts (1 for husbands, 1 for wives) only; Model 1 added the individual’s own background characteristics and those of the couple; Model 2 added the individual’s own health-related characteristics. Alternate (Alt.) Model 2 substituted the 7 specific disease conditions in place of the number of disease conditions. All parameter estimates and the robust SEs for the fixed effects were pooled across the 8 multiply-imputed data sets. Throughout the table, shading indicates a statistically significant effect at $p < .05$.

$^a$PRE = Proportion reduction in error measure, calculated as the proportion of variance explained by each model as compared to the total (explainable) variance from Model 0.

$^b$Model deviance estimates were pooled across the 8 multiply-imputed data sets. A chi-square test of statistical significance compared the deviance of each model to that of the prior nested model (note: the deviance of Alternate Model 2 was compared to Model 1). The resulting $\chi^2 (df)$ and $p$-values were as follows: Model 1 = 18.92 (12), $p = .090$; Model 2 = 42.59 (4), $p < .001$; Alternate Model 2 = 61.04 (16), $p < .001$. 
measure (PRE) was calculated separately by gender for each model. The PRE can be roughly interpreted as the proportion of variance explained, compared to the total (explainable) variance estimated in Model 0. Since models were nested, the relative fit of each model for the total sample was evaluated against that of the prior model using a chi-square test of model deviance (note: Alternate Model 2 was compared to Model 1). The parameter estimates (i.e., the unstandardized fixed effect coefficients and the random effect variances after Model 0), the robust standard errors for the fixed effects, and the deviance values for each model were pooled across the eight MI datasets. PRE measures and tests of model deviance were calculated using the (pooled) random effects and model deviance values.

The intercepts estimated in Model 0 represent the estimated mean pain intensity rating of DP husbands (0.84 ~ between “mild” and “moderate”) and DP wives (1.00 ~ “moderate”). Note that these are equivalent to the means presented earlier in Table 6. Both intercepts were significantly different from zero ($p < .001$). In Model 0, the random effects represent the amount of variance to be explained. The variances in Table 12 are slightly smaller than the observed variances ($SD^2$) reported in Table 6, as they now represent the amount of “explainable” variance (i.e., variance with estimated error removed). Variances for husbands and wives were both significantly different from zero ($p < .001$), indicating the presence of considerable explainable variance in spouses’ pain intensity ratings.

Model 1 added the set of individual-level and couple-level background variables. Of the six background variables, only household wealth (ihs-transformed) was a statistically-significant, independent predictor of pain intensity, and only for wives.
controlling for the effects of all other background variables, a one-unit increase in household wealth was associated with a very small decrease in DP wives’ pain intensity ratings ($\gamma = -0.01, p = .022$). Household wealth was estimated to have a similarly small effect on husbands’ pain intensity ratings ($\gamma = -0.02$), but the variability around this estimate was more pronounced, rendering the estimated effect non-significant ($p = .068$).

None of the other couple-level variables was independently associated with either husbands’ or wives’ pain intensity ratings. Contrary to expectations, none of the individual-level background characteristics (age, non-White race, years of education) was significantly related to pain intensity in husbands or wives. Together, the six background characteristics explained only 2% of the variance in husbands’ pain intensity ratings and only 3% of the variance in wives’ ratings. The overall deviance of Model 1 was not significantly different from that of Model 0, $\chi^2(12) = 18.92, p = .090$. The statistically significant random effects for both husbands and wives indicated that substantial amounts of variance in their pain intensity ratings remained unexplained.

Health-related characteristics were added as predictors in Model 2 (column 4). Surprisingly, body weight (measured as BMI) was not a statistically-significant predictor of pain intensity for either husbands or wives. Number of disease conditions, however, was a significant predictor of pain intensity for both spouses. Consistent with expectations, after controlling for individual and couple-level background characteristics, having more disease conditions was associated with greater pain intensity in DP husbands and DP wives. Model-based prediction equations were used to illustrate the nature and size of these estimated effects in the “average” DP husband and wife below.

The “average” DP husband was 67 years-old, white, had an 11th-grade education,
had average income and wealth (as compared to other DP couples), lived only with his spouse, and had a BMI in the upper end of the “overweight” category. Based on Model 2, an otherwise average DP husband with no disease conditions was predicted to have a pain intensity rating of 0.60 ($\gamma$ intercept in Model 1, roughly midway between “mild” and “moderate”). The parameter estimate for the number of disease conditions ($\gamma = 0.12, p < .001$), indicates that each additional disease condition was associated with a very small increase in pain intensity—equivalent to about two tenths of a standard deviation in husbands’ pain intensity rating (note: compare to the $SD$ of the random effect from Model 0 in Panel A of Table 11, 0.61). Although the estimated effect of a single condition was small, consider the additive effect of multiple disease conditions: If an otherwise average husband had 3 disease conditions, his expected pain intensity rating would be 0.96 (more clearly “moderate”). In this instance, the predicted pain intensity for the average husband with 3 conditions would be more than one half of a standard deviation higher than that of the average husband with no disease conditions.

The unstandardized fixed effect coefficient for number of disease conditions was of similar direction and size for DP wives. The Model 2 intercept ($\gamma = 0.76$, between “mild” and “moderate,” but closer to “moderate”) represents the predicted pain intensity for a DP wife with no disease conditions who was otherwise “average.” The average DP wife was 63 years-old, white, had just shy of a high school education, had average income and wealth (as compared to other DP couples), lived only with her spouse, and had a BMI toward the upper end of the “overweight” category. As with husbands, a single additional disease condition was associated with only a small increase wives’ pain intensity. Yet, if an otherwise average wife had 3 disease conditions, her expected pain
intensity would be 0.39 higher—a change of roughly two thirds of a standard deviation in wives’ pain intensity ratings (note: compare to the $SD$ of the random effect from Model 0 in Panel B of Table 11, 0.59).

The chi-square test of model deviance indicated that the overall fit of Model 2 was significantly better than that of Model 1, $\chi^2(4) = 42.59, p = .090$. Adding the two health-related variables increased the model PRE by .06 for husbands and .07 for wives. This increase was almost certainly attributable to the number of disease conditions. None of the individual- or couple-level background characteristics was a significant predictor of pain intensity in Model 2. As can be seen in Table 12, the estimates for these variables barely changed from Model 1 to Model 2. Notably, however, the parameter estimate for household wealth was no longer a statistically significant predictor of wives’ pain intensity ($p = .108$). Overall, Model 2 explained relatively modest proportions of variance in the pain intensity ratings of DP husbands (8%) and wives (10%). That both husbands’ and wives’ random effects were statistically significant suggested that sizeable amounts of residual variance remained unexplained.

In Alternate Model 2 (shown in the last column of Table 12), the seven specific disease conditions were substituted in place of the number of disease conditions. Arthritis, lung disease, and heart disease each had a statistically significant effect on the pain intensity ratings of husbands after controlling for background characteristics, BMI, and each of the other disease conditions. Estimates for the effects of heart disease and arthritis were of similar direction and magnitude. Compared to the otherwise average DP husband (refer to pp. 5-54 to 5-55 for description) with no disease conditions, those with heart disease or arthritis were estimated to have pain intensity ratings about one third of a
standard deviation higher. In contrast, the pain intensity ratings for those with lung
disease were estimated to be 0.35 points higher than the ratings of otherwise average
husbands—representing a more moderately-sized effect.

Among DP wives, only arthritis and lung disease remained independently
associated with pain intensity in Alternate Model 2. Based on the estimated parameters
in this model, pain intensity ratings of wives with arthritis were expected to be more than
one quarter of a standard deviation higher than the ratings of otherwise average wives
who had no disease conditions (see pp. 5-55 to 5-56 for description). Lung disease was
estimated to have a slightly larger effect than arthritis in wives, increasing ratings of pain
intensity more than one third of a standard deviation.

Alternate Model 2 was a significantly better-fitting model than Model 1, as
indicated by the model deviance test, $\chi^2(16) = 61.04, p < .001$. The alternate model
ultimately explained 12% of the variance in husbands’ and 11% of the variance in wives’
pain intensity. Replacing the number of disease conditions with the set of individual
disease conditions had no substantive effect on the final parameter estimates for
individual- and couple-level background characteristics or body weight. As before, none
of these variables was independently related to pain intensity in either DP husbands or
wives. The estimated random effects for both husbands and wives were statistically
significant (both $p < .001$), suggesting that considerable variance remained unexplained.

In summary, with one notable exception (number of disease conditions),

**Hypothesis 2** was generally not supported by the results of these analyses. Although
older age, non-White race, and lower SES were all expected to be associated with greater
pain intensity, none of the individual- or couple-level background characteristics emerged
as a significant, independent predictor of the pain intensity of DP husbands and wives. Moreover, except for selected measures of SES, most background characteristics were unrelated to the pain intensity ratings of DP spouses at even the bivariate level. It was also hypothesized that higher pain intensity among DP husbands and wives would be related to both greater body weight and the presence of more disease conditions. Of these two health-related characteristics, only the number of disease conditions evidenced a consistent relationship with pain intensity in both bivariate and multivariate analyses. For both DP husbands and wives, each additional disease condition was associated with a statistically significant (albeit small) increase in pain intensity—a relationship that held even when controlling for body weight and background characteristics.

These analyses addressed Exploratory Question 2 concerning the disease conditions most strongly related to pain intensity among DP husbands and wives. In bivariate analyses, four disease conditions (arthritis, lung disease, heart disease, and high blood pressure) had small positive relationships with pain intensity in husbands. In the multivariate analysis, arthritis and heart disease were each associated with slightly higher pain intensity and lung disease was associated with moderately higher pain intensity in DP husbands, but high blood pressure was no longer a significant predictor. In bivariate analyses of DP wives’ data, five disease conditions (all except cancer and stroke) had small, positive relationships with pain intensity. Only arthritis and lung disease remained significant in a multivariate context; both were associated with modestly higher pain intensity in DP wives. Multi-morbidity was found to be relatively common in both DP husbands and wives. The possibility that such multi-morbidity may account for some of the discrepancies between the bivariate and multivariate results is discussed in Chapter 6.
Relationship Between Pain Intensity and Depressive Symptomatology (Path A)

Bivariate Results

At the bivariate level, pain intensity was significantly related to depressive symptomatology (as measured by the CES-D8) in both DP husbands and wives (see Tables 9 and 10). For both spouses, this relationship was in the expected direction.
Higher pain intensity was associated with greater depressive symptomatology. This relationship was slightly stronger for wives ($r = .30$) than for husbands ($r = .18$).

Number of disease conditions was positively related to higher depressive symptomatology in both DP husbands and DP wives, although these relationships were of modest strength. BMI was not significantly related to depressive symptomatology levels of either DP husbands or wives. Several background characteristics evidenced significant bivariate relationships with the CES-D8 scores of DP spouses. Specifically, higher levels of depressive symptomatology were associated with non-White race, fewer years of education, lower household income, and lower household wealth in both husbands and wives. Having other(s) living in the household was associated with slightly greater depressive symptomatology among husbands; however, for wives, this relationship was not statistically significant. Age was not significantly related to CES-D8 scores in either DP husbands or wives.

**Multivariate Results**

Table 13 presents the results of multilevel analyses used to examine the intra-individual relationship between pain intensity and depressive symptomatology in DP spouses. In these analyses, CES-D8 depressive symptom scores of DP husbands and wives were regressed first on the set of individual- and couple-level background characteristics, then on the respondent’s health-related characteristics, and, finally, on his (or her) pain intensity. As before, a dual-intercept hierarchical linear model was used to examine the pain intensity-depressive symptom relationship in husbands and wives separately, but simultaneously. Results for husbands are presented in Panel A, and wives’ results are presented in Panel B. In order to permit model estimation, reliability
Table 13. Intra-Individual Predictors of Depressive Symptomatology in Dual-Pain Husbands and Wives (N = 423)

<table>
<thead>
<tr>
<th>Panel A: Husbands (N = 423)</th>
<th>Model 0</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fixed Effects [unstand. coefficient (SE)]:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>2.21 (.10)</td>
<td>2.10 (.12)</td>
<td>1.52 (.19)</td>
<td>1.31 (.21)</td>
</tr>
<tr>
<td>Age</td>
<td>&lt;-0.01 (.01)</td>
<td>-0.01 (.01)</td>
<td>-0.01 (.01)</td>
<td></td>
</tr>
<tr>
<td>Non-white race</td>
<td>-0.06 (.32)</td>
<td>-0.03 (.31)</td>
<td>-0.03 (.32)</td>
<td></td>
</tr>
<tr>
<td>Yrs. education</td>
<td>-0.08 (.03)</td>
<td>-0.07 (.03)</td>
<td>-0.08 (.03)</td>
<td></td>
</tr>
<tr>
<td>HH income (log)</td>
<td>-0.38 (.13)</td>
<td>-0.33 (.13)</td>
<td>-0.32 (.13)</td>
<td></td>
</tr>
<tr>
<td>HH wealth (ihs)</td>
<td>-0.04 (.02)</td>
<td>-0.03 (.02)</td>
<td>-0.03 (.02)</td>
<td></td>
</tr>
<tr>
<td>Others in HH</td>
<td>0.37 (.24)</td>
<td>0.40 (.24)</td>
<td>0.43 (.24)</td>
<td></td>
</tr>
<tr>
<td>Health-related characteristics</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. disease conditions (0 – 4+)</td>
<td></td>
<td>0.27 (.08)</td>
<td>0.22 (.08)</td>
<td></td>
</tr>
<tr>
<td>Body weight (BMI)</td>
<td></td>
<td>-0.04 (.02)</td>
<td>-0.04 (.02)</td>
<td></td>
</tr>
<tr>
<td>Pain</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain intensity (0 - 2)</td>
<td></td>
<td></td>
<td></td>
<td>0.35 (.14)</td>
</tr>
<tr>
<td>Random Effect [variance (SD)]:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PRE²</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<p>| PRE² | .13 | .17 | .19 |</p>
<table>
<thead>
<tr>
<th>Panel B: Wives (N = 423)</th>
<th>Model 0</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fixed Effects [unstand. coefficient (SE)]:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>2.61 (.11)</td>
<td>2.61 (.14)</td>
<td>2.18 (.24)</td>
<td>1.51 (.26)</td>
</tr>
<tr>
<td><strong>Background characteristics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>-0.01 (.01)</td>
<td>-0.02 (.01)</td>
<td>-0.02 (.01)</td>
<td></td>
</tr>
<tr>
<td>Non-white race</td>
<td>-0.18 (.37)</td>
<td>-0.17 (.36)</td>
<td>-0.15 (.35)</td>
<td></td>
</tr>
<tr>
<td>Yrs. of education</td>
<td>-0.16 (.04)</td>
<td>-0.16 (.04)</td>
<td>-0.15 (.04)</td>
<td></td>
</tr>
<tr>
<td>HH income (log)</td>
<td>-0.35 (.11)</td>
<td>-0.34 (.11)</td>
<td>-0.33 (.11)</td>
<td></td>
</tr>
<tr>
<td>HH wealth (ihs)</td>
<td>-0.04 (.03)</td>
<td>-0.04 (.03)</td>
<td>-0.03 (.03)</td>
<td></td>
</tr>
<tr>
<td>Others in HH</td>
<td>0.08 (.27)</td>
<td>0.11 (.27)</td>
<td>0.11 (.26)</td>
<td></td>
</tr>
<tr>
<td><strong>Health-related characteristics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. disease conditions (0 – 4+)</td>
<td></td>
<td>0.22 (.10)</td>
<td>0.11 (.09)</td>
<td></td>
</tr>
<tr>
<td>Body weight (BMI)</td>
<td>-0.02 (.02)</td>
<td>-0.02 (.02)</td>
<td>-0.02 (.02)</td>
<td></td>
</tr>
<tr>
<td><strong>Pain</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain intensity (0 - 2)</td>
<td></td>
<td></td>
<td></td>
<td>0.86 (.15)</td>
</tr>
<tr>
<td><strong>Random Effect [variance (SD)]:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PRE^a</td>
<td></td>
<td>.13</td>
<td>.15</td>
<td>.22</td>
</tr>
<tr>
<td>Model Deviance^b</td>
<td>3695.29</td>
<td>3558.14</td>
<td>3538.68</td>
<td>3505.10</td>
</tr>
</tbody>
</table>

Note. HH = Household; log = Log transformation; ihs = Inverse hyperbolic sine transformation. Depressive symptomatology was measured using the CES-D8 (an 8-item version of the Center for Epidemiologic Studies-Depression scale); scores could range from 0 (no symptoms) to 8 (endorsed all 8 symptoms). With the exception of the number of disease conditions, pain intensity, and physical activity limitations, all individual-level continuous variables were mean-centered by gender and all couple-level continuous variables were centered across the 423 couples. To permit estimation, initial error variances were fixed by gender (details in text and Appendix C). Model 0 estimated the dual intercepts (1 for husbands, 1 for wives) only; Model 1 added the individual’s own background characteristics and those of the couple; Model 2 added the individual’s own health-related characteristics; Model 3 added the individual’s own pain intensity. All parameter estimates and the robust SEs for the fixed effects were pooled across the 8 multiply-imputed data sets. Throughout the table, shading indicates a statistically significant effect at p < .05.

^aPRE = Proportion reduction in error measure, calculated as the proportion of variance explained by each model as compared to the total (explainable) variance from Model 0.

^bModel deviance estimates were pooled across the 8 multiply-imputed data sets. A chi-square test of statistical significance compared the deviance of each model to that of the prior nested model. The resulting χ² (df) and p-values were as follows: Model 1 = 137.16 (12), p < .001; Model 2 = 19.45 (4), p < .001; Model 3 = 33.59 (2), p < .001.
estimates for the CES-D8 (calculated by gender) were used to fix the initial error variances (see Appendix C).

As before, the results of these multiple nested models are summarized in the columns of the table. Model 0 served as a baseline, estimating the dual intercepts (one for husbands, one for wives) and the associated variances on the CES-D8 scale. Predictors were added in blocks. Individual- and couple-level background characteristics were entered in Model 1, health-related characteristics were added in Model 2, and pain intensity was added in Model 3. The pain intensity measure had a meaningful zero value (i.e., mild pain), and thus was not centered in these analyses. Except for number of disease conditions, all other continuous predictors were centered as before. Gender-specific PRE (proportion reduction in error) measures, deviance values, and tests of model fit were calculated for each model. Results were pooled across the eight MI datasets using procedures already described.

Model 0 in Table 13 shows that both intercepts and variances were significantly different from zero. The intercepts represent the estimated (and unadjusted) mean CES-D8 scores for DP husbands (2.21) and for wives (2.61). Note that these are equivalent to the mean scores presented in Table 6. The estimated random effects (i.e., the variances) in Table 13 are slightly smaller than the observed variances ($SD^2$) reported in Table 6, as they now represent the amount of “explainable” variance. The statistical significance of these values signals the presence of sizeable amounts of variability in husband and wife CES-D8 depressive symptomatology scores. Model 3 addressed RQ3 and Hypothesis 3 (H3). Since the substantive findings of Models 1 - 3 were equivalent, the narrative below focuses on findings from Model 3. Any changes observed across models are discussed.
As posited by H3, pain intensity was a significant predictor of depressive symptomatology in both DP husbands and wives, even after controlling for the effects of background and health-related characteristics. In husbands, each 1-unit increase in pain intensity was associated with an increase of roughly one third of a point on the CES-D8 ($\gamma = 0.35$). This was a fairly small effect, corresponding to roughly one fifth of a standard deviation (note: compare to the $SD$ of the random effect from Model 0 for husbands in Table 13, 1.79). In wives, each 1-unit increase in pain intensity was associated with nearly a 1-point increase on the CES-D8 scale ($\gamma = 0.86$). This increase corresponded to two fifths of a standard deviation (note: compare to the $SD$ of the random effect from Model 0 for wives in Table 13, 2.09), representing a small-to-moderate effect.

The Model 3 prediction equation was used to construct the graph in Figure 7. This graph illustrates the estimated intra-individual effect of pain intensity on the depressive symptomatology of DP husbands and wives, controlling for background and health-related characteristics. Data points on the graph represent those of otherwise average DP husbands and wives. For these illustrations, the average DP husband remains as described previously (see pp. 5-54 to 5-55), except that he also had over two disease conditions (2.12). The average DP wife remains as described earlier (see pp. 5-55 to 5-56), except that she now reported an average of two disease conditions (1.93).

Both lines in Figure 7 have a positive slope, indicating that increased pain intensity was associated with increased depressive symptomatology in both spouses. However, the graph also suggests that the intra-individual effect of pain intensity on depressive symptomatology may have differed by gender. Based on Exploratory Question 5a, this apparent gender difference was evaluated statistically. A model in
Figure 7

Estimated Intra-Individual Effect of Pain Intensity on Depressive Symptomatology in Dual-Pain Husbands and Wives

Note. Based on results from Model 3 in Table 13, a multi-level model of CES-D8 scores of Dual-Pain husbands and wives (423 couples). The CES-D8 has a maximum value of 8. Effects of pain intensity are illustrated for the “average” husband and wife (refer to text for details).
which husbands’ and wives’ coefficients for pain intensity were constrained to be equal
did not provide as good a fit to the data as did Model 3 without such constraints
(deviance = 3510.93; deviance test vs. Model 3, \( \chi^2(1) = 5.84, p < .016 \)). The intra-
individual effect of pain intensity on depressive symptomatology was, in fact, more
pronounced in DP wives than in DP husbands (i.e., gender moderated this effect).

Adding pain intensity to the multivariate model (Model 3, Table 13) explained
significant amounts of variance in both spouses’ depressive symptomatology levels. The
PRE increased by .02 for husbands and .07 for wives. The size of the unstandardized
coefficient for pain intensity can be compared to those of other variables in Model 3,
provided that such comparisons are gender-specific and take into account the estimated
standard deviation of the CES-D8 from Model 0. When viewed in this context, pain
intensity had the largest estimated independent effect on the depressive symptomatology
of DP husbands and DP wives.

In addition to pain intensity, several other variables were found to be significant
predictors of depressive symptomatology in Model 3. Of the six individual- and couple-
level background variables, only those relating to socioeconomic status had significant
independent effects on depressive symptomatology. For both spouses, higher education
and higher income were associated with fewer depressive symptoms. The estimated
effect of education on depressive symptomatology appeared slightly greater in DP wives
as compared to DP husbands, but both effects were small. Among wives, for instance,
each additional year of education was associated with scores that were, on average, less
than two tenths of a point lower on the CES-D8. Compared to education, household
income was estimated to have a slightly greater, yet still modest, effect on depressive
symptomatology. For both husbands and wives, each additional logged income unit (note: one logged unit was roughly $35K) was associated with average scores that were about one third of a point lower on the CES-D8. Household wealth, did not have an independent effect on levels of depressive symptomatology for either husbands or wives. Lastly, after controlling for the effects of other variables in the model, neither age nor race was significantly related to depressive symptom levels in either spouse.

For DP husbands, both health-related characteristics were significant predictors of depressive symptomatology. Based on Model 3, each additional disease condition was estimated to increase husbands’ CES-D8 scores by roughly one quarter of a point. Although not significantly related at a bivariate level, body weight (measured as BMI) did have a weak negative relationship with husbands’ depressive symptomatology after controlling for other variables. Among husbands, each one-unit increase in BMI was associated with a slightly lower score on the CES-D8.

Looking at wives’ results in Panel B, neither number of disease conditions nor BMI was significantly related to DP wives’ depressive symptomatology levels. Note that the parameter estimate for number of disease conditions was statistically significant in Model 2, with each additional disease condition estimated to increase wives’ average CES-D8 scores by one quarter of a point. The effect of number of disease conditions was appreciably reduced in size and statistical significance from Model 2 to Model 3, suggesting that pain intensity may mediate the relationship between disease conditions and depressive symptomatology. For DP husbands, the estimated effect for number of disease conditions on depressive symptomatology did decrease from Model 2 to Model 3, but this decrease was not very large and number of disease conditions still evidenced a
sizeable effect on husbands’ pain intensity.

The goodness of fit of the model to the data improved significantly with each subsequent model in Table 13. Each set of predictors explained some unique variance in the CES-D8 depressive symptomatology of DP spouses. Individual- and couple-level background characteristics (Model 1) explained approximately 13% of the variation in both husbands’ and wives’ depressive symptomatology levels, $\chi^2(12) = 137.16, p < .001$. Number of disease conditions and body weight (Model 2) explained an additional 2 to 4%, $\chi^2(4) = 19.45, p < .001$. Most importantly, the model deviance test comparing Model 3 to Model 2 was statistically significant, $\chi^2(2) = 33.59, p < .001$, indicating that adding pain intensity substantially improved the fit of the model to the data. The set of background variables, health-related characteristics, and pain intensity ratings together explained respectable amounts of variance in the depressive symptom levels of husbands (19%) and wives (22%). As indicated by the statistical significance of the random effects in Model 3, however, considerable variation in husbands’ and wives’ CES-D8 scores remained unexplained.

**Relationship Between Pain Intensity and Physical Limitations (Path B)**

**Bivariate Results**

Physical limitations (the measure of activity limitation) were hypothesized to at least partially mediate the relationship between pain intensity and depressive symptomatology (Research Question 4, Hypothesis 4c). As noted in the Analysis Plan, testing mediation hypotheses requires evaluating the intra-individual relationship between pain intensity and physical limitations (i.e., Path B). Tables 9 and 10 contain the bivariate correlation between pain intensity and physical limitations in DP husbands and
wives, respectively. In both spouses, higher pain intensity was associated with more physical limitations. The observed correlations were both of moderate size and were markedly similar for husbands and wives ($r = .33$ and $r = .35$, respectively).

Tables 9 and 10 also contain the correlations between physical limitations and the background and health-related characteristics of each spouse. Note that all measures of SES were significantly related to physical limitations in both husbands and wives. With the exception of a more moderately-sized correlation between household income and wives’ physical limitations ($r = -.32$), most SES variables were weakly related to (fewer) physical limitations. In general, relationships involving measures of SES were slightly stronger in wives than in husbands. For instance, higher levels of education were more strongly associated with fewer physical limitations in DP wives ($r = -.26$) than in DP husbands ($r = -.17$).

Greater physical limitations were observed among wives of non-White race, as well as those of increased age. Neither age nor race was significantly related to physical limitations in husbands. The presence of others living in the household was not related to husbands’ or wives’ physical limitations. In terms of health-related characteristics, number of disease conditions was moderately related to more physical limitations among both husbands and wives. Increased body weight was significantly (albeit modestly) associated with more physical limitations among wives, but not among husbands.

**Multivariate Results**

Table 14 summarizes the series of nested, dual-intercept multi-level models that examined the relationship between pain intensity and physical limitations in DP husbands and wives. Physical limitations were regressed first on the set of individual- and
<table>
<thead>
<tr>
<th>Fixed Effects [unstand. coefficient (SE)]:</th>
<th>Model 0</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>4.43 (.14)</td>
<td>4.51 (.17)</td>
<td>2.96 (.27)</td>
<td>2.30 (.28)</td>
</tr>
<tr>
<td><strong>Background characteristics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>0.01 (.01)</td>
<td>0.01 (.01)</td>
<td>&lt;-0.01 (.01)</td>
<td></td>
</tr>
<tr>
<td>Non-white race</td>
<td>-0.70 (.48)</td>
<td>-0.67 (.45)</td>
<td>-0.69 (.43)</td>
<td></td>
</tr>
<tr>
<td>Yrs. education</td>
<td>-0.06 (.05)</td>
<td>-0.06 (.05)</td>
<td>-0.07 (.04)</td>
<td></td>
</tr>
<tr>
<td>HH income (log)</td>
<td>-0.49 (.20)</td>
<td>-0.42 (.19)</td>
<td>-0.38 (.17)</td>
<td></td>
</tr>
<tr>
<td>HH wealth (ihs)</td>
<td>-0.06 (.03)</td>
<td>-0.05 (.03)</td>
<td>-0.04 (.03)</td>
<td></td>
</tr>
<tr>
<td>Others in HH</td>
<td>0.04 (.32)</td>
<td>0.09 (.31)</td>
<td>0.19 (.29)</td>
<td></td>
</tr>
<tr>
<td><strong>Health-related characteristics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. disease conditions (0 – 4*)</td>
<td></td>
<td></td>
<td>0.72 (.11)</td>
<td>0.59 (.11)</td>
</tr>
<tr>
<td>Body weight (BMI)</td>
<td>&lt;-.01 (.02)</td>
<td>.01 (.02)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Pain</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain intensity (0 - 2)</td>
<td></td>
<td></td>
<td></td>
<td>1.08 (.19)</td>
</tr>
</tbody>
</table>

**Random Effect [variance (SD)]:**

| PRE³ |         |         |         |         |
|      | 6.93 (2.63) | 6.36 (2.52) | 5.53 (2.35) | 5.00 (2.24) |

PRE³
Note. HH = Household; log = Log transformation; ihs = Inverse hyperbolic sine transformation. Physical limitations could range from 0 (none) to 11 (limited in all basic physical activities). Effects for husbands and wives were estimated separately, but simultaneously, via a dual-intercept hierarchical linear model. With the exception of the number of disease conditions and pain intensity, all individual-level continuous variables were mean-centered by gender and all couple-level continuous variables were centered across the 423 couples. To permit estimation, initial error variances were fixed by gender (details in text and Appendix C). Model 0 estimated the dual intercepts (1 for husbands, 1 for wives) only; Model 1 added the individual’s own background characteristics and those of the couple; Model 2 added the individual’s own health-related characteristics; Model 3 added the individual’s own pain intensity. All parameter

<table>
<thead>
<tr>
<th></th>
<th>Model 0</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fixed Effects [unstand. coefficient (SE)]:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>5.48 (.15)</td>
<td>5.51 (.18)</td>
<td>4.16 (.30)</td>
<td>3.34 (.31)</td>
</tr>
<tr>
<td>Background characteristics</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>0.03 (.01)</td>
<td>0.02 (.01)</td>
<td>0.02 (.01)</td>
<td></td>
</tr>
<tr>
<td>Non-white race</td>
<td>0.25 (.49)</td>
<td>0.10 (.46)</td>
<td>0.12 (.43)</td>
<td></td>
</tr>
<tr>
<td>Yrs. of education</td>
<td>-0.14 (.05)</td>
<td>-0.10 (.05)</td>
<td>-0.09 (.05)</td>
<td></td>
</tr>
<tr>
<td>HH income (log)</td>
<td>-0.74 (.17)</td>
<td>-0.70 (.14)</td>
<td>-0.68 (.13)</td>
<td></td>
</tr>
<tr>
<td>HH wealth (ihs)</td>
<td>-0.06 (.04)</td>
<td>-0.03 (.04)</td>
<td>-0.02 (.04)</td>
<td></td>
</tr>
<tr>
<td>Others in HH</td>
<td>-0.21 (.33)</td>
<td>-0.18 (.30)</td>
<td>-0.17 (.29)</td>
<td></td>
</tr>
<tr>
<td>Health-related characteristics</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. disease conditions (0 – 4+)</td>
<td></td>
<td></td>
<td>0.70 (.12)</td>
<td>0.59 (.12)</td>
</tr>
<tr>
<td>Body weight (BMI)</td>
<td>0.06 (.02)</td>
<td>0.06 (.02)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain intensity (0 - 2)</td>
<td></td>
<td></td>
<td></td>
<td>1.03 (.21)</td>
</tr>
<tr>
<td>Random Effect [variance (SD)]:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PRE</td>
<td>7.68 (2.77)</td>
<td>6.37 (2.52)</td>
<td>5.36 (2.32)</td>
<td>4.88 (2.21)</td>
</tr>
<tr>
<td>Model Deviance b</td>
<td>3764.95</td>
<td>3689.02</td>
<td>3597.11</td>
<td>3538.94</td>
</tr>
</tbody>
</table>
estimates and the robust SEs for the fixed effects were pooled across the 8 multiply-imputed data sets. Throughout the table, shading indicates a statistically significant effect at $p < .05$.

\(^{a}\)PRE = Proportion reduction in error measure, calculated as the proportion of variance explained by each model as compared to the total (explainable) variance from Model 0.

\(^{b}\)Model deviance estimates were pooled across the 8 multiply-imputed data sets. A chi-square test of statistical significance compared the deviance of each model to that of the prior nested model. The resulting $\chi^2 (df)$ and $p$-values were as follows: Model 1 = 75.93 (12), $p < .001$; Model 2 = 91.91 (4), $p < .001$; Model 3 = 58.17 (2), $p < .001$. 
couple-level background characteristics, then on respondent’s health-related characteristics, and finally, on his/her own level of pain intensity. As before, model estimation was made possible by fixing the initial error variances using (gender-specific) estimates of the reliability of the physical limitations scale (see Appendix C). Aside from pain intensity and number of disease conditions, all other continuous predictors were mean-centered (by gender where appropriate). Results were pooled across the eight MI datasets. Gender-specific PRE measures, deviance values, and tests of model fit were calculated for each model. Model results for DP husbands are shown in Panel A, those for wives in Panel B.

In Model 0, the baseline model, the intercepts and variances were all significantly different from zero \((p < .001)\). The intercepts represent the estimated (and unadjusted) average physical limitations scores for DP husbands (4.43) and for wives (5.48). The statistical significance of the random effects indicates the presence of sizeable amounts of variation in the physical limitations scores of husbands and wives. Given that Model 3 specifically addressed **RQ4a** and **H4a** (i.e., the relationship between pain intensity and physical limitations), the narrative below is focused on Model 3. Any changes observed between models are discussed within the context of the main findings from Model 3.

As predicted by **Hypothesis 4a**, pain intensity was a significant predictor of physical limitations in both DP spouses, even after controlling for the effects of background and health-related covariates. Among husbands, each one-unit increase in pain intensity was associated with an increase of about one point on the physical limitation scale \((\gamma = 1.08, p < .001)\). The estimated intra-individual effect of pain intensity in DP wives was similar. Both effects were of small to moderate size,
corresponding to one third of a standard deviation in husbands and two fifths of a
standard deviation in wives. Notably, pain intensity had the largest independent effect on
the physical limitations of both husbands and wives. Adding pain intensity to the model
of physical limitations increased the PRE by .08 for husbands and .06 for wives.

Several other variables were significantly associated with physical limitations in
DP husbands and wives. Household income was the only background characteristic that
remained independently related to physical limitations of both spouses in Model 3. In
both husbands and wives, higher household income was associated with slightly fewer
physical limitations. Although both estimated effects were fairly small, the effect of
household income was more marked in wives ($\gamma = -0.68$) than in husbands ($\gamma = -0.38$),
corresponding to one quarter and one sixth of a standard deviation for wives and
husbands, respectively. Among wives, increased age and less education were
significantly associated with more physical limitations, but only in early models. These
effects were reduced to nonsignificance when additional predictors were included in
subsequent models.

Number of disease conditions was consistently related to physical limitations in
both spouses, but the estimated size of the effect was reduced once pain intensity was
added in Model 3. Although small, the estimated effect was notable; an increase of two
disease conditions was associated with one additional physical limitation in both
husbands and wives. Among DP wives only, BMI was a significant predictor of more
physical limitations, although this effect was also fairly small in size.

The goodness of fit of the model to the data improved significantly with each
subsequent model in Table 14. In Model 1, individual- and couple-level background
characteristics explained 8% of variance in the physical limitation scores of DP husbands and 17% of the variance in wives’ scores, \( \chi^2(12) = 75.83, p < .001 \). Health-related characteristics explained an additional 12-13% of the variance in husbands’ and wives’ physical limitations, resulting in improved fit for Model 2, \( \chi^2(4) = 91.91, p < .001 \). Adding pain intensity (in Model 3) resulted in a significant improvement in the overall fit of the model to the data, \( \chi^2(2) = 58.17, p < .001 \). The entire set of variables included in Model 3 explained roughly 28% of the variance in the physical limitations of DP husbands, and a more sizeable amount of variance (37%) in the physical limitations of DP wives. Nonetheless, the statistical significance of the random effects in Model 3 indicated that a substantial amount of residual variation in the physical limitations of DP husbands and wives remained unexplained.

**Relationship Between Physical Limitations and Depressive Symptomatology (Path C) and Test of Mediation**

**Bivariate Results**

As can be seen in row 1 of Tables 9 and 10, physical limitations were significantly related to CES-D8 scores in husbands and in wives. Among husbands, greater physical limitations were moderately associated with higher levels of depressive symptomatology. The relationship was essentially identical for wives.

**Multivariate Results**

A final *intra*-individual model (Model 4) examined the relationship between each spouse’s physical limitations and his/her depressive symptomatology in a multivariate context. Table 15 presents the results of Model 4 for husbands (Panel A) and wives (Panel B). Since Model 4 was an extension of Model 3 (from Table 14), the results of
Table 15. Test of Physical Limitations as Mediators of the Intra-individual Relationship between Pain Intensity and Depressive Symptomatology in Husbands and Wives in Dual-Pain Couples (N = 423)

<table>
<thead>
<tr>
<th></th>
<th>PANEL A: HUSBANDS</th>
<th></th>
<th>PANEL B: WIVES</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Model 3</td>
<td>Model 4</td>
<td></td>
<td>Model 3</td>
</tr>
<tr>
<td><strong>Fixed Effects [unstand. coefficient (SE)]:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>1.31 (.21)</td>
<td>0.84 (.20)</td>
<td>1.51 (.26)</td>
<td>0.87 (.28)</td>
</tr>
<tr>
<td><strong>Background characteristics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>-0.01 (.01)</td>
<td>-0.01 (.01)</td>
<td>-0.02 (.01)</td>
<td>-0.03 (.01)</td>
</tr>
<tr>
<td>Non-white race</td>
<td>-0.03 (.32)</td>
<td>0.11 (.30)</td>
<td>-0.15 (.35)</td>
<td>-0.17 (.34)</td>
</tr>
<tr>
<td>Yrs. education</td>
<td>-0.08 (.03)</td>
<td>-0.06 (.03)</td>
<td>-0.15 (.04)</td>
<td>-0.13 (.04)</td>
</tr>
<tr>
<td>HH income (log)</td>
<td>-0.32 (.13)</td>
<td>-0.24 (.11)</td>
<td>-0.33 (.11)</td>
<td>-0.20 (.11)</td>
</tr>
<tr>
<td>HH wealth (ihs)</td>
<td>-0.03 (.02)</td>
<td>-0.02 (.02)</td>
<td>-0.03 (.03)</td>
<td>-0.02 (.03)</td>
</tr>
<tr>
<td>Others in HH</td>
<td>0.43 (.24)</td>
<td>0.39 (.23)</td>
<td>0.11 (.26)</td>
<td>0.14 (.25)</td>
</tr>
<tr>
<td><strong>Health-related characteristics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. disease conditions (0 – 4⁺)</td>
<td>0.22 (.08)</td>
<td>0.10 (.07)</td>
<td>0.11 (.09)</td>
<td>&lt;-0.01 (.10)</td>
</tr>
<tr>
<td>Body weight (BMI)</td>
<td>-0.04 (.02)</td>
<td>-0.04 (.02)</td>
<td>-0.02 (.02)</td>
<td>-0.03 (.02)</td>
</tr>
<tr>
<td><strong>Pain</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain intensity (0 - 2)</td>
<td>0.35 (.14)</td>
<td>0.13 (.14)</td>
<td>0.86 (.15)</td>
<td>0.67 (.16)</td>
</tr>
<tr>
<td><strong>Activity Limitation</strong></td>
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<td></td>
</tr>
<tr>
<td>Physical limitations (0 – 11)</td>
<td>0.21 (.04)</td>
<td></td>
<td>0.19 (.04)</td>
<td></td>
</tr>
<tr>
<td><strong>Random Effect [variance (SD)]:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PRE</td>
<td>.19</td>
<td>.28</td>
<td>.22</td>
<td>.27</td>
</tr>
<tr>
<td>Model Deviance</td>
<td></td>
<td></td>
<td>3505.10</td>
<td>3447.61</td>
</tr>
</tbody>
</table>

Note: For PREF and Model Deviance, the values in Model 4 are lower than those in Model 3, indicating better model fit.
Note. HH = Household; log = Log transformation; ihs = Inverse hyperbolic sine transformation. Depressive symptomatology was measured using the CES-D8 (an 8-item version of the Center for Epidemiologic Studies-Depression scale); scores could range from 0 (no symptoms) to 8 (endorsed all 8 symptoms). Effects for husbands and wives were estimated separately, but simultaneously, via a dual-intercept hierarchical linear model. To permit estimation, initial error variances were fixed by gender (details in text and Appendix C). With the exception of the number of disease conditions, pain intensity, and physical limitations, all individual-level continuous variables were mean-centered by gender and all couple-level continuous variables were centered across the 423 couples. Estimates and robust SEs for the fixed effects were pooled across the 8 multiply-imputed data sets. Shading (gray) indicates statistically significant effects at \( p < .05 \). Model 3 (from Table 13) is presented here for comparison purposes.

\(^a\)PRE = Proportion reduction in error measure, calculated as the proportion of variance explained by each model as compared to the total (explainable) variance from Model 0 (from Table 12).

\(^b\)Deviance values are presented for each dual-intercept model (i.e., Model 3 and Model 4, encompassing both husbands and wives). These deviance estimates were pooled across the 8 multiply-imputed data sets. A chi-square test of statistical significance compared the deviance of Model 4 to that of Model 3, \( \chi^2 = 57.49 \) (2), \( p < .001 \).
Model 3 are shown again here for comparison. In Model 4, each respondent’s CES-D8 depressive symptomatology score was regressed on the set of individual- and couple-level background characteristics, health-related characteristics, his/her own pain intensity, and his/her own physical limitations. Centering of continuous independent variables by gender, pooling of results across the eight MI datasets, and calculations of PRE measures, deviance values, and tests of model fit were handled as previously described. In addition to pain intensity, husbands’ and wives’ physical limitations were not centered in these analyses because they also had a meaningful zero value.

As predicted by Hypothesis 4b, more physical limitations were significantly associated with greater depressive symptomatology, even after controlling for relevant background and health-related characteristics and pain intensity. Among DP husbands, each additional physical limitation was associated with an increase of 0.21 on the CES-D8 scale. The estimated intra-individual effect of DP wives’ physical limitations was similar ($\gamma = .19$). The effect of a single physical limitation on depressive symptomatology was relatively small, corresponding to only 12% of a standard deviation for DP husbands and 9% of a standard deviation for wives (note: compare to the $SD$s of the random effects from Model 0 in Table 13. However, the additive effect of multiple physical limitations would be more substantial. A husband with three physical limitations would be predicted to have a CES-D8 score almost two thirds of a point higher, and one with five physical limitations would have a CES-D8 score over one point higher, than a husband with no physical limitations.

Model 4 also provided evidence supporting Hypothesis 4c—that the relationship between each spouse’s pain intensity and his/her own depressive symptomatology would
be at least partially mediated by his/her physical limitations. Figure 8 contains a path
diagram that summarizes the main findings regarding this mediation effect. Panel A
summarizes the findings for DP husbands and Panel B summarizes those for DP wives.
Paths in the diagram are labeled with unstandardized fixed effect estimates, marked with
asterisks to denote levels of statistical significance. Estimates for all paths have been
adjusted for the effects of the background and health-related characteristics that were
included in all analyses.

Together, these paths summarize the main findings from Tables 13, 14, and 15.
In Panel A for example, Path A shows the estimated effect of a husband’s pain intensity
on his own depressive symptomatology, controlling for his background and health-related
characteristics (i.e., Table 13, Panel A, Model 3). Path A’ shows the estimate for this
same effect after controlling for the effect of his physical limitations on depressive
symptomatology (i.e., Table 15, Panel A, Model 4). Path B shows the estimated effect of
the husband’s pain intensity on his own physical limitations (i.e., Table 14, Panel A,
Model 3). The effect of his own physical limitations on his depressive symptomatology,
controlling for his pain intensity and all background and health-related characteristics is
shown in Path C (i.e., Table 15, Panel A, Model 4).

Figure 8 shows that, for husbands (Panel A), the relationship between pain
intensity and depressive symptomatology was completely mediated by their physical
limitations. Physical limitations were significant, independent predictors of husbands’
depressive symptomatology (Path C). Moreover, once physical limitations were added to
the equation predicting husbands’ depressive symptomatology, pain intensity was no
longer statistically significant (Path A’ = 0.13, p = .347). The extent of mediation
Figure 8
Path Diagram of the Intra-Individual Relationship Between
Pain Intensity and Depressive Symptomatology in
Husbands (Panel A) and Wives (Panel B) in Dual-Pain Couples (N = 423)

Panel A: Husbands

Panel B: Wives

Note. Paths are labeled with unstandardized coefficients from dual-intercept multilevel analyses that estimated effects for husbands and wives separately, but simultaneously. All analyses controlled for several individual- and couple-level background characteristics (age, non-white race, years of education, household income, household wealth, others living in household) and two health-related characteristics (number of disease conditions, body mass index). Path A is the initial relationship between pain intensity and depressive symptomatology. Path A' is the same relationship, after controlling for physical limitations (i.e., the hypothesized mediator). The data in Panel A show evidence of mediation for husbands (i.e., Path A’ drops to nonsignificance after adding physical limitations). The data in Panel B provide evidence of partial mediation for wives (i.e., Path A’ drops in magnitude, but pain intensity still has a statistically significant direct effect on depressive symptomatology).

The coefficients for Path A and Path A’ differ significantly between husbands and wives at p < .05.

*p < .05. *** p < .001.
The indirect effect of pain intensity on depressive symptomatology was 0.22 (generally calculated as the product of Paths B and C, here = 1.08 * 0.21; but theoretically equivalent to the difference between Paths A and A'), with an estimated standard error of .059 (based on Aroian’s formula, \( SE_{\text{indirect}} = \sqrt{(\text{Path } C^2 \cdot SE_{\text{Path } B}^2) + (\text{Path } B^2 \cdot SE_{\text{Path } C}^2) + (SE_{\text{Path } B}^2 \cdot SE_{\text{Path } C}^2)} \)). The indirect effect was found to be statistically significant (\( z = 3.82, p < .001 \)), and a 95% confidence interval for the effect ranged from 0.10 to 0.34. Note that the initial (total) effect of pain intensity on depressive symptomatology (given here by Path A, 0.35) can be decomposed into the (remaining) direct effect (given by Path A', 0.13) and the indirect effect (calculated as Path B * Path C, 0.22).

The findings for DP husbands are consistent with what Baron and Kenny (1986) labeled “perfect” mediation, although others (e.g., James & Brett, 1984; Preacher & Hayes, 2004) prefer “complete” mediation. Kenny (2013) proposed that the ratio of the indirect effect to the total effect be used to provide a sense of the magnitude of a mediated effect. This quantity indicates the portion of the total effect that is mediated by the intervening variable(s). For DP husbands, this ratio was .63 (0.22/0.35). In other words, 63% of the total effect of husbands’ pain intensity on their depressive symptomatology was mediated by their physical limitations. The majority of the effect of husbands’ pain intensity on their depressive symptomatology was indirect, through its effect on their physical limitations.

For DP wives (Panel B or Figure 8), the intra-individual relationship between pain
intensity and depressive symptomatology was partially mediated by their physical limitations. As previously noted, physical limitations were a significant, independent predictor of wives’ CES-D8 scores (Path C). Once physical limitations were added to the model, the magnitude of the estimated effect of pain intensity was reduced, but was still statistically significant (Path A′ = 0.67, \( p < .001 \)). The indirect effect was 0.20 (Path B * Path C = 1.03 * .19, \( SE = .058 \)) and was statistically significant, \( z = 3.38, p = .001, 95\% CI [0.08 to 0.30] \).

The pattern of results observed for DP wives has been labeled “partial” mediation by Kenny and colleagues (e.g., Kenny, Kashy, & Bolger, 1998). Using Kenny’s (2013) formula, the ratio of the indirect to the total effect was .23 (0.20/0.86). Thus, only 23% of the effect of wives’ pain intensity on their depressive symptomatology was mediated through their physical limitations.

**Exploratory Question 5b** concerned possible gender differences in the indirect effect of pain intensity on depressive symptomatology via physical limitations. The pattern of findings summarized above and in Figure 8 certainly suggests that the extent of mediation varied by gender. Physical limitations completely mediated the relationship between husbands’ pain intensity and their depressive symptomatology, but physical limitations only partially mediated this relationship in wives. To formally test this difference, husbands’ and wives’ coefficients for pain intensity (i.e., Path A′, \( \gamma = 0.13 \) for husbands, \( \gamma = 0.67 \) for wives) were constrained to be equal in a final model (results not shown). The constrained model did not provide as good a fit to the data as did Model 4 (constrained model deviance = 3454.10; deviance test vs. Model 4, \( \chi^2(1) = 6.49, p = .011 \)), indicating that the coefficients could not be assumed to be equal. The remaining
The direct effect of pain intensity on depressive symptomatology was larger in DP wives than in DP husbands. This result can also be interpreted to mean that the indirect or mediated effect of pain intensity was smaller in wives than in husbands, generating some support for Exploratory Question 5b.

The gender difference in the size of the (remaining) direct effect of pain intensity can be seen most clearly in Figure 9. Data from Model 4 were used to graph lines showing the estimated effect of pain intensity on the depressive symptomatology of DP husbands and wives after controlling for the effects of all other variables in the model. The effects of other variables were held constant by setting them to zero (for centered continuous and categorical variables) or to their respective gender-specific mean values (for uncentered continuous variables). Figure 9 shows the (remaining) direct effect of pain intensity on the depressive symptomatology of otherwise average DP husbands and wives. The average DP husband and wife are as described previously (see pp. 5-54 to 5-56), except that now each also had an average number of physical limitations (roughly 4.5 for husbands and 5.5 for wives).

The slope of the line for DP wives in Figure 9 is positive and fairly strong, while the slope of the line for DP husbands is relatively flat. This illustrates that the direct effect of pain intensity was larger in wives than in husbands. When contrasted with Figure 7, Figure 9 also provides graphical evidence of the mediating effect of physical limitations on the relationship between pain intensity and depressive symptomatology—an effect that is discernible for wives, but is especially pronounced for husbands.

Looking at Model 4 as a whole (Table 15), several additional findings deserve mention. For example, adding physical limitations to the multivariate model predicting
Figure 9
Estimated Remaining Direct Effect of Pain Intensity on Depressive Symptomatology in Dual-Pain Husbands and Wives

Note. Based on results from Model 4 in Table 15, a multi-level model of CES-D8 scores of Dual-Pain (DP) husbands and wives (N = 423 couples), as well as the path diagram in Figure 8. The CES-D8 has a maximum value of 8. Effects of pain intensity are illustrated for the "average" DP husband and wife (i.e., controlling for all other variables in the model, including physical limitations; refer to text for details).
depressive symptomatology increased the PRE over Model 3 by .09 for husbands and .05 for wives. Model 4 also provided significantly improved fit to the data, $\chi^2(2) = 57.49, p < .001$. After controlling for all other predictors in Model 4, only education remained significantly associated with depressive symptomatology levels in both DP husbands and wives. Higher levels of education were associated with lower CES-D8 depressive symptomatology scores. Similar to the pattern observed in Model 3, this effect appeared larger in wives than in husbands. However, both effects were small, with each additional year of education associated with decreases of less than one tenth of a standard deviation on the CES-D8.

Among husbands (but not wives), household income and body weight also remained significantly associated with depressive symptomatology (the change observed in the parameter estimate between Models 3 and 4 will be discussed shortly). The estimated effect of body weight (BMI) remained stable, but small: Each additional BMI unit was associated with a small decrease in the CES-D8 score of DP husbands.

The apparently beneficial association of increased BMI and lower depressive symptomatology was counter-intuitive. Thus, supplemental analyses explored this relationship in more depth. Analyses using the WHO-recommended BMI categories suggested that there may be a curvilinear relationship between husbands’ body weight and levels of depressive symptomatology. The average CES-D8 scores of husbands by BMI category were: 2.61 ($SD = 2.25, n = 111$) for underweight/normal weight, 1.94 ($SD = 1.97, n = 171$) for overweight, and 2.21 ($SD = 2.00, n = 141$) for obese husbands. An analysis of variance yielded a significant omnibus test of group differences, $F(2, 420) = 3.64, p = .027$. The Levene test for homogeneity of variances was not statistically
significant, $F_{\text{Levene}}(2, 420) = 1.85, p = .158$. Consequently, post hoc pairwise comparisons utilized a Bonferroni correction. Only the mean difference between the CES-D8 scores of underweight/normal weight and overweight husbands (-0.68) was large enough to be statistically significant ($SE = .25, 95\% \text{ CI} [-0.07 \text{ to } -1.28])$.

Table 15 reveals three noticeable differences in the fixed effect estimates for the background and health-related characteristics in Model 4, as compared to those from Model 3. First, estimates of the salutary effect of household income on depressive symptomatology were reduced in Model 4. The estimated effect for husbands was reduced about 25\% in size (from $\gamma = -0.32$ to $\gamma = -0.24$). Although household income still had a significant beneficial effect on husbands’ depressives symptomatology levels, the effect size was fairly small: Each additional (logged) unit of income was associated with CES-D8 scores that were roughly one quarter of a point lower on the scale, a difference of less than one fifth of a standard deviation. For wives, the estimated effect was reduced over 30\% in size between Models 3 and 4, and household income was no longer a significant independent predictor of wives’ CES-D8 scores.

Second, a decrease was also observed in the size of the parameter estimates for number of disease conditions in both spouses. Reductions in the absolute size of these coefficients were more marked (e.g., more than 50\% for husbands, from $\gamma = 0.22$ to $\gamma = 0.10$). For husbands, number of disease conditions was no longer a significant independent predictor of CES-D8 scores. Even though the change in the absolute size of this coefficient was even greater in wives, the consequence was negligible: Number of disease conditions was not statistically significant in either Models 3 or 4.

Third, and lastly, the addition of physical limitations in Model 4 for wives
increased the size of the parameter estimate for age. Although the change in the estimate was very slight (from $\gamma = -0.02$ to $\gamma = -0.03$), it was enough to statistically distinguish the effect from zero. In DP wives, each additional year of age was associated with a slight decrease in their CES-D8 scores.

Together, the set of individual- and couple-level background variables, health-related characteristics, pain intensity ratings, and physical limitations explained about 28% of the variance in DP husbands’ depressive symptomatology levels. Approximately 27% of the variance in wives’ depressive symptomatology was explained by this same set of variables. Note that sizeable amounts of residual variation in depressive symptomatology still existed for both husbands and wives.

In summary, the results of these analyses supported Hypothesis 4c for both DP husbands and DP wives—one’s own physical limitations at least partially mediated the intra-individual relationship between pain intensity and depressive symptomatology. The results also showed that the extent of mediation varied significantly by gender. Although physical limitations completely mediated the relationship between one’s own pain intensity and depressive symptomatology for husbands, they only partially mediated this relationship for wives. Greater pain intensity in wives was still associated with an (albeit slightly reduced) increase in depressive symptomatology, even after controlling for physical limitations. Thus, the results of Model 4 also provided support for Exploratory Question 5b regarding gender differences in the indirect effect of pain intensity on depressive symptomatology via physical limitations. In other words, gender moderated the mediation effect.

**Model checking and tests of model assumptions.** Little guidance is available
regarding the testing of assumptions specific to dual-intercept hierarchical models with
dyadic data. In addition, HLM 6.08 produces relatively few measures that can be used
directly to check model assumptions and evaluate model specification errors.

Consequently, model checking for this final intra-individual model (Model 4) was guided
by strategies commonly recommended within the context of linear modeling (e.g.,
analysis of residuals, assessment of multicollinearity). These strategies were adapted to
the hierarchical structure of the data and were supplemented as necessary. Several of
these procedures do not currently support the pooling of results across MI datasets, so
one imputed dataset was selected at random for use in these analyses (imputation 8).

HLM generates residuals at both Level 1 (L1) and Level 2 (L2). The residuals at
Level 2 are those most closely associated with the set of predictors used in dual-intercept
models (i.e., they are the empirical-Bayes residuals associated with the husband and wife
intercepts). Following this, most diagnostic procedures were directed toward the L2
residuals. The L2 residuals from Model 4 were examined by gender. The L2 residuals
for husbands were relatively normally distributed, with $M = 0.00$ ($SD = 1.26$) and $Mdn =
-0.22$. Values ranged from -2.33 to 4.06, and skewness (0.81) and kurtosis (0.38) values
were within normal limits (see Curran et al., 1996). The distribution of L2 residuals for
wives was also relatively normal, and slightly more symmetrical, $M = 0.00$ ($SD = 1.54$),
$Mdn = -0.32$, and Range = -3.46 to 4.14 ($skewness = 0.59$ and $kurtosis = -0.46$). When
the L2 residuals were graphed against the L2 fitted values (i.e., those predicted by the L2
predictor variables), no obvious curvature or patterning of the variance across the range
of predicted scores was detected. Some mild clustering was observed when L2 residuals
were graphed against two predictor variables—BMI for husbands and age for wives,
suggesting that the model might be further refined by exploring alternate functional forms for these predictors.

As the handling of univariate outliers was described in the prior chapter, the discussion here focuses on potential outliers within a multivariate context. In order to identify potential multivariate outliers and influential cases, the L2 residuals were standardized by gender. Roughly 5% of husbands \((n = 22)\) had L2 standardized residuals greater than \(\pm 2.00\). Within this group of husbands, values ranged from 2.52 to 4.06. Less than 4% of wives \((n = 16)\) had large standardized residuals (i.e., greater than \(\pm 2.00\)). The residual of one wife was negative (-2.25), while those of remaining wives ranged between 2.01 to 2.70. In general, the HLM model with these predictors tended to underestimate CES-D8 scores in DP spouses.

The Model 4 L2 standardized residuals were also examined at the couple level. High-low charts were constructed for the standardized residuals of 50 husband-wife pairs selected randomly from the sample. No discernible pattern (e.g., husbands’ residuals always higher than wives’) was observed for either set of residuals. In the total sample, either the husband or the wife had a large L2 standardized residual \((> \pm 2.00)\) in 34 couples (8%). Large L2 standardized residuals were observed for both the husband and the wife in only two couples; for these two couples, the L2 predictors did not do a good job of predicting the depressive symptomatology of either spouse. Although these two couples differed from each other on several background variables (e.g., race, income, educational levels), they did share some characteristics that distinguished them from other DP couples. The husbands and wives in these couples reported a large number of physical limitations (8 to 10, out of 11 total) and more disease conditions than average (3
to 4 or more, out of 4 or more). Yet, despite this multi-morbidity and high levels of physical impairment, these spouses reported relatively low pain intensity (three reported mild pain intensity, while one wife reported moderate pain intensity). Both couples were also uniquely distinguishable from their peers. The spouses in one couple were much older than average (husband was 80, wife was 79), had BMI levels considerably lower than average (albeit in the normal weight range), and reported total net assets well below the median household wealth of DP couples. In the other couple, both spouses were slightly younger than average (husband was 65, wife was 61); in addition, the wife had very high BMI. The L2 standardized residuals for all 4 spouses were positive (ranging from 2.01 to 2.91), indicating that Model 4 considerably underestimated their depressive symptomatology.

Three additional diagnostic procedures were employed to detect potential model specification errors and/or violations of model-based assumptions. First, model-based standard errors associated with the fixed effect coefficients were compared to the robust standard errors. For Model 4, these differences were small (e.g., ≤ .001) and seldom substantive. According to Raudenbush & Bryk (2002), the “similarity [of these SEs] does imply that inferences about the precision of the regression coefficient estimates are not sensitive to departure of the data from those assumptions” (p. 303). Thus, even though the distributions of several model variables were mildly to moderately-skewed (e.g., wealth, CES-D8), this non-normality likely had little impact on model estimation.

Second, the Level 1 fitted values were compared to both the observed CES-D8 scores and the theoretical range of possible scores (both 0 - 8). The distribution of the L1 fitted values for husbands closely resembled their observed scores, and the same was true
for wives. L1 fitted values also covered the majority of the possible range of CES-D8 scores: Fitted values for husbands ranged from 0.07 to 7.30; values for wives ranged from -0.04 to 7.45. These observations suggest that model-based predicted values provided a reasonable fit to both the observed and possible scores on this measure.

Third, and finally, the potential for multicollinearity among predictor variables was assessed. Although lack of multicollinearity among predictor variables is not an assumption required for linear modeling, it is of concern because it can make models (and estimated coefficients) less robust—i.e., more sensitive to small changes in the data and to violations of assumptions of linear modeling (Allison, 1999). HLM 6.08 software does not provide any mechanism to assess possible multicollinearity among predictor variables. Consequently, this assessment was conducted using the collinearity diagnostics available within the OLS Regression procedure in SPSS. Diagnostics were calculated separately by gender for the set of predictor variables included in Model 4. Variance inflation factors (VIFs) were calculated for each predictor. Eigenvalues (and associated condition number), condition indexes (CIs), and variance decomposition proportions (VDPs) were calculated for the set of Model 4 predictors.

VIF measures the extent to which a predictor is linearly dependent on other predictors in the model (Allison, 1999, Chapter 7). It is this linear dependency that creates instability in parameter estimates. The Model 4 predictor variables for DP husbands had VIF values that ranged from a low of 1.15 for pain intensity to a high of 1.35 for household income (logged and centered). For wives, Model 4 predictor VIF values ranged from 1.16 for pain intensity to 1.42 for physical limitations. No VIF value exceeded a conventional cutoff of $VIF \geq 10$, which would signal the existence of serious
multicollinearity (Cohen et al., 2003). It is also noteworthy that no VIF exceeded a more conservative cutoff value of $VIF \geq 2.5$ proposed by Allison (1999). VIF can be used to estimate the extent to which the standard error of an affected parameter is “inflated” (estimated by taking the square-root of the VIF and examining the digits to the right of the decimal point). The highest VIF value reported above (1.42, for wives’ physical limitations) yielded a SQRT quantity of 1.19, indicating that the corresponding standard error was inflated by less than 20%. This suggests that no substantive multicollinearity-related inflation of variance occurred in the parameter estimates obtained in Model 4.

Callaghan and Chen (2008) suggested that social scientists make use of additional “best practice” methods for diagnosing multicollinearity borrowed from econometricians and biostatisticians. In general, these methods involve analyzing the interrelationships among a set of predictor variables vis-à-vis the characteristics of a matrix that partitions the variances of the parameter estimates of these predictors (i.e., variance decomposition proportions [VDPs]). One summary measure, the condition number, is the ratio of the largest to the smallest eigenvalue derived from the matrix. The condition number for Model 4 was 35.84 for DP husbands and 44.63 for DP wives. Both numbers are well below a suggested threshold of 100 for moderate collinearity (Callaghan & Chen, 2008). Condition indexes (CIs) are more detailed measures calculated for each eigenvalue derived from the matrix. CIs over 30 signal the presence of severe collinearity, while those over 10 indicate moderate collinearity (Callaghan & Chen, 2008). If collinearity is detected, VDP values can be used to identify the variables that are involved in (and the estimates that are negatively affected by) any near linear dependencies. The Model 4 CIs for DP husbands ranged from 1.00 to 5.98, and those for wives ranged from 1.00 to 6.67.
Together, these measures suggested that multicollinearity was not a problem in Model 4. CES-D8 depressive symptomatology scores were somewhat skewed in both DP husbands and wives. Consequently, a sensitivity analysis was conducted to examine the potential impact of using log-transformed CES-D8 scores on the results of Model 4. Analysis of the log-transformed CES-D8 scores yielded the same substantive results as those reported here for scores in the original metric. The relationship between pain intensity and depressive symptomatology was fully mediated by physical limitations in DP husbands, and only partially mediated in DP wives.

**Inter-Individual (Cross-Spouse) Relationships**

This section presents the results of analyses that examined the relationships among key study variables at the inter-individual (or cross-spouse) level in DP husbands and DP wives. Effects at the inter-individual level are considered “cross-spouse” effects because a characteristic or construct in one spouse is thought to influence a characteristic or construct in the other spouse. Prior to reviewing results pertaining to specific research questions and hypotheses, consideration is first given to the bivariate correlations that assessed the relationships between husbands’ and wives’ characteristics and their responses on various measures. The subsequent section reviews the results of analyses that examined the inter-individual relationship between one spouse’s pain intensity and his/her partner’s depressive symptomatology, including whether this relationship was mediated by the spouse’s physical limitations.

**Overview of Cross-Spouse Bivariate Relationships**

Table 16 presents the bivariate (Pearson) correlation coefficients calculated between husbands’ and wives’ individual-level background characteristics, health-related
Table 16. (Cross-spouse) Correlations Between Individual-level Model Variables of Husbands and Wives in Dual-Pain Couples (N = 423)

<table>
<thead>
<tr>
<th>HUSBANDS' VARIABLES</th>
<th>CES-D8 (0 - 8)</th>
<th>Pain intensity</th>
<th>Physical limitations</th>
<th>No. disease conditions</th>
<th>Body weight (BMI)</th>
<th>Age</th>
<th>Non-white race&lt;sup&gt;a,b&lt;/sup&gt;</th>
<th>Yrs. education</th>
</tr>
</thead>
<tbody>
<tr>
<td>CES-D8 (0 - 8)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-.18</td>
</tr>
<tr>
<td>Pain intensity (0 - 2)</td>
<td>.11</td>
<td>.10</td>
<td>.11</td>
<td>-.03</td>
<td>.02</td>
<td>.02</td>
<td></td>
<td>-.04</td>
</tr>
<tr>
<td>Physical limitations (0 - 11)</td>
<td>.19</td>
<td>.18</td>
<td>.20</td>
<td>.03</td>
<td>.04</td>
<td>&lt;-.01</td>
<td>-.12</td>
<td></td>
</tr>
<tr>
<td>No. disease conditions (0 - 4+)</td>
<td>.05</td>
<td>.05</td>
<td>.18</td>
<td>.02</td>
<td>.18</td>
<td>-.01</td>
<td>-.14</td>
<td></td>
</tr>
<tr>
<td>Body weight (BMI)</td>
<td>.01</td>
<td>.07</td>
<td>-.02</td>
<td>-.01</td>
<td>-.28</td>
<td>.07</td>
<td>-.01</td>
<td></td>
</tr>
<tr>
<td>Age (yrs.)</td>
<td>-.01</td>
<td>.08</td>
<td>.11</td>
<td>.14</td>
<td>-.26</td>
<td>-.10</td>
<td>-.08</td>
<td></td>
</tr>
<tr>
<td>Non-white race&lt;sup&gt;a,b&lt;/sup&gt;</td>
<td>.10</td>
<td>.06</td>
<td>.08</td>
<td>.05</td>
<td>.19</td>
<td>-.13</td>
<td>-.27</td>
<td></td>
</tr>
<tr>
<td>Yrs. education (0 - 17+)</td>
<td>-.18</td>
<td>-.15</td>
<td>-.27</td>
<td>-.14</td>
<td>-.11</td>
<td>-.06</td>
<td>-.22</td>
<td></td>
</tr>
</tbody>
</table>

Note. Correlations were pooled across the 8 multiply-imputed data sets. Wives’ variables are coded in the same manner as husbands’ variables. Correlations in shaded cells are statistically significant at p < .05. CES-D8 = 8-item Center for Epidemiologic Studies-Depression Scale. BMI = Body mass index. Data from cells on the diagonal (i.e., ICCs) were presented in Table 7.

<sup>a</sup>Dichotomous (1 = yes).

<sup>b</sup>Non-white race includes individuals who reported themselves to be African American/Black or of other race.
characteristics, and key study variables. Note that the values on the left-to-right diagonal of this matrix—the bivariate correlations between husbands’ and wives’ values on the same variables—have been omitted. Those intraclass correlations (ICCs) were presented earlier (see Table 7) and reviewed in relation to Research Question 6; recall that those ICCs provided evidence of spousal covariation in pain intensity (Path 1), depressive symptomatology (Path 2), and physical limitations (Path 3), and established a rationale for the use of multilevel modeling in all multivariate analyses. Household-level covariates were also excluded from Table 16 because they did not vary between husbands and wives.

For variables with missing data in the original file, correlation coefficients were pooled across the eight MI datasets. Correspondingly, all correlations were based on $N = 423$ couples. Shading denotes correlation coefficients that were statistically significant at $p \leq .05$. This correlation matrix is reviewed here in general terms only—mainly with an eye toward identifying variables and/or relationships that might create problems in subsequent analyses. Bivariate correlations for specific cross-spouse relationships are discussed in greater detail in conjunction with findings from relevant multivariate analyses.

In the present context, two general observations can be made about the correlations in Table 16. First, none of the correlations in Table 16 approach values that might portend a potential problem with multicollinearity in planned multivariate analyses (e.g., .70 or .80). Second, although a number of cross-spouse correlations are statistically significant, most are of fairly small magnitude. Indeed, there are no correlations of moderate strength or effect size. Due to the large sample size, even relatively weak
correlations (e.g., $r = .10$) are statistically significant at $p < .05$. The most sizeable correlations were observed for one spouse’s age and his/her partner’s body weight (e.g., $r = -.28$ between wife age and husband BMI) and those involving the education level of either spouse (e.g., $r = -.27$ between husbands’ non-White race and wives’ years of education). Many of these relationships are likely attributable to the marked similarity of spouses in terms of age, race, and level of education.

The cross-spouse relationships of most interest here are concentrated in the upper-left corner of the correlation matrix. The largest correlations involved physical limitations (e.g., $r = .19$ between husband physical limitations and wife CES-D8, and $r = .18$ between wife physical limitations and husband CES-D8, both $p < .05$). However, even these correlations indicate relationships of modest strength: For example, husband physical limitations accounted for less than 5% of the variance in wife depressive symptomatology ($r^2 = .036$). Although other bivariate cross-spouse correlations involving pain intensity, physical limitations, and depressive symptomatology were consistent with the direction of hypothesized relationships, they were also fairly small. Together, these observations suggest that it is necessary to control for the intra-individual relationships known to exist among key study variables in order to adequately evaluate the nature and strength of any possible cross-spouse effects.

**Pain in Relation to Psychological Distress at the Inter-Individual Level**

Broadly speaking, **Research Questions 7 through 10** concerned the relationship between the pain and psychological distress (i.e., pain intensity and depressive symptomatology) of DP spouses at the *inter-*individual level. Several sets of analyses
were required to test the hypotheses associated with these questions, so results are organized in four subsections. In the first subsection, findings are reviewed from analyses addressing **Research Question 7**, which posited a direct relationship between each spouse’s pain intensity and the depressive symptomatology of his/her partner. The next subsection presents the results of analyses for **Research Question 8**, which addressed the possible mediating role of the spouse’s physical limitations in the cross-spouse pain intensity-depressive symptomatology relationship. Both RQ7 and RQ8 related to Study Aim 3. The third subsection reviews the results of analyses that explored the potential joint or interactive effect of both spouses’ pain intensity on each spouse’s depressive symptomatology (**Exploratory Question 10**). In the fourth and last subsection, a final multivariate model illustrates the additive effects of several independent variables on the depressive symptomatology of both spouses.

In each subsection, the results of bivariate analyses addressing the proposed relationships are reviewed first, followed by the results of the multivariate analyses. Findings regarding gender differences in any of these relationships (**Research Question 9, addressing Study Aim 4**) are discussed within each subsection. If formal statistical tests of gender differences were warranted, those results are presented within the summary of the relevant multivariate analyses. The relationship between one spouse’s pain intensity and his/her partner’s depressive symptomatology was a primary interest of the present study. Therefore, findings involving other model variables are reviewed in detail only if: (a) the variable represented a new effect or was a new addition to a multivariate model (e.g., relationships involving spouse health characteristics), or (b) the estimated effect of the variable differed substantially from that observed in prior, intra-
individual models. One notable exception involves Model 9; results for all exogenous variables included in this final trimmed model will be reviewed.

Relationship Between Each Spouse’s Pain Intensity and His/Her Partner’s Depressive Symptomatology (Path 4)

In the conceptual model guiding this study (Figure 5, Chapter 3), the relationship between one spouse’s pain intensity and the depressive symptomatology of his/her partner is represented by Path 4. This inter-individual pathway is further distinguished in Figure 5 as two separate paths—Path 4_H and Path 4_W. Subscripts are used to convey the presumed source of the effect (Path 4_H represents the effect of the husband’s pain intensity on the wife’s depressive symptomatology; Path 4_W represents the effect of the wife’s pain intensity on the husband’s depressive symptomatology).

Bivariate Results

The bivariate correlations shown in Table 16 indicate that the pain intensity of each DP spouse was significantly related to his/her partner’s depressive symptomatology (as measured by the CES-D8). Specifically, higher pain intensity in the spouse was associated with more depressive symptoms in the partner. Note, though, that both correlations were fairly weak.

The health-related characteristics of each spouse were essentially unrelated to the depressive symptom levels of the other spouse at a bivariate level. Neither the number of disease conditions nor the body weight of the husband or the wife was significantly related to the depressive symptomatology of his/her partner. All of these cross-spouse correlations fell below \( r = .10 \), indicating that neither spouse’s health characteristics explained more than 1% of the variability observed in the partner’s depressive symptom
levels (see Table 16).

Statistically significant bivariate relationships were observed between some of the background characteristics of each spouse and the depressive symptomatology of his/her partner. Wives’ CES-D8 scores were negatively related to the education level of husbands, and a relationship of the same magnitude and direction was also observed between husbands’ CES-D8 scores and the education level of wives ($r = -0.18, p < .001$). Each partner’s depressive symptomatology had a small, positive relationship with the non-White race of the spouse. Although both correlations were small, the relationship between wives’ depressive symptomatology and husbands’ non-White race was slightly larger ($r = 0.15, p = .002$) than the reverse ($r = 0.10, p = .042$). Spouse age was not significantly related to the depressive symptomatology of partners.

**Multivariate Results**

Table 17 presents the results of analyses that examined the relationship of each spouse’s pain intensity to the partner’s depressive symptomatology within a multivariate context. Models 5-7 were constructed by extending Model 4, the final intra-individual model (from Table 15). Recall that Model 4 regressed the CES-D8 scores of each DP spouse on his/her own: background characteristics (both individual- and couple-level characteristics), health-related characteristics, pain intensity, and physical limitations. Characteristics of the other spouse were added beginning in Model 5.

Being extensions of Model 4, the models presented in Table 17 are also dual-intercept hierarchical linear models that examined the depressive symptomatology of husbands and wives separately, but simultaneously. Results for DP husbands are presented in Panel A, and those for DP wives are displayed in Panel B. Predictors are
Table 17. Test of the Cross-Spouse Relationship of Each Spouse’s Pain Intensity to His/Her Partner’s Depressive Symptomatology and Mediation by the Spouse’s Physical Limitations in Dual-Pain Husbands and Wives

<table>
<thead>
<tr>
<th></th>
<th>Model 5</th>
<th>Model 6</th>
<th>Model 7</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PANEL A: Husbands (N = 423)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Fixed Effects  [unstand. coeff. (SE)]:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>0.96 (.25)</td>
<td>0.84 (.27)</td>
<td>0.86 (.27)</td>
</tr>
<tr>
<td><strong>Respondent characteristics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>-0.01 (.01)</td>
<td>-0.01 (.01)</td>
<td>-0.01 (.01)</td>
</tr>
<tr>
<td>Non-white race</td>
<td>0.09 (.30)</td>
<td>0.09 (.30)</td>
<td>0.09 (.30)</td>
</tr>
<tr>
<td>Yrs. education</td>
<td>-0.06 (.03)</td>
<td>-0.06 (.03)</td>
<td>-0.06 (.03)</td>
</tr>
<tr>
<td>HH income (log)</td>
<td>-0.24 (.11)</td>
<td>-0.24 (.11)</td>
<td>-0.24 (.11)</td>
</tr>
<tr>
<td>HH wealth (ihs)</td>
<td>-0.02 (.02)</td>
<td>-0.02 (.02)</td>
<td>-0.02 (.02)</td>
</tr>
<tr>
<td>Others in HH</td>
<td>0.37 (.23)</td>
<td>0.37 (.23)</td>
<td>0.37 (.23)</td>
</tr>
<tr>
<td>No. disease conditions (0 – 4*)</td>
<td>0.09 (.08)</td>
<td>0.10 (.07)</td>
<td>0.09 (.07)</td>
</tr>
<tr>
<td>Body weight (BMI)</td>
<td>-0.04 (.02)</td>
<td>-0.05 (.02)</td>
<td>-0.05 (.02)</td>
</tr>
<tr>
<td>Pain intensity (0 - 2)</td>
<td>0.14 (.14)</td>
<td>0.16 (.14)</td>
<td>0.16 (.14)</td>
</tr>
<tr>
<td>Physical limitations (0 – 11)</td>
<td>0.21 (.04)</td>
<td>0.21 (.04)</td>
<td>0.21 (.04)</td>
</tr>
<tr>
<td><strong>Spouse characteristics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Health-related characteristics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. disease conditions (0 – 4*)</td>
<td>-0.06 (.07)</td>
<td>-0.08 (.08)</td>
<td>-0.08 (.08)</td>
</tr>
<tr>
<td>Body weight (BMI)</td>
<td>0.01 (.01)</td>
<td>0.01 (.01)</td>
<td>0.01 (.01)</td>
</tr>
<tr>
<td>Pain</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain intensity (0 - 2)</td>
<td></td>
<td>0.14 (.15)</td>
<td>0.15 (.16)</td>
</tr>
<tr>
<td><strong>Activity Limitation</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical limitations (0 – 11)</td>
<td></td>
<td></td>
<td>0.01 (.04)</td>
</tr>
<tr>
<td><strong>Random Effect  [variance (SD)]:</strong></td>
<td>2.30 (1.52)</td>
<td>2.29 (1.51)</td>
<td>2.29 (1.51)</td>
</tr>
<tr>
<td><strong>PRE</strong></td>
<td>.29</td>
<td>.29</td>
<td>.29</td>
</tr>
</tbody>
</table>
Depressive symptomatology was measured using the CES-D8 (an 8-item version of the Center for Epidemiologic Studies-Depression scale); scores could range from 0 (no symptoms) to 8 (endorsed all 8 symptoms). Effects for husbands and wives were estimated separately, but simultaneously, via a dual-intercept hierarchical linear model. To permit estimation, initial error variances were fixed by gender (details in text and Appendix C). With the exception of the number of disease conditions, pain intensity, and physical limitations, all individual-level continuous variables were mean-centered by gender and all couple-level continuous variables were centered across the 423 couples. Estimates and robust SEs for the fixed effects were pooled across the 8 multiply-imputed data sets. Shading (gray) indicates statistically significant effects at $p < .05$.

\textit{Note.} HH = Household; log = Log transformation; ihs = Inverse hyperbolic sine transformation. Depressive symptomatology was measured using the CES-D8 (an 8-item version of the Center for Epidemiologic Studies-Depression scale); scores could range from 0 (no symptoms) to 8 (endorsed all 8 symptoms). Effects for husbands and wives were estimated separately, but simultaneously, via a dual-intercept hierarchical linear model. To permit estimation, initial error variances were fixed by gender (details in text and Appendix C). With the exception of the number of disease conditions, pain intensity, and physical limitations, all individual-level continuous variables were mean-centered by gender and all couple-level continuous variables were centered across the 423 couples. Estimates and robust SEs for the fixed effects were pooled across the 8 multiply-imputed data sets. Shading (gray) indicates statistically significant effects at $p < .05$.

\texttt{PRE} = Proportion reduction in error measure, calculated as the proportion of variance explained by each model as compared to the total (explainable) variance from Model 0 (from Table 13).

<table>
<thead>
<tr>
<th></th>
<th>Model 5</th>
<th>Model 6</th>
<th>Model 7</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PANEL B: Wives (N = 423)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Fixed Effects [unstand. coeff. (SE)]:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>0.95 (.35)</td>
<td>0.85 (.36)</td>
<td>0.82 (.36)</td>
</tr>
<tr>
<td><strong>Respondent characteristics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>-0.02 (.01)</td>
<td>-0.02 (.01)</td>
<td>-0.02 (.01)</td>
</tr>
<tr>
<td>Non-white race</td>
<td>-0.17 (.30)</td>
<td>-0.18 (.34)</td>
<td>-0.15 (.34)</td>
</tr>
<tr>
<td>Yrs. education</td>
<td>-0.13 (.04)</td>
<td>-0.13 (.04)</td>
<td>-0.13 (.04)</td>
</tr>
<tr>
<td>HH income (log)</td>
<td>-0.20 (.12)</td>
<td>-0.20 (.12)</td>
<td>-0.19 (.12)</td>
</tr>
<tr>
<td>HH wealth (ihs)</td>
<td>-0.02 (.03)</td>
<td>-0.02 (.03)</td>
<td>-0.02 (.03)</td>
</tr>
<tr>
<td>Others in HH</td>
<td>0.14 (.25)</td>
<td>0.15 (.25)</td>
<td>0.14 (.25)</td>
</tr>
<tr>
<td>No. disease conditions (0 – 4+)</td>
<td>-0.01 (.10)</td>
<td>-0.02 (.10)</td>
<td>-0.02 (.10)</td>
</tr>
<tr>
<td>Body weight (BMI)</td>
<td>-0.03 (.02)</td>
<td>-0.03 (.02)</td>
<td>-0.02 (.02)</td>
</tr>
<tr>
<td>Pain intensity (0 - 2)</td>
<td>0.67 (.16)</td>
<td>0.69 (.16)</td>
<td>0.69 (.16)</td>
</tr>
<tr>
<td>Physical limitations (0 – 11)</td>
<td>0.19 (.04)</td>
<td>0.19 (.04)</td>
<td>0.18 (.04)</td>
</tr>
<tr>
<td><strong>Spouse characteristics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Health-related characteristics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. disease conditions (0 – 4+)</td>
<td>-0.03 (.09)</td>
<td>-0.05 (.09)</td>
<td>-0.07 (.09)</td>
</tr>
<tr>
<td>Body weight (BMI)</td>
<td>&lt;0.01 (.02)</td>
<td>&lt;0.01 (.02)</td>
<td>&lt;0.01 (.02)</td>
</tr>
<tr>
<td><strong>Pain</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain intensity (0 - 2)</td>
<td>0.17 (.16)</td>
<td>0.13 (.16)</td>
<td></td>
</tr>
<tr>
<td><strong>Activity Limitation</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical limitations (0 – 11)</td>
<td>0.04 (.04)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Random Effect [variance (SD)]:</strong></td>
<td>3.18 (1.78)</td>
<td>3.17 (1.78)</td>
<td>3.16 (1.78)</td>
</tr>
<tr>
<td>PRE\textsuperscript{a}</td>
<td>.27</td>
<td>.28</td>
<td>.28</td>
</tr>
<tr>
<td><strong>Model Deviance\textsuperscript{b}</strong></td>
<td>3446.54</td>
<td>3444.36</td>
<td>3443.37</td>
</tr>
</tbody>
</table>
Deviance values are presented for each dual-intercept model (i.e., Models 5-7, encompassing both husbands and wives). These deviance estimates were pooled across the 8 multiply-imputed data sets. A chi-square test of statistical significance compared the deviance of each model to that of the prior nested model (note: Model 5 was compared to Model 4 from Table 15). The resulting \( \chi^2 \) (df) and \( p \)-values were as follows: Model 5 = 1.07 (4), \( p = .899 \); Model 6 = 2.18 (2), \( p = .366 \); Model 7 = 0.98 (2), \( p = .611 \).
organized under two broad headings in order to draw attention to focal relationships and enhance readability. An individual’s own variables are listed under “Respondent characteristics.” Since the contributions of specific intra-individual predictors were examined previously, these variables are not organized into subcategories. Variables that emanate from an individual’s spouse (i.e., inter-individual predictors) are listed under “Spouse characteristics.” These inter-individual predictors are organized under the subheadings drawn from the conceptual model in Figure 5 (Chapter 3).

Models 5-7 are nested models in which the inter-individual predictors (i.e., the spouse’s variables) were added in blocks. The spouse’s health-related characteristics were added in Model 5 as additional covariates. Model 6 added the spouse’s pain intensity. Model 7—in which the spouse’s physical limitations were added—will be discussed in the next section. Consistent with prior intra-individual models, those measures with meaningful zero values (e.g., pain intensity, number of disease conditions, physical limitations) were not centered in these analyses. All other continuous predictors were centered as before. Gender-specific PRE measures, deviance values, and tests of model fit were calculated for each model. Results were pooled across the eight MI datasets in accordance with the procedures already described.

As can be seen in Model 5, after controlling for the relevant background characteristics, health-related characteristics, pain intensity, and physical limitations of each respondent, the health-related characteristics of the spouse were not significant predictors of the respondent depressive symptomatology. This finding was consistent across both husbands and wives, with nearly identical parameter estimates for the spouse’s number of disease conditions (and also for body weight). Given this lack of
significance, the gender-specific PRE measures for Model 5 increased by less than 1 percent. Not surprisingly, Model 5 did not provide a significant improvement in model fit over Model 4, model deviance $\chi^2(4) = 1.07, p = .899$.

Model 6 specifically addressed Research Question 7, concerning the existence of a direct relationship between one spouse’s pain intensity and the depressive symptomatology of his/her partner (i.e., the respondent). The results of Model 6 are summarized in the middle column of Table 17. Hypothesis 7 (H7) posited that increased pain intensity in each spouse would be related to greater depressive symptomatology in the other partner (after controlling for the effects of relevant background and health-related characteristics of both the partner and the spouse). Contrary to H7, neither spouse’s pain intensity level was a significant predictor of his/her partner’s depressive symptom levels. The point estimate for each effect was positive and sizeable: $\gamma = .14$ for Path 4W (the effect of the wife’s pain on husband depressive symptomatology), and $\gamma = .17$ for Path 4H (the effect of the husband’s pain on wife depressive symptomatology). However, both estimated standard errors were relatively large, indicating that neither effect could be distinguished reliably from zero.

Despite the existence of weak bivariate relationships, no evidence was found in initial multivariate analyses for any cross-spouse effect of pain intensity within DP couples. H7 was thus not supported by these analyses. The gender-specific PRE measures and random effect estimates for Model 6 remained virtually unchanged from Model 5. Accordingly, the deviance value of Model 6 was not significantly different from that of Model 5, $\chi^2(2) = 2.18, p = .366$, indicating no improvement in model fit. Note, however, that subsequent analyses revealed the existence of a more complicated
relationship involving the cross-spouse effect of pain intensity than that originally proposed by H7. Those findings will be presented shortly.

**Research Question 9** dealt with the possibility of gender differences (Study Aim 4) in the relationship between each spouse’s pain intensity and his/her partner’s depressive symptomatology. **Hypothesis H9 (H9)** proposed that wives would be more strongly affected by their husbands’ pain intensity than the reverse. In other words, Path 4h was expected to be stronger than Path 4w. As no evidence of a cross-spouse relationship for pain intensity was found for either husbands or wives, H9 was also not supported by these analyses. As will be discussed shortly, however, H9 was re-examined on the basis of subsequent findings.

**Relationship Between Each Spouse’s Physical Limitations and His/Her Partner’s Depressive Symptomatology (Path 5) and Test of Cross-Spouse Mediation**

Recall that the Baron and Kenny (1986) strategy for establishing mediation requires a statistically significant relationship between the independent variable and the dependent variable. They argue that, in the absence of this relationship, there is no substantive effect to be mediated. Based on the findings from Model 6, in which spouse pain intensity was not a significant predictor of partner depressive symptomatology, strict adherence to the Baron and Kenny strategy would suggest that no further mediation-related analyses are necessary.

Other have argued that researchers should still investigate other relationships within the mediation triad, even if the initial X-Y relationship is not statistically significant (e.g., Collins, Graham, & Flaherty, 1998; MacKinnon et al., 2002; Shrout & Bolger, 2002). Continued examination may be warranted to rule out several relatively
unlikely—but theoretically possible—scenarios. In one scenario, the mediating variable acts to suppress the relationship between the independent and dependent variables. In another, a threshold of influence exists in the relationship between two variables in the mediation sequence (e.g., between the independent and mediating variables); mediation will only occur once this threshold is reached. Continued investigation of the mediation hypothesis in the present study was justified on the basis of the above arguments. This decision was further supported by the small, but statistically significant, correlations observed between each spouse’s pain intensity and the depressive symptomatology of his/her partner.

A note regarding the mediation pathway in the cross-spouse context. Prior to presenting the results for Path 5, it may be helpful to review the mediation pathway proposed in the conceptual model guiding this study. The relationship between a DP spouse’s pain intensity and his/her partner’s depressive symptomatology is thought to be mediated by the spouse’s own activity limitation (as measured by physical limitations). In other words, the husband’s pain intensity is thought to influence the wife’s depressive symptomatology (at least in part) through its influence on the husband’s physical limitations (and the converse for wives). This pathway, represented as Path B in Figure 5, is thus *intra*-individual in nature. Empirical support for this pathway was presented earlier in this chapter: Specifically, the respondent’s own pain intensity was a significant predictor of his/her own physical limitations, even after controlling for relevant background and health-related variables (see Model 3 in Table 14). Moreover, this relationship was observed in both DP husbands and DP wives. Thus, following the Baron and Kenny (1986) mediation testing procedure, findings from the present study
have already established the existence of a statistically significant relationship between the independent variable and the proposed mediating variable.

The final pathway to be tested in the mediation triad (via Baron & Kenny, 1986) is labeled Path 5 in the proposed conceptual model (Figure 5). Path 5 represents the inter-individual relationship between one spouse’s physical limitations and the depressive symptomatology of his/her partner. As before, subscripts further distinguish the presumed source of this hypothesized cross-spouse effect. Path $5_h$ represents the effect of the husband’s physical limitations on the wife’s depressive symptomatology; Path $5_w$ corresponds to the effect of the wife’s physical limitations on the depressive symptom level of the husband. Tests of these paths partially addressed Study Aim 3.

*Bivariate Results*

Table 16 (the cross-spouse correlation matrix) shows that statistically significant bivariate relationships were observed between the physical limitations of each spouse and his/her partner’s depressive symptomatology (as measured by the CES-D8). Physical limitations in wives were positively associated with slightly higher depressive symptomatology in husbands. A relationship of nearly identical direction and strength was also observed between husbands’ physical limitations and wives’ depressive symptomatology. Although these bivariate relationships were fairly weak in magnitude, they were in the expected direction.

*Multivariate Results*

The final column of Table 17 displays the results of Model 7, the multivariate model designed to evaluate whether the relationship between each spouse’s pain intensity and his/her partner’s depressive symptomatology was mediated by the spouse’s physical
limitations (Research Question 8). To this end, Model 7 extended Model 6 by adding the spouse’s physical limitations as predictors of the depressive symptomatology of the partner. Findings with respect to both underlying hypotheses are reviewed below.

**Hypothesis 8a (H8a)** proposed that greater physical limitations in each spouse would be related to greater depressive symptomatology in his/her partner, after controlling for relevant background characteristics and the health-related characteristics and pain intensity of both spouses. However, as can be seen in Table 17, neither spouse’s physical limitations emerged as significant predictors of his/her partner’s depressive symptomatology in a multivariate context. The point estimates for each effect were very small and not statistically significant. So, although weak bivariate relationships did exist between each spouse’s physical limitations and the partner’s depressive symptomatology, no evidence was found for a cross-spouse effect of physical limitations in multivariate analyses. Thus, **H8a** was not supported by these data.

**Hypothesis 8b (H8b)** proposed that that the relationship between each spouse’s pain intensity and his/her partner’s depressive symptomatology would be at least partially mediated by the spouse’s physical limitations. Together, the results of Models 6 and 7 offer no empirical evidence to support the existence of this proposed mediation pathway. No cross-spouse effects for pain intensity were found. In addition, there was no evidence of a relationship between one spouse’s physical limitations and the depressive symptomatology of his/her partner. In short, **H8b** was not supported by these data.

Model 7 did not offer a significant improvement in fit over Model 6, as evidenced by a nonsignificant test of model deviance, $\chi^2(2) = 0.98, p = .611$. Moreover, the gender-specific PRE measures and random effect estimates remained virtually unchanged from
Model 6 to Model 7. Terms representing potential cross-spouse effects of pain and physical limitations did little to reduce the residual variation in the depressive symptomatology of DP husbands or DP wives.

Recall that **Exploratory Question 9 (EQ9)** concerned the possibility of gender differences in the indirect (i.e., mediated) cross-spouse relationship between each spouse’s pain intensity and his/her partner’s depressive symptomatology. These analyses provided no evidence to suggest that the physical limitations of the spouse (i.e., the proposed mediator) were related to the depressive symptomatology of the partner. There was also no evidence that these (non)findings varied at all by gender.

**The Joint Effect of Spouses’ Pain Intensity**

**Exploratory Question 10 (EQ10)** concerned the possibility of a joint or interactive effect of husband and wife pain intensity on the depressive symptomatology of either spouse. Table 18 summarizes the results of Model 8, the dual-intercept hierarchical linear model that extended Model 7 by adding a respondent by spouse pain intensity interaction term as a predictor of the respondent’s depressive symptomatology. In subsequent discussions, this effect may be referred to as the husband by wife pain intensity interaction or the Husband X Wife effect of pain intensity in order to enhance clarity. Model 8 followed all of the estimation, pooling, and deviance testing procedures used in prior models with one exception: In contrast to prior models, pain intensity was mean centered by gender in these analyses. This was done in order to reduce problems with collinearity that arise between uncentered first-order terms and a higher level product term (Aiken & West, 1991). As before, results for DP husbands are presented in Panel A of the table, and the results for DP wives are presented in Panel B.
Table 18. Test of Respondent by Spouse Pain Intensity Interaction Effect on Depressive Symptomatology in Dual-Pain Husbands and Wives

<table>
<thead>
<tr>
<th>PANEL A: Husbands (N = 423)</th>
<th>Model 8</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fixed Effects [unstand. coeff. (SE)]:</td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>1.17 (.28)</td>
</tr>
<tr>
<td>Respondent characteristics</td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>-0.01 (.01)</td>
</tr>
<tr>
<td>Non-white race</td>
<td>0.10 (.30)</td>
</tr>
<tr>
<td>Yrs. education</td>
<td>-0.06 (.03)</td>
</tr>
<tr>
<td>HH income (log)</td>
<td>-0.26 (.11)</td>
</tr>
<tr>
<td>HH wealth (ihs)</td>
<td>-0.02 (.02)</td>
</tr>
<tr>
<td>Others in HH</td>
<td>0.36 (.22)</td>
</tr>
<tr>
<td>No. disease conditions (0 – 4+)</td>
<td>0.09 (.07)</td>
</tr>
<tr>
<td>Body weight (BMI)</td>
<td>-0.04 (.02)</td>
</tr>
<tr>
<td>Pain intensity(^a)</td>
<td>0.18 (.14)</td>
</tr>
<tr>
<td>Physical limitations (0 – 11)</td>
<td>0.18 (.04)</td>
</tr>
<tr>
<td>Spouse characteristics</td>
<td></td>
</tr>
<tr>
<td>Health-related characteristics</td>
<td></td>
</tr>
<tr>
<td>No. disease conditions (0 – 4+)</td>
<td>-0.07 (.08)</td>
</tr>
<tr>
<td>Body weight (BMI)</td>
<td>0.01 (.01)</td>
</tr>
<tr>
<td>Pain</td>
<td></td>
</tr>
<tr>
<td>Pain intensity(^a)</td>
<td>0.16 (.15)</td>
</tr>
<tr>
<td>Activity Limitation</td>
<td></td>
</tr>
<tr>
<td>Physical limitations (0 – 11)</td>
<td>-0.01 (.04)</td>
</tr>
<tr>
<td>Interaction effect</td>
<td></td>
</tr>
<tr>
<td>Respondent X Spouse Pain Intensity</td>
<td>-0.43 (.22)</td>
</tr>
<tr>
<td>Random Effect [variance (SD)]:</td>
<td>2.25 (1.50)</td>
</tr>
<tr>
<td>PRE(^b)</td>
<td>.30</td>
</tr>
</tbody>
</table>
### PANEL B: Wives (N = 423)

**Fixed Effects [unstand. coeff. (SE)]:**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Coefficient</th>
<th>SE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Intercept</strong></td>
<td>1.61</td>
<td>.36</td>
</tr>
<tr>
<td><strong>Respondent characteristics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>-0.02</td>
<td>.01</td>
</tr>
<tr>
<td>Non-white race</td>
<td>-0.16</td>
<td>.34</td>
</tr>
<tr>
<td>Yrs. education</td>
<td>-0.13</td>
<td>.04</td>
</tr>
<tr>
<td>HH income (log)</td>
<td>-0.18</td>
<td>.12</td>
</tr>
<tr>
<td>HH wealth (ihs)</td>
<td>-0.02</td>
<td>.03</td>
</tr>
<tr>
<td>Others in HH</td>
<td>0.15</td>
<td>.25</td>
</tr>
<tr>
<td>No. disease conditions (0–4*)</td>
<td>-0.03</td>
<td>.10</td>
</tr>
<tr>
<td>Body weight (BMI)</td>
<td>-0.02</td>
<td>.02</td>
</tr>
<tr>
<td>Pain intensity(^a)</td>
<td>0.68</td>
<td>.16</td>
</tr>
<tr>
<td>Physical limitations (0–11)</td>
<td>0.21</td>
<td>.04</td>
</tr>
<tr>
<td><strong>Spouse characteristics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Health-related characteristics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. disease conditions (0–4*)</td>
<td>-0.07</td>
<td>.09</td>
</tr>
<tr>
<td>Body weight (BMI)</td>
<td>&lt;-0.01</td>
<td>.02</td>
</tr>
<tr>
<td><strong>Pain</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain intensity(^a)</td>
<td>0.12</td>
<td>.16</td>
</tr>
<tr>
<td><strong>Activity Limitation</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical limitations (0–11)</td>
<td>0.04</td>
<td>.04</td>
</tr>
<tr>
<td><strong>Interaction effect</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Respondent X Spouse Pain Intensity</td>
<td>0.07</td>
<td>.20</td>
</tr>
</tbody>
</table>

**Random Effect [variance (SD)]:**

| PRE\(^b\) | 3.16 | 1.78 |

**Model Deviance\(^c\):**

| 3437.79 |

---

**Note.** HH = Household; log = Log transformation; ihs = Inverse hyperbolic sine transformation. Depressive symptomatology was measured using the CES-D8 (an 8-item version of the Center for Epidemiologic Studies-Depression scale); scores could range from 0 (no symptoms) to 8 (endorsed all 8 symptoms). Effects for husbands and wives were estimated separately, but simultaneously, via a dual-intercept hierarchical linear model. To permit estimation, initial error variances were fixed by gender (details in text and Appendix C). With the exception of the number of disease conditions and physical limitations, all individual-level continuous variables were mean-centered by gender and all couple-level continuous variables were centered across the 423 couples. Estimates and robust SEs for the fixed effects were pooled across the 8 multiply-imputed data sets. Shading (gray) indicates statistically significant effects at \( p < .05 \).

\(^a\)Pain intensity was mean-centered by gender in these analyses in order to reduce the potential for multicollinearity between the uncentered first-order terms and the higher-order product term.
PRE = Proportion reduction in error measure, calculated as the proportion of variance explained by each model as compared to the total (explainable) variance from Model 0 (from Table 13).

Model deviance estimates were pooled across the 8 multiply-imputed data sets. A chi-square test of statistical significance compared the deviance of Model 8 to that of Model 7 (from Table 17), $\chi^2(2) = 5.59$, $p = .061$. 
Of most interest in Table 18 are the parameter estimates for the Respondent X Spouse Pain Intensity interaction term. Consider first the results for DP husbands: As shown in Panel A, the interaction term was a statistically significant predictor of husbands’ depressive symptomatology. Broadly speaking, the significance of this term indicated the presence of a complex relationship between husbands’ CES-D8 scores and the pain intensity level of both spouses.

To facilitate interpretation, the prediction equation from Model 8 was used to decompose the interaction effect. A graph based on this decomposition is provided as Figure 10. The graph shows the estimated effect of different levels of wives’ pain intensity on the CES-D8 depressive symptomatology of husbands, after controlling for the effects of all other variables in the model. As the effects of other variables were held constant by setting them to zero and/or to their respective mean values, the effects illustrated in Figure 10 are those for the average DP husband. The characteristics of the average DP husband were described earlier (see pp. 5-54 to 5-55). Note that Model 8 confers an additional attribute: the average DP husband was also married to an average DP wife (she, too, also was described earlier, see pp. 5-55 to 5-56).

As Figure 10 makes clear, the effect of wife pain intensity on husband depressive symptomatology varied as a function of the husband’s pain intensity. The three lines are considered simple regression lines, and depict the regression of husband depressive symptomatology on wife pain intensity within each level of husband pain intensity. When husband pain intensity was average (i.e., just shy of “moderate”), the estimated simple slope for wife pain intensity on husband depressive symptom level was slightly positive (0.16, displayed as the solid line). When husband pain intensity was lower
Figure 10
Respondent by Spouse Pain Intensity Interaction Effect on Depressive Symptomatology in Dual-Pain Husbands

Note. Based on results from Panel A of Model 8 in Table 18, a multi-level model of CES-D8 scores of Dual-Pain husbands and wives (N = 423 couples). The CES-D8 has a maximum value of 8. Effects of pain intensity are illustrated for the otherwise "average" Dual-Pain husband and wife (i.e., controlling for all other variables in the model; see text for details). The estimated simple slope for wife pain intensity on husband CES-D8 is -0.27 when husband pain is higher (+1), 0.16 when husband pain is average, and 0.59 when husband pain is lower (-1).
(i.e., roughly “mild”), the estimated slope for wife pain intensity was also positive, but much more pronounced (0.59, shown as the dotted line). Lastly, when husband pain intensity was higher (i.e., roughly “severe”), the estimated simple slope for wife pain intensity was actually negative (-0.27, displayed as the dashed and dotted line). The coefficient for the interaction effect in Panel A ($\gamma = -0.43$) can be interpreted directly as the difference in the slope of wife pain intensity on husband depressive symptomatology between one level of husband pain intensity and the next (e.g., between lower and average pain intensity). The effect sizes associated with these simple slopes were fairly small, ranging from 10% to 33% of a standard deviation unit. The interaction effect was disordinal in nature, with the crossing point occurring toward the severe range of wife pain intensity.

Figure 10 shows that husband depressive symptomatology was highest in husbands with higher than average pain intensity who had wives with lower than average pain intensity, as well as in husbands with lower than average pain intensity who had wives with higher than average pain intensity. Depressive symptomatology scores were lowest among husbands who, along with their wives, had lower than average pain intensity. Among husbands with higher than average pain intensity, depressive symptomatology levels actually decreased with increasing levels of wife pain intensity; in this group, levels of depressive symptomatology were lowest in couples where both spouses had higher than average pain intensity.

The significant Respondent X Spouse Pain Intensity interaction effect in husbands justified revisiting Research Question 7, concerning the cross-spouse effect of pain intensity. Recall that Hypothesis 7 (H7) posited a steady increase in one partner’s
depressive symptomatology associated with increasing pain intensity levels in his/her spouse. The predicted relationship was observed, but only among husbands with lower than average pain intensity. Depressive symptom levels of husbands with average pain intensity were only minimally related to wife pain intensity, and this effect was not statistically significant \( (p = .279) \). Among husbands with higher than average pain intensity, wife pain intensity was negatively related to husband depressive symptomatology—a finding that directly contradicted H7. Therefore, the results of Model 8 for husbands provided very mixed support for H7.

Panel B of Table 18 presents the results of Model 8 for DP wives. The Respondent X Spouse Pain Intensity interaction effect was not a statistically significant predictor of wives’ depressive symptomatology. A model-based graph was created for wives in order to compare their results with the results of husbands. Figure 11 shows the estimated effects of different levels of husband pain intensity on the CES-D8 depressive symptomatology of wives, after controlling for the effects of all other variables. Effects are illustrated for the average wife, whose characteristics were described earlier (see pp. 5-55 to 5-56). Note, too, that the average DP wife was also married to an average DP husband (see description on p. 5-54 to 5-55).

Figure 11 displays the simple regression lines derived from the Model 8 equation. The three lines depict the regression of wife CES-D8 scores on husband pain intensity within each level of wives’ pain intensity. The three lines are basically parallel, indicating that the effect of husbands’ pain intensity did not vary as a function of the pain intensity level of wives. The estimated simple slope of husband pain intensity on wife CES-D8 scores was slightly positive, regardless of whether wife pain intensity was lower.
Figure 11
Respondent by Spouse Pain Intensity Interaction
Effect on Depressive Symptomatology in Dual-Pain Wives

Note. Based on results from Panel B of Model 8 in Table 18, a multi-level model of CES-D8 scores of Dual-Pain husbands and wives (N = 423 couples). The CES-D8 has a maximum value of 8. Effects of pain intensity are illustrated for the otherwise "average" Dual-Pain husband and wife (i.e., controlling for all other variables in the model; see text for details). The estimated simple slope for husband pain intensity on wife CES-D8 is 0.19 when wife pain is higher (+1), 0.12 when wife pain is average, and 0.05 when wife pain is lower (-1).
than average (0.05, displayed as the dotted line), average (0.12, shown as the solid line), or higher than average (0.19, displayed as the dashed and dotted line). The coefficient for the interaction effect in Panel B ($\gamma = 0.07$) represents the difference in the slope of husband pain intensity on wife CES-D8 depressive symptomatology between one level of wife pain intensity and the next (e.g., between average and higher). This coefficient was not statistically significant ($p = .738$), indicating that that these simple slopes did not differ from each other. Note that the estimated standard error of the interaction effect was more than twice the size of the coefficient ($SE\gamma = .20$), indicating a fair amount of imprecision in this estimate. This issue will be addressed in detail in the section on model checking that follows. Because no evidence for a cross-spouse effect of pain intensity was found in wives, $H7$ was not supported for DP wives.

Figure 11 does illustrate the sizable effect of wives’ pain intensity on their own depressive symptomatology—an effect that remained significant in Model 8. This effect is reflected in the systematic increase in wives’ CES-D8 depressive symptomatology scores associated with each incremental increase in wives’ own pain intensity. Regardless of husband pain intensity level, wives’ predicted CES-D8 scores increased with their own pain intensity.

It is important to note that the effects of a respondent’s own pain and his/her spouse’s pain were observed while controlling for his/her own physical limitations and those of his/her spouse. As can be seen in Table 18, the respondent’s own physical limitations remained a significant predictor of (higher) depressive symptomatology in both husbands and wives. Thus, the *intra-*individual effect of the respondent’s own pain on his/her own depressive symptoms (as discussed above) was still at least partially
mediated by his/her own physical limitations. Again in Model 8, however, the spouse’s physical limitations were not significantly related to the depressive symptomatology of either DP husbands or wives. The fixed effect estimates obtained for other predictor variables in Model were very similar to those in Model 7. Neither of the health-related characteristics of either spouse (BMI, number of health conditions) was significantly related to depressive symptomatology in the other partner. Education was still the only individual-level background characteristic independently associated with (slightly lower) depressive symptomatology levels in both spouses. For husbands, greater household income and higher BMI remained significant predictors of (lower) depressive symptomatology.

Additional data regarding the overall performance of Model 8 is presented in Table 18. Adding the Respondent X Spouse Pain Intensity interaction effect to the multivariate model predicting depressive symptomatology increased the PRE over Model 7 by a small amount for husbands (.01), but not for wives. Together, the set of individual- and couple-level background variables and the health-related characteristics, pain intensity ratings, and physical limitations of both the respondent and his/her spouse explained roughly 30% of the variance in husbands’ depressive symptomatology and 28% of the variance in wives’ depressive symptomatology. However, sizeable amounts of variation in the CES-D8 depressive symptomatology of both spouses remain unexplained, as evidenced by the statistical significance of both random effects.

When the model deviance value associated with Model 8 was compared to that of Model 7, the difference was only marginally statistically significant, $\chi^2(2) = 5.59, p = .061$. This likely reflects the fact that the Respondent X Spouse Pain Intensity interaction
effect was a statistically significant predictor only for husbands and not for wives.
Consider, though, that Model 7 itself was not statistically significant, and therefore may
not have provided the best model for comparison. It would thus be premature to infer
that the Respondent X Spouse Pain Intensity interaction effect was of little value in
explaining DP husbands’ CES-D8 scores. Further refinement of the predictive model
might be necessary to adequately isolate the contribution of this effect.

Although no explicit hypothesis was developed concerning the potential
interactive effect of the pain intensity levels of DP spouses, the contrast in findings
between husbands and wives warranted reconsideration of Research Question 9 (RQ9).
RQ9 dealt with gender differences in the cross-spouse relationship between each
spouse’s pain intensity and his/her partner’s depressive symptomatology. Recall that the
associated hypothesis, Hypothesis 9 (H9), proposed that wives would be more strongly
affected by their husbands’ pain intensity than the converse. The results of Model 8, in
conjunction with the graphs in Figures 10 and 11, provide clear evidence of a gender
difference in the cross-spouse effect of pain. Surprisingly, the nature of this difference
was inconsistent with the direction of effects predicted by H9: Husbands’ depressive
symptomatology was related to wives’ pain intensity (in complex ways), but wives’
depressive symptomatology was not associated with the pain intensity of husbands.
Thus, evidence from Model 8 (i.e., that husbands were affected by wives’ pain intensity,
but wives were not affected by husbands’ pain intensity) clearly contradicted H9.

Model Checking and Tests of Model Assumptions

Two sensitivity analyses were performed. First, Model 8 was tested using log-
transformed CES-D8 scores. The results of this model yielded substantively identical
results. Specifically, the Husband X Wife Pain Intensity interaction effect remained a statistically significant predictor of DP husbands’ depressive symptomatology, but was not significantly related to wives’ depressive symptomatology. One additional finding from this analysis deserves mention. The standard error of the pain intensity interaction effect in wives was considerably smaller ($\gamma = -.04, SE_{\gamma} = .06$) when logged CES-D8 scores were used. Recall that the number of DP husbands reporting severe pain was fairly small ($n = 70$). Even though these husbands appeared to be amply distributed across the three levels of wife pain intensity (18.6%, 45.7%, and 35.7%, respectively), the absolute number of couples in each group was small, especially couples in which the husband had severe pain and the wife had mild pain. These small numbers resulted in similar amounts of imprecision in the estimated Husband X Wife Pain Intensity interaction effect on the original CES-D8 scores of both spouses. Although this imprecision did not hamper the sizeable interaction effect estimated in husbands, the large standard error for the interaction effect in wives was a concern. That the analysis of logged CES-D8 scores yielded a more reasonable standard error for the estimated interaction effect in wives suggests that the distribution of wives’ depressive symptom scores in one of the three groups may have been non-normal. Adjusting for this non-normality, however, did not alter the outcome: No joint husband-wife pain intensity effect was observed for wives’ depressive symptomatology.

A second sensitivity analysis addressed whether the observed pain intensity interaction effect for husbands could have been a statistical artifact. Regression scholars warn about the danger of falsely concluding that an interaction exists when, in fact, one of the independent variables has a nonlinear relationship with the outcome variable (e.g.,
Several alternative models were tested in order to evaluate different configurations of linear, quadratic, and product terms for husband and wife pain intensity. No model emerged as clearly superior in these analyses; in fact, some of the best fitting models suggested the presence of both non-linear and interactive effects for husband and wife pain intensity. These analyses provided no compelling evidence that the pain intensity interaction effect observed for DP husbands was attributable to a statistical artifact. This conclusion was further supported by one additional finding. According to Cohen et al. (2003), such spurious interaction effects are most likely to occur when the two component variables are at least moderately correlated (pp. 299-300). Recall that DP husband and wife pain intensity ratings were only modestly related ($ICC_{adj} = .10$).

Additional model checking of Model 8 was done using the same strategies used to check the final intra-individual model (i.e., Model 4). Model-based residuals and fitted values were examined in order to identify potential multivariate outliers and influential cases. Multicollinearity diagnostics were computed for the full set of predictors in the model. As before, model-checking was done using data from one imputed dataset selected at random (imputation 4).

The Level 2 (L2) residuals (i.e., empirical-Bayes residuals for the random intercepts) were first examined by gender. Both husbands’ and wives’ L2 residuals were relatively normally distributed. The L2 residuals for husbands ranged from -2.35 to 3.93 and evidenced minor positive skewness ($M = 0.00, SD = 1.24, Mdn = -0.23$). Calculated skewness and kurtosis values were well within normal limits, however ($skewness = 0.79; kurtosis = 0.37$). The distribution of wives’ L2 residuals was more symmetrical ($M = \ldots$)
0.00, SD = 1.53, Mdn = -0.32, skewness = 0.58, kurtosis = -0.43), with values ranging from -3.64 to 4.05.

No noteworthy patterns were observed when the L2 residuals were graphed against the L2 fitted values (i.e., dependent variable values predicted by the L2 variables) for either spouse. The L2 residuals were also graphed against the continuous predictors in Model 8. Unusual patterning was observed in only two graphs. The graph of husbands’ L2 residuals against their BMI values revealed a cluster of cases toward the upper end of the BMI range with negative residuals. Some clustering was also observed in relation to wives’ age: L2 residuals seemed slightly larger for wives at both ends of the age range, compared to those of average age. This clustering suggests that it may be useful to explore alternate functional forms of these predictor variables. For instance, incorporating a term to represent a non-linear effect of husband BMI on husband depressive symptomatology might improve model fit.

The L2 residuals were standardized by gender, in order to identify potential multivariate outliers and influential cases. Less than 5% of husbands (n = 19) had L2 standardized residuals greater than ± 2.00. Within this group of husbands, values ranged from 2.62 to 3.93. Less than 4% of wives (n = 15) had large standardized residuals (i.e., greater than ± 2.00). The residual for one wife was negative (-2.37), while those of remaining wives ranged between 2.07 to 2.64. Based on these values, the model mainly under-estimated the depressive symptom levels of these DP spouses.

The standardized L2 residuals were also examined at the couple level. High-low charts were constructed for the standardized residuals of 50 husband-wife pairs selected randomly from the sample. No discernible pattern was observed for either set of
residuals. Just under 8% of couples \( n = 32 \) had either a husband or a wife with a large L2 standardized residual. Large standardized L2 residuals were observed for both husband and wife in only one couple, indicating that the L2 predictors did not do a good job of predicting the depressive symptomatology of either spouse in this couple. This couple was one of the couples identified as a potential outlier in the analysis of the residuals for Model 4. Both spouses in this couple were older than average (husband was 80, wife was 79), had BMI levels considerably lower than average (albeit in the normal weight range), and reported total net assets well below the median household wealth of DP couples. Although both reported a large number of physical limitations (8 out of 11 total) and more disease conditions than average (3 out of 4 or more), they reported only mild pain intensity. The L2 standardized residual for each spouse was positive (2.99 for the husband, 2.18 for the wife). Thus, the Model 8 predictor variables underestimated the depressive symptomatology scores of this husband and his wife.

Additional diagnostic procedures were used to detect potential model specification errors and/or violations of model-based assumptions. The model-based and robust standard errors associated with the fixed effect coefficients were compared. In Model 8, differences between model-based and robust standard errors \( (SEs) \) for the fixed effect coefficients were fairly small (e.g., \( \leq .01 \)). The trend was for the robust \( SEs \) to be slightly larger than the model-based \( SEs \), which generally reduced the statistical significance of the affected predictors. These small differences rarely resulted in a substantive difference with respect to whether a predictor was significant or not. Thus, observed departures from normality in the outcome and in some of the predictor variables likely had little impact on model estimation (Raudenbush & Bryk, 2002).
The Level 1 fitted values for husbands and wives were compared to both the possible range of scores and the observed range of CES-D8 scores (both 0 - 8). L1 fitted values for husbands ranged from 0.04 to 7.35, whereas values for wives ranged from -0.07 to 7.51. The L1 fitted values covered the majority of the possible range of CES-D8 scores. In addition, both distributions closely resembled the range of observed scores.

Multicollinearity diagnostics were calculated for the set of predictor variables included in Model 8. In accordance with the use of the dual-intercept multilevel models, diagnostics were run separately for husbands and wives. VIF values for the predictors in Model 8 for husbands ranged from 1.03 for the Husband x Wife Pain Intensity interaction term to 1.50 for wife physical limitations. For wives, VIF values for the predictors ranged from 1.03 for the Husband x Wife Pain Intensity interaction term to 1.50 for both wife age and wife physical limitations. No VIF value exceeded either the conventional VIF ≥ 10 cutoff (Cohen et al., 2003) or a more stringent cutoff (VIF ≥ 2.5; Allison, 1999). The square root of the highest VIF value yielded a quantity of 1.22. The fact that this value was relatively close to 1 suggests that very little multicollinearity-related inflation occurred in the standard errors of the parameter estimates in Model 8.

Additional multicollinearity diagnostics were also examined. The condition number for Model 8 was 69.79 for both husbands and wives; this is below a suggested threshold of 100 used to signal moderate collinearity (Callaghan & Chen, 2008). Condition indexes (CIs) ranged from 1.00 to 8.34 for husbands and from 1.00 to 8.37 for wives; none exceeded a suggested cutoff of 10 for moderate collinearity (Callaghan & Chen, 2008). In short, all diagnostic measures indicated that no substantive problems with multicollinearity occurred in Model 8.
A Final Trimmed Model and Illustration of Additive Effects

Table 19 presents a final multivariate model of DP spouses’ depressive symptomatology—Model 9. Although there is debate in the literature regarding the advisability and necessity of model trimming, as well as whether model refinement should occur only within the context of an extended program of research (e.g., Kutner, Nachtsheim, & Neter, 2004; Pedhauzer, 1997), there are limitations associated with over-specification in linear models. Most importantly, the inclusion of predictor variables with no relationship to the dependent variable can reduce the precision of the parameter estimates for all variables in the model (Allison, 1999). Given that dual-intercept hierarchical models essentially contain twice as many predictors as single-level regression models (e.g., demographic variables like age are modeled for both husbands and wives), trimming was regarded as especially appropriate in the present study.

Two criteria were used to determine if a predictor variable was retained in the final model. First, a variable was retained if it had been a statistically significant predictor in any prior model for either husbands or wives. Second, a variable was retained as a predictor if it represented a key construct in the proposed conceptual model (e.g., pain intensity of both the respondent and the spouse). Application of these criteria resulted in the removal of three background characteristics (Others living in the household, household wealth, and non-White race) and the two health-related characteristics of the spouse (number of disease conditions and BMI). The final, trimmed model thus remained comparable by gender (i.e., same predictors included for husbands and wives) and was also consistent with the study’s conceptual framework. The structure and specifications of Model 9 were essentially the same as those described for Model 8,
Table 19. Final Trimmed Model Predicting Depressive Symptomatology in Husbands and Wives in Dual-Pain Couples (N = 423)

<table>
<thead>
<tr>
<th>Model 9 (Final Trimmed Model&lt;sup&gt;a&lt;/sup&gt;)</th>
<th>PANEL A: HUSBANDS</th>
<th>PANEL B: WIVES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fixed Effects [unstand. coeff. (SE)]:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>1.16 (.25)</td>
<td>1.49 (.29)</td>
</tr>
<tr>
<td>Respondent characteristics</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>-0.02 (.01)</td>
<td>-0.03 (.01)</td>
</tr>
<tr>
<td>Yrs. education</td>
<td>-0.07 (.01)</td>
<td>-0.13 (.01)</td>
</tr>
<tr>
<td>HH income (log)</td>
<td>-0.32 (.10)</td>
<td>-0.21 (.11)</td>
</tr>
<tr>
<td>No. disease conditions (0–4+)</td>
<td>0.10 (.07)</td>
<td>-0.01 (.10)</td>
</tr>
<tr>
<td>Body weight (BMI)</td>
<td>-0.04 (.02)</td>
<td>-0.03 (.02)</td>
</tr>
<tr>
<td>Respondent Pain &amp; Activity Limitation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain intensity&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.16 (.14)</td>
<td>0.70 (.16)</td>
</tr>
<tr>
<td>Physical limitations (0–11)</td>
<td>0.21 (.04)</td>
<td>0.18 (.04)</td>
</tr>
<tr>
<td>Spouse Pain &amp; Activity Limitations</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain intensity&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.17 (.15)</td>
<td>0.11 (.15)</td>
</tr>
<tr>
<td>Physical limitations (0–11)</td>
<td>-0.01 (.03)</td>
<td>-0.03 (.04)</td>
</tr>
<tr>
<td>Interaction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Respondent X Spouse Pain Intensity</td>
<td>-0.44 (.22)</td>
<td>-0.06 (.20)</td>
</tr>
<tr>
<td>Random Effect [variance (SD)]:</td>
<td>2.31 (1.52)</td>
<td>3.18 (1.78)</td>
</tr>
<tr>
<td>PRE&lt;sup&gt;c&lt;/sup&gt;</td>
<td>.28</td>
<td>.27</td>
</tr>
</tbody>
</table>

Model Deviance | 3446.34 |

Note. HH = Household; log = Log transformation; ihs = Inverse hyperbolic sine transformation. Depressive symptomatology was measured using the CES-D8 (an 8-item version of the Center for Epidemiologic Studies-Depression scale); scores could range from 0 (no symptoms) to 8 (endorsed all 8 symptoms). Effects for husbands and wives were estimated separately, but simultaneously, via a dual-intercept hierarchical linear model. To permit estimation, initial error variances were fixed by gender (details in text and Appendix C). With the exception of the number of disease conditions and physical limitations, all individual-level continuous variables were mean-centered by gender and all couple-level continuous variables were centered across the 423 couples. Estimates and robust SEs for the fixed effects were pooled across the 8 multiply-imputed data sets. Shading (gray) indicates statistically significant effects at p < .05.

<sup>a</sup>This is a trimmed version of Model 8 (Table 18). A predictor variable was retained in the final model if (a) it had been statistically significant in any prior model (Models 1-8) for either husbands or wives, or (b) it represented a key construct in the conceptual model (e.g., pain intensity, physical limitations).

<sup>b</sup>Pain intensity was mean-centered by gender in these analyses in order to reduce the potential for multicollinearity between the uncentered first-order terms and the higher-level product term.

<sup>c</sup>PRE = Proportion reduction in error measure, calculated as the proportion of variance explained by the model as compared to the total (explainable) variance from Model 0 (from Table 13).
but the results are presented in a slightly different format. In order to facilitate the
comparison of results by gender, Panels A and B are presented side by side in Table 19.

The PRE values shown in Table 19 for both husbands and wives were a bit
smaller in Model 9, as compared to Model 8. This was most likely a consequence of
removing several predictor variables from the model. Yet, Model 9 still explained a
reasonable amount of variance in husbands’ and wives’ CES-D8 scores (28% and 27%,
respectively). The model deviance value for Model 9 is listed at the bottom of Table 19.
However, the change in model deviance between Models 9 and 8 was not tested
statistically, as Model 9 was intended mainly to illustrate (not test) effects. Notably,
though, the trimming of nonsignificant predictors did not seriously impair or degrade the
model. In fact, most remaining parameters exhibited remarkable stability, despite
changes to the number and composition of predictor variables. Consistent with all prior
models, the random effects in Model 9 remained significant, confirming that considerable
amounts of variance remained unexplained. Compared to Model 8, Model 9 was more
parsimonious—a desirable quality in explanatory models (Jaccard & Jacoby, 2010).

The fixed effect estimates from Model 9 were markedly similar to those from
Model 8. The only substantive differences occurred in the estimated effects of age and
household income on depressive symptomatology. Whereas age had not been a
significant covariate in prior models for husbands, it was now a significant (albeit weak)
predictor of lower depressive symptomatology. Age also re-emerged as a weak, but
statistically significant, predictor of lower depressive symptomatology in wives; the
coefficient for age had not reached significance in Model 7, but had been significant in all
prior intra-individual and cross-spouse models. Depressive symptomatology in wives
was now also inversely related to household income—a relationship that had only been significant in early intra-individual models. Household income remained a significant predictor of (lower) depressive symptomatology in husbands. Education remained independently associated with (lower) depressive symptomatology levels in both DP husbands and wives. For husbands, higher BMI also remained a significant predictor of (lower) depressive symptomatology. As in most prior models, number of disease conditions was not significantly related to the depressive symptoms of either spouse.

After controlling for all other predictors, one’s own physical limitations remained a significant predictor of (higher) CES-D8 depressive symptomatology for both spouses. One’s own pain intensity retained its sizeable, independent relationship with higher depressive symptomatology in wives, but had no independent, straightforward association with husbands’ depressive symptom levels. These results were again consistent with the main findings from prior intra-individual models. For example, they suggest that the respondent’s own physical limitations at least partially mediated the relationship between his/her own pain intensity and depressive symptomatology.

The estimated coefficients for the Respondent X Spouse Pain Intensity interaction effect were very stable; for example, the coefficient for the interaction in husbands changed from $\gamma = -0.43$ in Model 8 to $\gamma = -0.44$ in Model 9. The joint effect of husband and wife pain intensity remained a significant predictor of husbands’ depressive symptomatology, but was still unrelated to wives’ depressive symptoms. As was the case in the prior model, the standard error for the pain intensity interaction effect in DP wives was fairly large. The conditional cross-spouse effect of wives’ pain intensity on husbands’ depressive symptomatology remained the same as in Model 8 (i.e., the effect
depended on the level of the husbands’ pain intensity). These findings provided mixed support for Hypothesis 7 (concerning the existence of a cross-spouse effect for pain intensity), but the fact that a cross-spouse effect was found only in husbands and not in wives contradicted Hypothesis 9. The physical limitations of the spouse were not significantly related to the depressive symptom levels of either spouse. Moreover, there was no evidence to suggest that the spouse’s physical limitations mediated the relationship between his/her pain intensity and the depressive symptomatology of his/her partner, resulting in a lack of support for Hypotheses 8a and 8b.

As in prior models, many of the parameter estimates for the predictor variables in Model 9 were relatively small. The CES-D8 depressive symptom scores predicted by prior models and graphed in Figures 7, 9, 10, and 11 were also low, relative to the range of possible values on the scale (i.e., 0 - 8). However, those graphs were designed to isolate specific effects by holding other predictors constant. While helpful, this strategy often fails to convey adequately the additive impact of a set of predictor variables. Because predictor variables tend to be inter-related, increases in one are typically accompanied by increases in another. Additionally, it can be more compelling to consider the effects of substantial (and often more realistic) variation in the values of predictor variables—for example, the effect on depressive symptomatology of a 1-standard deviation increase in physical limitations vs. a one-unit increase. In the current context, such illustrations might help to identify the spouses and couples who (based on Model 9) were most at risk for elevated depressive symptomatology.

The equation derived from Model 9 illustrates the additive impact of multiple predictors on the depressive symptomatology of DP spouses. A collection of predicted
CES-D8 scores for DP husbands and wives are displayed in Figures 12 and 13, respectively. Here, the effects of the respondent’s own pain intensity, the spouse’s pain intensity (if any), and their interaction (if any) have been graphed in the presence of other additive risk factors. These predicted CES-D8 scores reflect the effects of pain intensity and the effects of other predictor variables. To illustrate the additive nature of these effects, the values of the other predictor variables were systematically varied away from their averages—generally 1 SD higher or lower, depending on the nature of the effect.

Compared to average DP couples depicted in earlier graphs, couples at risk for increased depressive symptomatology had lower HH income and were comprised of spouses who were younger, had less education, more disease conditions, slightly lower body weight, and more physical limitations. These characteristics are discussed in more detail below.

Figure 12 displays the predicted CES-D8 scores of husbands in DP couples with additive risk factors (i.e., a constellation of characteristics that placed them at risk for elevated depressive symptomatology). Compared to average DP husbands, these husbands were about a decade younger (56.50 yrs.). They had only an 8th grade education (11.44 - 1SD [3.38] = 8.06 yrs.), and reported lower household income (~ 10th percentile). These husbands reported an average of 3.4 disease conditions (2.12 + 1SD; ~ 83rd percentile). Their BMI was 22.7 (27.93 – 1SD [5.24] BMI units), placing them in the normal, rather than over-weight, category. These husbands also had a high number of physical limitations (7.37 = 4.43 + 1SD). Notably, husbands with additive risk factors also had wives with additive risk factors (see description below).

The most notable feature of Figure 12 is the elevation in husbands’ predicted CES-D8 scores resulting from the additive impact of multiple risk factors. Their scores
Figure 12
Respondent by Spouse Pain Intensity Interaction Effect for Dual-Pain Husbands in the Presence of Additive Risk Factors

Note. Based on results from Panel A of Model 9 in Table 19, a multi-level model of CES-D8 scores of Dual Pain (DP) husbands and wives (N = 423 couples). Lines show the estimated effect of wife pain intensity on husband CES-D8 within each level of husband pain intensity (lower, average, higher). Effects are estimated for DP couples who, compared to "average" DP couples, have additive risk factors (e.g., lower HH income and are comprised of spouses who are younger and have less education, more disease conditions, slightly lower body weight, and greater physical limitations; see text for details).
Figure 13
Respondent by Spouse Pain Intensity Interaction Effect for Dual-Pain Wives in the Presence of Additive Risk Factors

Note. Based on results from Panel A of Model 9 in Table 19, a multi-level model of CES-D8 scores of Dual Pain (DP) husbands and wives (N = 423 couples). Lines show the estimated effect of husband pain intensity on wife CES-D8 within each level of wife pain intensity (lower, average, higher). Effects are estimated for DP couples who, compared to “average” DP couples, have additive risk factors (e.g., lower HH income and are comprised of spouses who are younger and have less education, more disease conditions, slightly lower body weight, and greater physical limitations; see text for details).
are now clustered around the mid-point of the scale (i.e., 4 vs. 2). The graph also illustrates the interaction between a husband’s own pain intensity and the pain intensity of his wife; the substantive interpretation of the simple slopes is as previously described.

Figure 13 displays the predicted CES-D8 scores of DP wives with additive risk factors. Like their husbands, these DP wives were roughly a decade younger than average DP wives (52.34 yrs.; roughly the 14th percentile). They also had less education, having not quite finished 9th grade (~ 16th percentile). They had an average BMI of 22.1 (28.51 – 1SD; ~ 14th percentile), placing them in the normal weight range. These wives reported having roughly 3 disease conditions (1.93 + 1SD), and more physical limitations than average DP wives (8.69 vs. 5.48). These wives had husbands with additive risk factors, and lived in households with lower than average income.

The main contribution of Figure 13 is an illustration of the striking negative impact of multiple risk factors on wives’ CES-D8 depressive symptomatology scores. DP wives’ scores now cluster above the midpoint of the scale (i.e., 4.5 vs. 2.5). Figure 13 reiterates that wives’ depressive symptom levels were not significantly affected by the pain intensity levels of their husbands. Instead, the parallel lines in the graph emphasize the negative effect associated with increased levels of their own pain intensity.

Model Checking and Tests of Model Assumptions

As with Model 8, two sets of sensitivity analyses were conducted for Model 9. Analysis of the log-transformed CES-D8 scores yielded the same substantive results as those reported for scores in the original metric. Although the standard error for the pain intensity interaction effect in wives was significantly reduced in models using the log-transformed CES-D, the interaction effect was still not a significant predictor of wives’
depressive symptomatology. The second set of sensitivity analyses evaluated different combinations of linear, quadratic, and product terms for husband and wife pain intensity. No model emerged as a superior-fitting model. Thus, these results provided no evidence that the observed pain intensity interaction effect in DP husbands was an artifact produced by a non-linear relationship involving one or both spouse’s pain intensity ratings.

Model checking of Model 9 also involved examination of residuals and an evaluation of potential multivariate outliers and influential cases. Multicollinearity diagnostics were also computed. As before, model checking was done using one imputed dataset selected at random (imputation 5).

The Level 2 (L2) residuals from Model 9 were examined separately by gender. Both husbands’ and wives’ L2 residuals had relatively normal distributions. Some positive skewness was observed in the distribution of husbands’ L2 residuals ($M = 0.00$, $SD = 1.26$, $Mdn = -0.28$, $Range = -2.51 – 4.06$). However, the calculated values for skewness (0.79) and kurtosis (0.32) were well within normal limits. Wives’ L2 residuals were more symmetrically distributed ($M = 0.00$, $SD = 1.55$, $Mdn = -0.36$, $skewness = 0.60$, $kurtosis = -0.41$), with values ranging from -3.49 to 4.32.

When the L2 residuals were graphed against the L2 fitted values (i.e., those predicted by the L2 predictor variables) for husbands and wives, no noteworthy patterns were observed. The clustering observed in Model 8 when L2 residuals were graphed against L2 predictor variables was also observed for Model 9 (i.e., husband BMI and wife age). Again, this clustering may suggest that altering the functional form of these predictor variables could improve the overall fit of the multivariate model.
The L2 residuals were standardized by gender, in order to identify potential multivariate outliers and influential cases. Twenty-three (5.4%) husbands had L2 standardized residuals greater than ± 2.00. Within this group of husbands, L2 residual values ranged from 2.53 to 4.06. Sixteen (3.8%) wives had large standardized residuals (i.e., greater than ± 2.00). One wife in this group had a negative L2 residual (-2.25), but L2 residuals for remaining wives ranged between 2.03 to 2.79. Thus, the model leaned toward under-prediction of CES-D8 scores in DP husbands.

The standardized L2 residuals were also examined at the couple level. The standardized residuals of 50 randomly-selected husband-wife pairs were examined using high-low charts. No discernible pattern was observed for either set of residuals. Large L2 standardized residuals were observed for either the husband or wife in only eight percent of couples (n = 35). Both spouses had large standardized L2 residuals in two couples, indicating that the L2 predictors in Model 9 did not do a good job of predicting the depressive symptom levels of either spouse in these couples. Notably, these were the same two couples who had been identified as possible outliers based on the standardized L2 residuals from Model 4. A detailed description of these couples was provided earlier (p. 5-83). In short, the spouses in both couples had more disease conditions and more physical limitations that other DP spouses, but reported low levels of pain intensity. One couple was further distinguished by their advanced age and normal body weight; the other couple was slightly younger than average and the wife in this couple had very high body weight. Here again, the L2 standardized residuals for all 4 spouses were positive (ranging from 2.05 to 2.89), indicating that the predictor variables in Model 9 underestimated their CES-D8 depressive symptomatology scores.
Additional diagnostic procedures were used to detect potential model specification errors and/or violations of model-based assumptions. The model-based and robust standard errors were compared. Much like the pattern observed in Model 8, differences between model-based and robust standard errors (SEs) for the fixed effect coefficients in Model 9 were fairly small (e.g., \( \leq .01 \)). Robust SEs tended to be slightly larger than the model-based SEs, reducing the statistical significance of the affected predictors, but rarely resulting in a substantive difference with respect to conclusions. Observed departures from normality in the outcome and in some of the predictor variables likely had little impact on model estimation (Raudenbush & Bryk, 2002).

The Model 9 Level 1 fitted values for husbands and wives were compared to both the possible and the observed range of CES-D8 scores (both 0 - 8). For husbands, L1 fitted values ranged from 0.02 to 7.36, whereas values for wives ranged from -0.35 to 7.48. Thus, the L1 fitted values covered the majority of the theoretical range of CES-D8 scores and closely resembled the range of observed CES-D8 scores.

Multicollinearity diagnostics were calculated for the set of predictor variables included in Model 9. Diagnostics were run separately for husbands and wives. For husbands, predictor VIF values ranged from a low of 1.02 for the Husband x Wife Pain Intensity interaction term to a high of 1.37 for wife physical limitations. Model 9 VIF values for wives ranged from a low of 1.03 for the Husband x Wife Pain Intensity interaction term, to a high of 1.50 for wife physical limitations. No VIF value exceeded either conventional or more stringent cutoff values (e.g., \( \geq 10 \) per Cohen et al., 2003, or \( \geq 2.5 \) per Allison, 1999, respectively). The square root of the highest VIF value yielded a quantity of 1.22, suggesting that little multicollinearity-related inflation had occurred in
the standard errors of the parameter estimates. The Model 9 condition number was 43.41 for husbands and 42.57 for wives, both of which were below a suggested threshold of 100 (Callaghan & Chen, 2008). Condition indexes ranged from 1.00 to 6.60 for husbands and 1.00 to 6.53 for wives; none exceeded a cutoff value of 10 that might suggest a problem with multicollinearity in Model 9 (Callaghan & Chen, 2008).
# CHAPTER 6: DISCUSSION

## TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Summary/Overview</td>
<td>6-1</td>
</tr>
<tr>
<td>Consideration of Major Findings</td>
<td>6-3</td>
</tr>
<tr>
<td>How Common Are Reports of Pain in Middle-Aged and Older Married Couples?</td>
<td>6-3</td>
</tr>
<tr>
<td>Dual-Pain Couples: Who Are They and What Are Their Characteristics?</td>
<td>6-7</td>
</tr>
<tr>
<td>How Are Husbands and Wives in Dual-Pain Couples Different and Similar?</td>
<td>6-11</td>
</tr>
<tr>
<td>Gender Differences</td>
<td>6-11</td>
</tr>
<tr>
<td>Pain Intensity</td>
<td>6-11</td>
</tr>
<tr>
<td>Physical Limitations</td>
<td>6-14</td>
</tr>
<tr>
<td>Depressive Symptomatology</td>
<td>6-17</td>
</tr>
<tr>
<td>Other Characteristics</td>
<td>6-20</td>
</tr>
<tr>
<td>Putting Gender Differences in Context</td>
<td>6-22</td>
</tr>
<tr>
<td>Similarities/Cross-Spouse Covariation (Paths 1, 2, and 3)</td>
<td>6-24</td>
</tr>
<tr>
<td>Pain Intensity, Depressive Symptomatology, and Physical Limitations</td>
<td>6-25</td>
</tr>
<tr>
<td>Other Characteristics</td>
<td>6-26</td>
</tr>
<tr>
<td>Explaining Observed Covariation</td>
<td>6-27</td>
</tr>
<tr>
<td>Lack of Predictors of Pain Intensity</td>
<td>6-35</td>
</tr>
<tr>
<td>The Predictive Value of Individual Disease Conditions</td>
<td>6-40</td>
</tr>
<tr>
<td>The Intra-Individual Relationship Between Pain Intensity and Depressive Symptomatology</td>
<td>6-43</td>
</tr>
<tr>
<td>Pain Intensity Is Associated with Depressive Symptomatology (Path A)</td>
<td>6-43</td>
</tr>
</tbody>
</table>
Pain Intensity Is Associated with Physical Limitations (Path B) ................................................................. 6-48

Physical Limitations Are Associated with Depressive Symptomatology (Path C) ............................................. 6-50

Physical Limitations (at Least Partially) Mediate This Relationship (Path A’) ..................................................... 6-51

Gender Differences ..................................................................... 6-51

Consistent Evidence of Partial Mediation.................................. 6-54

Shared Study and Sample Characteristics................................ 6-56

Differences in Study and Sample Characteristics ................. 6-57

Findings Regarding Covariates .................................................. 6-69

Background Characteristics ...................................................... 6-70

Health-Related Characteristics .................................................. 6-80

The Inter-Individual Relationship between Pain Intensity and Depressive Symptomatology (Paths 4 and 5) .............................................................. 6-87

The Husband by Wife Pain Intensity Interaction Effect in DP Husbands (Path 4W) ............................................. 6-89

Pattern 1: Husbands With Mild Pain Intensity ......................... 6-90

Pattern 2: Husbands With Moderate Pain Intensity ............... 6-90

Pattern 3: Husbands With Severe Pain Intensity .................... 6-91

Lack of a Husband by Wife Pain Intensity Interaction Effect and Any Straightforward Cross-Spouse Effect of Pain Intensity in DP Wives (Path 4H) ................................................ 6-100

Methodological Considerations ................................................. 6-104

The Gender Difference in the Cross-Spouse Effects of Pain Intensity (Path 4W vs. Path 4H) ............................................ 6-106

Conclusion .................................................................................. 6-107
Absence of Cross-spouse Mediation by Physical Limitations (Paths 5_w & 5_H) ................................................................. 6-108
  No Mediation ............................................................................ 6-108
  No Cross-Spouse Effects Involving Physical Limitations ............................................................................. 6-109
Findings Regarding Covariates .......................................................... 6-110
  Lack of Cross-Spouse Effects Involving Health-Related Characteristics ........................................................ 6-112
Implications for Practice, Education, & Policy .................................................. 6-113
  Social Work Practice Implications ........................................................ 6-113
  Implications for the Education & Training of Social Workers and Other Health Care Professionals ........................................... 6-121
  Social Policy Implications ........................................................................ 6-129
Study Limitations .......................................................................................... 6-135
  Limitations of Secondary Data .............................................................. 6-135
    Pain and Pain Intensity .................................................................... 6-135
    Depressive Symptomatology ............................................................ 6-138
    Physical Limitations (Activity Limitation) .......................................... 6-139
    Other Measures ............................................................................... 6-141
  Model Misspecification: Conceptual & Statistical Issues ....................... 6-143
    Cross-sectional Data ........................................................................ 6-143
    Omitted Constructs and Relationships ................................................. 6-145
      Intra-individual Level .................................................................... 6-145
      Inter-individual Level ..................................................................... 6-147
      Other Issues ............................................................................... 6-149
  Generalizability and Limitations of the Sample ....................................... 6-156
Limitations Associated with Sample Selection Criteria ...................... 6-159

Uniqueness of DP Couples ................................................................. 6-161

Strategic Directions for Research & Theory Building............................ 6-162

Confronting the Limitations of Activity Limitation Theory:
The Need for Theory Building and Additional Research on the
Cross-spouse Effects of Pain ................................................................. 6-163

Refine the Construct of Activity Limitation for Application
at the Inter-Individual Level ................................................................. 6-167

Consider Alternative and/or Additional Mediators ....................... 6-168

Might Mediation Take Place at a Different Level? .............................. 6-169

Other Possible Mediators ................................................................. 6-170

Explore (Other) Potential Moderating Constructs ......................... 6-172

Is it His Pain, Her Pain, or Our Pain? .............................................. 6-173

Do We Cope With Pain as Individuals, as a Couple,
or Both? .................................................................................... 6-174

Do Couple-Level Patterns Matter; and, If so, How? .................... 6-176

Validating and Explaining Gender Differences in the
Relationships Between Pain and Psychological Distress .................... 6-179

Balancing Specificity and Generality in Replicating and
Extending This Study ....................................................................... 6-181

Evaluating the Relevance of the Chronic Versus Acute Pain
Distinction in Mid and Later Life ....................................................... 6-187

Conclusion: Study Strengths and Contributions ............................... 6-190
CHAPTER 6: DISCUSSION

Summary/Overview

This study examined pain and its relationship to psychological distress in middle-aged and older community-dwelling married couples. A preliminary sample of 5,386 couples was drawn from the 1998 wave of the Health and Retirement Study (HRS)—a national study of adults over age 50 and their spouses (regardless of age). Study Aim 1 sought to examine reports of pain in this initial sample of couples. Each spouse responded independently to a general HRS question about pain. The responses of spouse pairs were used to identify a focal sample of 423 “dual-pain” (DP) couples in which both spouses reported pain. To further address Aim 1, these DP couples were compared to the other couples (i.e., couples in which neither, or only one, spouse reported pain) in terms of background characteristics and the health and well-being of husbands and wives.

A conceptual model was developed to guide the study of pain and its relationship to psychological distress in these DP couples. Building on prior theoretical and empirical work (e.g., Williamson & Schulz, 1996), the model proposed that the pain-distress relationship was mediated by activity limitation. Tenets from models of the disablement process (e.g., Verbrugge & Jette, 1994), interdependence theory (Kelley & Thibaut, 1978), and interpersonal theories of emotion (e.g., Hatfield et al., 2002) were used to expand the model to consider these relationships within the marital dyad.

Capitalizing on the unique context provided by DP couples and the availability of comparable data from both spouses, research questions and hypotheses were developed and tested at both the intra-individual (i.e., within person) and the inter-individual (i.e., cross-spouse) level. Study Aim 2 concerned relationships at the intra-individual level
and sought to determine (a) which demographic and health-related characteristics were associated with reports of pain in these spouses; (b) the relationship between each spouse’s pain and his/her own psychological distress; (c) and whether this relationship was mediated by activity limitation. Study Aim 3 was concerned with relationships at the inter-individual level and investigated (a) the covariation between spouses’ reports of pain, activity limitation, and psychological distress; (b) the relationship between each spouse’s pain and the psychological distress of his/her partner; and (c) whether this relationship was mediated by the spouse’s activity limitation.

Key constructs were operationalized thusly: Pain was assessed as pain intensity and measured with a single-item verbal descriptor scale (rated from mild to severe); psychological distress was measured as depressive symptomatology and assessed using a multi-item scale (CES-D8); and activity limitation was specifically defined as physical limitations, which were measured as difficulties with basic physical abilities (e.g., lifting, stooping). Because of the dyadic nature of these data, dual-intercept multilevel models were used for all multivariate analyses. Wives’ and husbands’ data were modeled separately, but simultaneously. This also provided a mechanism to evaluate whether any intra- or inter-individual relationships varied by gender (Study Aim 4).

Except for hypotheses concerning variables—other than gender—associated with pain intensity (for which few significant predictors emerged), support was found for most study hypotheses at the intra-individual level. The expected relationships between pain intensity and depressive symptomatology were observed, and the pattern of findings generally supported the hypothesized mediating role of physical limitations. Findings at the inter-individual level were mixed. Husbands’ and wives’ reports of pain intensity,
depressive symptomatology, and physical limitations evidenced both similarities and differences. A significant cross-spouse effect was observed; however, this effect was gender-specific and depended on the pain intensity levels of each spouse. In addition, no evidence was found to suggest that spouses’ physical limitations mediated any cross-spouse effects of pain.

In this chapter, the major findings are reviewed and placed within the context of existing theory and prior empirical evidence. Competing explanations for several findings are offered and discussed. Where appropriate, key findings are tempered by specific study limitations and tied to precise directions for future research. Implications for social work practice, social work and health professional education, and institutional and public policy are reviewed. More general study limitations and strategic directions for future research and scholarship are outlined toward the end of this chapter. The chapter ends with an overall summary of the study’s strengths and contributions.

**Consideration of Major Findings**

*How Common Are Reports of Pain in Middle-Aged and Older Married Couples?*

A unique contribution of this study is that pain reports were examined at the couple level. In the preliminary sample of 5,386 HRS couples, a sizeable percentage of husbands (22%) and wives (28%) reported being “often troubled with pain.” These numbers are both higher than, and lower than, rates reported in prior studies.

Some studies of community-dwelling middle-aged and older adults have documented much higher rates of pain (e.g., 66% among those age 50 and older in Thomas et al., 2004; and 86% in those age 65 and over in Mobily et al., 1994). Lower
rates of pain have been reported in other studies (e.g., 20-40% in Crook et al., 1984; 33% of those age 65 and older in NHANES 1999-2000, as reported in IOM, 2011). According to one review, prevalence rates for pain among older adults in the community tend to fall between 25% and 50% (AGS Panel, 2002). However, rates depend largely on how questions are asked and how pain is defined by investigators. As noted in Chapter 2, rates tend to be higher when respondents are asked to report “all” or “any” pain versus specific types or locations of pain. With the notable exception of “musculoskeletal” pain (e.g., 73% in Scudds & Robertson, 2000), estimates tied to specific pain types or locations tend to be lower (e.g., 17% for abdominal pain in Von Korff et al., 1990).

The prevalence rates observed in this preliminary sample of couples appear most consistent with those reported in studies of chronic or persistent pain. As reviewed in Chapter 2, prevalence rates for chronic or persistent pain in community-dwelling adults vary widely, and rates are often not broken out by age. One early review by Verhaak & Kerssens (1998) found reported prevalence rates for chronic pain in adults of all ages ranged from 2% to 40% across 15 different epidemiological studies. The authors computed a median estimate of 15%. More recent studies have attempted to limit reports of chronic or persistent pain by asking about pain that has lasted for several months (generally 3 or 6 months). In these studies, prevalence rates for chronic pain in community-dwelling adults have ranged from 17% to 38% (see Chapter 2 for review). Studies using the 3-month criterion tend to yield higher estimates—for example, two population-based studies reported overall prevalence rates for chronic pain of 24% (Rustoen, 2004) and 35% (Mantyselka et al., 2003). When definitional criteria are more conservative or specific, estimated prevalence rates tend to decrease. For example, “pain
experienced every day for at least 3 months out of the prior 6 months” resulted in a
prevalence estimate of 18.5% (Blyth et al., 2001). As noted in Chapter 2, most studies
report increased prevalence of chronic pain with age—at least up to a certain point (see
review by Helme & Gibson, 1999). However, there is little consensus regarding whether
rates level off or decrease in later years and the age at which such transitions occur.

Although the HRS pain question does not directly assess the duration or
frequency of pain, the question wording (“often troubled with pain”) implies that the pain
is both recurrent and bothersome. A similarly-worded item has been used in several
often troubled with pain?,” but participants also had to report experiencing “any
noteworthy pain within the past 2 weeks” Overall prevalence estimates for “pain” was
11% for all adults; rates increased steadily with age from 13% in 41-50 year-olds to 29%
for those 71-80 years-old. In a footnote, the authors noted: “The word often was
emphasized in order to get at the more problematic and persistent complaints” (p. 301).

In a random sample of households in one province, 27% of adults endorsed
having “a troublesome pain in the past 2 weeks” on the Canadian Health Survey (e.g.,
Health Survey employed slightly different wording to ask respondents “Are you usually
free of pain and discomfort?” Using these data, Rashiq and Dick (2009) reported a crude
prevalence rate for “chronic non-cancer pain” of 14% for adults age 15 and over;
prevalence rates increased steadily with age, with estimates for middle-aged and older
adults ranging from 16% among those 45-54 years of age to 24% in those age 75 and
over. These figures are entirely consistent with the rates of pain reported here for HRS
husbands and wives.

The pain question in HRS does not appear to be simply measuring the occasional aches and pains that accompany common aging-related diseases, such as arthritis. This study found that a surprising number of husbands and wives who did not report being “often troubled by pain” item did report pain on follow-up questions about disease-specific pain. For example, 85% of wives with arthritis reported experiencing arthritis-specific pain; however, only 47% of wives who reported arthritis pain endorsed the “often troubled by pain” question.

It is possible that the prevalence rates for pain in the current study are unique to this sample of middle-age and older married couples. This is the first known study to examine general reports of pain among couples in a large, national sample of community-dwelling aging adults. Thus, there are no studies with which to directly compare these figures. It is notable, however, that prevalence figures from the present study closely resemble those reported for all respondents (regardless of marital status) in the 2000 wave of HRS—25% for men, 31% for women (Reyes-Gibby et al., 2007).

Because the general pain question was posed to each spouse in married couples, the 5,386 couples in the preliminary sample were categorized according to the pattern of responses given by spouse pairs. Forty-three percent, or roughly 4 out of 10 couples, were affected by pain in one or both spouses. Couples in which only the wife reported pain were more common (20% of couples) than couples in which only the husband reported pain (15% of couples). These proportions, along with the gender-specific prevalence rates in the preliminary HRS couples sample (reported above), are consistent with an abundance of studies documenting gender differences in reports of pain. As
documented in Chapter 2, women generally have higher prevalence rates for most disease conditions that are associated with pain (e.g., osteoarthritis, rheumatism), as well as conditions typically identified as chronic pain disorders (e.g., migraine headaches, fibromyalgia, orofacial pain; e.g., LeResche, 2011; Unruh, 1996). Studies have also found that women are more likely than men to report pain associated with specific disease conditions (e.g., arthritis; Felson, 1998). This gender difference is typically found in older samples as well (see Miller & Talerico, 2002). Reports of chronic pain in population-based samples also tend to be split along gender lines—e.g., 17% of males and 20% of females reported chronic pain in the Blyth et al. (2001) study cited above.

**Dual-Pain Couples: Who Are They and What Are Their Characteristics?**

This is the first known study to document the existence of a sizeable number of community-dwelling aging couples in which both spouses have problems with pain. These “Dual-Pain” couples comprised roughly 8% of married couples who participated in the 1998 HRS. Although the absolute size of this group (423 couples), along with their proportion relative to all couples, is relatively small, their numbers are compelling when extrapolated to the population level.

Existing research on pain in married couples has been limited to studies of chronic pain patients and their spouses, or couples in which only one spouse suffers from a painful health condition (e.g., arthritis, lupus). Researchers have largely ignored the possibility that patients’ spouses may also be experiencing pain. Most prior work has only hinted at the existence of “dual-pain” couples, with references made only hypothetically or in clinical anecdotes (Roy, 1994, 2001) or incidentally, by noting the
presence of pain among the spouses of identified “pain patients” (e.g., Cano, 2005; Sharp & Nicholas, 2000). As reviewed in Chapter 3, only a handful of studies have included information about the spouse’s pain in any substantive analyses. These studies have also been limited in terms of sample size and composition, with most using convenience samples of chronic pain patients and their spouses (e.g., Flor et al., 1987) or patients with specific disease conditions and their caregiving spouses (e.g., rheumatoid arthritis in Jacobi et al., 2003). In contrast, couples in the present study were identified within a large, nationally-representative survey of community-dwelling middle-aged and older adults. This context is important, because survey respondents were not selected on the basis of any pre-existing disease condition or the receipt of clinical services.

One aim of the present study was to describe these DP couples and compare them to the other couples in HRS. DP couples were generally comprised of middle-aged and older spouses who had been married for over three decades. Most were living in couple-only households in the community. DP couples had a median annual household income of just over $30,000, and their median assets totaled roughly $120,000. The average DP husband was 67 and his wife was 63. Most couples were of the same race (87% White) and ethnicity (97% non-Hispanic). Both spouses reported completing, on average, slightly less than a high school education. Post-secondary education was reported by only 31% of husbands and 28% of wives. On average, DP husbands and wives each reported 2 (of 7) doctor-diagnosed disease conditions, with arthritis, high blood pressure, and heart disease being most common. Both spouses tended to be overweight; notably, 33% of husbands and 38% of wives had BMI values in the obese range. The average DP husband and wife each reported a moderate number of physical limitations and a modest
number of depressive symptoms, although both spouses displayed considerable variability on these measures.

DP couples differed in some important ways from other couples in HRS. Compared to couples in which neither spouse reported pain, DP couples had lower household income and less accumulated wealth. DP husbands and wives were older, less educated, and in worse health than the spouses in couples in which neither had pain; DP spouses also endorsed greater depressive symptomatology. Although smaller in magnitude, many of these differences were observed between DP spouses and their same-gendered peers in couples in which only the husband, or the wife, reported pain.

The origin of these differences is unclear, as is the nature of the relationship between such differences and reports of pain. On one hand, the aforementioned differences could have contributed to the dual-pain status of these husbands and wives. For example, having lower SES may have uniquely disadvantaged these couples, perhaps by reducing their access to health care and/or the quality of the health care they have received over the years. Lower SES may also have limited the employment options of these spouses, relegating them to lower-paying jobs with intense physical demands. Low SES may also have resulted in lower levels of health literacy, compromising the ability of these couples to effectively advocate for, and utilize, needed care. As a result of these and other related mechanisms, these husbands and wives may have been at increased risk for developing pain in old age.

On the other hand, it is possible that the reduced health and lower SES of these couples are a consequence of pre-existing pain. Problems with pain earlier in life may have led to gaps or reductions in employment. Chronic health problems (including pain)
may even have forced these individuals to leave the workforce prematurely. The employment status of one or both spouses may also have limited the availability of health insurance and preventive care for one or both partners, further contributing to the poor health of the spouses. Bouts of illness or uncontrolled pain may also have depleted any accumulated savings of the couple. Maybe as a result of ongoing problems with pain, these couples carry poorer health and financial instability into their later years.

Observed differences in SES and health between DP spouses and those in couples in which neither spouse reported pain could be due to differences in other characteristics. Considering that both DP husbands and wives were, on average, significantly older than their peers in couples not reporting pain, increased age might account for the lower levels of education, income, wealth, and health observed in DP spouses. Note, however, that cohort differences are inextricably entwined with age in HRS. Accordingly, cohort differences in factors such as SES could also contribute to observed differences in health.

It must be acknowledged that the a priori contrasts used in the present study did not specifically compare all husbands (or wives) with pain to all husbands (or wives) without pain. Thus, caution is warranted in attributing observed differences directly to pain. On the other hand, findings from the present study are in-line with other studies that have compared the characteristics of persons with pain to those of persons without pain. For example, analyses of data from the 2004 wave of HRS found that participants reporting moderate or severe pain differed significantly from those without such pain on many of these same variables (e.g., education level, comorbid conditions, depressive symptoms; Covinsky et al., 2009). Nonetheless, future studies may wish to utilize post-hoc comparisons or create different groups of husbands and wives in order to make the
basis of such comparisons less ambiguous.

The present study is limited in its ability to address many of the competing explanations offered for the differences between DP couples and other couples in HRS. Future studies might provide additional insights by re-examining group differences while controlling for relevant covariates. For example, in order to rule out possible age and cohort-based explanations, comparisons should control for age differences between groups. Longitudinal research—especially that which documents the onset and course of pain in aging spouses—is also needed to clarify the nature of the associations between the pain status of these couples and various demographic and health-related factors.

*How Are Husbands and Wives in Dual-Pain Couples Different and Similar?*

The present study sought to examine potential differences and similarities between husbands and wives in DP couples with respect to key study constructs. These examinations helped to address portions of Study Aim 4 (to evaluate gender differences) and Study Aim 3 (to investigate covariation). Results revealed both differences and similarities between DP husbands and wives—not only on pain intensity, but on other key study variables as well.

**Gender Differences**

Support was found for hypothesized gender differences in all three key study variables—pain intensity, physical limitations, and depressive symptomatology.

*Pain Intensity*

On average, DP wives reported slightly higher levels of pain intensity than their husbands, with the average wife reporting more moderate pain and the average husband
reporting more mild pain. This finding is generally consistent with the bulk of existing research in this area. On the whole, women have been found to report higher levels of pain intensity than men (Fillingim et al., 2009; Unruh, 1996). This gender difference has been observed in several large-scale studies of community-dwelling adults (e.g., NCHS, 2006; Sammons, 2000; Unruh et al., 1999). Most studies examining specific types of pain have also found that, compared to men, women report greater pain intensity in musculoskeletal pain (Anderson et al., 1993; Bingefors & Isacson, 2004), migraine headache (Hasvold & Johnsen, 1993), and cancer pain (Green et al., 2011).

As reported in Chapter 2, evidence of gender differences in pain intensity has been more equivocal in two areas: (1) studies of pain in older adults; and (2) studies of persons with chronic pain. Although some large-scale studies of community-dwelling older adults have found that women report greater pain intensity than men (e.g., Scudds & Ostbye, 2001), others have not (e.g., Bradbeer, Helme, Yong, Kendig, & Gibson, 2003). Miller & Talerico (2002) concluded that inconsistency in the measurement of pain intensity across studies of older adults made drawing any formal conclusions about gender differences suspect. Findings regarding gender differences in pain intensity among those with chronic pain have also varied, with some studies finding the consistent pattern (e.g., Munce & Stewart, 2007), some finding no gender difference (e.g., Robinson, Wise, Riley, & Atchison, 1998), and others finding greater pain intensity in males (e.g., Koegh, McCracken, & Ecclestone, 2005).

A recently-published study by Ruau, Liu, Clark, Angst, and Butte (2012) provided what is arguably the most compelling evidence to date in support of women reporting greater pain intensity than men. These authors compared the pain intensity
scores of over 11,000 men and women recorded during hospital and clinic visits at a large academic medical center on the West coast. Women reported higher average pain intensity than men across the majority of primary diagnostic categories. In fact, when researchers restricted comparisons to the 22 diagnoses with a minimum of 69 visits per gender, women reported greater pain intensity in 21 (95%) of these categories. Gender differences were observed for both acute conditions (e.g., strains/sprains, respiratory infections) and chronic diseases (e.g., diabetes, rheumatoid arthritis, osteoarthritis, liver disease). The authors also noted that most gender differences persisted across adults in different age categories. Effect sizes across the most common diagnostic categories ranged from .21 to .51 (i.e., small to medium).

A host of biological, psychological, and social factors have been hypothesized to explain observed gender differences in pain prevalence and pain perception (Berkley, 1997, 2000; Keefe et al., 2000; Robinson, Riley, & Myers, 2000). Studies have increasingly identified various genetic and hormonal factors that seem to play a role in sensitivity to pain (Greenspan et al., 2007). However, despite an increase in research on gender differences in pain and pain intensity during the past two decades, no clear consensus has emerged. Most experts agree that more study is still needed (Fillingim et al., 2009; LeResche, 2011).

Although the present study cannot address the origins of the observed differences in pain intensity by gender, it contributes a potentially valuable piece of evidence to the debate. In prior studies of pain in middle aged and older adults, gender has often been confounded by differences in marital status and/or living arrangements (e.g., Mobily et al., 1994; NCHS, 2006). The demographic trends associated with population aging
indicate that women tend to dominate the ranks of the unmarried (e.g., widowed, divorced, never married) and those living alone (Lagulia, 1998). Being unmarried (e.g., Crook et al., 1984; Eriksen et al., 2003) and living alone (Jordan et al., 2007) have also been associated with reports of greater pain intensity, making it difficult to determine the extent to which observed differences in pain intensity can be reasonably attributed to gender. For example, Bradbeer et al. (2003) demonstrated that gender differences in community-dwelling older adults’ reports of pain (and in ratings of moderate to severe pain) disappeared once marital status and living arrangement were controlled. Findings from the present study suggest that gender differences may still remain, even when other characteristics are controlled. By selecting respondents who were married (to each other) and living together, the current study successfully eliminated these potential confounds. The nature of the sample also ensured equally-sized groups for comparison.

As reported in Chapter 2, studies of married individuals with chronic pain or a chronically-painful condition (e.g., arthritis) have generally not found evidence of a gender difference in pain intensity. To date, only the study by Keefe et al. (2000) found a significant gender difference in the ratings of pain intensity of married individuals. In that study, married women with knee osteoarthritis rated their pain intensity significantly higher than did married men. The present study is the first known study to document a gender difference in the pain intensity ratings of spouses in community-dwelling, aging couples.

Physical Limitations

As hypothesized, DP wives reported significantly more physical limitations than their husbands. It is somewhat challenging to compare this finding to those of prior
studies because of variation in the conceptualization, definition, and measurement of physical limitations. Similar to HRS, the majority of large-scale studies rely on self-reported difficulties or limitations. However, there is a general lack of comparability in terms of the specific items/tasks included, the wording of items, the number and types of response categories presented, and the categorization schemes used to define limitation or disability based on responses. Comparisons are also complicated by inconsistencies in nomenclature. Many of the same physical difficulty questions used in HRS have been labeled “lower body difficulties” (e.g., Clark et al., 1998), “mobility difficulties” (e.g., Ieozzini et al., 2001), “physical disability” (e.g., Murtagh & Hubert, 2004), and “functional limitations—[including] mobility limitations and range of motion limitations” (Merrill, Seeman, Kasl, & Berkman, 1997). As related in Chapter 2, it is also common to encounter various different types of difficulties grouped together under the rubric “functional limitations” or “functional disabilities.”

Variations in item composition and nomenclature aside, the majority of studies of community-dwelling middle-aged and older adults have documented greater physical limitations among women as compared to men (e.g., Ieozzini et al., 2001; Liang et al., 2008; Murtaugh & Hubert, 2004; Oman et al., 1999). The current findings are also consistent with those from studies limited to individuals with pain. Ample evidence suggests that among chronic pain patients, women typically report greater levels of physical impairment (LeResche, 2000). Studies of older persons with pain living in the community also find that reports of activity limitation (e.g., mobility, daily tasks and activities) tend to be higher in women than in men (e.g., Scudds & Ostbye, 2001). In the study of married individuals with knee pain by Keefe, Lefebre et al. (2000), women
reported higher levels of disability (measured as a broad composite, including difficulties with ADLs, mobility, dexterity, and completing common household tasks) than the men.

Some have suggested that the gender difference in physical limitations in mid and later life can be explained by underlying differences in the prevalence in disabling health conditions (Louie & Ward, 2010). Although some evidence exists to support this contention (e.g., Murtaugh & Hubert, 2004), it is unlikely to fully account for all observed differences (Leveille, Resnick, & Balfour, 2000). Additionally, some empirical findings appear to dispute the underlying differences in prevalence argument. For example, several studies have shown that, even among men and women suffering from the same disease condition (e.g., arthritis), women report greater levels of physical disablement (see review by Greenspan et al., 2007).

Is it possible that the observed gender difference in physical limitations is due to differences between men and women in their willingness and/or inclination to report health-related difficulties? Or perhaps differences reflect a gender bias in reporting, such that women tend to exaggerate or over-report their physical limitations or difficulties? To date, numerous studies have compared self-reports of physical limitations to performance on objective tests of physical function. The findings of most studies suggest that observed differences in self-reports tend to mirror differences in physical functioning—i.e., women perform worse than men. Studies have also found that, on the whole, self-reports of physical and functional limitations are fairly accurate (compared to the criterion of observed performance). For example, Sainio et al. (2006) compared self-reported difficulty climbing a flight of stairs to performance on a stair-climbing apparatus in a sample of community-dwelling adults aged 55 and over. Overall, a greater
proportion of women reported stair-climbing difficulty than did men, but the women also performed more poorly than men on the objective test. In addition, the gender difference in both self-reported and observed performance widened with increasing age. When the authors compared self-reports of stair-climbing difficulty to assessed performance, disagreement was observed in only 17% of cases, and over-reporting was far less common than under-reporting of difficulty. Some systematic patterns were observed in the direction of disagreement: Over-reporting was more common among women and older persons, while younger persons and men tended to under-report difficulty.

The slight tendency for women to over-report and men to under-report physical limitations has also been observed in other studies, including a study limited to adults over age 70 (Merrill, Seeman, Kasl, & Berkman, 1997). Merrill et al. also found that physical performance was able to account for much, but not all, of the gender difference in self-reported limitations. Thus, findings from prior studies suggest that the majority of self-reported physical limitations are accurate, and that a large portion of the gender difference in reporting of limitations reflects actual differences in performance. Evidence also suggests a slight difference in the perception and reporting of difficulty—with women perhaps more attuned to, and willing to, report physical difficulties than men. A variety of other explanations have been offered to explain gender differences in physical functioning, including lower muscle strength and greater age-associated loss of muscle, higher BMI, and lower levels of activity among women (Leveille et al., 2000). To date, however, no single explanation has gained unanimous support.

Depressive Symptomatology

Consistent with the bulk of existing research, DP wives endorsed a greater
number of depressive symptoms than did their husbands. Epidemiological studies have generally found that women suffer from depressive disorders (including dysthymia, minor depression, and major depression) at roughly twice the rate of men (Kessler, 2003). As reported in Chapter 2, numerous studies of community-dwelling middle-aged and older adults document this gender difference in levels of depressive symptomatology (see Beekman et al., 1999 for review). Studies using HRS data have consistently found greater depressive symptomatology in women, as compared to men (e.g., Steffick et al., 2000). This pattern has also been observed among individuals with chronically-painful conditions (e.g., Munce & Stewart, 2007), as well as in studies comprised of married individuals (e.g., Mirowsky & Ross, 1989).

Unlike other key variables in this study, several prior studies have examined and compared the depressive symptomatology levels of husbands and wives within later life couples. For example, wives reported significantly higher levels of depressive symptomatology than husbands in samples of 3,700 midlife couples and 1,600 older adult couples (Townsend et al., 2001). Not all studies document gender differences, however. Differences were not found between husbands’ and wives’ scores on the Geriatric Depression Scale in a small sample ($N = 144$) of community-dwelling adults in Florida (e.g., Robb, Small, and Haley, 2008). And, in one of the only studies to compare the depressive symptomatology levels of married (though not to each other) chronic pain patients, Cano, Weisberg, et al. (2000) also found no evidence of gender differences. Nonetheless, the findings from the present study are consistent with those from other large-scale studies of older, community-dwelling married couples here in the U.S. (e.g., Bookwala & Schulz, 1996) and abroad (e.g., in Australia in Hoppman et al., 2008).
That women report more depressive symptomatology than men is one of the most reliable findings in all of social science research (Nolen-Hoeksema, 1990). A growing body of evidence suggests that this gender difference emerges relatively early in life, perhaps even in adolescence (e.g., Hankin & Abramson, 2001; Nolen-Hoeksema et al., 1994). Although some research suggests that the gender difference in depressive symptomatology may be reduced or disappear in advanced old age (e.g., above age 70; Barefoot et al., 2001), the majority of existing research documents higher levels of depressive symptomatology in women across the lifespan (e.g., Nolen-Hoeksema, 2001).

Theories have been developed around many putative causes of depression—biological, psychological, and social. Most attempt to account for the development of greater depressive symptomatology and depressive disorders in women, as compared to men. At present, evidence exists to support selected elements of each of these major processes (Nolen-Hoeksema, 2001; Hyde, Mezulis, & Abramson, 2008). The most tenable theories emphasize the intersection of multiple causal processes—for example, the presence of vulnerabilities (e.g., biological, neurochemical, psychological, cognitive) that are influenced by a variety of external forces and experiences throughout the life course (e.g., Hyde et al., 2008). As yet, however, no existing theory has been able to fully explain the observed gender differences (Nolen-Hoeksema, 2001).

Interestingly, gender disparities in depressive symptomatology and disorders have been observed in most cultures, despite broad variation in absolute prevalence rates across countries (e.g., Maier et al., 1999; Zunzunegui et al., 2007). Moreover, when these studies have controlled for marital/partnership status, living arrangements, SES, age, and other factors, gender differences in rates of depressive disorders are only
modestly reduced, suggesting that socio-cultural factors cannot fully account for the increased burden of depressive symptomatology among women. Results in the present study are in keeping with such findings: Wives evidenced greater depressive symptomatology than husbands, despite sharing many established socio-cultural risk factors.

Ultimately, it is likely that the higher levels of depressive symptomatology observed among the DP wives in this study result from a confluence of factors, as they do for women in general. Although findings from the present study may be consonant with several different proposed explanatory processes, they are unlikely to provide support favoring any specific explanation. In-depth, longitudinal studies will be required to make substantive contributions to this debate.

*Other Characteristics*

Gender differences in an unexpected direction were also observed in the present study. Most notably, DP husbands reported a greater number of disease conditions than did DP wives. This is at odds with much existing research showing that women report a greater number of disease conditions than men (e.g., McDonough & Walters, 2001; Murtaugh & Hubert, 2004; Wolff, Starfield, & Anderson, 2002). Some studies, however, have found no difference in the number of disease conditions reported by men and women (e.g., Macintyre et al., 1996; Soldo et al., 2006). Variation in study findings may be at least partially attributable to the number and types of conditions that are queried in particular studies. For example, if the list of conditions is long and includes conditions of a less serious nature (e.g., allergies, skin disorders), as well as more general symptoms (e.g., migraines, hemorrhoids), women will generally report more chronic conditions than
men (Verbrugge, 1984). Overall, studies suggest that women tend to report more nonfatal chronic conditions (e.g. musculoskeletal conditions, digestive disorders), whereas men report more fatal chronic conditions (e.g., cardiovascular disease, emphysema; Verbrugge, 1989). The HRS interview included only seven disease conditions, the balance of which appears more heavily weighted toward the latter.

Prior findings regarding gender and the number of chronic conditions also appear to depend on the age of the population under study. Prevalence rates for most of the chronic conditions assessed in HRS have been shown to increase with age (e.g., Melzer et al., 2005; Verbrugge, 1984; 1989). However, comparisons of males and females within specific age ranges often yield conflicting findings. One study found that among those in their 60s, women report fewer conditions than men; among those in their 70s and early 80s, women report only slightly more conditions than men; and among those over age 85, women report substantially more conditions than men (e.g., Arber & Cooper, 1999). It is thus possible that the age difference observed between DP husbands and wives in the present study contributed to finding a greater number of chronic conditions in DP husbands. Alternatively, by controlling for a variety of socio-demographic factors (e.g., SES, marital status) via the selection of married couples in which both reported pain, these analyses may have uncovered an instance of what Verbrugge (1989) labeled the “male health disadvantage” (p. 295). That is, the heightened vulnerability of males is evident only when gender differences on other factors (e.g., role statuses, SES, age, etc.) are controlled.

In this study, DP husbands had a higher prevalence of heart disease and stroke, whereas DP wives reported more arthritis. These findings are consistent with long-
standing trends observed among aging adults, gleaned from analyses of data from HRS (Fisher et al., 2005) and other large-scale studies in the U. S. (e.g., CDC/NCHS, 2002; 2011). The findings are also consistent with the results of studies in Canada (e.g., Rochon et al., 2011; Walters et al., 2002). Although gender differences in rates of hypertension (women higher) and cancer (men higher) have also been reported in some studies (e.g., FIFARS, 2010), such differences were not observed in this study.

*Putting Gender Differences in Context*

In addition to considering the construct-specific explanations reviewed above, these findings should also be interpreted in light of more general explanations that have been offered to account for observed gender differences in health. These more general accounts may be especially relevant, given that the gender differences observed within DP couples are largely consistent with the findings of prior studies, independent of variations in sample composition.

Verbrugge (1985) grouped hypotheses regarding gender differences in health and health care into five categories: (1) biological risks, including genetic, physiological, and hormonal factors; (2) acquired risks that arise from work and social roles; (3) psychosocial factors relating to awareness of physical health issues, as well as the willingness and ability to pursue treatment; (4) health reporting behaviors, including willingness, but also other aspects of reporting (e.g., memory, language, and level of detail); and (5) health care history and experiences, including previous interactions with providers. The first two categories overlap a great deal with several of the hypotheses suggested to account for gender differences in depressive symptomatology. The latter categories offer some novel constructs for consideration. In reviewing the available
evidence, Verbrugge concluded that differences in acquired risks appeared to offer the
best account of gender differences in health and health care, followed by psychosocial
factors, and then prior health care. In general, women tend to have more frequent,
disabling (but not life-threatening) illnesses, while men typically suffer from more
serious (and often fatal) health conditions. Psychosocial factors appear to make women
more attentive to physical health changes and symptoms and propel them to seek initial
care sooner (for non-fatal, chronic conditions only) and to pursue more extensive and
prolonged care. Finally, more frequent and ongoing medical contacts may contribute to
earlier diagnosis, reduced illness severity, and ultimately reduced mortality in women
(Verbrugge, 1985).

The extent to which the gender differences observed between DP spouses can be
attributed to differences in these various mechanisms is unknown. This study was not
designed to address a research question of this scope, nor was HRS. It is, however, worth
reflecting briefly on how some of these latter mechanisms might operate in DP spouses.
For example, DP wives may report greater pain, physical limitations, and depressive
symptomatology because they are more attuned to physical symptoms. Moreover,
attending to these symptoms may actually convey (socio-biologically speaking) a
survival advantage to women, or at least increase the chances of having a health care
professional attend to their concerns.

Recall that several potential confounding factors were controlled by the design of
the present study, including marital status, shared living environment, economic well-
being, and the presence of pain. Yet, gender differences were observed across all key
study variables. This suggests that the gender differences observed in the pain intensity,
physical limitations, and depressive symptomatology levels of DP spouses are unlikely to be attributable to inadequate control of these known confounds. Note that study findings regarding relationships among pain intensity, physical limitations, and depressive symptomatology—both at the intra- and inter-individual level—have not yet been considered. Accordingly, it is possible that findings regarding these relationships could contribute valuable information to this debate.

Although differences between DP husbands and wives on key study variables (i.e., pain, physical limitations, and depressive symptomatology) were consistent with the bulk of prior empirical findings and also with proposed hypotheses, most differences were of fairly small magnitude. The largest effect size was observed on the measure of physical limitations ($d = .34$); yet, even this would be considered in the “small” range, according to Cohen’s guidelines (1977). So, although statistically significant, the observed differences may be of limited practical significance. The existence of such differences, however, provided ample justification for exploring whether the relationships between or among these variables might differ by gender (Study Aims #2 and #4). Key multivariate findings will be reviewed after first considering those with respect to the covariation of key study variables between spouses.

**Similarities/Cross-Spouse Covariation (Paths 1, 2, and 3)**

Despite the existence of mean (and proportional) differences by gender, DP husbands and wives also displayed significant amounts of shared variability on most key study variables. These findings provided support for the three hypotheses under Research Question 6 concerning the existence of Paths 1, 2, and 3 in the proposed conceptual model (Figure 5). Effect sizes for these estimates of covariation (adjusted for
gender differences) were generally in the small to moderate range.

Pain Intensity, Depressive Symptomatology, and Physical Limitations

Although the magnitude of the (adjusted) intraclass correlation for DP couples’ ratings of pain intensity was fairly small (.10), additional analyses showed that husbands and wives reported the same level of pain intensity in almost half (45%) of DP couples. Furthermore, few couples (< 5%) displayed extreme differences in pain intensity ratings (i.e., one spouse with severe, the other with mild pain). As this is the first known study of a large sample of community-dwelling aging couples in which both spouses report pain, these estimates cannot be directly compared to those in previous research.

As described in Chapter 3, a handful of prior studies have documented the existence of co-occurring pain within samples of community-dwelling couples. However, such couples have typically been recruited on the basis of one spouse—the “target” spouse—suffering from chronic musculoskeletal pain or a painful disease condition (e.g., rheumatoid arthritis). In these studies, estimates of pain among non-target spouses have ranged from 26% (Cano, 2003) to just under 50% (e.g., Jacobi et al., 2003). Estimates of the co-occurrence of pain in the spouses of persons with chronic pain have been slightly lower (e.g., 21% in Sharp & Nicolas, 2000). The findings from prior research, together with those from the current study, suggest that the phenomenon of co-occurring pain in aging spouses warrants further investigation. Future research should also specifically examine spousal similarity in ratings of pain intensity.

The estimated level of shared variability in the depressive symptomatology of DP spouses (.28) was largely consistent with the findings from prior research. Studies to date have generally reported moderate levels of similarity between spouses on most measures
of mental health. Correlation estimates for spouses on measures of psychological or emotional distress generally have ranged between .20 and .30 (e.g., Bookwala & Schulz, 1996; Butterworth & Rodgers, 2006; Galbaud du Fort et al. 1994; Pruchno, Wilson-Genderson, & Cartwright, 2009; Townsend et al., 2001). That the level of covariation in depressive symptom levels of DP spouses is toward the upper end of this range might reflect the long-standing nature of these relationships—e.g., 80% of DP couples had been married for more than 20 years.

With respect to key study variables, DP spouses shared the most variation in their reported physical limitations (.32). As related in Chapter 3, few studies have examined spousal covariation for the measure of physical limitations used in the present study. A limited number of studies have included several basic physical limitations items within a broad measure of functional difficulties or disability. In a sample of 1,700 couples drawn from an early wave of the AHEAD study, Hoppman et al. (2011) reported a small correlation (r = .08) between husbands’ and wives’ scores on a measure of functional limitations comprised of IADLs and a handful of the physical limitation items. Wilson (2001, 2002) created a “physical health index” that included all of the physical limitation items in HRS and basic ADLs. The correlations between the scores of spouses in over 4,000 couples from wave 1 of HRS ranged from .17 to .25, depending on the age range of the spouses and whether the measure was treated as an interval- or ordinal-level measure. It is possible that the level of covariation observed among DP couples was slightly higher than that seen in prior studies because of greater construct specificity in, and/or greater variability in scores derived from, the physical limitations measure.

Other Characteristics
Significant levels of shared variability were also observed in terms of the number of disease conditions reported by the spouses (.17). Patterns of multi-morbidity with respect to individual disease conditions also appeared to overlap substantially in DP husbands and wives. These finding are consistent with those of most prior studies. For example, a weighted index of chronic conditions was correlated at .21 between the spouses drawn from wave 1 of HRS (Wilson 2001, 2002). In some prior studies, couple-level concordance has been found for cancer, (e.g., Stimpson & Peek, 2005), arthritis (e.g., Stimpson & Peek, 2005), and hypertension (e.g., Hippisley-Cox et al., 2002; Peek & Markides, 2003).

DP spouses were similar in terms of body weight, as measured by BMI (.22). This level of similarity has been observed in other studies. For example, small, but significant, levels of covariation in BMI were observed in a panel of over 1,300 young and middle-aged couples in Canada; $r = .14$ at baseline (Katzmarzyk, Hebebrand, Bouchard, & Katzmarzyk, 2002), and $r = .17$ at 6-year follow-up (Katzmarzyk, Pe’russe, Rao, & Bouchard, 1999). A meta-analysis by DiCastelnuovo, Quarcquaruccio, Donati, de Gaetano, and Iacoviello (2008) concluded that significant (although fairly modest) levels of similarity exist between spouses for most cardiovascular risk factors—including hypertension, high cholesterol, and body weight/body fat, with overall correlation estimates ranging from .06 to .23.

**Explaining Observed Covariation**

In general, findings regarding covariation in DP spouses are in keeping with the conclusion reached by several systematic reviews: Spouses in older married couples tend to display considerable similarity in terms of their emotional and physical well-being.
A variety of different mechanisms have been proposed to explain the similarity observed between spouses across various constructs—most often mental or physical health. Most proposed mechanisms are theoretically based, but a few have developed in response to empirical observation. Since the majority of these mechanisms were discussed in Chapter 3, they are reviewed here only briefly.

Like most of the existing studies reporting on spousal concordance of physical and mental health, data limitations prevent any direct assessment of which mechanism(s) are most likely responsible for the observed similarities in DP spouses. These data were cross-sectional, and no comparative analyses were done with respect to marital duration. HRS contains few, if any, measures of the pre-marital health and well-being of respondents—information necessary to rule out competing explanations. Additionally, participating couples were not asked to provide a historical account of their marriage, and questions about current marital functioning and interactions are sparse. Consequently, a variety of competing theories and mechanisms could be applied to explain the similarities observed between DP husbands and wives on key study variables.

**Substantive mechanisms.** Proposed mechanisms fall into three basic categories: assortative mating, shared environment, and social and/or interpersonal interaction and interdependence. Assortative mating refers to the tendency of individuals to select a mate who has similar characteristics (Thiessen, 1999). In assortative mating, spousal similarity can result from direct selection (i.e., selection on the basis of similar characteristics), indirect selection (i.e., as a secondary consequence of selection based on other characteristics), and/or constraints on selection imposed by social forces (i.e., social
stratification, social homogamy; Lykken & Tellegen, 1993).

Married couples also share the same environment and, presumably, are exposed to many of the same risk factors for various physical and mental health disorders. Most couples share similar lifestyles, diet, and health behaviors and habits. They also share many of the same events over the life course. And, just as important, they generally face these events with shared financial, social, and institutional resources (e.g., Smith & Zick, 1994). Thus, shared risks, resources, and exposure to the same collection of life events constitute another major explanatory mechanism for spousal similarity.

The final category of explanatory mechanisms can be loosely assembled under theories of interpersonal/spousal interaction and/or the influence of the social context of marriage. As reviewed in Chapter 3, such theories formed the basis of several features of the proposed conceptual model guiding the present study, including the cross-spouse effects of pain and spousal similarity in key study constructs. Marriages typically involve considerable interdependence between partners, as spouses rely on each other to fulfill important needs (Kelley, 1981). This interdependence provides a foundation for many important interactions and interpersonal processes that occur within the marital relationship. Spouses comprise an integral part of married persons’ social networks, and these networks tend to become smaller with increased age, resulting in the potential for increased influence of spouses in later life (e.g., Baltes & Carstensen, 1998). One partner may even attempt to control the spouse’s health behaviors in an effort to keep him/her healthy (Lewis & Rook, 1999). Emotional or affective contagion (e.g., Coyne et al., 1987; Hatfield et al., 1992) has also been assumed to operate in spouse pairs.

Few studies of spousal similarity or concordance in mental health status identify a
specific theory a priori. Most appear to seek only post-hoc explanations to account for observed findings (Myler et al., 2007). Of studies that do reference theory (either a priori or post hoc), most tend to endorse interpersonal and/or interactional explanations and/or those involving shared risks and resources. Evidence from longitudinal studies appears to bolster such assertions. In general, studies have found that similarity in the mental health status (often measured as symptomatology) of spouses tends to increase over time (Butterworth & Rodgers, 2006; Tower & Kasl, 1996). This positive association between relationship duration and couple-level variation in mental health status was observed by Butterworth and Rodgers in one of the largest comparative cross-sectional analyses conducted to date ($N = 3,808$ couples). These authors found that the level of spouse similarity within relationships of relatively short duration (< 5 yrs.) could be largely explained by their similarity on other characteristics (e.g., age, education, physical health). However, a large proportion of the mental health similarity between spouses in longer-term relationships remained unexplained, even after controlling for those initial similarities. Together, these findings suggest that, although assortative mating may account for initial levels of covariation in the mental health symptomatology of spouses, the levels of covariation seen in more long-term spouses is more likely attributable to the impact of factors such as the shared experiences of, and/or interactions within, the couple (Butterworth & Rodgers, 2006). This kind of complex mechanism appears to provide very plausible account of the levels of covariation observed between DP spouses.

Within-couple similarity in the number of physical limitations, body weight (BMI), and the number and types of disease conditions might be explained by assortative mating. Similarity in background characteristics could be the result of, or even a by-
product of, selection effects. Alternatively, such similarity could reflect the effect of
social stratification on the availability of potential mates. However, these physical health
characteristics are also likely to be influenced by shared environment, health behaviors,
and lifestyle factors (e.g., environmental exposure, eating habits, and activity levels).
Moreover, similarity between DP husbands and wives on BMI and disease conditions
also may be at least partially attributable to the general level of similarity they display in
their demographic/background characteristics (e.g., .54 for education, .84 for age).

The observed similarity in the pain intensity ratings of DP spouses is more
difficult to explain. Several possibilities that have been mentioned in the literature were
reviewed in Chapter 3. For example, some chronic pain researchers have suggested that
persons with a family history of pain may seek out others with a similar history. Family
has also been discussed as a potential source of learned pain behavior (e.g., Fillingim et
al., 2000). Other scholars have offered that pain might be contagious (e.g., Morse &
Mitcham, 1997). Still others have suggested that symptoms of pain among the spouses of
chronic pain patients could represent expressions of emotional distress (e.g., Roy, 2001).
As reported in Chapter 3, these explanations are largely speculative and have amassed
little empirical support. And, although some potentially could account for spousal
concordance in reports of pain, they have limited ability to account for covariation in
ratings of pain intensity. It may be more reasonable to attribute similarity in pain
intensity to the shared health risks, behaviors, and resources of the couple.

It is possible that a mechanism unique to these couples underlies the levels of
similarity observed between the responses of DP husbands and wives. For instance,
because both spouses report being troubled with pain, they may also have similar levels
of physical limitations and depressive symptomatology. It is not clear, though, whether this would actually constitute a unique mechanism, specific to these couples. The general comparability of the present findings to those reported in studies of diverse samples of couples, along with the existence of significant gender differences that also fit with those found in other samples, appear to argue against the existence of a mechanism unique to these couples. Nevertheless, it would be interesting to compare the levels of similarity observed in DP couples to the levels seen in other couples, including those in which only one spouse reports pain and those in which neither spouse reports pain.

**Methodological influences.** In terms of alternative explanations that are not theoretically-based, the similarity observed between DP spouses could be (at least partially) attributable to methodological features of the study. Three possible methodological influences are mentioned below; most relate directly to features of the broader study from which these data were drawn (i.e., HRS).

**Contamination of responses.** Although the HRS interview protocol specified that each spouse was to be interviewed separately, it is possible that some respondents may have censored or otherwise altered their responses because their spouse was present, or within hearing distance, during the interview. Note, though, that this phenomenon would have had to occur in a sizeable number of households, and would have needed to affect the responses of both spouses in similar ways, in order to artificially inflate the level of shared variability observed in the responses of the DP spouse pairs.

**Selective attrition.** The present study utilized data from couples identified within one wave of an ongoing panel study. Consequently, selective attrition of respondents within the HRS panel could potentially contribute to increased spousal similarity in study
constructs. This seems especially likely with respect to constructs related to physical health. For instance, if one spouse experienced a significant decline in health prior to the 1998 survey, it may have limited the representation of couples in the sample in which spouses had more disparate levels of health and functioning. Such circumstances would also have reduced the likelihood of the couple meeting various criteria used to select the initial sample of eligible couples for the present study (e.g., requiring that the couple be residing together in the community at the time of the interview; exclusion of couples in which one, or both, spouses required a proxy interview).

It is also possible to think about selective attrition operating at an even broader level; accordingly, a more accurate label might be “selective marital survival (or attrition).” Empirical data suggest that declining health in one partner is a risk factor for relationship dissolution (e.g., Wilson & Waddoups, 2003). Thus, couples who survive into old age are likely to be relatively similar in terms of their general health and well-being. This argument does not imply that only the most healthy couples survive into old age, only that there may be differential rates of “survival” for marriages in which spouses are more or less congruent in health status. Differential rates of marital survival would certainly have influenced the eligibility of individual HRS respondents for the present study. It is unclear how selective survival/attrition (in marriage and/or within HRS) might specifically have affected reports of pain. The lack of prior study with dual-pain couples also makes it difficult to anticipate how such mechanisms might affect the composition and characteristics of samples of aging DP couples.

*Shared method bias.* DP husbands’ and wives’ responses may exhibit substantial covariation simply because they were assessed using the same data collection method
(e.g., self-report), mode (e.g., phone interview), or instrument (e.g., an 8-item measure with dichotomous response options). However, if shared method bias were the fundamental cause of the covariation observed between DP spouses, one might expect the observed ICCs to be of relatively equal strength across all constructs (they are not).

**Evaluating alternative explanations.** Several of the observations mentioned above suggest that, although methodological factors may have contributed to some level of shared variability in the responses of DP spouse pairs, they are not likely to be the primary explanatory mechanism. However, sorting out the relative contributions of various theoretically- and methodologically-based mechanisms would require prospective studies of DP couples. Ideally, studies would examine couple-level variability on key constructs early in the relationship and at specified intervals over a long period of time. Detailed assessments of each partner’s family history, as well as his/her own health history, would be necessary to better evaluate the role of assortative mating in couple-level similarity. Evaluation of social and marital interaction explanations would require frequent, in-depth measurement of couple-level interactions and marital functioning. It should be noted that some research teams have pioneered the design of observational studies and subsequent micro-analysis of the interactions of couples in the context of pain (e.g., Cano, Barterian, & Heller, 2008; Newton-John & Williams, 2006). To date, such methods have not been directed with intention toward DP couples. Future studies might also benefit from investigating covariation between DP spouses across a more extensive range of data collection methods and measures—for example, tests of physical performance (e.g., timed get-up-and-go test, gait speed), the use of compensatory devices and strategies (i.e., use of canes, adaptive aides), and multidimensional measures of pain.
Regardless of the precise underlying mechanism(s), the significant levels of covariation observed between DP spouses—especially on key study constructs—reinforce the need to analyze data from married couples using techniques appropriate for nested data. In recognition of overlapping husband-wife variability on key endogenous variables (e.g., pain intensity, physical limitations, and depressive symptoms), the present study utilized dual-intercept multilevel models in all multivariate analyses. These models allowed data from DP spouses to be analyzed simultaneously, while also providing the ability to test for gender differences. Findings from this study further strengthen recommendations regarding the use of statistical procedures that take into account the non-independence of spousal data.

*Lack of Predictors of Pain Intensity*

Few background or health-related characteristics emerged as significant predictors of pain intensity in DP husbands and wives (part of Study Aim 2). The majority of hypothesized relationships involving background and health-related characteristics and pain intensity were not supported in this study. Increased age, minority race/ethnicity, and lower SES were all expected to be associated with greater pain intensity in DP husbands and wives. Yet, none of the individual- or couple-level background characteristics in this study were significant predictors of pain intensity in a multivariate context. Together, the six background characteristics (including another couple-level variable, others residing in the household) explained only 2% of the variance in husbands’ pain intensity ratings and only 3% of the variance in wives’ ratings.

Only disease conditions (as a count and as individual conditions) evidenced a consistent relationship with pain intensity in both bivariate and multivariate analyses.
When analyzed as a total count, each additional condition was associated with a small, but statistically significant increase in pain intensity in both DP husbands and wives. In an alternate model substituting individual disease conditions for the count, only arthritis and lung disease were found to independently predict higher pain intensity in both husbands and wives. Heart disease was also associated with greater pain intensity, but only for husbands.

The general lack of findings with respect to the background and health-related characteristics related to pain intensity could provide valuable information about DP couples. Perhaps the pain intensity of the spouses in dual-pain couples is unique. Since these couples have not been previously studied, more focused research is needed to confirm or dispute these findings.

A variety of alternative explanations for the lack of findings could be offered, only a few of which are discussed here. One possibility is that some background variables were rendered nonsignificant once other variables were added in multivariate analyses. Selected measures of SES (education, household wealth) displayed significant bivariate correlations with the pain intensity ratings of DP husbands and wives. For example, lower pain intensity levels were observed in DP husbands who reported higher levels of education, as well as those with greater household wealth. Yet, no SES variables were independently related to pain intensity when (a) they were entered together in a multivariate model (for wives), and/or (b) disease conditions were added to a multivariate model (for husbands). The present study employed considerably more covariates than have been included in many prior studies. Thus, the statistical controls provided through multivariate analyses may legitimately explain the lack of significance.
of some SES variables in predicting pain intensity. This pattern of findings could suggest that observed bivariate relationships were spurious in nature. Alternatively, it might suggest pathways of indirect influence: For example, reduced SES affects pain intensity via increased risk for specific, or a greater number of, disease conditions. This latter possibility seems more likely, given that numerous prior studies have linked SES to pain intensity (e.g., Avis et al., 2003; Eachus et al., 1999; Krueger & Stone, 2008)

Increased statistical controls in multivariate analyses are unlikely to explain the absence of expected relationships between pain intensity and several other background and health-related characteristics. Neither increased age nor minority (non-White) race was related to pain intensity in DP spouses at the bivariate or multivariate level. This contrasts with findings from several studies showing increased pain intensity among older adults (e.g., Jakobsson et al., 2003; Scudds & Ostbye, 2001) and those of minority race (e.g., Avis et al., 2003; Portenoy et al., 2004). Although evidence from prior studies has been more equivocal about the relationship between body weight and pain intensity, several studies (e.g., Heo et al., 2003; Hitt et al., 2007; Sach et al., 2007) have found that greater body weight (especially in the obese range) is linked to greater pain intensity, especially among women. Yet in the present study, BMI was not related to the pain intensity of either DP spouse in bivariate or multivariate analyses.

The lack of expected findings regarding age, race, and body weight could be linked to how these constructs were measured and/or analyzed. Perhaps these constructs were measured with greater error and/or operationalized in substantially differently ways than in prior studies. For example, BMI was calculated from the self-reported height and weight of HRS participants, rather than being measured more directly or calculated using
measurements obtained from physical examination. There may also have been
differences in how some variables were modeled in analyses. For instance, several prior
studies have categorized BMI and used those categories in analyses, rather than using
BMI as a continuous measure (e.g., Hitt et al., 2007).

In the present study, pain intensity was examined only in those respondents who
indicated “being often troubled by pain.” Thus, all findings regarding predictors of pain
intensity rating need to be viewed in this context. The average DP wife rated her pain as
“moderate.” Perhaps the pain intensity of DP husbands and wives is already located
within the upper levels of an underlying continuum of pain intensity. A limited range of
pain intensity may have attenuated relationships with background and health variables.
Or, perhaps the demographic and health characteristics identified as predictors in prior
studies are, in fact, unrelated to variability within the (already relatively high) levels of
pain intensity reported by DP husbands and wives. Further research is needed to better
identify predictors of pain intensity within dual-pain couples. Additional comparative
studies may also help to determine if the unique characteristics of DP couples (e.g.,
advanced age, lower SES, and poorer health) limit the extent to which their pain intensity
can be explained by factors identified in previous research.

Methodological features of the present study could also account for the lack of
significant predictors of pain intensity in DP spouses. For instance, pain intensity was
modeled as a continuous variable using statistical techniques that assume linearity in
relationship to predictor variables. This decision was justified on conceptual and
methodological grounds, and was consistent with prior empirical treatment of such
measures (see Chapter 4). However, alternative conceptualizations or statistical
modeling strategies may have been equally viable.

At a conceptual level, it is worth considering whether the anchors on the HRS pain intensity measure could be regarded as qualitatively distinct categories. Relatedly, perhaps DP husbands and wives are able to distinguish only two levels of intensity. It is further possible that the different categories or levels of pain intensity might be associated with different background and health-related characteristics. In short, modeling pain intensity as a continuous variable could have contributed to a lack of significant predictors.

Several other methodological features of the current study may help to explain the lack of significant predictors of pain intensity. By selecting only those couples in which both spouses reported pain, this study distinguished pain intensity from the presence of pain. In prior studies, especially those with community-dwelling adults (e.g., Hitt et al., 2007; Krueger & Stone, 2008), measures of pain intensity have often included a zero-point (i.e., no pain). Depending on how researchers analyze the resulting data, pain intensity can be confounded by pain presence. Consequently, the effects reported in prior studies for some demographic and health characteristics may have reflected differences between respondents with pain (at any level of intensity) and those without pain.

The age range of the sample was limited (by design) to those represented in HRS. Although HRS included spouses regardless of age, the majority of husbands and wives in DP couples fell within a twenty-five-year age range, from roughly the mid-50s to the late 70s. Some, though not all, studies limited to older adults have found no association between pain intensity and increased age (e.g., Lavsky-Shulan, Wallace, Kohout et al., 1985; Mobily et al., 1994). It is therefore possible that age is not related to pain intensity
within the range of ages represented in these DP couples.

The design of the present study controlled for both marital status and living with one’s spouse, thereby limiting any variability in pain intensity associated with these characteristics. Although in some studies (e.g., Bradbeer et al., 2003), controlling for these characteristics has been found to reduce differences in pain intensity by gender, there is limited evidence that these characteristics can account for the associations between pain intensity and other background variables. It seems plausible, however, that controlling for marital status and living with one’s spouse resulted in greater homogamy in the sample with respect to age, household income, and household wealth.

Finally, consistent with Study Aim 4, predictors of pain intensity were examined separately, but simultaneously, for DP husbands and wives. This analytic approach goes beyond simply demonstrating that gender is a significant predictor of pain intensity. It provides an opportunity to detect and model gender-specific relationships between other predictor variables and pain intensity—an area neglected in much prior research. Although the estimated effects of most predictors were observed to be of similar significance and strength in both DP husbands and wives, it is important to recognize that these effects were tested within gender-specific models. This has generally not been the case in most prior research. It is therefore possible that some of the design features of the current study—either alone, or in combination—accounted for the lack of significant predictors of pain intensity. This is an area ripe for future research.

The Predictive Value of Individual Disease Conditions

Analyses designed to address Exploratory Question 2 provided some valuable information about the conditions most strongly related to pain intensity in DP spouses.
Although several self-reported disease conditions were associated with greater pain intensity in bivariate analyses, only arthritis and lung disease (for both spouses) and heart disease (for husbands only) emerged as significant predictors in a multivariate context. Surprisingly, lung disease was the condition estimated to have the strongest effect on pain intensity—an effect size corresponding to one half of a standard deviation for husbands and one third of a standard deviation for wives. Existing research related to pain in lung disease is sparse, and most studies have examined only reports of pain or chronic pain, not pain intensity. A recent study in Norway found that 45% of patients diagnosed with chronic obstructive pulmonary disease reported pain, as compared to only 34% of adults from the general population (Bentsen, Rustoen, & Miaskowski, 2011). However, no difference was found in actual ratings of pain intensity. In a sample of community-dwelling adults in Australia, lung disease was one of the disease conditions that had the highest proportion of sufferers reporting the existence of chronic pain (41%), falling just behind osteoporosis (29%) and arthritis (45%; Dominick et al., 2012). However, lung disease was not independently associated with reports of chronic pain in multivariate models. In addition to arthritis, heart/lung disease was independently associated with higher pain ratings in the study by Kahana et al. (1997). However, because heart and lung diseases were combined into a single category, their results offer only qualified support for the findings in the present study.

Only the recent study by Ruau et al. (2012; see p. 12, this chapter) offers potentially comparable data, derived from the pain intensity ratings provided during the medical visits of over 11,000 patients. In their study, the average pain intensity ratings of patients with respiratory diseases as a primary diagnosis were very similar to those of
patients with osteoarthritis. And, although these pain ratings were not as high as those given by patients with rheumatoid arthritis or chronic liver disease, they were higher than those of patients presenting with acute conditions such as lower leg fracture and knee injury. Findings from the current study, along with those of Ruau et al. (2012), suggest the need for additional research on pain within the context of lung disease. Additionally, the lack of any significant association between diabetes and pain intensity in the present study is somewhat surprising, especially in light of data reported in Ruau et al.

Overall, results from this study suggest that, in terms of accounting for variability in the pain intensity ratings of DP spouses, incorporating information about the presence of individual disease conditions may be more advantageous than using a count of disease conditions—especially for husbands. The final model that included the number of disease conditions explained roughly 8% of the variance in husbands,’ and 10% of the variance in wives,’ pain intensity. However, when individual disease conditions were substituted for the number of conditions, the model explained roughly 12% of the variance in the pain intensity ratings of husbands and 11% of variance in the ratings of wives. Additionally, the estimated effect sizes of specific conditions (lung disease in both, arthritis in husbands) were generally larger than those associated with the count of disease conditions.

Findings regarding the explanatory benefits associated with individual disease conditions are consonant with findings from a recent study from Australia. Dominick, Blyth, and Nicholas (2012) found that six doctor-diagnosed, self-reported disease conditions were associated with reports of chronic pain. The odds associated with the conditions varied (adjusted odds ratios ranged from 1.4 for bowel disease to 3.9 for
arthritis), but each was independently linked to increased reports of chronic pain.

Although Dominick et al. (2012) examined reports of chronic pain (not pain intensity), their findings did highlight the benefit of examining individual disease conditions.

The Intra-Individual Relationship Between Pain Intensity and Depressive Symptomatology

Prior to reviewing study findings regarding the relationship between pain and depressive symptomatology at the couple (or inter-individual) level, it is important to first consider how these constructs were related at the intra-individual level (part of Study Aim 2). In short, support was found for each of the lettered pathways illustrated in the conceptual model that guided this study (i.e., Paths A, B, and C in Figure 5). The overall pattern of findings was also consistent with the activity limitation mediation model. Thus, support was found for all hypotheses under Research Questions 3 and 4.

**Pain Intensity Is Associated with Depressive Symptomatology (Path A)**

As hypothesized, greater pain intensity was associated with increased depressive symptomatology in both DP husbands and DP wives. This relationship was evident at the bivariate level and, more importantly, remained significant after controlling for relevant background and health-related characteristics. Although estimated effect sizes were in the small to moderate range, pain intensity was one of the strongest independent predictors of depressive symptom levels in both spouses. Pain intensity alone accounted for 2% of the variance in the depressive symptomatology reported by DP husbands, and a more sizeable amount of variation (7%) in the depressive symptomatology of DP wives.

As might be surmised from the above numbers, the strength of the (initial) direct relationship between pain intensity and depressive symptomatology did vary by gender:
After controlling for relevant background and health characteristics, pain intensity was more strongly related to depressive symptomatology in DP wives than in DP husbands. Moreover, the results of a formal test of statistical significance indicated that the size of the difference in the strength of Path A was unlikely to have occurred by chance. This finding jointly addressed Study Aims 2 and 4. Specifically, in answer to Exploratory Question 5a: Within DP couples, gender moderated the strength of the (initial) direct relationship between pain intensity and depressive symptomatology.

As discussed in Chapter 2, gender differences in the relationship between pain intensity and depressive symptomatology among community-dwelling aging persons have generally received little attention from researchers. Additionally, prior studies have varied widely in study populations and the measurement of key variables. Variations notwithstanding, gender differences in the pain-depressive symptom relationship have been documented in the majority of prior investigations. The relationship between pain intensity and depressive symptomatology has been observed to be stronger in women than in men within samples of chronic pain patients (e.g., Haley et al., 1985; Unruh, 1996), adults with pain living in the community (e.g., Munce & Stewart, 2007), and older adults receiving home care in the community (Onder et al., 2005).

Not all prior studies have found gender differences in the pain-depressive symptom relationship. For example, higher pain severity was associated with greater depressive symptomatology in both male and female frail older adults receiving home care (Landi et al., 2005). Similar findings have been reported in studies of chronic pain patients (e.g., Edwards et al., 2000; Keogh et al., 2006), as well as married (though not to each other) chronic pain patients (e.g., Cano et al., 2000). Some studies have even found
a stronger relationship between pain and depressive symptomatology in males, as compared to females. For example, in one of the previous mediation studies, Geerlings et al. (2002) found that the longitudinal relationships between levels of pain and depressive symptoms were stronger in men than in women in a sample of community-dwelling older adults. However, this difference was significant only at the highest level of pain. In addition, when Geerlings et al. ran alternate models predicting “depression” (using dichotomized CES-D scores), the gender difference was not statistically significant. Although a trend in this same direction was observed in another mediation study, the difference was reportedly not statistically significant (Parmelee et al., 1991).

As discussed in Chapter 2, differences in sample composition and the operationalization of both independent and dependent variables likely account for the differences in findings across studies with regard to gender and the association between pain and depressive symptomatology. It is also noteworthy, however, that findings from the current study more closely parallel those from studies comprised of individuals with chronic and/or long-lived pain. For example, Munce & Stewart (2007, cited above) was based on a sample of adults in the Canadian Community Health Survey who had suffered for more than six months from arthritis, migraine/ headaches, back problems, or other “chronic pain conditions.”

In this study, DP wives reported higher pain intensity than did DP husbands. It is therefore possible that pain intensity is more strongly related to symptoms of depression in DP wives simply because they experience greater pain intensity than their male partners. This suggestion has been offered by numerous scholars (e.g., Hoffman & Tarzian, 2008; Unruh et al., 1999). Unfortunately, reliance on self-report in pain research
would make it almost impossible to design an empirical study that could validate this proposed explanation.

The stronger pain-depressive symptom relationship observed in DP wives might indicate that their pain is less well assessed and/or treated that that of their husbands. Because data regarding pain treatment were not included in the present study, the extent to which DP wives’ pain was un- or under-treated is unknown. There is, however, substantial evidence that women, generally, have less well-treated pain than do men. In the case of joint replacement in severe arthritis, for example, women typically endure greater and more prolonged pain and functional decline before pursuing surgical treatment (e.g., Hawker et al., 2000; Holtzman, Saleh, & Kane, 2002). This difference is observed in spite of the fact that women are more likely to use health care services in general and to seek treatment for arthritis and other pain-related conditions (e.g., Verbrugge, 1989; Fillingim, 2000). The underlying reasons for observed gender differences in pain management are unclear. Although evidence suggests that women may actually be more aggressive in seeking treatment for pain (Fillingim, 2000), studies also show that some health professionals are more likely to dismiss or downplay the pain complaints of women (e.g., Hoffman & Tarzian, 2008; LeResche, 2011). It is also possible that available pain medications and treatments are less effective in women than in men (Greenspan et al., 2007).

Alternatively, women may be more negatively affected by pain intensity than men, and this may be true in DP couples, as well. There are many potential sources of such a difference. For example, the disparity may reflect a greater propensity for women to internalize stressors, in contrast to the externalizing tendencies of men (Meana, 1998).
Drawing on the stress and coping paradigms offered by Folkman and Lazarus (1984) and Pearlin et al. (1991), it is also possible that women and men differ along several key points in the stress and coping process. For example, women and men may differ in the expectancies they have about pain, how they cognitively evaluate or appraise it, the coping strategies they apply to deal with it, and the internal (e.g., self-efficacy, mastery) and external resources (e.g., social support, help-seeking) they bring to the experience. Although studies have examined gender differences in several of these behaviors and responses, there is little evidence that these factors can explain the gender difference in the pain-depression relationship (Fillingim et al., 2009; LeResche, 2011). Because these constructs were not included in the present study, their potential explanatory power with respect to the findings in DP spouses cannot be assessed.

In the present study, the search for a substantive explanation underlying the gender difference in the pain intensity-depressive symptomatology relationship must also be tempered by consideration of the distributional properties of husbands’ and wives’ responses on these measures. As a group, DP wives displayed greater variability in both pain intensity and depressive symptomatology than did DP husbands. Wives also had higher average scores on both measures. Accordingly, the increased strength in the pain intensity-depressive symptomatology relationship observed in DP wives, as compared to husbands, could be related to a combination of these two factors.

Gender differences notwithstanding, pain intensity was significantly and positively associated with depressive symptomatology in both DP husbands and wives. This finding is consistent with the results of most published studies, including research with community-dwelling older adults (Bair et al., 2003; Magni et al., 1990), patients
with painful disease conditions (e.g. Bookwala et al., 2003), and chronic pain patients (e.g., see review by Fishbain et al., 1997; also see Chapter 2). These findings also yielded empirical support for Path A and provided a basis for testing the proposed mediation model involving activity limitation (e.g., Williamson & Schulz, 1992a).

**Pain Intensity Is Associated with Physical Limitations (Path B)**

Following recommended procedures for evaluating mediation hypotheses (e.g., Baron & Kenny, 1986), a series of multivariate analyses established that pain intensity was a substantial predictor of physical limitations in both DP husbands and wives. Consistent with expectations and with previous research (e.g., Leveille et al., 2001; Moss, Lawton, & Glicksman, 1991; Williamson, 2000b), greater pain intensity was associated with more physical limitations. Pain intensity explained a sizeable amount of variance in the physical limitations of both spouses—roughly 6% in DP wives and 8% in DP husbands. These findings established the necessary evidence in support of Path B.

Although a significant relationship between pain intensity and physical limitations is an essential component of the proposed mediation pathway, this finding is also worthy of independent consideration. Increased pain intensity was a strong predictor of greater physical limitations, even after controlling for the effects of background characteristics and health-related variables such as the number of disease conditions. Pain often has been overlooked as an independent contributor to physical limitations. For example, in a review of known risk factors for mobility limitation in community-dwelling older adults (Yeom et al., 2008), pain was mentioned as a risk factor, but was discussed only as a symptom of chronic disease conditions. Moreover, the only evidence cited for this known risk was a publication of early results from the Women’s Health and Aging Study
(WHAS) in which knee pain was linked to mobility limitations within a sample of older women living in the community (Lamb, Guralnik et al., 2000). Numerous studies have neglected to consider pain, even as a covariate, in analyses that examined functional limitations (e.g., Davison et al., 2002; Merrill et al., 1997). Others have examined pain as a risk factor for functional decline only within the context of specific diseases (e.g., arthritis, heart disease; e.g., Stuck et al., 1999). In studies that have included pain as a risk factor for functional or physical difficulties, consideration has often been limited to certain types or locations of pain (e.g., pain in lower back or in lower limbs; Ettinger et al., 1994; Idler et al., 2000; Leveille et al., 2002; Melzer et al., 2005). Chapter 2 established that pain has received relatively scant attention within many existing theoretical models of physical disablement (e.g., Nagi, 1979; Verbrugge & Jette, 1994). Findings from this study suggest that studies of the disablement process would benefit from increased attention to pain, independent of its association with specific diseases.

It is not possible to determine whether the physical limitations reported in the present study resulted specifically from respondents’ pain. Muscle weakness, reduced endurance, and breathing difficulties might also have contributed to difficulties with tasks such as bending, lifting, or climbing stairs (e.g., Landers, Hunter, Wetzstein, Bammam, & Weinsier, 2001; Leveille et al., 2004; Whitson et al., 2009). It would be helpful in future research to examine the extent to which respondents attribute these limitations to pain and/or changes in pain intensity. Prospective studies may also help to more clearly establish the role of pain in the onset and progression of basic physical limitations.
Physical Limitations Are Associated with Depressive Symptomatology (Path C)

Physical limitations independently predicted depressive symptomatology in both DP husbands and wives. Although the effect of each individual physical limitation was estimated to be fairly small, the additive effect of multiple limitations was shown to be more substantial. Physical limitations explained roughly 5% of the variance in wives’ depressive symptomatology and 9% of the variance in the depressive symptom levels reported by husbands. These findings are consistent with those from several lines of prior research, including studies of persons with pain in the community (e.g., Magni et al., 1994) and in clinical samples (e.g., Smith et al., 1986). Considerably more evidence exists with respect to the relationship between activity limitation (a.k.a., functional or physical disability) and depressive symptomatology when activity limitation has been defined more broadly (e.g., including ADL and/or IADL difficulties). Numerous studies over the years have documented the relationship between functional disability and depressive symptomatology (e.g., Aneshensel, Frerichs, & Huba, 1984; Berkman et al., 1986; Geiselmann et al., 2001; Zeiss et al., 1996). This relationship has also been found in studies that have examined physical limitations specifically (e.g., Hays et al., 1998; Yang & George, 2005). The presence of mobility limitation (as indicated by a subset of items similar to those used in this study) has also been associated with depressive symptomatology in studies of both middle-aged and older community-dwelling adults (e.g., Ormel, et al., 2002; Melzer et al., 2005). A few studies also document significant associations between greater physical limitations and higher depressive symptomatology in both husbands and wives in samples of community-dwelling older married couples.
These findings provided support for Path C—a pathway necessary to establish the mediating effect of physical limitations in the relationship between pain and depressive symptomatology. Findings with respect to mediation are discussed next.

**Physical Limitations (at Least Partially) Mediate This Relationship (Path A’)**

**Gender Differences**

One central finding from this study is that the extent to which the relationship between pain intensity and depressive symptomatology was mediated by physical limitations differed by gender. This finding jointly addressed Study Aims 2 and 4, and specifically Exploratory Question 5b, which sought to evaluate gender as a potential moderator of the mediating effect of activity limitation. Among DP husbands, physical limitations were found to completely mediate the relationship between pain intensity and depressive symptomatology. However, physical limitations only partially mediated this relationship in DP wives. In other words, controlling for the relationships between pain intensity and physical limitations (Path B) and between physical limitations and depressive symptomatology (Path C), the initial relationship observed between pain intensity and depressive symptomatology was reduced to non-significance in DP husbands (Path A’ was effectively zero). In DP wives, however, although the initial relationship was significantly reduced in strength, pain intensity still had a significant direct effect on depressive symptomatology (i.e., Path A’ < Path A, but was still non-zero). This suggests that, in wives, at least part of the relationship between pain intensity and depressive symptomatology is independent of the association between physical
limitations and depressive symptomatology.

One obvious explanation for this pattern of results is that the fundamental relationship between pain intensity and depressive symptomatology may differ by gender. Perhaps in men, pain intensity increases depressive symptomatology solely through its effect on increased physical limitations. For women, in addition to its effect via increased physical limitations, pain intensity may also invoke depressive symptoms directly. This difference could also potentially account for the stronger relationship observed between pain intensity and depressive symptomatology among DP wives. It might also help to explain the plethora of gender differences found in various studies—for example, that comorbid chronic pain and clinical depression have been found more often in women than in men (Meana, 1998). Of course, this explanation is only viable if other competing explanations (e.g., both are simply more prevalent in women) could be ruled out; unfortunately, the current data are not adequate to perform such judgments.

Alternatively, the gender difference in the mediating effect of activity limitation in this study could reflect underlying differences in the value or importance that men and women ascribe to basic physical abilities. For instance, men may place greater value on maintaining independence in these basic tasks. For them, increased physical limitations may prompt feelings of dependency and negatively affect their sense of self-worth. Perhaps they also fear becoming a burden to others, including their wives. Men might be especially prone to these feelings in marital relationships that are characterized by more traditional gender roles. The 11 items comprising the physical limitations scale reflect more large-muscle abilities that may be of greater concern to males. In contrast, women may consider these limitations to be a normative part of the aging process and/or have
less difficulty asking for, and accepting, help. Women may place more value on other
types of abilities or tasks, such as the ability to engage in social activities. Perhaps
women are more negatively affected by activity limitation in these areas. It is also
possible that such limitations further mediate the pain-depressive symptomatology
relationship in women. All of these possibilities await testing in future studies.

As noted in Chapter 2, few activity limitation mediation studies have explicitly
examined gender differences in mediation. Numerous studies have examined gender
differences in reports of pain. A good number have also controlled for the effect of
gender on depressive symptomatology (e.g., Bookwala et al., 2003). A few studies have
even included gender as a covariate in the analysis of physical limitations (e.g., Kahana et
al., 1997). Valuable as these strategies are, however, they test only for a main effect of
gender, not a gender difference in the relationship(s) between variables. Such differences
can be assessed only through the use of gender-based interaction terms, separate group
analyses, or—in the case of dyadic data—dual-intercept models.

Only the study by Geerlings et al. (2002) attempted to explore gender differences
in mediation by activity limitation. Recall that Geerlings et al. (2002) found the
longitudinal relationship between levels of pain and depressive symptoms to be stronger
in men than in women, even after controlling for the effects of age and physical
limitations. However, this effect was significant only when pain symptoms reached high
levels, and the gender difference was not statistically significant when they ran a parallel
model predicting “probable depression” (using a dichotomized CES-D score). As
previously discussed, although findings from Geerlings et al. might tentatively suggest
that no gender difference exists in mediation by activity limitation, lack of a statistical
evaluation of the mediation effect, incomplete reporting of key relationships, and mixed findings depending on the coding of the outcome variable make it difficult to draw a firm conclusion from their data. It can be said, however, that no prior study has provided evidence of a gender difference in the mediation of the pain-depressive symptomatology relationship by activity limitation. Findings from this study regarding the moderating role of gender are thus provocative.

*Consistent Evidence of Partial Mediation*

Although a gender difference in mediation was found in DP couples, this difference was largely a matter of degree. The overall pattern of results provided compelling evidence of partial mediation—another central finding of this study. Results supported the activity mediation hypothesis articulated in Research Question 4c. Specifically, when the relationships between pain intensity and physical limitations (Path B) and between physical limitations and depressive symptomatology (Path C) were controlled, the initial relationship between pain intensity and depressive symptomatology was significantly reduced in both husbands and wives (i.e., Path $A' < Path A$). This pattern of results indicates that pain intensity affects depressive symptomatology in DP spouses, at least in part, because it contributes to more physical limitations. In turn, increased physical limitations contribute to greater depressive symptomatology.

These findings provide support for the proposed conceptual model. They also bolster support for Williamson’s activity limitation/restriction model of depressed affect: Activity limitation (measured as physical limitations) at least partially mediated the (intra-individual) pain-depressive symptomatology relationship in DP husbands and wives. These results closely mirror those of the five previous studies that reported partial
mediation, including those by Williamson and colleagues (Williamson & Schulz, 1992a; Williamson & Schulz, 1995) and others (Arnstein et al., 1999; Goodland, 2002; and Gureje et al., 2001). Likewise, because the current study found evidence of at least partial mediation in both spouses, these findings are also generally consistent with the three prior studies that reported total mediation (Rudy et al., 1988; Turk et al., 1995; and Williamson, 2000a). In sum, results from the current study are in agreement with the findings from the majority of previous studies that have examined activity limitation as a mediator of the relationship between pain and depressive symptomatology.

On the other hand, the current results are inconsistent with the five prior studies that found no evidence of mediation (Bookwala et al., 2003; Cohen-Mansfield & Marx, 1993; Geerlings et al., 2002; Kahana et al., 1997; and Parmelee et al., 1991). This inconsistency is especially germane because the sampling frame from which DP couples were drawn (i.e., the panel of HRS respondents) most closely resembles the samples studied by Geerlings and colleagues, Kahana and colleagues, and Bookwala and colleagues. Hence, the ensuing discussion largely focuses on plausible explanations for the difference in mediation results between the current study and these three large-scale studies of community-dwelling older adults. When relevant, the features and findings of the other previous mediation studies (as discussed in Chapter 2) are also considered.

The studies by Geerlings et al. (2002), Kahana et al. (1997), and Bookwala et al. (2003) all failed to find evidence that activity limitation mediated the association between pain and psychological distress. In all three studies, pain was significantly related to psychological distress, with higher levels of pain predicting greater distress in each sample. So, all three studies found evidence supporting Path A in the conceptual model.
In these studies, support for the mediating role of activity limitation was linked to a breakdown in either Path B or Path C (in fact, this was true of all studies in the no mediation group, with the exception of Cohen-Mansfield & Marx, 1993 in which neither Path B nor Path C was supported). In the Kahana et al. and Bookwala et al. studies, the relationship between activity limitation and psychological distress (Path C) was not statistically significant in multivariate models. In the Geerlings et al. study, both activity limitation and pain were significant predictors of psychological distress, but the addition of activity limitation did not appreciably reduce the association between pain and distress. Geerlings et al. did not report data regarding the strength of the relationship between pain and activity limitation (i.e., Path B).

**Shared Study and Sample Characteristics**

The current study shared several characteristics with these three studies. All but Bookwala et al. had samples drawn using random or stratified random sampling of a population of community-dwelling aging persons. Of course, the populations did vary somewhat: Sampling in HRS (from which the present sample was derived) was based on U.S. households with members over age 50; Geerlings et al. sampled adults age 55-85 in the Netherlands; and Kahana et al. sampled residents over age 71 from three different retirement communities in Florida. Participants in Bookwala et al. were all living in the community, but were volunteers recruited through arthritis clinics, primary care practices, and newspaper ads.

In all four studies, psychological distress was conceptualized as depressive symptomatology (Kahana et al. also included negative affect). Moreover, all used some version of the CES-D to measure depressive symptoms. Except for Geerlings et al. (who}
analyzed both logged and dichotomized CES-D scores), the studies analyzed CES-D scale scores as continuous, interval-like variables. Although the measures used to assess pain varied (an issue to be discussed shortly), all except Bookwala et al. (who examined only knee pain) used a fairly general definition of pain that (e.g., “pain from all sources over the past year” in Kahana et al.; “I am in pain [or have pain]…sitting, standing, walking…” in Geerlings et al.; and “often troubled with pain” in the present study). The samples in these four studies were similar in terms of overall size (Ns ranged from 367 in Bookwala et al. to 804 in Kahana et al.) and gender representation (ranging from 50% female in the present study to 66% female in Kahana et al.). Additionally, all four studies tested mediation within multivariate models that included several relevant background characteristics, including gender and (except for Bookwala et al.) age. In fact, three of the four studies (not Geerlings et al.) included a number of other covariates in most models, with all incorporating measures of race, SES, and disease burden. The inclusion of multiple covariates actually distinguishes all four studies from many studies in the partial and total mediation categories (Turk et. al., 1995; Williamson & Schulz, 1992a; Williamson & Schulz, 1995; Goodland, 2002), which often had few or no control variables in analyses testing mediation. This distinguishing characteristic does not, however, offer any explanation for the differences observed in mediation findings.

Differences in Study and Sample Characteristics

This study differed in some notable ways from the three most comparable studies in the no mediation category. It is difficult, however, to pinpoint the difference(s) most likely to account for the disparity in study findings regarding mediation. The discussion below will therefore be limited to just a few candidates regarded by this author as
plausible explanatory factors.

Two key differences are apparent when this sample is compared to those in the studies by Bookwala et al. (2003), Kahana et al. (1997), and Geerlings et al. (2002). First, by design, 100% of the present sample was married. In contrast, roughly half of respondents were married in each of these no mediation studies. The percentage of married respondents was 47% in Kahana et al., 52% in Bookwala et al., and 53% in Geerlings et al. Second (and also by design), 100% of participants in the present study reported having pain. This figure differs markedly from prevalence data reported in two of the no mediation studies. Little or no pain was reported by 49% of the Kahana et al. sample, and 73% of the Geerlings et al. sample reported no pain. (Neither the percentage reporting pain nor average pain intensity in the Bookwala et al. sample could be determined from published data.)

In order to evaluate the potential importance of these two differences in sample composition, it is also useful to consider whether these (or other) features are shared with the majority of studies in the partial and total mediation categories. With respect to marital status, there was a distinct trend for studies in the partial and total mediation categories to have a larger proportion of married participants (a few studies also counted domestic partnerships). Among samples in the total mediation studies, the percent married ranged from 66% (Rudy et al., 1988) to 72% (Williamson, 2000a). The percentage of married respondents in the samples of the partial mediation studies ranged from 66% (Arnstein et al., 1999) to 81.3% (Goodland, 2002). An exception to this trend was the study by Williamson & Schulz (1992a), in which only 33% of respondents were married. (Information regarding marital status was not provided for the sample in
Williamson & Schulz, 1995.) However, attributing the difference in mediation findings to marital status based only on these data would be imprudent, especially given the absence of prior studies in which samples were homogeneous in terms of marital status.

Two of the no mediation studies (Bookwala et al., and Kahana et al.) did control for marital status in their mediation analyses. Additionally, Williamson (2000a) reported that in preliminary analyses, marital status was unrelated to any key variables in her study. However, no prior study has examined whether the relationships among pain, activity limitation, and depressive symptomatology differ based on marital status. It is difficult to conceive of many reasons why having a larger proportion of married persons in a sample would lead to a greater likelihood of finding mediation by activity limitation. One possibility is that having a greater percentage of married persons in the sample contributed to greater homogeneity among respondents on other (observed or potentially unobserved) characteristics. This homogeneity may have helped to reduce some of the “noise” that has interfered in the analysis of key relationships in prior studies.

It is important to note that these two sample characteristics (all married and all reporting pain) were inexorably linked in the present study—by design, all participants reported pain and were married to a partner who also reported pain. There is no indication that the samples in any previous mediation studies included respondents who were married to each other. Additionally, although some of these studies were part of larger research programs that did include the spouses of married respondents (e.g., Rudy et al., 1988) none of these studies reported whether (or to what extent) those spouses also suffered from pain. Consequently, no data exist to support any speculation regarding some joint influence of marriage and spouse pain on the study findings with respect to
mediation of the pain-depressive symptomatology relationship at the intra-individual level. However, this possibility does exist and requires further study.

In contrast to the percentage of respondents who reported pain in the studies by Geerlings et al. (27%) and Kahana et al. (51%), roughly half of the prior studies that found at least partial mediation had samples in which (presumably) 100% of respondents reported pain. Two total mediation studies (Rudy et al., 1988; Turk et al., 1995) and one partial mediation study (Arnstein et al., 1999) had samples comprised of patients newly-referred to chronic pain clinics. Injured workers with chronic pain constituted the sample in Goodland (2002; a partial mediation study). Lack of comparability in terms of which dimensions of pain were assessed and how they were measured make it hard to accurately estimate the percentage of respondents with pain in the remaining four studies that also found support for mediation. However, given that two of the studies (Williamson, 2000a; and Williamson & Schulz, 1995) had samples comprised of cancer patients, it may be reasonable to assume that pain was fairly prevalent in these samples. Thus, the majority of studies that found evidence of mediation appear to have had samples in which most, if not all, respondents reported pain.

Could simply having a larger proportion of respondents with pain influence the likelihood of finding support for mediation? Quite possibly, yes. In general, a larger number of respondents with pain should yield greater variability on measures of most dimensions of pain (e.g., intensity, frequency, etc.). In contrast, the variability, and possibly the overall range, of responses on pain measures would be restricted in samples comprised of smaller proportions of persons with pain. The distributions on these measures would also be significantly positively skewed and kurtotic. In general, these
factors tend to reduce associations between the affected measure and other variables in a study. Reduced variability and/or restricted range and/or non-normal distributions on measures of pain may have been a contributing factor in some of the studies that did not find evidence of mediation. Use of the word “contributing” here is intentional—these factors, especially reduced variability, typically attenuate all relationships involving that variable. So, while this proposed explanation might reasonably account for some (non-) findings (e.g., the lack of significance of Path B in Geerlings et al., 2002), it is inconsistent with others (e.g., a significant pain-depressive symptomatology relationship in all no mediation studies). Another possibility is that, in studies in which a large proportion of respondents did not have pain, observed relationships involving pain intensity may have actually been reflecting associations with the presence of pain. It would thus be interesting to re-examine the activity limitation mediation hypothesis in these studies using only the subsample of respondents who reported pain.

Alternatively, it is possible that the feature that most distinguishes studies supporting mediation from non-supporting studies is not the proportion with pain, but some qualitative difference in the pain reported by different samples. Studies varied widely in the type(s) or locations of pain reported by participants. As previously reported, two studies (Williamson, 2000a; and Williamson & Schulz, 1995) had samples comprised solely of cancer patients, and both found evidence supporting mediation (total and partial, respectively). Only two studies assessed disease-specific pain—subjects in Bookwala et al. (2003) were asked to report knee osteoarthritis pain only, and Williamson and Schulz (1995) queried respondents about cancer-related pain. Yet, these two studies yielded contradictory results (no and partial mediation, respectively). As
discussed in Chapter 2, no other patterns with respect to the type(s) or location of pain were evident across studies. For example, musculoskeletal pain and/or arthritis were common in both studies that found mediation—including the present study—and those that did not.

Results from the present study do appear to buttress an earlier observation regarding the tendency to find evidence of mediation in studies in which pain could be regarded as chronic, persistent, or recurrent or pain that was more current or recent. Because this pattern was noted during the review of prior studies in Chapter 2, the discussion here is limited to similarities between the present study and Gureje et al. (2001), a study that found partial mediation. Gureje et al. (2001) sampled adults from primary care settings and identified respondents with “persistent pain,” as defined by several specific criteria. At the outset of the study, 22% percent of participants were classified as having “persistent pain.” This percentage is notable for two reasons. First, although the percentage of the sample with pain was low, Gureje et al. found evidence of partial mediation by activity limitation. This may be attributable to the fact that Gureje et al. tested the mediation hypothesis using persistent pain status as the independent variable. Second, the rate of endorsement of the general HRS pain item within the preliminary couples sample in the present study (21% and 24% for husbands and wives, respectfully) are highly consistent with the figure reported by Gureje. As has been mentioned, even though pain was not explicitly defined as persistent or chronic within HRS, the phrasing of the general pain question appears to tap more than incidental or occasional pain. Thus, with the addition of the present study, more than half of studies with evidence of at least partial mediation (6 of the now 9) either explicitly or implicitly
defined pain as chronic, persistent, and/or recurrent. Also as previously noted, participants in the remaining three studies (Williamson, 2000a; Williamson & Schulz, 1992a, 1995) reported pain that was experienced more recently (e.g., past week or two) than the pain reported by participants in the majority of studies that did not find evidence of mediation (e.g., Bookwala et al., 2003; Kahana et al., 1997).

Focusing on chronic/persistent or recurrent pain and/or pain that was experienced more recently (hereafter referred to as “persistent and/or current pain”) may have helped to strengthen the relationships between and among pain, activity limitation, and symptoms of depression in many studies, thereby increasing the possibility of finding at least partial mediation. There are numerous potential mechanisms through which such strengthening could occur. It is possible that the relationships among variables within the activity limitation mediation model are fundamentally stronger in the context of persistent and/or current pain (as compared to pain that is not persistent or recent). It is conceivable that the pain-activity limitation-depressive symptomatology relationship exists only within the context of persistent and/or recent pain. A “threshold effect” may also be in operation, such that pain needs to reach a certain level of persistence/chronicity and/or occur within a recent time frame in order to produce a sizeable effect on activity limitation and depressive symptoms. Respondents’ ratings of persistent and/or more recent pain are likely to be more reflective of their current experience than are ratings of more occasional and/or past pain; this might enhance associations with other variables collected via measures that specified a recent time frame for reporting (e.g., depressive symptoms in the past week). Alternatively, the presence of persistent and/or current pain may signal reduced overall health and well-being in respondents, resulting in higher
levels of both activity limitation and depressive symptomatology—something that could also enhance the relatedness of key variables. Regrettably, many of these competing explanations cannot be evaluated in either the current or prior studies due to the lack of information about the chronicity and recency of respondents’ pain.

**Differences in the Conceptualization and Measurement of Activity Limitation**

In addition to differences with respect to sample composition and reports of pain, the present study differed markedly from most studies in the no mediation group in terms of how the mediating construct—activity limitation—was conceptualized and measured. As observed earlier, most of the “failures” in prior mediation studies seemed to result from a lack of expected relationships involving activity limitation. Variability in the definition and operationalization of activity limitation was identified previously as one potential source of inconsistent findings regarding mediation (see Chapter 2). While some studies used measures that included fundamental physical abilities (physical limitations), others targeted limitations in independent activities required for daily living (IADLs), and still others emphasized difficulties with basic self-care (i.e., ADLs). A few studies even expanded the concept into the social realm; for example, Kahana et al. (1997) included a measure of “social disability” that captured the extent of (dis)engagement in valued social activities. Williamson and colleagues included several different types of activity limitation in one broad measure (e.g., Williamson, 2000a; Williamson & Schulz, 1995). Recall that three trends with respect to activity limitation were identified in Chapter 2: (a) Studies with measures of activity limitation that emphasized ADLs tended to NOT find evidence of mediation; (b) Evidence for mediation appeared more often in studies that used pain-specific measures of activity limitation; and
(c) When social functioning/limitation was defined and measured separately from, yet evaluated within the same multivariate statistical model as, limitations of a more physical nature, study results tended to NOT support the mediation hypothesis.

Partly because of the first trend, activity limitation was operationalized and measured in the present study as basic physical limitations (e.g., difficulty stooping, lifting, climbing stairs). Use of a measure exclusively focused on basic physical limitations distinguished the present study from previous research. Although basic physical limitations had been included in the measures of activity limitation used in several prior studies (Bookwala et al., 2003; Geerlings et al., 2002; Williamson, 2000a), they never dominated the content of those measures. Results from this study provided clear support for the activity limitation mediation hypothesis when activity limitation was defined as basic physical limitations. These results also suggest that the previous inconsistency in mediation study results may have resulted, at least in part, from variations in the measurement of activity limitation. However, because this study did not compare these results with those based on a measure of ADLs, findings from this study can only indirectly corroborate the first trend. One clear direction for future research is to compare mediation results across different definitions and measures of activity limitation within this sample. It would be interesting to see if results would conform to those of Williams and Schulz (1992a), who reportedly were unable to replicate their mediation findings using only the ADL limitation items within their measure of activity limitation (Williamson, 2000a).

Because the HRS physical limitation questions were not specific to difficulties arising from pain, findings from the present study cannot directly address the second
trend observed among prior studies—i.e., a tendency to observe mediation when activity limitation was assessed specific to pain. Note, however, that because all respondents in the present study had pain, it would be especially difficult to determine the extent to which physical limitations were attributable to pain versus other possible sources. Future studies might attempt to clarify this issue, although whether or not individuals can (accurately) identify a distinct source of such limitations is, itself, an empirical question that requires additional attention (e.g., Ettinger et al., 1994).

The present study did not include limitations in social functioning. As a result, this study cannot directly address the third trend—i.e., a tendency to not find evidence of mediation by (physical) activity limitation when a measure of social limitations was also included in multivariate models. In the discussion of the gender difference in the extent of mediation found in this study, it was suggested that the wives in DP couples might be more distressed by limitations affecting their ability to engage in social activities than by basic physical limitations. One potentially fruitful direction for future research is to clarify the role of limitations in social functioning within the pain-activity limitation-depressive symptomatology framework. Such efforts may yield important insights into the gendered relationships between pain and depressive symptomatology that were observed among DP spouses.

It is noteworthy that in the present study, significant mediation of the pain-depressive symptomatology relationship by physical limitations was observed in a sample of advanced age. These findings offer some evidence to dispute one argument that has been advanced by several authors to help explain the lack of mediation found in some studies. For example, Geerlings et al. (2002) contended that their mobility-related
activity limitation items were “more closely related to personal capabilities” than the items used by Williamson and colleagues (p. 28). As such, these limitations may not have been relevant to their older adult sample (all over age 55, with a median of 70), perhaps because they viewed such limitations as age-normative. Because these authors tested for, but did not find, an age-related difference in the pain-depressive symptom relationship in their sample, they suggested that the change in expectations regarding functional status might have already occurred in their respondents. This suggestion was first introduced by Williamson (e.g., Williamson, 2000b), and fueled by some of the cross-sectional findings in the study by Williamson and Schulz (1995). In that study, the mediating effect of activity restriction was stronger among younger cancer patients (under age 65 or between 60 and 64) than among older cancer patients (over 65 or between 65 and 70). Notably, however, this pattern was not observed longitudinally.

Explanations referencing potential age- and/or experience-normative expectations regarding activity limitation, pain, and depressive symptoms (alone or in in various combinations) have also been proposed to explain the lack of mediation found in studies by Parmelee et al. (1991) and Bookwala et al. (2003). Although DP husbands and wives were, on average, a few years younger, the age distributions of DP spouses appear to overlap substantially with those of respondents in Bookwala et al. (2003) and Geerlings et al. (2002); for example, fewer than 2% of DP husbands and 7% of DP wives were under 50 years of age. Evidence of mediation by physical limitations in the present sample thus presents a serious challenge to age-related explanations for a lack of mediation, as do the findings of Williamson & Schulz (1992a) who found partial mediation in an outpatient sample of older adults ($M = 72$).
Age-based expectations and norms do seem slightly more plausible when considering the findings of Cohen-Mansfield & Marx (1993), Parmelee et al. (1991) and Kahana et al. (1997), given the advanced age of these samples (all means above 80 years). However, as noted above, these studies shared other characteristics (e.g., measures of pain and/or activity limitation) that might better explain the lack of mediation; in addition, some of these characteristics were also shared with the studies by Bookwala et al. (2003) and Geerlings (2002). Because age-moderated mediation was not explicitly tested in the present study, it cannot be dismissed as a potential factor contributing to the variation across studies with respect to mediation findings. Additional, and more carefully designed, studies will be required in order to clarify the contributions that age and age-normative or experience-based expectations make within the pain-activity limitation-depressive symptomatology relationship. At the very least, however, findings from the present study suggest that current explanations may not be adequate.

In summary, findings from the present study regarding (at least partial) mediation of the pain intensity-depressive symptomatology relationship by activity limitation at the intra-individual level are consistent with those from the bulk of prior mediation studies. These findings provide initial support for the proposed conceptual model (Figure 5), and also offer further support for Williamson’s activity limitation/restriction model. In addition, these findings make four important contributions to the existing literature. First, mediation was demonstrated using a measure of activity limitation comprised solely of physical limitations (as opposed to IADL and/or ADL limitations). Second, gender was found to moderate the extent to which activity limitation mediated the pain-depressive
symptomatology relationship, with more complete mediation observed in husbands as compared to wives. Third, mediation was found in a sample of married couples in which both partners reported problems with pain. And, although this sample could be distinguished on several levels from those of prior mediation studies (e.g., genders were equally represented, all were married, all were married to another person with pain), study findings regarding mediation were largely consistent with the majority of prior findings. Fourth, and finally, mediation was found in a sample of adults of advanced age.

When viewed in conjunction with the findings and contributions of prior mediation studies, findings from the present study raise important questions about the types of activity limitation that specifically mediate the pain-depressive symptomatology relationship at the intra-individual level. Other closely-related, and equally-important, questions raised include “for whom?” and “to what extent?” Strategies to address some of these questions are presented later in this chapter.

Findings Regarding Covariates

The background and health-related characteristics that were found to be significant within intra-individual multivariate models warrant some discussion. Findings regarding covariates in models of DP husbands’ and wives’ physical limitations are reviewed briefly below, followed by the covariates that emerged in models of the depressive symptomatology of DP spouses. These findings are then jointly considered in relation to the existing literature.

Besides pain intensity, few covariates were associated with the physical limitations of DP husbands and wives in multivariate models at the intra-individual level. With the exception of body weight, significant covariates and their relative contributions
were similar by gender. Household income (logged) was the only background variable uniquely associated with physical limitations in both husbands and wives. In both spouses, lower household income predicted higher physical limitations. In terms of health-related variables, more doctor-diagnosed disease conditions were tied to higher physical limitations in both husbands and wives. Greater body weight was also associated with more physical limitations in DP wives.

Depressive symptomatology, too, was related to a limited number background and health-related characteristics in the intra-individual multivariate models. In both spouses, lower educational attainment was associated with greater depressive symptomatology. Lower household income was also linked to more depressive symptoms, but this finding was only consistently observed in DP husbands. Age was related to depressive symptoms in wives only, with younger DP wives displaying slightly higher levels of depressive symptomatology than older DP wives. Finally, higher body weight (BMI) was associated with fewer depressive symptoms among DP husbands.

**Background Characteristics**

Findings in this study with respect to the influence of socioeconomic status are generally consistent with the bulk of findings from prior studies that have examined risk factors for poor physical and/or mental health. A considerable body of research documents a positive association between higher SES and better physical functioning. As established in Chapter 2, existing studies have varied widely in the definitions and measures of activity limitation or physical functioning, with a majority of studies relying on measures mainly comprised of IADLs and ADLs. However, similar relationships have been documented in studies that have included, or focused more specifically on,
basic physical limitations (e.g., mobility limitations, impairments in physical functioning). For example, even after controlling for the effects of age and gender, low income and low education were significantly associated with greater levels of functional impairment (measured with a mix of IADL, ADL, and physical limitation items) in community-dwelling older adults between ages 70 and 79 (Berkman et al., 1995). In a community-based sample of midlife and older adults, Verbrugge, Gates, and Ike (1991) found that low education was a significant independent predictor of higher levels of physical limitations. Even among very old, community-dwelling adults (e.g., those over 80), less education has been associated with more and worsening levels of physical limitations (e.g., Harris et al., 1989; Parker et al., 1996).

Some studies have found differences among measures of SES. For example, in a sample of older adults, Murtaugh & Hubert (2004) found that only (low) income, not education, was associated with greater physical limitations. In one of the mediation studies, income was the only background variable with a direct effect on levels of personal disability: Increased income was associated with decreased personal disability (Kahana et al., 1997). In contrast, some studies have found level of education to be the most consistent socio-demographic predictor of physical functioning in community-dwelling adults in mid and later life (e.g., Pinsky et al., 1987).

In studies of older adults, both low income (e.g., Blazer et al., 1991) and low education (e.g., Blazer et al., 1994) have been associated reliably with higher depressive symptomatology. One of the mediation studies (Bookwala et al., 2003) also reported that lower education remained associated with increased depressive symptomatology in multivariate analyses. A limited number of studies have examined predictors separately
for men and women. For example, Choi & Bohman (2007) analyzed CES-D8 data separately for men and women ages 65 and older drawn from HRS 1998 and 2000. They found that even after adjusting for covariates (e.g., number of medical conditions, ADL & IADL impairments, health behaviors, marital status, and age), years of education and household income were consistently negatively associated with higher depressive symptomatology in women; only years of education was consistently negatively associated with depressive symptoms in men. Different patterns have been observed in other studies. A recent study examined predictors of depressive symptomatology levels separately for community-dwelling men and women aged 60 to 85 in Germany (Glaesmer, Ridel-Heller, Braehler, Spangenberg, & Luppa, 2011). In multivariate analyses, level of education was not significantly related to depressive symptomatology in either men or women. Household income was a significant predictor of (fewer) symptoms of depression, but only for men—a finding echoed in the present study. Supplemental analyses by Glaesmer et al., 2011) showed that household income would have been identified as a significant predictor for the entire sample, had the two groups been combined in single multivariate analysis with gender as a covariate.

Some researchers have observed that the relationships between SES variables and depressive symptomatology are diluted when the variables are added concurrently to predictive models (e.g., Dunlop et al., 2004). Others have reported that the effects of SES variables are often reduced with the addition of other background variables. For instance, Choi & Bohman (2007) reported that the effects of education and income were significantly reduced once other covariates were added to multivariate models of depressive symptomatology; moreover, this pattern was observed in both men and
women. Although the specific associations observed in the present study between the
different indicators of SES and depressive symptomatology differ somewhat from those
reported in previous studies, the overall patterns observed while building the multivariate
predictive models are largely consonant with existing research. Overall, results from this
study also support the notion that economic disadvantage is associated with higher levels
of depressive symptomatology.

It is somewhat surprising that age was not significantly related to physical
limitations in either DP husbands or wives. Most studies document a positive association
between increasing age and greater activity limitation among community-dwelling adults
(e.g., Kivela & Pahkala, 2001; also see review by Seeman, 1994). Studies limited to the
consideration of physical limitations have also reported this trend (e.g., Clark et al., 1998;
Cronnin-Stubbs et al., 2000; Geerlings et al., 2001; Verbrugge, Gates, & Ike, 1991). An
increase in physical limitations with age has also been found within older adult samples.
For example, one study showed that reports of difficulty with most basic physical tasks
(including lifting, climbing steps, etc.) increased steadily across three 10-year age cohorts
of community-dwelling adults over age 65 (Fried et al., 1991). Even among the oldest-
old, increased age has been linked to more and/or worsening physical limitations (e.g.,
Harris et al., 1989; Ostchega et al., 2000; Parker et al., 1996). Some researchers have
observed that incorporating covariates, such as specific disease conditions, cognitive
functioning, and physiological measures (e.g., blood pressure), often reduces the
association between age and physical limitations (e.g., Fried et al., 1991). This
observation might suggest that the effects of age are mediated through increased
incidence of disease or disease processes. Even though age was associated with a greater
number of disease conditions in DP husbands (and also in DP wives), the lack of a
significant bivariate relationship between age and physical limitations in husbands raises
questions about this as a possible explanation for the findings in the present study.
Clearly, these relationships merit further research.

Age was a significant predictor of (lower) depressive symptomatology only in DP
wives. As reported in Chapter 2, conflicting patterns related to age and depressive
symptomatology have been observed across studies. In general, studies have documented
a linear relationship between age and depressive symptomatology, with depressive
symptoms increasing with increased age (e.g., Blazer, 1993; Blazer et al., 1994).
However, there is continued debate about whether the pattern is completely linear across
all of adulthood, and even across all of older adulthood. In some studies, the oldest-old
(e.g., above 70 or 75) display the highest levels of depressive symptomatology among all
adults (e.g., Blazer et al., 1994; Gatz & Hurwicz, 1990; Schnitker, 2005). In other
studies, older adults display the lowest levels of depressive symptomatology of all age
groups (e.g., Nolen-Hoeksema & Ahrens, 2002), suggesting a more curvilinear
relationship. Unfortunately, longitudinal studies have not sufficiently clarified this
relationship (see Djernes, 2004, for review).

Gender differences in the relationship between age and depressive symptom-
atology have been observed in some studies. The pattern of findings, however, has not
been consistent. For example, some studies of community-dwelling adults have found
age to be unrelated to depressive symptomatology in women, but positively associated
with symptomatology in men (e.g., Barefoot et al., 2002). Other studies have observed
gender differences of a different nature. In a study of 119 community-dwelling older
married couples (aged 60-84), older age was positively associated with greater depressive symptomatology in wives, but was not a significant predictor in husbands (Robb, Small, & Haley, 2008). Beekman et al. (1995) found a similar result when they examined the presence of significant depressive symptomatology (labeled “minor” depression) in a representative sample of community-dwelling older adults. In women, older age was related to a linear increase in the incidence of minor depression. The relationship was more complex in males, with their results suggesting a nonlinear relationship: Greater incidence of minor depression was found among both younger (under age 60) and older (over age 75) men, as compared to those age 60 to 75.

As is the case with many socio-demographic factors—when other variables are controlled in multivariate analyses, the relationship between age and depressive symptomatology may be reduced (e.g., Berkman et al., 1986) or even reversed (e.g., Blazer et al., 1991). For example, in multivariate analyses controlling for health and functional limitations, Dunlop et al. (2004) found that the odds of depressive symptom levels indicative of probable major depression among participants in the HRS 1996 sample were greater for adults in the late-50s age group than for those in the 60-65 age group. In the study by Choi and Bohman (2007), age was found to be negatively associated with depressive symptomatology in their sample of older women. Most notably, these authors reported that this age effect appeared to strengthen as more covariates were added to multivariate predictive models.

The presence of mixed findings throughout the literature suggests that the relationship between age and depressive symptomatology is complex. It is likely that age has both positive and negative effects on depressive symptomatology, perhaps through
pathways involving health-related characteristics. The mediation study by Kahana et al. (1997), for example, documented both indirect and direct effects of age on depressive symptomatology. Age was the only background variable with an independent association with depressive symptomatology: greater age was associated with slightly fewer depressive symptoms. At the same time, however, greater age was associated with musculoskeletal and heart/lung diseases, which were, in turn, related to greater pain, which was subsequently linked to higher levels of depressive symptomatology. It is thus possible that age influences depressive symptomatology through multiple pathways in DP wives; however, this possibility requires further study.

Three background characteristics—household wealth, non-white race, and the presence of others in the household—were not significantly related to either the physical limitations or the depressive symptomatology of DP husbands or wives in any intra-individual multivariate model. In general, these variables displayed only weak bivariate correlations with physical limitations and depressive symptomatology in both spouses. Of the three, household wealth was most strongly and consistently (but still only weakly) tied to each outcome variable. Household wealth is not commonly used as a covariate in studies of physical or mental health. When it has been used, most studies report an inverse relationship between wealth and health. For example, lower levels of accumulated wealth were associated with an increased prevalence of depressed mood in a large sample of community-dwelling midlife and older persons in England (Wikman et al., 2010). A study of couples drawn from early waves of HRS and AHEAD, Townsend et al. (2001) found that household wealth explained a small, but significant amount of variation in spouses’ depressive symptomatology levels, above and beyond that explained
by household income. The fact that household income and wealth were moderately correlated in DP couples suggests that the relationship between wealth and each outcome variable was largely shared with income. The lack of significance for both wealth and income in relation to DP wives’ depressive symptomatology might be attributable to overlapping variance with other background variables. On the other hand, it is also possible that in DP wives, the effect of income (and perhaps wealth) on depressive symptoms was mediated by its effect on physical limitations. This potential explanation is bolstered by the fact that household income had been a significant predictor of wives’ depressive symptoms until physical limitations were added in Model 4.

In bivariate analyses, DP spouses of non-White race evidenced slightly more physical limitations (wives only) and depressive symptoms (both spouses) than did White spouses. This is consistent with most prior research showing that older Whites tend to experience less physical limitations than their African American counterparts (e.g., Fried et al., 1991; Clark & Gibson, 1997; Stump et al., 1997; Seeman et al., 1994). As noted by several authors, however, the risk of greater physical limitations among those of minority race may be conveyed indirectly, through increases in other known risk factors, such as chronic disease or SES (Clark et al., 1997; Zsembik, Peek, & Peek; 2000).

The nature and extent of racial differences in depressive symptomatology among aging community-dwelling adults remains unclear, despite decades of study (Blazer et al., 1994; George & Lynch, 2003). In most studies, older Blacks have been found to report more depressive symptomatology than older Whites (e.g., Blazer, Landerman, Hays, Simonsick, & Saunders, 1998; Ellison, 1995; Skarupske et al., 2005). Similar racial differences have been observed in middle-aged samples (e.g., Freeman et al., 2001). In
contrast, Whites have been found to display higher levels of certain depressive symptoms (e.g., sadness) than Blacks (e.g., Gallo, Cooper-Patrick, & Lesikar, 1998). Other studies have found greater depressive symptomatology among those of minority race, but only among women (e.g., Swenson et al., 2000) or only within middle-aged participants (e.g., Fiori, McIlvane, Brown, & Antonucci, 2006). When relevant covariates (e.g., gender, age, marital status, SES indicators, health status) are controlled in multivariate models, differences in depressive symptomatology by race have been eliminated in some (e.g., Bromberger et al., 2003), but not all studies (e.g., Jang et al., 2005; Skarupski et al., 2005). The Bookwala et al (2003) mediation study found that, after controlling for pain, activity limitation, health problems, and other background variables, aging Whites actually had higher levels of depressive symptomatology than did non-Whites.

In the present study, it is possible that the variance in physical limitations and depressive symptomatology that was associated with race may have been explained by differences in other background variables and/or mediated through other variables included in multivariate models. In support of this possibility, recall that non-White race was associated with most other background characteristics in expected ways (e.g., modestly negatively correlated with SES variables). The significant bivariate correlation between non-White race and body weight in DP wives could also support a potential mediation hypothesis. Alternatively, it is also possible that combining Black and other races into a single “non-White” category may have masked important differences among these races. Additional study is required to disentangle these various possibilities.

Shared variance with other background variables may be a plausible explanation for the lack of significant findings for the presence of others living in the household.
Note, however, that this variable was related only weakly at a bivariate level to husbands’ depressive symptom levels. The presence of others in the household was intended in the present study as a gross indicator of household structure and composition. It is possible that more refined measures of these constructs (e.g., presence of a child under 18, presence of an aging parent or other relative, the actual count of others in the household) might show greater associations with physical limitations and depressive symptoms in DP spouses. For instance, in one study, married women residing in spouse-only households or in households including just the spouse and their child(ren) evidenced significantly lower depressive symptom levels than did married women living in more complex households (i.e., those including the spouse along with non-relatives or relatives other than the couple’s children; Hughes & Waite, 2006). On the other hand, even those indicators might prove to be inadequate proxies for underlying constructs of interest, such as caregiving arrangements, availability of instrumental and emotional support, and opportunities for positive and negative social interactions. For example, a plethora of studies have shown that social isolation is a risk factor for poor physical health (e.g., Cornwell & Waite, 2009), greater physical and functional limitations (e.g., Verbrugge, Reoma, & Gruber-Baldini, 1994) and declines in functional status over time (e.g., Cacioppo & Hawkley, 2003; Unger et al., 1999). Many of these factors have also been linked to depressive symptomatology. For example, in multivariate analyses controlling for health conditions, functional limitations, and other common socio-demographic variables, Dunlop et al. (2004) found greater risk of probable depression among HRS 1996 respondents who reported providing some type of instrumental support for an aging parent or parent-in-law. Availability of and satisfaction with social support has also been
associated with lower depressive symptomatology in several studies of community-dwelling aging persons (e.g., Glaesmer et al., 2011; Heikkinen, Riitta-Liisa, & Kauppinen, 2004; see also review by Djernes, 2004). Future studies could examine household composition, caregiving arrangements, and other exchanges in more detail.

Health-Related Characteristics

Of the two health-related characteristics in the conceptual model, only body weight evidenced any unique relationship with the physical limitations or depressive symptomatology of DP spouses. Even then, the nature of the observed relationships differed by gender: BMI was positively related to physical limitations in wives, but was unrelated in husbands. In addition, BMI was inversely related to depressive symptomatology in husbands, but was not related to wives’ depressive symptomatology.

The finding that BMI was positively related to greater physical limitations in wives is consistent with the findings of many prior studies. Among studies limited to women, higher BMI and/or obesity, specifically, have been regularly associated with impairment in physical functioning in later life (e.g., Apovian et al., 2002; Lamb et al., 2000), as well as in middle-age (e.g., Evers-Larsson & Mattsson, 2001; Sach et al., 2007; Stafford et al., 1998). The Evers-Larsson & Mattsson (2001) study found that the relationship between higher BMI and more physical limitations remained significant, even after controlling for the effects of age and lower body pain; this finding is notable because, similar to the present study, both pain and BMI were independent predictors of difficulties in physical functioning. Findings from this study suggest that increased body weight contributes, along with greater pain intensity, to heightened physical limitations among DP wives.
Body weight was not a significant predictor of physical limitations in DP husbands—a finding inconsistent with the findings of many prior studies. For example, in a community-based sample of over 1,600 middle-aged and older adults Sternfeld, Ngo, Satariano, and Tager (2002) found that, in both women and men, higher body fat was associated with deficits in both self-reported physical limitations (e.g., climbing stairs, stooping, lifting) and observed physical performance (e.g., walking speed). Numerous other studies have found cross-sectional (e.g., Han et al., 1998; Murtaugh & Hubert, 2004; Yan et al., 2004) and longitudinal (e.g., Clark et al., 1998; Daviglus et al., 2003; Lee et al., 2005) relationships between obesity and greater limitations in physical functioning in both genders in mid and later life. Unlike for DP wives, no significant bivariate relationship was observed between DP husbands’ BMI and physical limitations. It is therefore unlikely that the lack of an effect of BMI within a multivariate context was due to mediating variables. And, although BMI had a moderate negative relationship to age in DP husbands, age was not a significant predictor of physical limitations either.

Surprisingly, DP husbands with higher BMI evidenced less depressive symptomatology than those with lower BMI. This finding appears to offer support for the “jolly fat” hypothesis first articulated by Crisp and colleagues in Great Britain (Crisp & McGuiness, 1975; Crisp, Queenan, Sittampalm, & Harris, 1980). Crisp et al. found that, especially among middle-aged and older men, obesity was associated with lower levels of psychological distress, including fewer symptoms of depression. These authors speculated that obesity might offer some protection against psychological distress, the nature of which could be rooted in biochemical, characterological, or experiential differences between obese and non-obese persons. Findings from subsequent studies
have been very mixed. Several studies in the U. S. have garnered support for the jolly fat hypothesis. For example, Palinkas et al. (1996) found that the odds of scoring in the depressed range on the BDI were decreased among both overweight and obese older men, as compared to men with normal or low body weight. While this relationship trended in the opposite direction for older women, differences did not reach statistical significance; Palinkas and colleagues offered that this was perhaps due to the greater stigma associated with excessive weight in women as compared to men.

On the other hand, numerous studies have found either no relationship between body weight and depressive symptomatology (e.g., Dunlop et al., 2004; Hach et al., 2007; Stewart & Brook, 1983) or evidence that directly contradicts the jolly fat hypothesis (e.g., Daviglius et al., 2003; Roberts, Strawbridge, Deleger, & Kaplan, 2002). In some studies, findings have varied by gender: Obesity has most often been linked to higher depression and/or depressive symptomatology in women (e.g., Carpenter, Hasin, Allison, & Faith, 2000; Istvan, Zavela, & Weidner, 1992). However, it is challenging to compare study findings, given marked differences in study methodology (e.g., cross-sectional vs. prospective), measurement (e.g., BMI as a continuous vs. categorical measure), and analytic strategies (e.g., incorporation of many vs. a limited number of covariates).

Although findings from the current study seem most consistent with the jolly fat hypothesis (for husbands), the post hoc analyses of residuals did suggest that additional exploration of potential non-linear effects of BMI on depressive symptomatology may be warranted. Such exploration is also supported by the findings of some studies that have reported a “J”-shaped relationship between body weight and measures of psychological distress (e.g., Heo et al., 2003; Johnston et al., 2004). It may also be worthwhile to
investigate whether any differences emerge in the association between body weight and depressive symptomatology in DP spouses when *categories* of body weight are used instead of the continuous BMI measure.

In light of the differential associations observed in the present study between BMI and two key study variables (physical limitations and depressive symptomatology) by gender, one potential direction for future research might be to examine potential interactive effects. For instance, perhaps body weight (or specific levels thereof—e.g., obesity) interacts with pain intensity or other variables to influence physical limitations and/or depressive symptomatology in DP spouses. This notion is consistent with some of the findings from Lamb et al.’s (2000) study of older women. These authors found that obesity interacted with severe knee pain to increase the odds of mobility limitation beyond those associated with either factor individually. Such findings suggest that the relationships among BMI, pain, physical limitations, and depressive symptomatology are complex and warrant further study.

Number of disease conditions was not a significant independent predictor of physical limitations or depressive symptomatology in either spouse. These findings are at odds with many prior studies that have documented these relationships in samples of community-dwelling adults. Number of disease conditions has been regularly associated with functional impairment in both cross-sectional and longitudinal studies (see review by Gijsen, Hoeymans, Schellevis, Ruwaard, Satariano, & van den Bos, 2001). This association has also been verified when basic physical limitations are used as indicators of functional impairment (e.g., Groll et al., 2005; Hart, Wang, Stratgord, & Mioduski, 2008; Verbrugge, Lepkowski, & Imanaka, 1989). Furthermore, a greater number of
comorbidities has been associated with worsening in physical task performance over time among older adults (e.g., Bryant et al., 2007).

Findings from the present study are also discordant with a large body of research showing a connection between a number of disease conditions and higher levels of depressive symptomatology among community-dwelling adults in later life (e.g., Black, Goodwin, & Markides, 1998; Choi & Bohman, 2007; Mills, 2001; Palinkas et al., 1990; Penninx et al., 1996). In fact, number of disease conditions was found to be the strongest predictor of (greater) depressive symptomatology in both aging men and women in the study by Glaesmer et al. (2011). In community samples of middle aged and older adults, a greater number of disease conditions has also been associated with the presence of “significant” depressive symptomatology or “depressed mood” as indicated by high scores on measures of depressive symptomatology (e.g., Beekman et al., 1997; Wikman et al., 2010), as well as “probable” major depression assessed via screening interviews (e.g., Dunlop et al., 2004).

One potential explanation for the lack of significance in this study emerges from consideration of the pattern of results for number of disease conditions across all intra-individual multivariate models in this study. Recall that a greater number of disease conditions had been significantly related to both greater physical limitations and higher depressive symptomatology in both DP spouses in early models (e.g., Models 2 and/or 3). These relationships then became non-significant when pain intensity (in the case of physical limitations) or physical limitations (in the case of depressive symptoms) were added to the model. Together with evidence that number of disease conditions was related to pain intensity in both spouses, these findings suggest that the effect of a
respondent’s disease burden on his/her depressive symptomatology is potentially mediated through his/her pain intensity and/or physical limitations. They additionally suggest that pain intensity may mediate the relationship between number of disease conditions and physical limitations.

Support for this proffered explanation is enhanced by the observation that many prior studies have not controlled for pain or physical limitations. When studies do control for these variables, they tend to report similar patterns to those observed in this study. For instance, Dunlop et al. (2004) reported that when functional limitations (especially physical limitations) were added to multivariate models predicting high depressive symptomatology, the strength of the effect associated with multiple disease conditions was reduced considerably. Note that the findings of some prior activity limitation studies may not support such an explanation. In several of these studies, a greater number of disease conditions/health problems remained associated with higher depressive symptomatology even after controlling for the effects of pain and activity limitation (Bookwala et al., 2003; Cohen-Mansfield & Marx, 1993; and Parmelee et al., 1991). It is interesting that all three of these studies failed to find evidence of activity limitation mediation, which might suggest that the type of activity limitation is important in other mediation pathways, as well. Other prior mediation studies have incorporated disease conditions in varied ways, making it difficult to draw firm conclusions about possible mediation. However, findings from several prior studies seem to suggest that the effect of disease conditions on depressive symptomatology is largely indirect. Williamson and Schulz (1992a), for instance, found that activity limitation fully mediated the relationship between disease conditions and depressive symptomatology in their
geriatric outpatient sample. It might, therefore, be valuable in future studies to explicitly test the extent to which pain and/or physical limitations mediate the relationship between disease conditions and depressive symptomatology.

The present study examined only a count of disease conditions in relation to physical limitations and depressive symptomatology. One clear direction for future research is to explore the relationships between specific disease conditions and the physical limitations and depressive symptom levels of DP spouses. Just as specific conditions were related to increased pain intensity, there may be certain conditions—or patterns of conditions—that independently contribute to greater physical limitations and/or elevated depressive symptomatology. Indeed, evidence of some differential effects of selected health conditions on depressive symptomatology has been found within some samples, including the HRS 1996 wave (Dunlop et al., 2004), and the mediation study by Kahana et al. (1997). Some research even suggests that specific disease conditions might differentially affect the depressive symptomatology of older men and women (e.g., Palinkas et al., 1990). Research has also shown that some disease conditions, and certain combinations of conditions, are associated with physical limitations of various types and/or different levels of severity (e.g., Fried et al., 1999; Fultz et al., 2003; Verbrugge et al., 1994; Williamson & Schulz, 1995). Careful study of diseases and disease patterns in relation to pain intensity among community-dwelling middle-aged and older adults is likely to provide valuable information to epidemiologists, public health officials, and healthcare practitioners.

Overall, several of the patterns observed for the covariates in this study suggest that there may be indirect—or even numerous—pathways of influence for some of the
demographic and health-related characteristics of DP spouses. For example, increased BMI may affect depressive symptomatology in DP wives via increased physical limitations. In both spouses, more disease conditions may contribute to greater depressive symptomatology by way of greater pain intensity and more physical limitations. Meanwhile, some characteristics—such as age—may be differentially related to physical limitations and/or depressive symptomatology by gender. Most of these potential indirect pathways will require formal testing in future studies. Future research should also explore potential non-linear relationships, especially those involving age and BMI. The collection of findings (and non-findings) involving the majority of covariates lends some additional support to the proposed conceptual model (Figure 5), as the existence of multiple pathways is entirely consistent with the intra-individual relationships depicted in the model.

*The Inter-Individual Relationship between Pain Intensity and Depressive Symptomatology (Paths 4 and 5)*

This study’s conceptual model proposed a straightforward inter-individual (i.e., cross-spouse) relationship between each spouse’s pain intensity and his/her partner’s depressive symptomatology. Specifically, it was expected that higher levels of pain intensity in each spouse would be related to greater depressive symptomatology in his/her partner. Moreover, such straightforward cross-spouse effects for pain intensity were expected even after controlling for the effects of each partner’s own pain intensity as well as his/her other background and health-related characteristics. Investigation of this inter-individual relationship was part of Study Aim 3.

Initial analyses found no evidence of a straightforward cross-spouse effect for
pain intensity. After controlling for DP husbands’ own background and health characteristics, pain intensity, and physical limitations, DP wives’ pain intensity was not significantly related to husbands’ depressive symptomatology. The pattern of findings was identical for DP wives: Husbands’ pain intensity was not independently related to wives’ depressive symptomatology. Thus, no support was found initially for the existence of Paths 4_H and 4_W in Figure 5 (i.e., Hypothesis 7).

However, the results of a planned analysis based on Exploratory Question 10 revealed a more complex inter-individual relationship between the pain intensity of both spouses and the depressive symptomatology of one of the partners—husbands. Wives’ pain intensity did have an effect on husbands’ depressive symptom levels, but the nature and size of this effect depended on husbands’ own pain intensity. This effect will be discussed in greater detail shortly. DP wives’ depressive symptom levels were only related to their own level of pain intensity and not to the pain intensity levels of their husbands. These findings addressed both Study Aims 3 and 4. The observed cross-spouse effect of wives’ pain intensity on husbands’ depressive symptomatology provided qualified support for the existence of Path 4_W (part of Hypothesis 7). However, no evidence was found for a cross-spouse effect of husbands’ pain—i.e., Path 4_H.

This is the first known study to report that the pain intensity levels of both spouses were jointly associated with the psychological well-being of one of the partners. In fact, this is the first known study to investigate such an interaction, largely because this is the first known study to identify a sizeable sample of couples in which both partners report problems with pain. As reported in Chapter 3, Leonard and Cano (2006) did examine an interaction involving the presence of pain in the spouse in a sample of 113
community-residing couples recruited on the basis of one partner having chronic musculoskeletal pain. However, the other construct involved in the interaction was the spouse’s cognitions about the partner’s pain.

Research questions about a potential interaction between spouses’ pain intensity can, of course, only be addressed within such “dual-pain” (DP) couples. Because the investigation of a cross-spouse pain intensity interaction was exploratory, no explicit hypotheses were developed. Findings regarding this interaction effect are discussed next.

The first subsection offers potential interpretations of, and explanations for, the interaction effect observed in DP husbands. The second section considers the pattern of findings observed for DP wives—specifically, the lack of a respondent by spouse pain intensity effect, as well as the lack of any cross-spouse effect of husbands’ pain intensity.

The Husband by Wife Pain Intensity Interaction Effect in DP Husbands (Path 4W)

Depressive symptomatology in DP husbands was associated with the pain intensity of wives, but the direction and strength of this association varied according to the level of pain intensity of husbands. Husbands on each end of the pain intensity spectrum appeared to be influenced by the pain intensity of their wives in opposing ways. Husbands reporting more mild pain displayed increasingly *higher* depressive symptomatology with each higher level of wife pain intensity, whereas husbands with more severe pain showed increasingly *lower* levels of depressive symptomatology with each higher level of wife pain intensity. Among husbands with moderate pain, the depressive symptom levels of those whose wives had higher pain intensity were only slightly, and not significantly, higher than those whose wives had lower pain intensity. In
the absence of directly comparable prior studies, it is difficult to do more than speculate about the potential mechanisms or processes that might explain these findings. Note that any comprehensive explanation for these findings must not only account for each of these three patterns, but must also address the distinctions among them.

**Pattern 1: Husbands With Mild Pain Intensity**

This pattern involves a deleterious effect of wives’ pain intensity on the depressive symptomatology of husbands with mild pain severity. These husbands displayed the cross-spouse effect of pain intensity that had been expected based on prior research—i.e., greater pain intensity in one spouse would be associated with higher depressive symptomatology in the partner, even after controlling for the effects of the partner’s own pain intensity, physical limitations, and other characteristics (the so-called “straightforward” cross-spouse effect of pain intensity). This finding is consistent with the bulk of findings regarding the deleterious effects of one spouse’s pain on the psychological well-being of the partner (e.g., Otis, Cardella, & Kerns, 2004; Roy, 2001), as well as studies showing that increased pain intensity in one spouse is associated with higher depressive symptomatology in the partner (e.g., Kerns & Turk, 1984; Schwartz et al., 1991). However, this expected effect was observed only in husbands with mild pain.

**Pattern 2: Husbands With Moderate Pain Intensity**

Although the depressive symptom levels of husbands with moderate pain evidenced a slight trend in the same (expected) direction, these husbands appeared largely unaffected by the level of pain intensity of their wives. What could account for these two distinct patterns? Perhaps spouses (in this case, husbands) only notice, attend to, or are affected by, their partners’ (wives’) pain when their own pain is at a relatively
low level of intensity. Once one’s own pain reaches a certain level of intensity, there may be an inability to attend to the pain of the partner. This explanation would suggest that cross-spouse effects will be most profound, and perhaps only detectable, when the partner is not experiencing significant problems of his/her own. An *attention threshold* could account for the difference in the depressive symptom levels between husbands with mild and those with moderate pain intensity. It is also consistent with the finding that, among DP husbands, the lowest depressive symptom levels were seen in those with mild pain whose wives also reported mild pain. However, this explanation cannot account for the wholly contradictory pattern seen in husbands with severe pain. Additionally, embracing this *attention threshold* explanation would require developing a convincing argument for why this phenomenon appears to operate only in husbands, and not in wives—this issue will be addressed in more detail in a later section.

*Pattern 3: Husbands With Severe Pain Intensity*

In the third pattern, the depressive symptomatology of DP husbands with severe pain was inversely related to the pain intensity of their wives. This relationship runs counter to the bulk of available evidence regarding the generally negative cross-spouse effects of pain (e.g., Kerns & Turk, 1984; Otis, Cardella, & Kerns, 2004; Roy, 2001). Yet, in DP husbands with severe pain, it would appear that greater wife pain intensity has a salutary effect on husband psychological well-being. If this pattern is taken at face value, two explanations seem plausible. Each is discussed below.

**Explanation 1.** The association of lower levels of husband depressive symptomatology with higher levels of wife pain severity might reflect a “*misery loves company*” perspective among husbands with severe pain. From a theoretical standpoint,
this adage is most closely connected to social comparison theory (Festinger, 1954). According to this theory, individuals have a basic need to evaluate themselves and they achieve this through comparison with others. Schachter (1959) extended social comparison to the realm of emotions. When confronted with a novel threat, uncertainty motivates individuals to look to others to help evaluate their own emotional reactions. Schachter hypothesized that this need for emotional self-evaluation would be most clearly met through affiliation with others who were also facing that threat:

[M]isery doesn't love just any kind of company, it loves only miserable company. (Schachter, 1959, p. 24)

Schachter’s hypothesis has received support from decades of research regarding stress and affiliate selection. Although most research on affiliate choice has focused on the emotions of anxiety and fear, it seems reasonable that these processes could extend to pain, as well. In the current study, DP husbands with severe pain may benefit through affiliation with wives who are also experiencing pain. Perhaps even greater benefits are accrued to husbands whose wives are also experiencing severe pain—potentially accounting for the pattern of reduced psychological distress observed among these husbands. However, such an explanation is speculative, given the lack of information regarding the onset of pain in DP husbands (to evaluate whether it is a “novel” experience), along with their cognitive appraisals of the pain (to evaluate whether it is perceived as a “threat”). The present findings are also challenged by an additional element of Schachter’s extension of social comparison theory. Although this proposition has not been well-tested (Gump & Kulik, 1997), Schachter further hypothesized that individuals seeking affiliation would be influenced by the emotional reactions of those
with whom they affiliate. This suggests that DP husbands with severe pain and their wives should exhibit some level of emotional congruence. However, the pattern of depressive symptoms in these wives is markedly dissimilar to that of their husbands.

Explanation 2. The pattern of decreasing depressive symptomatology associated with increasing wife pain intensity among husbands with severe pain might be explained by a different extension of social comparison theory. Perhaps husbands with severe pain are engaging in *downward (social) comparison* with their wives. The theory of downward comparison holds that individuals who experience negative affect, especially in situations that are difficult to improve through personal agency, can increase their subjective well-being through comparison with a less fortunate other (Wills, 1981). Downward comparison can be an active process (e.g., denigrating, scapegoating, or aggressing against others), or it can occur on a more passive basis (e.g., comparing oneself to a less fortunate other; see Taylor, 1983, for example).

This author was unable to locate any existing literature concerning the use of downward comparison within the marital relationship. Yet, it is reasonable to suggest that it might occur. Applied to the present study, a husband with severe pain may derive a psychological benefit from comparing himself to his wife who is also struggling with pain. For example, a husband may think, “I must not be doing that bad if my wife is also having problems with pain.” Note that the current findings do not completely conform to a conventional downward comparison effect, in which a person (i.e., husband) feels better from observing that someone else (i.e., his wife) is worse off (e.g., “Compared to my wife, I’m doing OK”). Within a conventional framework, a psychological benefit would be expected only in those husbands whose pain intensity was less than the level reported
by his wife—a pattern that was not consistently observed across DP husbands with severe pain. Additionally, because the depressive symptom levels of husbands with mild and moderate pain fail to conform to such expectations, adopting this explanation would require arguing that only husbands with severe pain engage in downward comparison.

**Alternative 1.** Alternative explanations for this third pattern involve making some novel assumptions. For instance, perhaps the wives of DP husbands with severe pain are actively shielding or hiding their pain from their spouses. Such behavior is a form of relationship-focused coping called *protective buffering*, broadly characterized as efforts to protect one’s partner from upset and burden by “hiding concerns, denying worries, and yielding to the partner to avoid disagreements” (Coyne & Smith, 1991, p. 405). Contrary to popular belief, increased communication within the marital relationship does not always contribute to improved spousal well-being. In fact, some studies have shown that protective buffering can confer a psychological benefit to the unaware spouse. For example, wives’ protective buffering has been linked to increased self-efficacy in husbands recuperating after a heart attack (Coyne & Smith, 1994).

Protective buffering has been studied in married couples, although investigations generally have been limited to couples in which one spouse is the designated “patient”—for instance, cancer patients and their partners (Manne et al., 2007), and heart patients and their caregivers (e.g., Suls, Green, Rose, Lounsbury, & Gordon, 1997). Protective buffering might reasonably occur in DP couples. Wives of husbands with severe pain may be motivated to protect their husbands by hiding their own pain. Presumably, these husbands would then display reduced levels of depressive symptomatology.

When applied to the present findings, a protective buffering explanation
encounters difficulties. If the wives of DP husbands with severe pain were engaged in protective buffering, one would expect these husbands to be less negatively affected by increased pain intensity in wives. This suggests more of a flattened slope for depressive symptomatology among DP husbands with severe pain, not a slope that decreases with increased wife pain intensity (the observed pattern). A decreasing slope would only be possible if these wives were not only engaging in protective buffering to hide their own pain, but also over-compensating in some way (e.g., lavishing extra attention on their husbands, pretending to be in better health or spirits than they really are, etc.).

A flattened slope for depressive symptomatology consistent with protective buffering does appear among husbands with moderate pain. Thus, is possible that wives within these couples are engaged in protective buffering. However, protective buffering cannot account for the pattern of depressive symptoms observed in husbands with mild pain. Moreover, protective buffering has been consistently associated with negative consequences for the spouse doing the buffering (e.g., Coyne & Smith, 1991; Langer, Brown, & Syrjala, 2009). When the depressive symptom levels of DP wives are examined in relation to the pain intensity levels of their husbands, the observed patterns do not fully support a protective buffering interpretation—that is, the wives supposedly engaged in protective buffering do not appear to suffer negative consequences.

**Alternative 2.** Other plausible explanations for the joint effect of husband and wife pain intensity on husbands’ depressive symptomatology might arise from considering the patterns more broadly. Note that the joint distribution of the pain intensity ratings of husband and wife creates 9 distinct categories of couples (e.g., Wife-mild/ Husband-moderate; Wife-severe/Husband-mild, etc.; see, Table 8, Results chapter).
When these 9 categories of couples are arranged along a continuum representing DP husbands’ depressive symptom scores, several interesting observations can be made.

As previously noted, the lowest levels of depressive symptomatology were observed among husbands with mild pain whose wives also reported mild pain. Yet, relatively low levels of depressive symptomatology were also seen in husbands with either mild or moderate pain whose wives reported either moderate or mild pain, as well as in husbands with severe pain whose wives also reported severe pain. In contrast, the highest levels of depressive symptomatology were observed in husbands with mild pain whose wives reported severe pain and also in husbands with severe pain whose wives reported mild pain.

When observations about the relative positioning of couples along the continuum of predicted depressive symptom levels in husbands are viewed in conjunction with the three patterns identified earlier, one additional trend is apparent. The negative effect of wives’ pain on husbands’ depressive symptomatology appears to be higher among men whose wives are more dissimilar from them in terms of pain intensity. Conversely, men whose wives report levels of pain intensity more similar to their own appear to be less negatively affected by their wives’ pain. In fact, men who have severe pain appear to experience less depressive symptomatology as wives’ pain intensity ratings become more similar to their own.

Decades of research have led to some adjustments to Schachter’s “misery loves company” hypothesis. Researchers have observed that when given a choice, most people facing a novel threat prefer to associate with others facing the same or a very similar threat, rather than be alone, with others facing no threat, or with others facing a different
threat. These and other findings prompted Gump and Kulik (1997) to offer a clarification of Schachter’s hypothesis:

[M]isery does not love just any company, or just any miserable company.
More precisely...misery loves the company of those in the same miserable situation.
(Gump & Kulik, 1997, p. 317).

Perhaps all DP husbands benefit psychologically from sharing the same or a similar experience (i.e., level of pain intensity) as their wives. A number of interpersonal and/or intra-personal processes could underlie such an effect. For example, husbands may experience an elevation in mood as a result of an enhanced sense of solidarity or a perception that pain is a “shared stress” in the marriage (Zimbardo & Formica, 1963). Perhaps DP couples in which both husband and wife have severe pain have learned how to adapt and are able to provide each other with empathic understanding and mutual support. In these couples, husbands might be encouraged to communicate more openly about their pain. Alternatively, husbands’ psychological well-being may be bolstered by a belief that, since their wives are also suffering from similar levels of pain intensity, they are not “overburdening” the relationship with their own pain. Given that the majority of couples in HRS hail from generations in which gender roles were fairly traditional, these husbands might be especially concerned about the level of burden and dependence that they impose on their wives.

Similar pain intensity levels in spouses might reflect congruence between husbands and wives in terms of their general health status. Congruence in health status may result in greater similarity in the care and support needs of each partner. Exchange theory (e.g., Thibaut & Kelley, 1959) posits that the more equal the exchanges in a
relationship, and the more reciprocal they are, the better the relationship quality and the psychological well-being of both partners. Conversely, Thibaut and Kelly suggested that greater dissimilarity and less reciprocity in exchanges have a negative impact on the relationship and the well-being of each spouse (1959). Lack of reciprocity in the marital relationship—in terms of both receiving and providing support—has been associated with increased psychological distress in both spouses (e.g., Gleason, Bolger, & Shrout, 2003). Thus, greater similarity in pain intensity between partners may provide an emotional uplift to DP husbands.

**Conclusion.** None of the proffered explanations appears capable of explaining all aspects of the interaction between husband and wife pain intensity with respect to DP husbands’ depressive symptomatology. It is possible that each pattern arises from a different mechanism, or even multiple mechanisms. It appears that the psychological well-being of DP husbands is (a) negatively affected by greater pain intensity in either spouse; (b) but the negative impact of the wife’s pain could be: (b1) tempered in some husbands, perhaps as a result of preoccupation with his own pain or wives’ protective buffering, or (b2) exacerbated in husbands with mild pain; and (c) improved in some husbands because their wives also suffer a similar level of pain, or because their wives engage in both protective buffering and overcompensation. Unfortunately, limitations of the available data preclude further evaluation of these explanations.

**Methodological Considerations**

Before moving on, several methodological issues merit mention. First, the husband by wife pain intensity interaction effect on husbands should be interpreted within the context of the range of their depressive symptom scores. On the whole, DP
husbands had fairly low levels of depressive symptomatology, especially when compared to the levels observed in DP wives.

Second, readers should bear in mind that the different patterns of depressive symptomatology identified above were observed across different groups of husbands (defined by their own and their wives’ pain intensity levels). These patterns were not displayed by a single group of husbands observed across different (i.e., changing) levels of their own wives’ pain intensity. Clearly, these findings must be validated in longitudinal studies.

Third, the dual intercept multilevel models used here assume that the underlying interaction of spouses’ pain intensity ratings is “bilinear,” that is, the pain intensity effect of the husband is modeled as a linear function of wife pain intensity and vice-versa (Jaccard & Dodge, 2004). It is entirely possible that spousal pain intensity levels interact in non-linear ways. Along these lines, regression scholars have warned of the danger of falsely concluding that an interaction exists when, in fact, one of the independent variables has a nonlinear relationship with the outcome (e.g., Cohen et al., 2003; Darlington, 1990). Follow-up analyses found no evidence to suggest that the observed interaction effect in husbands was an artifact produced by a non-linear relationship involving one or both spouse’s pain intensity ratings. However, additional modeling using alternative functional forms of pain intensity is warranted.

Fourth, it is noteworthy that the respondent by spouse pain intensity interaction effect was tested in a model that contained respondents’ own physical limitations. Thus, the interaction was observed even in the presence of mediation at the intra-individual level. Recall that husbands’ physical limitations completely mediated the relationship
between their own pain and their own depressive symptomatology. However, the significant interaction effect suggests that husbands own pain still did directly affect their own depressive symptomatology, but the nature of the effect was contingent on the pain intensity level of their wives. It would be useful in future studies to examine the respondent by spouse pain intensity interaction effect in a model without respondents’ own physical limitations.

Fifth and finally, the joint effect of spouses’ pain intensity ratings explained an additional 1% of the variability in the depressive symptomatology of DP husbands. This should not, however, be dismissed as trivial. In most research, interaction effects rarely approach Cohen’s proposed “moderate” effect size level (15%; Cohen, 1988). Effect sizes for interaction effects in the social sciences are typically very small, contributing only 1 to 3% additional explained variance (Aiken & West, 1991, pp. 169-170). Thus, the respondent by spouse interaction effect observed in the present study is of a size that is consistent with social science research. Nevertheless, it will be important to confirm this interaction in future samples of couples in which both spouses experience pain.

**Lack of a Husband by Wife Pain Intensity Interaction Effect and Any Straightforward Cross-Spouse Effect of Pain Intensity in DP Wives (Path 4H)**

No joint effect of husband-wife pain intensity was observed for DP wives. Depressive symptom levels of DP wives were related only to their own level of pain intensity. These findings are inconsistent with expectations developed from prior research documenting the deleterious cross-spouse effects associated with pain in general (e.g., Otis, Cardella, & Kerns, 2004; Roy, 2001), and also specifically with greater pain intensity (e.g., Kerns & Turk, 1984; Schwartz et al., 1991).
Recall, however, that the review in Chapter 3 located a number of studies in which the pain intensity of one spouse was not significantly related to the partner’s depressive symptomatology. Lack of such an association has been documented in studies of chronic pain patients (e.g., Capitolo, 1998; Flor, Turk, & Scholz, 1987) and studies involving community-dwelling older couples selected on the basis of pain and/or a specific disease condition in one spouse (e.g., Druley et al., 2003; Leonard, 2004). As discussed earlier, the sample characteristics and/or methodological features of many of these studies limit their comparability with the present study. For example, the pain intensity ratings of older wives with arthritis were not related to depressive symptom levels of husbands in the study by Druley et al. (2003); however, their sample was comprised of only female-patient couples. Studies in both camps (i.e., those with significant cross-spouse findings and those without) have also varied in their use of the patient’s own pain intensity rating or the partner’s perception of the patient’s pain intensity, but no clear pattern was evident in terms of study findings. It is thus possible that findings of the present study mirror the mixed findings within this area of research.

Nevertheless, the lack of any significant cross-spouse effect involving husbands’ pain intensity in DP wives merits further attention—especially in light of the (conditional) cross-spouse effect observed for wives’ pain intensity in DP husbands. Several plausible explanations for these non-findings are reviewed below. Ultimately, the most tenable explanation(s) must necessarily address both the lack of a joint husband-wife pain intensity effect (i.e., interaction effect) on wives’ depressive symptoms, and the apparent absence of any significant cross-spouse pain intensity effect (i.e., first-order effect for husbands’ pain intensity) on wives’ depressive symptomatology.
Several of the explanations offered for the interaction effect in DP husbands could potentially be applied to the lack of a cross-spouse effect for husbands’ pain intensity on DP wives. Take, for instance, the notion of a possible *attention threshold*—i.e., that one’s ability to perceive and/or attend to pain in the spouse is dependent on one’s own level of pain. DP wives, on average, reported higher levels of pain intensity than their husbands. It is possible that DP wives were so focused on their own pain that they were not able to attend to, and were therefore not affected by, the pain intensity of their husbands. This possibility has been acknowledged by others, as evidenced by a statement attributed to the wife of former President Lyndon B. Johnson:

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It’s odd that you can get so anesthetized by your own pain or your own problem that you don’t quite fully share the hell of someone close to you.
(Claudia A. “Lady Bird” Johnson, n.d.)
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An attention threshold effect may well explain the lack of any cross-spouse effect for husbands’ pain among wives with moderate or severe pain. However, this explanation seems less suitably applied to wives with mild pain. Nonetheless, it is conceivable that all of the levels of pain intensity experienced by DP husbands fell below some threshold of detection (or, perhaps, concern) of DP wives.

Another potential explanation discussed previously in relation to the cross-spouse interaction effect observed in husbands—*protective buffering*—may be relevant to the lack of an observable effect of husbands’ pain intensity on DP wives. Possibly, DP husbands were actively hiding their pain from their wives; that is, perhaps these husbands were intentionally suppressing their expressions of pain, or minimizing their interactions with their wives. Although it was previously suggested that the wives of husbands with
severe pain may have engaged in protective buffering with respect to their own pain (and potentially overcompensating for it, as well), here the suggestion is that all DP husbands might have engaged in protectively buffering. DP husbands may have recognized that their wives were experiencing pain, and thus attempted to hide their own pain. Here again, though, protective buffering falls short of providing an all-encompassing explanation: It seems implausible that all DP husbands would engage in protective buffering. In addition, the pattern of husbands’ depressive symptom levels was not wholly consistent with a protective buffering interpretation (i.e., assuming that protective buffering would exact a noticeable toll on those who practice it).

Rather than intentionally hiding their pain from their wives, perhaps DP husbands do not express their pain in ways that visible to their wives. This might reflect a difference in how women and men exhibit pain, or it could be related to a more generalized gender difference in the extent to which men and women talk about and share emotions and feelings within close relationships (e.g., Brody, 1997). Alternatively, it is possible that DP wives may not be aware that their husbands are experiencing pain or its intensity. It is also possible that wives are aware of their husbands’ pain, but unconcerned by it. No data were available in the present study regarding the extent to which either spouse was aware of the other’s pain. Additionally, no information was collected about either spouse’s verbal and behavioral expressions of pain.

That DP wives’ depressive symptomatology appeared unaffected by the pain intensity of their husbands could also be tied to socially-prescribed gender roles and experiences, especially within the family. Women typically function as caregivers within the family unit (NAC & AARP, 2004; Stone, Cafferata, & Sangl, 1987). There are
numerous ways in which their role and experiences as caregivers might influence wives’ reactions to their husbands’ pain. For instance, wives may expect that their aging spouses will develop pain and may be better able deal with such pain as it arises. Wives may also have more social support (perceived or actual) available to them in dealing with illness and pain in their husbands. It is also possible that, by virtue of their previous caregiving experiences, wives are better able to adapt to their husbands’ pain. DP husbands were, on average, a decade older than their wives. Consequently, most wives probably had prior experience dealing with their husbands’ health issues (and potentially also their pain).

*Methodological Considerations*

It is important to consider whether any methodological features of the present study may have contributed to the lack of cross-spouse effects of husbands’ pain on DP wives. Based on findings from prior studies, it was expected that higher levels of pain intensity in one spouse would be associated with greater depressive symptomatology in his/her partner *even after* controlling for the effects of each partner’s own pain intensity (as well as his/her other background and health-related characteristics). This hypothesis was proposed *despite the fact that the partner’s own pain intensity has not been considered (or statistically controlled) in most prior studies.*

Within DP couples, it is hypothetically conceivable that the cross-spouse effect of one spouse’s pain is not detectable once the variability in the partner’s depressive symptomatology associated with his/her own pain intensity is partialled out. Under this scenario, spouses’ pain intensity ratings could be related to partners’ depressive symptomatology at a bivariate level, but are not associated with any unique variance after partners’ own pain intensity (and other characteristics) is controlled. This might occur if
there was too much overlap in the pain intensity ratings of spouses and in the variability shared with the depressive symptoms of the partner. In such instances, it would be nearly impossible to distinguish the source of any effect (especially using linear modeling).

In this study, the pain intensity ratings of husbands and of wives were significantly (although only weakly) correlated with the depressive symptomatology levels of their respective spouses. There are several sources of data that appear to refute a shared variability explanation for the lack of (or failure to detect) a significant cross-spouse effect of pain observed in wives. First, the adjusted ICC for the pain intensity ratings of spouses was small (.10), and collinearity diagnostics for multivariate models including both respondent and spouse pain intensity ratings revealed no problems. Second, the fixed effect coefficients and standard errors for pain intensity exhibited considerable stability across both the intra- and inter-individual models. This pattern of results is unlikely to have occurred if the pain intensity ratings of spouses were too highly correlated. Third, the small increases in PRE between the intra- and inter-individual models did not hint at the existence of any “hidden” explained variability resulting from substantial shared variability among predictor variables. Fourth, and finally, if a problem with overlapping variability among predictors really did exist, it would be extremely unusual to observe a significant effect involving both spouses’ pain intensity ratings for one spouse and not the other.

The lack of a significant pain intensity interaction effect for wives may be related to an issue related to the joint distribution of husband and wife pain. The number of DP husbands reporting severe pain was fairly small (n = 70), and less than 20% of these husbands had wives who reported mild pain. These small numbers resulted in
considerable imprecision around the estimates of the interaction term coefficient in the prediction of both husbands’ and wives’ depressive symptomatology. Although this imprecision did not impact the statistical significance of the sizeable coefficient estimated in husbands, the SE for wives was large (roughly 3 times the estimated coefficient). This is suggestive of difficulties in the estimation of this effect. Although sensitivity analyses using the logged CES-D8 yielded the same substantive findings (i.e., significant interaction effect in husbands, but not in wives), the SE of the interaction effect estimate was considerably smaller. This suggests that the distribution of the depressive symptomatology of wives within one or more of the joint distribution of pain intensity categories may have been very non-normal. It is possible that a different, or larger, sample of DP couples could yield different findings for the joint effect of spouses’ pain intensity ratings on wives’ depressive symptoms.

The Gender Difference in the Cross-Spouse Effects of Pain Intensity

(Path 4W vs. Path 4H)

The overall pattern of results in the present study suggests that gender moderates the cross-spouse effects of pain intensity. However, the nature of the moderation effect observed in DP couples was quite unexpected. Recall that Hypothesis 9 posited that wives would be more strongly affected by their husbands’ pain (i.e., Path 4H > Path 4W) than vice-versa. Instead, the depressive symptomatology of husbands was associated (albeit in complex ways) with the pain intensity of their wives, but the depressive symptoms of wives’ was not significantly related to their husbands’ pain intensity. In other words, cross-spouse effects of pain intensity were observed in DP husbands, but not in wives. Hypothesis 9 was thus not supported; in fact, findings were in the contrary
direction: DP husbands appeared more affected by their wives’ pain than the reverse.

It is challenging to place this finding within the body of existing research. No prior studies have examined gender differences in the cross-spouse effects of pain intensity within dual-pain couples. Even within the more general literature on the effects of pain on the spouse, there has been little systematic investigation of the extent to which the inter-individual effects of pain vary based on the gender of the spouse (presumably) without pain. Many study only one gender (e.g., Druley et al., 2003). In addition, most prior studies of couples and pain have not been fully dyadic—that is, studies have not routinely examined the characteristics and experiences of both spouses and how they influence outcomes for each.

**Conclusion**

It is difficult to determine exactly how much of the difference in observed cross-spouse effects to attribute directly to gender. Within the couple, DP husbands and wives are potentially distinguishable on the basis of other characteristics, such as roles within the family. It is also important to interpret findings regarding the cross-spouse effects within a larger context that acknowledges the numerous gender differences observed in the present study. It is unclear whether any of the following might have affected the pattern of cross-spouse effects observed in the present study:

DP wives’ higher levels of pain intensity, physical limitations, and depressive symptomatology; a stronger initial direct relationship between their own pain intensity and depressive symptomatology; and partial (vs. total) mediation of this relationship by physical limitations. Finally, it is possible that the gender differences observed in the present study were uncovered because of the novel approach adopted in these analyses.
(i.e., dual intercept model). This is not to suggest that the findings were an artifact of the analytic strategy; rather, the strategy may have permitted a more refined investigation of gender differences than has been attempted in prior studies. More research is needed to investigate the provocative findings of the joint effects of the pain intensity of both spouses in predicting levels of psychological distress in husbands.

**Absence of Cross-spouse Mediation by Physical Limitations (Paths 5_w & 5_h)**

*No Mediation*

There was no evidence that spouses’ physical limitations were related to partners’ depressive symptom levels. Furthermore, including the spouse’s physical limitations in prediction models did not substantially alter the relationship between the spouse’s pain intensity and the partner’s depressive symptomatology. Consequently, these findings offered no support for Hypotheses 8a and 8b concerning the mediating role of the spouse’s activity limitation in the cross-spouse pain-depressive symptom relationship.

Recall that the review in Chapter 3 identified only one prior study that appeared to address the possible mediating role of activity limitation in the spouse. In the Flor et al. (1987) study of male pain patients at the VA and their wives (also cited above), multivariate results seemed to suggest that husbands’ activity limitation did not mediate the cross-spouse relationship between his pain intensity and wives’ depressive symptomatology. Also recall, however, that several methodological issues (e.g., no bivariate correlations presented; the inclusion of other potential mediating variables—e.g., husbands’ coping, wives’ perceptions of control, and wives’ marital satisfaction—in the model) raised doubts about the extent to which that study provided a reasonable test of the cross-spouse mediation by activity limitation.
Conclusions about the lack of mediation by the spouse’s physical limitations should be viewed with caution in light of the observed interaction effect between DP spouses’ pain intensity ratings. Each spouse’s physical limitations were included as predictors in the final models for the depressive symptomatology of each partner (i.e., Models 8 and 9). However this modeling strategy may not have adequately evaluated all possible mediation effects; for example, perhaps wives’ physical limitations mediated only one part of the complex relationship between wives’ pain intensity ratings and husbands’ depressive symptoms. It was beyond the scope of the present study to test for a potential mediated-moderation effect that was also moderated by gender. Future research might evaluate this possibility through a multi-group SEM model in which couples are grouped according to husbands’ pain intensity level. Additional insights might also emerge from varying the order in which partner and spouse characteristics are entered into multivariate prediction models. At the same time, however, it may be constructive to re-examine the utility of the inter-individual activity limitation model in light of the (non-)findings from the present study. This re-examination and suggestions for future research are presented later in this chapter.

No Cross-Spouse Effects Involving Physical Limitations

In addition to a lack of evidence supporting a mediating role for the spouses’ physical limitations, this study also did not observe any evidence of a cross-spouse effect involving the physical limitations of either DP husbands or wives. Lack of a relationship between the physical limitations of the spouse and the depressive symptomatology of the partner is surprising; however, this specific phenomenon has not been that well studied.

In light of the findings from studies in which activity limitation has been
considered more broadly, the lack of a cross-spouse effect for spouses’ activity limitation in the present study could be attributable to how this construct was conceptualized and measured. Activity limitation was intentionally conceptualized and operationalized as limitations in performing basic physical activities and abilities (e.g., climbing steps, stooping/ crouching, walking several blocks). Although such basic physical limitations appear to play an important role in DP spouses’ adjustment to pain at the intra-individual level, it is possible that such limitations are too “minor” to be of consequence to the partner. Perhaps limitations of this nature do not warrant the partner’s concern or distress. Or, perhaps they must reach a certain threshold before they merit the partner’s attention and concern. Evaluating these various possibilities should be a priority in future research. Details regarding specific study ideas are presented later in this chapter.

Findings Regarding Covariates

Generally speaking, little change was observed between the intra- and inter-individual models in terms of significant covariates of depressive symptoms in DP spouses. Neither of the health-related characteristics of either spouse (BMI, number of health conditions) was significantly related to depressive symptomatology in the other partner. Moreover, adding the spouse’s pain intensity and physical limitations and the joint pain intensity interaction term did little to alter the observed relationships involving individual and couple level background variables and the respondent’s own health characteristics.

Recall that significant predictors of depressive symptomatology in the final intra-individual models included: Years of education (in both husbands and wives); household income (in husbands only); age (in wives only); and body weight (in husbands only).
These same relationships were preserved in the final cross-spouse model that included all potential predictors (Model 8); the relative strength of these effects was also maintained. DP husbands’ depressive symptom levels were inversely related to their body weight (i.e., the “jolly fat” hypothesis discussed previously), household income, and years of education. Increased education and age significantly predicted (reduced) depressive symptoms in DP wives.

Findings for a few covariates differed slightly in the final, trimmed inter-individual model (i.e., Model 9 that included a reduced set of predictors—only key study variables and predictors that had been significant for either husbands or wives in a previous model). Results involving body weight (for husband) and years of education (for both) remained unchanged. Household income remained a significant predictor of (lower) depressive symptoms among husbands, but it also emerged as a significant predictor of DP wives’ (lower) depressive symptomatology. Increased age was still associated with lower depressive symptomatology for wives, but in this final model it also predicted lower depressive symptomatology in DP husbands.

The change involving household income between the full and trimmed inter-individual models was not surprising, given that household income was significantly related to DP wives’ depressive symptomatology in several early intra-individual models. Household income was weakly, but significantly, negatively correlated with non-White race and others living in the household. These two variables were trimmed from the final inter-individual model. Thus, it is likely that removing these variables also removed small amounts of overlapping variability between income and these two variables in relation to wives’ depressive symptomatology.
Although age had not been associated with DP husbands’ depressive symptomatology in any of the intra-individual models, age in husbands was slightly (and significantly) correlated with the two background variables trimmed from model 9 (non-White race and others living in the household). Thus, age may have emerged as a significant covariate of depressive symptoms in DP husbands because of the shared variance that was removed when these other background variables were trimmed from the final model. As reviewed previously, there is a lack of agreement in the literature regarding the true nature of the relationship between age and depressive symptomatology. However, the finding that increased age was associated with slightly lower depressive symptom levels among DP husbands in a multivariate context is consistent with some prior literature (e.g., Murrell et al., 1983), and also mirrors the pattern seen in DP wives.

Lack of Cross-Spouse Effects Involving Health-Related Characteristics

It is noteworthy that in these cross-spouse models, there was no evidence of any cross-spouse effects involving the health-related characteristics of either spouse (number of disease conditions, body weight) and the depressive symptomatology of his/her partner. Together, these health characteristics of the spouse accounted for no unique variance in the depressive symptomatology of either DP husbands or wives. Furthermore, including the spouse’s characteristics in these models did not substantially alter the relationships between the respondent’s own characteristics and his/her depressive symptomatology.

Unlike what was observed in the intra-individual models, the pattern of relationships observed across the multivariate cross-spouse models did not suggest that these spouse health characteristics affected the partner’s depressive symptomatology.
indirectly. That is, there was no evidence that the effects of each spouse’s health characteristics on the partner’s depressive symptoms were potentially mediated by the spouse’s pain intensity and/or physical limitations.

It is difficult, however, to equate these (non-)findings with those of prior studies. Rather than examining the effect of one spouse’s BMI or number of disease conditions on the depressive symptomatology levels of the partner, many prior studies have just examined the level of spousal concordance on these measures. Other studies appear to have not tested (or have not reported) the cross-spouse effects of such characteristics (e.g., Bookwala et al., 2008; Pruchno et al., 2009; Robb et al., 2008; Strawbridge et al., 2004). For example, in their study of end-stage-kidney patients and spouses, Pruchno et al. (2009) presented descriptive data regarding the health status of both partners (including number of comorbidities). However, the potential cross-spouse effects of these comorbidities were not included in multivariate models of patient and spouse depressive symptomatology levels. These omissions may reflect the fact that such constructs were not the primary cross-spouse effects of interest of these studies.

**Implications for Practice, Education, & Policy**

Several key findings from this study have implications for the practice of social work, the education and training of social workers and other health care professionals, and the formulation of social policy. Recommendations in each of these three areas are presented below.

**Social Work Practice Implications**

This is the first known study to empirically document the existence of co-occurring pain in married couples drawn from a representative sample of community-
dwelling adults in mid and later life. Social work practitioners need to be alerted to the possibility that pain may be a problem for both partners in aging couples. In the present study, dual-pain couples comprised eight percent of the married couples participating in the 1998 wave of HRS. Based on these data—and assuming that HRS couples are, indeed, representative of the population of community-dwelling, aging adults in the U.S.—a practitioner working with adults in mid and later life might reasonably expect to encounter 1 dual-pain couple among every 12-13 community-dwelling married couples that he/she serves. According to this research, these couples are likely to be slightly older, have more health problems, and have fewer resources (e.g., education, income, wealth) than couples in which only one or neither spouse reports pain.

Taken as a whole, the findings from the present study highlight the importance of expanding the psychosocial assessment of middle-aged and older individuals to focus on the experience of pain and its impact on quality of life—both for the individual and his/her marital partner. At the intra-individual level, clinicians need to assess the antecedents and risk factors for pain. Most are likely familiar with pain in the context of musculoskeletal diseases (e.g., osteoarthritis, rheumatoid arthritis). However, the present study also found increased pain intensity associated with lung disease (in both spouses) and heart disease (in husbands). These diseases may be overlooked sources of pain in later life, and practitioners need to be sure to assess pain among clients reporting these conditions. Moreover, heart disease should not be overlooked as a potential source of pain in women; the relatively small number of wives with heart disease may have made detecting a significant relationship more difficult. A similar caution applies to diabetes, as there was a trend toward higher pain intensity ratings among wives with diabetes in
this sample. At the same time, professionals should not assume that pain is always tied to a specific disease or health condition. Roughly 10% of husbands and 12% of wives in these dual-pain couples did not report the presence of any doctor-diagnosed disease condition. Furthermore, reports of arthritis- and heart-related pain were far from universal among the husbands and wives who reported these diseases.

Findings with respect to the relationships among pain, physical limitations, and depressive symptomatology at the intra-individual level may be useful in the design of prevention and intervention efforts aimed at reducing depressive symptomatology in middle-aged and older adults. Pain intensity was strongly related to physical limitations, and physical limitations at least partially mediated the relationship between pain intensity and depressive symptomatology in both husbands and wives. This mediation model (based on the proposed conceptual model, with roots in Williamson’s activity limitation theory) suggests two distinct opportunities to intervene “upstream” to prevent increased depressive symptomatology in adults aging with pain.

First, symptoms of depression are likely to be reduced by efforts to improve pain management. Social workers can play an important role here, not only by referring clients for appropriate medical evaluation and treatment, but also by advocating on behalf of clients (e.g., with physicians, health care institutions, and insurance companies) for prompt, aggressive, and appropriate pain management. With proper training, social workers can also provide a range of non-pharmacological interventions to clients with pain, including cognitive-behavioral and supportive psychotherapy, relaxation and meditation training, hypnosis, and biofeedback.

The second “upstream” intervention to prevent or reduce depressive
symptomatology among those with pain concerns the role of physical limitations as a mediator of the pain-depressive symptom relationship at the intra-individual level. Findings from the present study suggest that interventions to reduce physical limitations or improve physical functioning may help to reduce depressive symptomatology. In addition to providing clients with referrals to physical and occupational therapy, social workers can encourage clients to engage in appropriate levels of physical activity and exercise. Accumulating evidence now suggests that, in addition to helping to reduce depressive symptomatology (e.g., Blake, Mo, Malik, & Thomas, 2009), increased physical activity can also lead to reductions in pain and increased functional capacity among aging adults (Penedo & Dahn, 2005; Roddy et al., 2005). Some clients may benefit from counseling to reduce barriers to physical activity. Others may require support from social workers in problem-solving, breaking down tasks and pacing themselves in everyday activities.

Clients might also benefit from interventions that are more “downstream” in nature. When pain and physical limitations are already present, a variety of interventions may help to reduce depressive symptomatology in midlife and older clients. Cognitive-behavioral therapy, coping skills training, and non-pharmacological pain management techniques can be offered to clients in individual and/or group settings. The majority of social workers are already skilled in many of the general techniques that underlie such interventions. However, most would need supplemental education and training to apply these techniques with individuals and couples affected by pain. Recognizing that adaptation reflects interaction between person and his/her environment, social work practitioners may wish to balance interventions aimed at individuals with interventions
that are aimed at reducing the demands of the environment. To that end, clinicians working with later life couples should increase their familiarity with principles of universal design and the range of aids that can be used to support independence.

Regardless of the specific intervention target within the pain-physical limitations-depressive symptom relationship, social workers should understand that intervention is critical. Pain problems and depressive symptomatology are often observed concurrently among community-dwelling adults (e.g., Ohayon, 2004; Magni et al., 1990, 1993; McWilliams et al., 2003). Moreover, such comorbidity appears to increase with age (e.g., Onder et al., 2005; Mossey et al., 2000; Verhaak et al., 1998). Individuals who have both pain and depression have been shown to have worse clinical outcomes than those who have either pain or depression (Arnow et al., 2006; Geerlings et al., 2002; see also review by Bair et al., 2003). There is also increasing evidence that the co-existence of pain and depression makes treating each harder. Pain has been associated with reduced responsiveness to treatment for depression (Bair et al., 2004; Kroenke, Shen, Oxman, Williams, & Detrich, 2008), and depressive symptomatology has been linked to poorer treatment outcomes in pain patients (e.g., Burton, Til lostson, Main, & Hollis, 1995; Epker & Block, 2001). Consequently, early intervention may disrupt the associations among pain, physical limitations, and depressive symptoms and improve well-being.

Multiple findings from this study suggest that social workers should pay close attention to gender in their work with aging clients who experience pain. Practitioners need to recognize that there may be important gender differences in the experience of pain and at various points along the pathways through which pain influences depressive symptomatology. For example, the relationship between pain intensity and depressive
symptoms was considerably stronger in DP wives than in husbands. Moreover, physical limitations only partially mediated the pain-depressive symptom relationship in wives. Consequently, interventions may need to more aggressively target pain intensity in wives. Practitioners should also be alerted to the possibility that some gender differences in the effects of pain on depressive symptoms could also be due to inadequate assessment or treatment of pain in women. Finally, higher BMI in DP wives was associated with more physical limitations, even after controlling for pain intensity level, and the overall pattern of results raises the possibility that physical limitations may mediate the relationship between higher body weight and greater depressive symptomatology. Should this finding be replicated in future studies, clinicians may want to incorporate weight reduction into interventions designed for women with pain.

The intra-individual findings from this study have specific implications for treating husbands, as well. Analyses showed that physical limitations fully mediated the relationship between pain intensity and depressive symptomatology in husbands. This suggests that men may be especially psychologically vulnerable to declines in physical functioning. Practitioners working with middle-aged and older men in pain would do well to emphasize interventions that help to preserve physical functioning (where possible) and/or enhance coping and adaptation to physical declines.

Gender also emerged as an important factor with respect to the inter-individual (cross-spouse) effects of pain. Although husbands’ depressive symptomatology appeared to be influenced by their wives’ pain intensity, wives’ depressive symptomatology did not appear to be affected by the pain intensity of their husbands. Among husbands, the effect of wife pain intensity varied, depending on the level of their own pain intensity.
Husbands with lower pain intensity appeared to be negatively affected by increased levels of pain intensity in their wives. An opposite effect was seen in husbands with higher pain intensity—depressive symptom levels in these husbands appeared to decline with increasing levels of wife pain intensity.

The clinical implications of these finding may depend, in part, on which of several possible mechanisms accounts for these different effects. However, in the absence of any conclusive explanation, social work practitioners should regard these findings as a directive to assess pain in both partners in married couples. Failure to do so may result in clinicians missing potentially important effects of pain on both partners. Findings from this study raise the possibility that, in couples in which both spouses have pain, the effect of one spouse’s pain intensity on the well-being of the other partner may be anything but straightforward. In working with such couples, clinicians should be alert to the ways in which gender might influence the effects that one spouse’s pain has on the psychological well-being of the partner. These findings also suggest that the cross-spouse effects of pain might depend on the partner’s own level of pain. In addition to assessing pain in both spouses in aging couples, clinicians will want to explore if, and how, different pain intensity levels of spouses “play out” within the context of marriage. To this end, clinicians should examine each partner’s perceptions of, and reactions to, his/her own pain, as well as the pain of his/her partner.

In this study, both spouses were found to have pain in roughly 20% of those midlife and older couples in which one of the spouses reported pain. Social workers who routinely encounter aging adults who experience pain—in acute care settings, outpatient rheumatology and orthopedic clinics, and, of course, in pain management clinics and
hospice/palliative care programs—should be aware of the co-occurrence of pain in married couples. There is a good chance that a substantial number of the married patients on their caseloads have a spouse who is also experiencing significant pain. Based on the findings from the current study, spouses in dual-pain couples are likely to be of advanced age and suffer from numerous health conditions and physical limitations. These couples are also likely to have limited personal and financial resources. In these couples, both spouses may be at increased risk for elevated symptoms of depression.

Findings from this study suggest that practitioners consider targeting couples in the design and delivery of health promotion and disease prevention efforts. Spouses have been included in chronic pain treatment programs for several decades, but there is now accumulating evidence of the value of spouse involvement in chronic disease management programs (e.g., for arthritis, diabetes; see Martire et al., 2010, for review). For example, randomized-controlled studies with osteoarthritis patients have documented increased efficacy associated with programs that specifically target both patients and spouses (e.g., Martire, Lustig, Schulz, Miller, & Helgeson, 2004). The present study would suggest that, for a sizeable portion of couples, both spouses could be regarded as the “patient” or “person with pain” in such programs. Clinicians can help such couples to set reasonable expectations regarding pain and physical limitations—both for themselves, and for their spouses. By offering programs geared toward couples, clinicians can capitalize on, and potentially (re-)shape, the natural processes of social influence that operate within close relationships such as marriage (Lewis et al., 2004). Couple-level interventions may be especially helpful in increasing adoption of, and adherence to, healthy habits like increased exercise and improved diet. For some, including the spouse
may help to reduce barriers to physical activity.

Finally, social workers are often called upon to educate clients about basic mental and physical health matters. Findings from the current study underscore the need for social workers to educate their middle-aged and older clients about pain and the intra-individual relationships among pain intensity-physical limitations-and depressive symptomatology. In this author’s experience, providing information about the close association between pain and depressive symptomatology can sometimes encourage (initially reluctant) clients to consider talking to their physicians about taking an antidepressant medication to “help with pain.” Clients can also use this information to better advocate for their needs in encounters with medical professionals. In light of the findings from the present study, it is also important to make clients aware of the potential impact of one spouse’s pain on the other’s well-being. To this end, it may be helpful to solicit independent reports from each spouse regarding a recent occasion in which he/she was aware that his/her partner was experiencing pain (or an exacerbation of pain). Clinicians can then use these reports to help illustrate potential cross-spouse effects of pain. In this way, clinicians can build on the “lay” understanding—often intuited, but rarely articulated—among married persons regarding how each affects, and is affected by, what is going on with his/her partner.

Implications for the Education & Training of Social Workers and Other Health Care Professionals

Whereas the education of patients and the public help to shape the demand for quality pain care, issues related to the supply of appropriately-trained care providers require attention as well.
On the basis of the available evidence, the committee believes that practitioners are not adequately trained to manage patients with pain, despite increased attention to this area in recent years. (IOM, 1987, p. 283)

[Federal agencies], accrediting organizations, and undergraduate and graduate health professions training programs should improve pain education curricula for health care professionals. (IOM, 2011, Rec. 4.2, p. 210)

As the dates of the above quotations make clear, progress in improving the education of health care providers regarding pain has been slow. The Institutes of Medicine recently published a report entitled, *Relieving Pain in America: A Blueprint for Transforming Prevention, Care, Education, and Research*. (IOM, 2011). The report was commissioned by the Department of Health and Human Services, through the NIH, as part of the federal Patient Protection and Affordable Care Act of 2010 that was mentioned in Chapter 1 of this dissertation. The IOM convened a multidisciplinary committee of pain researchers and practitioners and charged them with assessing the current status of, and developing recommendations to improve, pain care, education, and research in the U.S. The committee authoring the 2011 report pointedly mentioned the existence of several prior reports on this issue, many of which had even been issued by the IOM. The committee further noted that, in completing the report, they “saw little evidence of progress toward [the] well-articulated goals and extensively documented findings of the past” (IOM, p. 23).

The 2011 IOM report recommended that pain education be improved across all undergraduate and graduate health professions training programs. Moreover, the need to improve pain education was specifically noted at all levels—undergraduate, graduate, and
post-graduate health professional training programs. The IOM called on the federal and state agencies that fund education and training programs, as well as the agencies and organizations that accredit programs, to direct increased resources and attention to improving pain education. The IOM also recommended the creation of specialized training programs to create cadres of professionals with advanced expertise in pain care. Although the discipline of social work was not mentioned by name, social work is clearly contained within several broad rubrics employed by the IOM, including “people in the healing professions,” (p. 1-4), “other health professionals who participate in the delivery of pain care” (p. 4-26), and “health care providers who help care for [people with pain]” (p. S-4). In addition, the Council on Social Work Education is identified as an accrediting body that should be involved in improving the pain-related education of health care professionals (IOM, 2011, Table 6-1, pp. 272-275).

The extent to which pain, pain management, and the relationships among pain, physical limitations, and depressive symptomatology are covered in contemporary social work education is currently unknown. As reported in Chapter 1, there is a startling lack of attention to pain in general within the social work literature, and references specific to education or training on pain and/or pain management are scarce. Mentions of pain and pain management in the social work literature occur almost exclusively within the broader contexts of oncology social work or end-of-life/palliative care—content areas that have also been under-represented within social work publications.

Although social work scholars often lament the lack of adequate pain-related education and training of social workers, few empirical studies document the extent to which pain is incorporated into social work education at any level. Data regarding social
worker knowledge, competencies, and/or evaluation of education/training related to pain and pain management are limited to a handful of studies conducted with licensed social work practitioners working in oncology (e.g., Jones, 2005; Zebrack et al., 2008) or hospice/palliative care (e.g., Oliver et al., 2009). Findings from these studies, along with those of an older survey of medical social workers in Canada (Sieppert, 1996), were reviewed in Chapter 1. Most importantly, all found evidence of serious deficits in the knowledge and skills of practicing social workers related to pain and pain management.

Available evidence suggests that pain and pain management are not topics routinely incorporated into post-baccalaureate social work education. In 2003, Kramer and colleagues reviewed 50 leading social work textbooks for content related to end-of-life care (Kramer, Pacourek, & Hovland-Scafe). As a critical element of end-of-life care, “pain, distress, and suffering management” was one of the content areas tracked in the review. Whereas less than three percent of the total content across all textbooks was devoted to end-of-life care, coverage specific to pain and symptom management was even more meager. Pain-related content was found in only 14% ($n = 7$) of textbooks. Forty-eight total pages were devoted to pain and symptom management, corresponding to less than one percent of all textbook pages. Existing content covered less than 5% of the topics outlined in published guidelines for education related to pain in end-of-life care, and only 1% of topics was judged by reviewers as “adequately covered.”

In order to inform the development of specific educational efforts targeted to social workers, a useful first step would be to survey Baccalaureate and Masters-level social work programs regarding pain-related content within existing curricula.
The incorporation of pain and pain management content into the social work curriculum can be aided by numerous existing resources, including a variety of curriculum guides and guidelines for clinical practice. Examples include:

1. *International Association for the Study of Pain (IASP) Model Core Curricula Regarding Pain and Symptom Management.* This inter-professional curriculum outline covers the multidimensional nature of pain, pain assessment, pain management, and specific clinical conditions. It is available at the IASP website: [http://www.iasppain.org/Content/NavigationMenu/GeneralResourceLinks/Curricula/Interprofessional/default.htm](http://www.iasppain.org/Content/NavigationMenu/GeneralResourceLinks/Curricula/Interprofessional/default.htm)

2. *Promoting Excellence in Pain Management and Palliative Care for Social Workers* (Otis-Green, Lucas, Spolum, Ferrell, & Grant, 2008). This training program emphasizes core competencies in palliative care (as presented in Gwyther et al., 2005). The program includes considerable content on pain and pain management.

3. Various clinical practice guidelines are available to health care professionals. These are published by a variety of sources, including the American Geriatrics Society (AGS, 2002, p. S-210), the American Psychological Association (e.g., APA, 2011), and others. Many include assessment tools and sample questions to ask in clinical interviews with persons who have pain.

Findings from the present study suggest some additional content areas that should be incorporated into curricula. First, social workers should be trained in the assessment of the three central constructs included in this study—pain, activity limitation (viz., physical limitations), depression and depressive symptomatology. While most social
workers are no doubt familiar with pain as a symptom of cancer and arthritis, findings from this study suggest that they be educated about other possible sources and types of pain (e.g., lung disease, heart disease). However, training should also emphasize that the experience of illness and disease varies across individuals, and that pain is not universally associated with certain disease conditions. Moreover, treatments that target a specific condition may not provide adequate pain management and, in some cases, might actually cause or contribute to a client’s pain (e.g., chemotherapy for cancer). The multimorbidity observed in DP spouses in this study might also serve as a caution that pain may be associated with multiple disease conditions and possibly certain combinations of conditions. It is important that information also be presented regarding other potential sources of pain in middle-aged and older adults, beyond the limited disease conditions assessed in the current study. At the same time, social workers should understand that pain in older adults may not be traceable to the existence of any (identifiable) disease condition. Regardless of known or presumed cause, social workers would do well to regard client reports of pain as valid and informative.

Evidence-based reviews of interventions that target pain might be introduced as required reading within courses related to social work in health care settings. If not already part of the content for courses that address social work practice with couples and families, the phenomenon of couple-level concordance or similarity across various aspects of health should be discussed. Additionally, effort should be made to expose social work students to theories related to the influence of social contextual factors on health. Social work practice courses could also be infused with examples of successful physical and mental health interventions that have been designed and used with aging
Most work done in contemporary health care is conducted in multidisciplinary settings. Thus, it is imperative that social workers be knowledgeable about the expertise and scope of practice of professionals in other disciplines. Inter- and multi-disciplinary teams have long been the standard model of care within specialty pain clinics and centers, and are prominently featured in the care provided to aging veterans at VA medical centers. There has also been an increasing emphasis on integrated and team-delivered care within primary and long-term care settings. Thus, training for social workers should include the skills necessary to participate effectively in multidisciplinary teams.

Continuing education represents one end of the continuum of social worker education and training. Michigan is currently the only state that requires social workers to pursue post-degree, continuing education regarding pain and pain management. Beginning in late 2009, individuals seeking to renew a social work license in Michigan must complete one hour of continuing education on the topic of pain and/or pain management (Michigan Social Workers Continuing Education Collaborative [MSWCEC], 2009). Course content suggested by MSWCEC includes the psychology of pain, behavior modification, and stress management.

The IOM report makes clear that pain management is a responsibility shared by all health care professionals:

**Effective pain management is a moral imperative, a professional responsibility, and the duty of people in the healing professions.**

(IOM, 2011, p. 22)

However, as expressed in the title of their 2011 report, the IOM Committee clearly believes that improving pain management in the U.S. will require a “cultural
transformation” in how pain is currently understood, treated, and prevented. Such a transformation will most certainly require improvements in the pain-related education and training of all health care providers. Substantial forward progress in this area has been made by NIH. In late 2011, the NIH Pain Consortium issued a Request for Proposals to establish at least 10 Centers of Excellence in Pain Education (CoEPEs) throughout the U.S. (Altarum Institute/Palladian Partners, 2011). Reacting to the perceived inadequacy of current education and training related to pain, the NIH envisioned CoEPEs as providing “leadership for change in the pain management education of health professionals nationwide” (p. 2). CoEPEs are tasked with developing, integrating, evaluating, and distributing inter-professional curriculum resources in pain management for medical and health professional schools. In funding “hubs” for curriculum development and dissemination, NIH appears to be modeling the CoEPEs after the successful Geriatric Education Centers (GEC) program established through the federal Bureau of Health Professions.

In May, 2012, 12 health professions schools were designated as CoEPEs, and will receive funding for three years (NIH Pain Consortium, 2012). Some centers will develop curricula focused on specific topics and/or populations; for example, the University of New Mexico CoEPE will focus on pain management in older adults (University of New Mexico, 2012). All centers are housed in schools of medicine, dental medicine, nursing, and/or pharmacy; however, some centers plan to build curricula that cut across several health professional training programs. Unfortunately, only one center specifically includes a school of social work—the University of Washington (University of Washington, 2012). Hopefully, faculty in schools of social work across the U.S. will take
advantage of the work being done at the CoEPEs, and adapt pain education materials for inclusion in social work education.

Social Policy Implications

Findings from the present study have several identifiable implications for the formation of social policy. This study contributes to the currently limited evidence base regarding the sheer number of families affected by pain. Over two-fifths of the aging married couples participating in one wave of a nationally-representative panel survey of midlife and older adults were affected by pain in one or both partners. These data could add to the groundswell of support for the development of a federal pain care policy.

Results from this study suggest that pain is a major contributor to physical limitations among married persons in mid and later life. Study findings at the intra-individual level were consistent with a model in which the effects of pain on depressive symptomatology are at least partially mediated through increased physical (or activity) limitations. These findings are in line with much prior research (e.g., Williamson & Schulz, 1992a; see also Table 1), including several longitudinal studies (e.g., Gureje et al., 2001). In the presence of continued empirical support, this mediation pathway may prove useful in the design of public health programs to promote physical and emotional well-being among older adults. A marked and consistent relationship was observed between pain intensity and physical limitations in both DP spouses in this study. This was true, even after adjusting for the effects of age, SES, and number of disease conditions. These findings suggest that increased attention paid to pain prevention and management efforts may yield benefits in terms of the maintenance of physical functioning and reduced psychological distress among aging persons. These findings
also highlight the importance of screening individuals with pain for depressive symptoms and assessing their physical limitations.

If future research can isolate pain as a causative factor in the onset and maintenance of impairments in physical functioning, pain will demand increased attention in policy making. Significant policy implications are tied to the impact of pain on workforce participation and on the provision of health and social services. To the extent that pain contributes to impairments in physical functioning among those in middle age, pain may alter workforce availability and participation rates. Pain may also limit the capacity of individuals to maintain employment throughout midlife.

Increased pain and physical limitations among those in middle age may increase the need for health care and other assistive services in later life. Although health care reform efforts are still evolving, it is possible that current decisions could negatively affect future cohorts as they progress through middle age. Efforts to curb health care spending have increasingly focused on revisions to the Medicare program. Serious consideration is likely to be given to raising the age of eligibility for Medicare, as this would also help to align Medicare eligibility with increases in the retirement age being implemented through Social Security. Should this occur, growing numbers of persons will face difficult choices regarding continued employment, and many could have trouble navigating the “eligibility gap” between substantive employment and access to Medicare benefits. And, as highlighted by the current study, policy makers need to consider not just the needs of aging persons in weighing different policy options, but also the needs of aging couples.

Adults in mid and later life are populations of immense importance from a public
policy perspective. Largely because of the aging of the baby boomers, the population of older adults in the U.S., is projected to more than double by 2050—to 88 million, from 40 million in 2010 (Vincent & Velkoff, 2010). The U.S. is facing a rapidly growing population of older adults, many of whom will suffer significant pain problems. This has implications for the utilization of health care resources and health care financing. It is likely that the majority of these individuals will receive their health care under the auspices of the Medicare program, and many more will be eligible for Medicaid. Accordingly, social workers should be aware that,

The federal Medicare program bears fully one-fourth of U.S. medical expenditures for pain; in 2008, this amounted to at least $65.3 billion, or 14 percent of all Medicare costs. In total, federal and state programs—including Medicare, Medicaid, the Department of Veterans Affairs (VA), TRICARE, workers’ compensation, and others—paid out $99 billion in 2008 in medical expenditures attributable to pain. (IOM, 2011, Executive Summary, p. 1)

Social workers should see it as their responsibility to advocate for and ensure effective pain management for their clients. To this end, social workers need to be knowledgeable of, and able to reference, current evidence-based standards and guidelines for pain management. Social workers need to make sure health care organizations, insurance companies, managed care organizations, and federal and state health care programs (e.g., Medicare, Medicaid) recognize the importance of adequate pain assessment and care. It is also important that social workers encourage awareness of pain as an important quality of life issue among those directing and staffing the numerous
federal, state, and local agencies and organizations that plan and coordinate services for aging adults (e.g., the federal Administration on Aging, state-level departments on Aging, multi-county coordinating and planning bodies such as Area Agencies on Aging, municipalities, senior centers, and public recreation programs).

Many institutions and accrediting bodies appear to have followed the lead of the Veterans Administration in adopting pain as a 5th vital sign—i.e., pain is something to be assessed, recorded, and monitored in all patient interactions. While assessment is important, it is critical to ensure that providers are adequately addressing pain when it is reported and not just measuring it because they have to, and then doing nothing about it. Despite widespread adoption of routinized assessment practices, the application of industry accreditation standards, and the dissemination of guidelines for the clinical management of pain, observers throughout North America have questioned whether pain is any better managed now than it was in prior decades (e.g., IOM, 2011; Michigan Advisory Committee on Pain and Symptom Management, 2010; Sawyer et al., 2008). Social workers can play an important role in acting as advocates for institutional change in this regard.

There is some evidence to suggest that the problem of pain may be growing within the adult population in the U. S. A recent analysis conducted for the IOM Pain Report (2011) revealed a steady increase in pain prevalence across three NHANES surveys, beginning in 1999/2000 and ending in 2003/2004 (when NHANES stopped collecting these data). Moreover, this trend was observed in almost all segments of the population, whether the data were broken out by age, gender, race, or SES. Experts also believe that several demographic and societal trends will result in the continued increase
in pain prevalence (IOM, 2011). Influential trends include the growth of the middle-aged and older adult populations; increases in the prevalence rates of several diseases and conditions associated with pain (e.g., arthritis, cardiovascular disease, obesity); improvements in modern medicine that allow people to live longer, even with serious illnesses; and greater public awareness of pain and treatment options (IOM, 2011).

Gender featured prominently in the results of the current study. In the initial sample of all HRS couples, wives were more likely to report being troubled by pain than husbands. Within DP couples, wives reported slightly higher ratings of pain intensity than husbands. Moreover, compared to their husbands, DP wives’ depressive symptomatology was more strongly affected by higher levels of their own pain intensity—an effect that remained sizeable even after controlling for the mediating effect of physical activity limitation. The bulk of findings from this study are consistent with prior work suggesting that women are disproportionately affected by pain and at increased risk for inadequate pain assessment and treatment (e.g., LeResche, 2011). In light of such data, social workers may want to ensure that institutional and organizational policies adequately address the pain care needs of midlife and older women. Findings related to the important role of gender, may lend support to advocacy efforts aimed at reducing disparities in pain management policies and practices that disproportionately affect vulnerable populations.

This is the first known study to document the co-occurrence of pain in a substantial number of community-dwelling marital partners in mid and later life. This finding should help to raise awareness of pain as a couple-level issue. Such awareness could be used to influence policies and procedures within social, health, and
governmental institutions in terms of providing more family-oriented assessment and treatment. This study contributes to the knowledge base regarding the prevalence of pain among midlife and older couples living independently in the community. This information might be used to help inform changes in policies and priorities surrounding pain assessment and treatment within health care systems.

Beyond the coexistence of a problem with pain, significant covariation was observed in DP spouse pairs on several indicators of physical health (e.g., disease conditions, BMI, physical limitations) and also in level of depressive symptomatology. These findings contribute to a growing body of literature regarding spousal concordance/covariation in physical and mental health. The existence of significant covariation in the health of spouses has implications for the design of health promotion and disease management programs. For example, it suggests that wellness and prevention efforts could have positive effects that extend beyond the individual program participants. This potential “ripple effect” might encourage employers, managed care organizations, and third party insurers to invest more resources in health promotion programs. Additionally, employee/member participation in wellness programs and educational groups for managing pain and/or chronic diseases might be improved if spouses are explicitly invited to participate. In addition, by focusing on pain and its potential role in the disablement process within the novel context of married couples, this study may be able to heighten concern about pain as an issue intimately connected to health care quality.

Findings from this study suggest the need to reinforce the “safety net” of social services available to families as they approach and enter later life. Couples in which both
spouses reported pain appear to be economically disadvantaged in comparison to couples in which one or neither spouse had pain. Regardless of whether such economic circumstances are factors contributing to, or consequences arising from, their pain status, DP couples may struggle to meet their basic needs. Policy makers may thus need to direct special attention and increased assistance to these couples.

**Study Limitations**

The practice, education, and policy implications reviewed above must be tempered by a number of study limitations. The principal limitations of this study are organized into three broad areas: Limitations associated with secondary data, conceptual and statistical issues related to model misspecification, and concerns about the generalizability and limitations of the study sample. Although some of these limitations may have been mentioned earlier, they are discussed in greater detail below.

*Limits of Secondary Data*

**Pain and Pain Intensity**

One acknowledged limitation of the present study is its reliance on the relatively weak measures of pain used in HRS. HRS was designed to examine broadly the personal, financial, and social issues that influence, and are influenced by, workforce participation and transitions in later life. Consequently, the HRS survey contains few questions about pain. In addition, some pain questions are asked only of those with specific diseases. The general pain question that is asked of all respondents employs unusual wording (i.e., “Are you often troubled with pain?”) and is not directly comparable to the measures of pain used in many other studies.

Despite the inherent limitations associated with self-reports, self-reported pain is
the only (currently available) way to assess pain. In fact, self-report is regarded as the most accurate and reliable evidence of the presence and intensity of pain (Turk & Melzack, 1992; Von Korff et al., 2000). Of course, self-report of pain can be subject to bias. Bias is most often in the direction of under-reporting—i.e., individuals (or groups) may not report pain for a variety of reasons (e.g., fear of looking weak, belief that pain is something to be expected with age, etc.). This may be especially relevant to reports of pain within HRS, as some studies have suggested that older individuals are more likely to under-report pain (Gibson, Katz, Corran, Farrell, & Helme, 1994). Reporting may also be tied to semantics; many older adults who deny the presence of “pain” are willing to acknowledge “discomfort,” “hurting,” or “aching” (Parmelee, 1994; Duggleby & Lander, 1994; Miller, et al., 1996). Such findings, when viewed together with evidence suggesting that the HRS general pain question may measure persistent pain, suggest that prevalence estimates derived from HRS may underestimate the number of community-dwelling aging couples experiencing problems with pain.

The pain intensity measure used in HRS also has irrefutable limitations. Respondents are asked to indicate “how bad (your) pain is most of the time” using a verbal descriptor scale with only three levels: mild, moderate, and severe. This item was likely selected because it is easy to administer both in person and over the phone and is not very (cognitively) demanding for older respondents. From the standpoint of a researcher interested in pain, however, this measure has three serious drawbacks.

First, the 3-point response scale constrains the amount of variability available for analysis. It is possible that constrained variability may have contributed to the lack of significant predictors of pain intensity in the current study. This scale is also likely to
lack responsivity to interventions and yield decreased variability in longitudinal contexts.

Second, a three-point scale for pain intensity is inconsistent with the majority of scales used to assess pain in adults across a variety of clinical settings. Research suggests that most people are able to distinguish at least 11 levels of pain intensity (Jensen et al., 1994). Thus, the 3-point scale may not have adequately assessed the pain intensity levels experienced by DP spouses.

Third, this item conveys relatively little detail about respondents’ pain intensity, as participants are asked to provide only a single, overall rating. In contrast, several established measures ask respondents to provide several distinct pain intensity ratings. For example, the Brief Pain Inventory (Cleeland, 1991) has respondents use an 11-point numeric scale to rate their pain during the past week at its worst, at its least, on average, and at its current level; an arithmetic average is then used as an indicator of overall pain intensity or severity. Averaged or aggregated pain intensity ratings have been found to offer improved reliability and responsivity over single-item measures (Von Korff et al., 2000). On the other hand, obtaining multiple pain intensity ratings is often impractical. Moreover, Von Korff et al. (2000) concluded that sufficient empirical support exists for asking respondents to report their usual or average pain intensity for up to a 3-month recall period. Available studies suggest that when bias in reporting does occur, it tends to be in the direction of overestimation of prior levels of pain. One known source of this bias is the severity of pain at the time of recall—that is, people will tend to rate their previous pain as worse when their present pain intensity is high than when their present pain intensity is low (e.g., Salovey et al., 1992). The pain intensity ratings provided by HRS respondents thus can be viewed as generally accurate and valid reports of their
average pain intensity over some recent period, but may be slightly biased by respondents’ level of pain intensity at the time of the interview. It is notable that a recency bias with respect to reported pain intensity would more closely match the reporting period for symptoms on the CES-D8 in HRS.

Several desired measures of pain, including the duration of pain and the specific location of a respondent’s pain, are not included in HRS. Information suggestive of the presumed cause of pain is missing as well. HRS respondents are provided with an opportunity to report other important health conditions (e.g., “Please name any medical diseases or conditions that are important to your health now that we have not talked about.”). These reports might provide additional information about the kinds or types of pain that DP husbands and wives experienced (e.g., shingles, TMJ, spinal stenosis, etc.). However, survey administrators “masked” these data in the public release data files to preserve anonymity. Responses were collapsed into roughly a dozen categories (e.g., endocrine and nutritional conditions; allergies; sinusitis; tonsillitis; digestive system, including stomach, kidney, and bladder; etc.). Unfortunately, the broad categories used by HRS were not sufficiently descriptive to identify other pain-relevant conditions.

**Depressive Symptomatology**

The current study relied on the measure of depressive symptomatology in HRS: A reduced, 8-item version of the CES-D. Like other commonly-used research measures of depressive symptomatology (e.g., BDI, Zung, etc.), the original CES-D is limited in terms of both the breadth and depth to which it covers all important dimensions of the construct. For instance, the clinical features of depressive disorders (e.g., pre-occupation with death, inappropriate guilt) are particularly under-represented in the CES-D.
Construct coverage is even more limited in the 8-item version of this measure.

The dichotomous response format used for the CES-D8 in HRS imposes additional conceptual limitations. Most notably, the frequency and duration of depressive symptoms are assessed only indirectly. Respondents were asked to indicate if symptoms were present “much of the time during the past week” (see Appendix A). Such wording imposes a relatively high threshold for symptom endorsement. Analyses by Steffick et al. (2000), suggest that respondents who have experienced recent depressive symptoms, but with low frequency, will typically under-report their depressive symptomatology on the dichotomous version of the scale. Thus, like pain, depressive symptomatology may have been underestimated in DP spouses in the current study.

**Physical Limitations (Activity Limitation)**

In this study, “activity limitation” was operationalized as basic physical limitations. Physical limitations in HRS were measured without regard to any underlying cause. It is thus impossible to know whether the physical limitations reported by DP spouses resulted specifically from pain. Although pain was strongly associated with, and therefore assumed to be a primary contributor to, the physical limitations of DP husbands and wives, the basis of this relationship and its causal direction cannot be confirmed using data from the present study.

HRS does routinely include a survey question that attempts to measure activity limitation due to pain, “Does the pain make it difficult for you to do your usual activities, such as household chores or work?” This item was initially slated for inclusion in the present study. However, serious estimation problems were encountered when attempting to jointly model DP husbands’ and wives’ responses to this question. Through
consultation with software and statistical experts (M. du Toit, Personal Communication, June 22-29, 2011; J. Z. Smith & A. G. Sayer, Personal Communication, June 29, 2011), it was determined that it was not feasible to model husbands' and wives' responses on this single, dichotomous item. Although it would have been possible to run separate models by gender, it was determined that (in addition to violating the assumption of independence) such models would introduce unacceptable levels of complexity and uncertainty into the larger mediation model. Future research should attempt to test the mediation hypothesis using an improved measure of pain-specific physical limitations.

The HRS measure of physical limitations has other limitations as well. First, respondents were asked to indicate the extent to which they had “difficulty” performing various physical tasks. Difficulty is a very subjective perception and can be interpreted differently by different people. Second, some studies have shown that in general, people tend to overestimate their functional abilities and report themselves to be less impaired or limited than others might judge them to be (e.g., Nagi, 1969; Rubenstein et al., 1984). This under-reporting of impairment or disability seems to be especially pronounced in older adulthood (e.g., Kelly-Hayes, Jette, & Wolf, 1992), perhaps because of normative expectations about aging and the loss of some abilities or due to a tendency to perceive oneself as being “better off” than their same-age peers (e.g., Rickabaugh, 1997). This would suggest that physical limitations, like pain and depressive symptomatology, may have been under-reported by DP spouses.

Some of the more “advanced” physical limitation items in HRS (e.g., climbing several flights of stairs without resting) may not be relevant to all respondents, especially those whose environments limit opportunities to execute the task (e.g., if they never
encounter multiple flights of stairs). It is unclear which response category these individuals might endorse for such items—no difficulty, can’t do, don’t do, or don’t know. In the present study, however, such individuals would have been coded as having difficulty with these items; as a result, physical limitations may have been potentially overestimated for these individuals.

In early waves of HRS, respondents were asked to rate the level of difficulty they experienced in doing each task/activity. The more restricted response options were utilized in all later waves of HRS, thereby limiting details concerning the amount of difficulty experienced by respondents, as well as the total amount of potential variability on the measure. Dichotomous scoring of the items (i.e., difficulty or no difficulty), as was done in the present study and is generally recommended by HRS (e.g., Fonda & Herzog, 2004), can also yield distributions that deviate substantially from normality. The distributions of physical limitations in DP spouses, while somewhat skewed, were still within normal limits and displayed a good deal of variability. It is conceivable, however, that relationships with other variables may have been constrained by the somewhat limited range of possible scores on the physical limitations measure (i.e., 0 – 11).

**Other Measures**

Some other measures used the present study were also less than ideal. BMI was constructed from self-reported height and weight, and thus is likely to have been underestimated in sample of middle-aged and older couples (e.g., Stommel & Schoenborn, 2009). Although BMI was found to be a significant predictor of some outcome variables in the present study (e.g., physical limitations in DP wives), additional relationships might have been observed if a more accurate measure of body weight had
been used (e.g., BMI calculated from measured height and weight). Researchers have also increasingly expressed concern that BMI does not adequately discriminate between excess body fat and lean muscle mass. Some have even suggested that poor specificity of BMI and/or misclassification errors arising from reliance on BMI without regard to other factors could account for the “obesity paradox,” i.e., evidence of more positive morbidity and mortality outcomes associated with increased BMI (e.g., Ashe, Miller, Eng, Noreau et al., 2009; Romero-Corral et al., 2006). Yet, there is continued debate over whether more accurate and valid measurement of excess body fat would be better achieved through alternative methodologies (e.g., bioimpedance, waist-to-hip circumference ratios, skin-fold thickness measurements), or the development of standards or statistical adjustments based on differences associated with factors such as age, gender, race, ethnicity, and method of measurement (e.g., Prentice & Jebb, 2001; Stommel & Schoenborn, 2009). Findings from this study regarding BMI should thus be viewed in light of these limitations.

The measure of disease conditions used in the present study was also limited. The measure relies on respondents’ self-report of conditions that were previously diagnosed by a physician. Consequently, any active but as yet undiagnosed conditions in respondents will be overlooked, along with any conditions for which a diagnosis either has not been provided or does not appear to map onto the disease conditions queried by HRS. The present study lacked detail about the severity of the disease conditions reported by DP spouses. HRS does solicit more detailed information regarding most of the disease conditions. However, follow-up questions vary widely by condition, making it difficult to construct a meaningful index of severity. HRS assesses a relatively limited
number and range of disease conditions. It would have been helpful in the current study to examine DP spouses’ reports of common chronic pain conditions (e.g., migraine, fibromyalgia, low back pain), as well as their reports of other disease conditions in which pain is a prominent feature (e.g., gout, osteoporosis, Crohn’s disease). HRS does include a limited number of other conditions in a “frequent symptom” checklist (e.g., back pain, stomach ulcer, headache). Unfortunately, the checklist is administered only every other wave and was not included in the 1998 HRS interview. Future research should explore the extent to which these other conditions are related to pain intensity in DP couples.

Model Misspecification: Conceptual & Statistical Issues

Cross-sectional Data

One major limitation of the present study, and of cross-sectional research in general, is its inability to establish cause and effect. In the present study there is no way to determine the exact causal ordering of the three key variables—pain intensity, physical limitations, and depressive symptomatology. The current study was guided by a conceptual model based on an assumption—driven by a wealth of available data—that pain leads to depressive symptomatology at the intra-individual level. Data from DP couples were found to be consistent with this hypothesized pathway. Given the cross-sectional nature of these data, however, alternative causal directions and explanations cannot be ruled out. The proposed conceptual model is recursive; that is, it assumes that all effects are unidirectional. However, it is likely that reciprocal effects exist between the key constructs in the model. For example, there is ample evidence that persons with depression report higher levels of pain intensity than persons without depression (e.g., cites). A more realistic model would incorporate feedback loops. Longitudinal studies
are needed to clarify the directionality of relationships and to evaluate the possibility of reciprocal effects.

The present study also found support for a model in which physical limitations (at least partially) mediate the intra-individual relationship between pain intensity and depressive symptomatology in DP spouses. However, a mediated causal model cannot be substantiated using data from a cross-sectional, non-experimental study. Ultimately, results from the current study can only suggest that this specific mediation model is *plausible*. That the findings from the present study regarding the intra-individual mediation pathway are consistent with the bulk of prior studies—both cross-sectional and longitudinal—provides added support for the proposed conceptual model, as well as the activity limitation model of depressed affect (Williamson & Schulz, 1992a). However, additional longitudinal data are required to clearly establish directionality.

Similar direct and indirect pathways were hypothesized to operate at the inter-individual level in DP couples: Pain in one spouse was assumed to lead to depressive symptomatology in the other partner, and this relationship was expected to be mediated by the spouse’s activity limitation. Qualified support was found for the existence of a cross-spouse effect of pain in this sample: Wives’ pain intensity was associated with husbands’ depressive symptoms, but the nature of the effect depended on the level of husbands’ own pain intensity. Again, these cross-sectional data cannot completely substantiate the directionality of this relationship. Evidence to support the presumed causal direction of effects will require longitudinal data from DP spouses. Suggestions to guide such future research will be discussed shortly.
**Omitted Constructs and Relationships**

This study did not examine all potentially important constructs in the relationship between pain and depressive symptomatology. There are a large number of additional constructs that were not considered in this study that could show stronger associations with pain intensity, physical limitations, and/or depressive symptomatology. There are potentially important relationships that were omitted as well. The section below considers a select few of these.

**Intra-individual Level**

In DP couples, gender was found to moderate the intra-individual relationship between pain and depressive symptomatology and also the extent to which this relationship was mediated by physical limitations. Moreover, these effects were found in multivariate models that controlled for a range of background and health characteristics. However, this study did not explore whether any of these other characteristics might moderate all or part of the pain-depressive symptomatology pathway.

One candidate worthy of exploration is age: Several authors have suggested that age may moderate the intra-individual relationship between pain and depressive symptomatology, and this suggestion has received some limited empirical support (e.g., Williamson & Schulz, 1995). Williamson (2000, Chapter 4) argued that observed age effects in terms of the levels of mediation provided by activity restriction are not due to differences in how activity restriction is operationalized, but rather due to experiences with pain that lead to habituation to pain and disability over time and less emotional distress. Other researchers have suggested that, once chronic pain is present in older adults, it may have a greater impact on their functioning and well-being (physical,
psychological, and social domains) than in younger adults (e.g., Gibson et al., 1994).

The present study lacked ancillary data related to pain. One notable omission is the history of the pain condition(s) reported by each spouse. Lacking such information, it is difficult to evaluate the extent to which respondents’ answers to the general pain question actually reflect pain that is chronic/persistent and/or recurrent in nature. Another prominent omission is the lack of data regarding the extent to which DP spouses were receiving treatment—pharmacological, physiological, behavioral, or otherwise—for their pain. Although it may seem reasonable to assume that pain was not being managed effectively in spouses who endorsed the “often troubled with pain” statement, this assumption should be tested explicitly in future research.

Unfortunately, HRS does not specifically assess interventions specific to pain. However, basic information about prescribed medications is routinely collected, and questions regarding the use of complementary therapies have been added in more recent years. Additional insights may be obtained in future research by including reported medication use, as well as respondents’ use of non-pharmacological and non-medical treatments. It would be interesting to explore whether controlling for pain treatment diminishes the moderating effects of gender on the pain intensity-depressive symptomatology relationship—at the intra-individual and/or the inter-individual level—in DP spouses. The impact of medication use on the relationship between pain and depressive symptomatology has not been well studied, and some existing research has yielded unexpected results. For instance, Leveille et al. (2007) found that accounting for daily analgesic use among older women did not alter their risk of developing physical limitations (viz., mobility limitations) due to pain.
Inter-individual Level

The present study lacked information regarding the extent to which husbands and wives expressed their pain to their partners, as well as each partner’s awareness and interpretation of his/her spouse’s pain intensity. Most of these interpretations assume that the partner is (or is not) affected by the spouse’s pain intensity as rated by the spouse him/herself. This assumption could be erroneous, and there is no way to address this in the current study. There are several possible sources of “disconnect” along this pathway. A spouse’s rating of pain intensity may not match what he/she shows or expresses to or displays around his/her partner. Alternatively, perhaps the partner lacks an awareness of the level of pain experienced by his/her spouse. It is also possible that the effect (i.e., the cross-spouse effect) lies in the partner’s perception of the spouse’s pain intensity, not in the spouse’s report of his/her own pain.

Two lines of research suggest that one partner’s perception of the other partner’s pain might complicate the study of cross-spouse effects of pain: First, across different studies, spouse (or caregiver) psychological distress has been related to their perceptions of patient pain (e.g., Ferrell et al., 1995; Manne & Zautra, 2003). Second, numerous studies have documented substantial discordance between patient and caregiver (usually spouse) ratings of the intensity of the patient’s pain. In general, spouses/caregivers tend to over-estimate the patient’s pain, relative to the patient’s own report (Cremeans-Smith et al., 2003; Miaskowski, Zimmer, Barrett, Dibble, & Wallhagen, 1997; Riemsma, Taal, & Rasker, 2000). Under-estimation has also been observed—this is especially likely when the source of the patient’s pain is unknown or is attributed to a contested/disputed diagnosis (e.g., chronic fatigue syndrome, fibromyalgia, and lupus; e.g., Druley et al.,

6-147
However, studies of these conditions confound gender with the nature of pain, as women tend to be over-represented in these diagnoses. In light of these limitations, future research with dual-pain couples would benefit from gathering information from each spouse regarding: his/her perception of the other partner’s pain, his/her reactions to the pain of the other partner, perceptions of the other partner’s reaction to his/her pain, and an evaluation of the marriage.

In the present study, both gender and one’s own pain intensity were found to moderate the cross-spouse effect of pain intensity. This study did not, however, explore whether the inter-individual relationship between pain and depressive symptomatology might be influenced by any of the other variables in the proposed conceptual model. The most likely candidate for a potential moderator is some measure of couple “closeness” or “connectedness.” A few studies have found that the cross-spouse effects may vary by the extent to which couples feel emotionally close (e.g., Tower & Kasl, 1995).

The quality of the marital relationship has also been shown to be important within the context of chronic pain. For example, numerous studies document high levels of marital dissatisfaction among both chronic pain patients and their spouses (e.g., Kerns & Turk, 1984). Studies have also found that marital satisfaction, closeness, and/or marital functioning can impact the psychological well-being of both patients and spouses (e.g., Mohamed et al., 1978; see also review by Leonard, Cano, & Johansen, 2006). Others have also proposed that marital quality may be particularly important in older couples and may impact the day-to-day management of their chronic illnesses (Berg & Upchurch, 2007; Fang, Manne, & Pape, 2001; Keicolt-Glaser & Newton, 2001). In the context of dual-pain couples, marital quality could function as a mediator or a moderator. Limited
measures of marital functioning or quality are available within HRS. Length of marriage is available, and was examined as a background variable in the present study. Alternatively, length of marriage could be examined as a proxy for marital quality or closeness. It would, however, be a poor approximation of the goal construct.

Other Issues

Handling of specific measures. In addition to the inherent limitations of several key measures, there are potential shortcomings associated with how some measures were treated in statistical analyses. As has been mentioned, pain intensity was measured using a verbal rating scale (VRS) with only three categories. Ratings of pain intensity were regarded as quasi-interval level data in most analyses. Although this is a relatively common practice in studies that use VRS data, questions can be raised about whether this is the most appropriate statistical approach to these data (Jensen & Karoly, 2001). This approach implicitly assumes that equal intervals exist between the descriptive adjectives (e.g., between mild and moderate pain, and between moderate and severe pain), which may not be the case. Treating pain intensity as a continuous, interval-level variable also presumes that its relationship with other variables in linear in nature. Analyses in the present study potentially could have missed effects specific to certain levels of pain intensity (e.g., severe pain) or misinterpreted non-linear effects as linear.

The main outcome—depressive symptomatology—was conceptualized as a uni-dimensional, continuous construct, and was measured using the CES-D8. The limited number of items, in combination with dichotomous response categories, has been known to yield scores that deviate substantially from a normal distribution (e.g., Gallo et al., 2000; Choi & Kim, 2007). Depending on the nature of the sample, this problem can be
further compounded by reduced or restricted range and variability. There is continued
debate regarding the most appropriate conceptual and statistical treatment of the CES-D8,
and lack of consensus has resulted in vastly different treatments of this measure in the
literature (see Method chapter, beginning p. 4-14). Some statisticians might argue for the
use of specialized, non-linear models with these data (viz., negative binomial or zero-
inflated Poisson regression). However, strategies to implement such models using a dual-
intercepts approach with cross-sectional, dyadic data have not been developed (J. Z.

The distributions of CES-D8 scores observed in DP husbands and wives
displayed good range and were notably less skewed and kurtotic than those typically
observed in community-based samples (e.g., Steffick, 2000; Keating et al., 2005).
Moreover, the sensitivity analyses conducted as part of this study found no substantive
differences in the results of bivariate or multivariate analyses when a log-transformed
version of the CES-D8 was substituted as the outcome variable. These findings are
consistent with the results of other sensitivity analyses conducted with the CES-D8 (e.g.,
Gallo et al., 2000; Siegel et al., 2004).

**Evaluation of alternative models.** Apart from potential errors in the causal
ordering of key constructs and omitted variables and relationships, there are other ways in
which the conceptual model guiding the present study could be mis-specified.
Alternative models could differ primarily at the intra-individual level, at the inter-
individual level, or both. At the intra-individual level, one alternative model that should
be evaluated is that physical limitations act, not as mediators, but as *moderators* of the
pain-depressive symptomatology relationship. Authors of some prior mediation studies
reportedly evaluated this possibility in their data and found no evidence in support of moderation (e.g., Williamson & Schulz, 1992a; Williamson & Schafer, 2005).

At the inter-individual level, one alternative model involves the pain in one spouse affecting his/her own depressive symptomatology which, in turn, directly influences the depressive symptomatology of his/her partner. In other words, perhaps the cross-spouse effect of pain intensity is mediated through its effect on the spouse’s depressive symptomatology. These are but two possible alternative models that are worthy of evaluation in future cross-sectional, and ultimately, longitudinal research.

Various criteria can be used to evaluate the fit of multilevel models. This study relied on the stability and reasonableness of the fixed effect coefficients, model deviance comparison tests, and model-based proportion reduction in error measures (PREs). It may be beneficial, however, to consider alternative measures, such as Akaike’s Information Criterion or Mallow’s $C_p$. Ideally, multiple diverse criteria should be used to compare competing conceptual models.

**Further model refinement.** It may be possible to further refine the final multilevel model presented in Chapter 5 (i.e., Model 9). One strategy might involve removing additional non-significant predictor variables from the model. Model trimming could also be done separately for husbands and wives. Such gender-specific trimming would, however, make it difficult to directly compare the estimated effects of specific variables in husbands and wives.

In the multilevel models tested here, predictor variables were assumed to have an independent and linear effect on the outcome of interest. Analyses of some model-based residuals suggested the possibility of non-linear relationships involving age and body
weight. Future analyses should compare alternate functional forms (e.g., quadratic, tetric) of these and other predictor variables. Incorporating nonlinear effects into these models may result in improved model fit.

**Dual-intercept multilevel model as a “double-edged sword.”** Dual-intercept multilevel models (MLM) provide some clear advantages—for example, the ability to examine and evaluate possible gender differences, not just in the direct effects, but also in mediated effects. However, use of a dual-intercept multilevel model also confers limitations. Three specific limitations are discussed below. Note that, although these limitations are described primarily from an analytic perspective, each has clear conceptual implications.

*Inability to partition variance.* MLM is conventionally used to understand (or control for) the influence of larger social units or forces on individual-level (or other smaller unit) outcomes. At a basic level, researchers can use MLM to partition the variance associated with each level of the hierarchy: For example, variance in student test scores could be decomposed into variability attributable to characteristics of the student, the classroom, and the school.

Theoretically, it would seem that data from married couples could be treated in the same manner—i.e., variance could be partitioned into individual-level and couple-level components. However, this is generally not the case when cross-sectional dyadic data involve an outcome measured using a single indicator. Because each dyad has only two pieces of outcome data (and therefore limited degrees of freedom), the number of effects that can be modeled is restricted. In the current study, a dual intercepts approach was used because of interest in gender differences within aging couples and a desire to
model both intra-individual and cross-spouse relationships involving pain intensity. From a statistical standpoint, this required that the intercept be identified as the random effect in the multi-level model. This allowed the CES-D8 depressive symptomatology scores to be modeled separately, but simultaneously, for husbands and wives. This parameterization required all predictor variables—whether measured at the individual-level or the couple-level—to be entered simultaneously, rather than hierarchically. As a consequence, it was not possible to partition (and potentially explain) variability in CES-D8 scores into couple-level and individual-level variance.

Apart from the possibilities of (a) ignoring the non-independence of spouses’ data and using traditional OLS regression, or (b) aggregating all data to the level of the couple (both of which have the potential for serious inferential flaws; see discussion of this issue in Chapters 3 and 4), few alternatives are available to model these data. If multilevel modeling is the technique used to account for non-independence in the data, two alternative strategies exist. First, gender could be identified as the random effect. This would permit either: (a) attending to the (in)congruence in CES-D8 scores between husbands and wives, and the predictors (or consequences) of such (in)congruence; or (b) controlling for a gender main effect, and examining the predictors of CES-D8 scores (it must be assumed, however, that the predictors have the same effect in husbands and wives). The second option would be to identify pain intensity as the random effect. Analyses would then focus on the differential effects of pain on CES-D8 scores. However, neither of these options would permit the examination of gender differences in either the direct or indirect effects of pain on depressive symptomatology.

Tenability of the assumptions underlying imposed constraints. As discussed in
the Method chapter (beginning p. 4-79), constraints must be imposed in order to estimate a dual-intercept, cross-sectional dyadic model when the outcome variable is measured using a single indicator. The constraint utilized in this study involved setting the error variance of the dependent variable to a pre-determined value. Details about the derivation and implementation of this constraint involved have been described elsewhere (e.g., see Appendix D). At issue here is the possibility that several assumptions underlying this constraint could be flawed.

Assuming that most researchers would concede that the error variance in an outcome variable can be accurately estimated from available data, disagreement might occur around the best method for deriving such estimates. In this study, reliability estimates—specifically, Cronbach’s alpha estimate of the internal consistency of the items on multi-item scales—were used to estimate the error variance of outcome variables, and separate estimates were calculated for husbands and wives. Because pain intensity was measured using a single item, an *a posteriori* strategy was adopted in which the reliability of this item was assumed to be roughly equal to the average of the observed reliabilities of the other outcome measures (i.e., physical limitations and depressive symptomatology). The logic and decisions underlying this strategy could be debated. Additionally, alternative methods of estimating “true” scores do exist. For example, item-response theory (IRT) could be used to accommodate differential item functioning and to provide estimates that are conditioned on the underlying levels of the construct (Hays et al., 2006). An IRT approach might be especially useful with married couples, as some have suggested that select CES-D items may be gender biased (e.g., Stommel et al., 1993). However, additional research is needed to demonstrate the feasibility and
soundness of using IRT to model cross-sectional, dyadic data.

**Danger of ecological fallacy.** Broadly speaking, ecological fallacy occurs when conclusions from the examination of one unit of analysis are inappropriately applied to another unit of analysis (Piantadosi, Byar, & Green, 1988). In their seminal paper on dyadic data, Thompson and Walker (1982) warned of ecological fallacy as a potential pitfall associated with the analysis of dyadic data. Specifically, when both dyad members provide primarily individual-level data, and analyses are generally done in the aggregate, researchers must be careful not to overstate conclusions at the relationship level. In the present study, several differences were observed between husbands and wives—e.g., wives had higher pain intensity levels and a stronger relationship between pain intensity and depressive symptomatology than did husbands. However, this does not necessarily mean that these differences exist within any given dual-pain couple.

Risk of ecological fallacy is heightened by several features in the present study. First, HRS primarily collects individual-level data from respondents (e.g., one’s own pain, depressive symptoms, etc.); few relationship-oriented measures (e.g., satisfaction with the marital relationship) are included. Second, few HRS measures are amenable to the creation of meaningful “second-order” dyadic data—i.e., the creation of dyadic-level data from information collected at the individual level (Thompson & Walker, 1982, p. 893). If, for example, HRS had asked each spouse to rate the pain intensity level of his/her partner, these ratings could be compared, potentially creating second-order measures of dyadic similarity or difference. Thompson and Walker assert that it is only from these measures that relationship properties, such as empathic accuracy or shared meaning, can potentially be inferred. Third, and finally, dual-intercept multilevel models
inherently aggregate data (in this study, data were aggregated by gender). Given the types of dyadic data available in HRS, the findings from these dual-intercept multilevel models should not be assumed to represent any intra-dyadic process, nor should they be thought of as applying to all dual-pain couples.

On the other hand, steps were taken in the present study to mitigate this potential risk. The study did not focus exclusively on gender differences in DP couples. For instance, couple-level similarity was examined for most study constructs. In addition, some constructs—e.g., the notion of dual-pain couples, the Husband X Wife Pain Intensity interaction effect—were conceptualized at the dyadic level and based on the pattern of responses within couples. Moreover, several planned and supplemental analyses moved beyond gender-aggregated results to explore couple-level variability (e.g., cross tabulations of pain intensity ratings and joint analysis of MLM residuals).

**Generalizability and Limitations of the Sample**

HRS was not designed to be a study of married couples. The inclusion of spouses and partners of married participants was originally conceived as an economical and efficient way to enlarge the sample and recruit new cohorts into the study. The success of HRS in recruiting the spouses/partners of age-eligible respondents (roughly 95%, as reported in Heeringa & Connor, 1995) and its commitment to collecting data from both partners (independently) has created a unique and valuable resource for the study of couples in mid and later life. However, the extent to which findings from studies of couples drawn from HRS can be generalized to the U.S. population of midlife and older married couples is not clear.

The sampling unit used by HRS is the household, and eligible households are
those with at least one resident falling within a specified age range. Reports show that HRS (and AHEAD) achieved initial baseline response rates over 80% at both the household and respondent levels (Heeringa & Connor, 1995). The proportion of married/coupled households participating at baseline closely matched estimates derived from available census data. For example, 1989/1990 census data estimated that 19.3% of U.S. households would have at least one resident who was age-eligible for participation in HRS, and that 64.1% of these households would be comprised of married/partnered couples. In the first wave, HRS recruited an average of 1.64 respondents per eligible household, clearly matching census data.

HRS utilizes multi-stage area probability sampling to ensure the geographic and age-related representativeness of recruited samples. Blacks, Hispanics, and residents of Florida have been oversampled at a rate of roughly 2 to 1. Reportedly, HRS has been very successful in recruiting and retaining participants of minority race and ethnicity. Baseline response rates and rates of attrition for minority participants are comparable to those of Whites, and the characteristics of minority participants closely parallel those of minority participants in other large national surveys (Ofstedal & Weir, 2011).

Analyses of cross-sectional data from HRS have generally found that the characteristics of age-eligible participants closely match those reported in other large-scale datasets. For example, age-eligible respondents in HRS 1992 displayed similar levels of education, rates of labor force participation, and marital status as their same-age peers in the 1992 Current Population Survey (Kapteyn, Michaud, Smith, & van Soest, 2006). One notable exception relates to household wealth: Like many other large-scale household surveys, HRS does not capture the full range of amassed wealth in the U.S.—
households with the greatest net worth are not well-represented in the study (Juster, Smith, and Stafford, 1999).

Participant-level response rates for the second and subsequent waves of HRS (up to 2002) have been in the low-mid 90%s (HRS, 2011). However, detailed analyses of the representativeness of the HRS sample over time have been somewhat limited. RAND did analyze attrition and non-response patterns for roughly 10,000 age-eligible HRS respondents over the first 5 waves, i.e., 10 years (Kapteyn et al., 2006). As has been found in other panel studies, non-response due to mortality was highest among respondents with low household SES (Kapteyn et al., 2006). Although some baseline characteristics (e.g., minority race and ethnicity, older age, lower education) and the occurrence of health and status changes (e.g., onset of certain health problems; divorce, in males only) were associated with non-response over time, the 2002 sample evidenced few significant differences from the initial sample (with the notable exception of increased cumulative attrition among immigrants and Hispanics and, to a lesser extent, Blacks). Instead, analyses revealed that it was the “temporary attritors” (i.e., respondents who did not respond at one or more interim waves, but who eventually returned to the study) who differed most from the initial sample of respondents (Kapteyn et al., 2006). HRS’ efforts to re-contact non-respondents at later waves has been credited by RAND with reducing cumulative attrition, attenuating attrition bias, and helping to maintain the representativeness of the HRS sample over time. As an example: 33% (n = 355) of HRS wave 3 non-respondents were successfully interviewed in wave 4 (1998). In addition, the number of participants who have voluntarily and permanently withdrawn from the study has been very small. By 1998, only 3% of HRS, and <1% of AHEAD respondents had
requested to be permanently dropped from the study (HRS, 2011).

The complex sampling and extensive follow-up procedures used by HRS have helped to ensure that the findings of studies using all HRS respondents can (with the use of appropriate weighting schemes) be generalized to the population of adults over age 50 in the U.S. (HRS, 2011). But, it is challenging to assess the representativeness of the couples included in HRS. To this author’s knowledge, there are no published data regarding the response and attrition rates of the married or partnered couples who have participated in HRS. And, because of the varied ages of the partners in these couples and the decision to use younger spouses to “fill in” the new cohorts recruited into the study (starting in 1998), HRS no longer publishes wave-specific household-level response rates.

**Limitations Associated with Sample Selection Criteria**

Nevertheless, based on the information provided above (e.g., overall HRS response, attrition, and mortality rates) and data gathered through this study’s sample selection process, some limited inferences can be made about the ways in which the preliminary sample of married couples drawn from HRS 1998 might differ from the population of later life couples in the U.S. Three important differences can be traced to one or more of the sample selection criteria used in the present study (refer to Figure 6).

First, a large number of couples ($n = 1,359$) were excluded from this study because one or both spouses required a proxy interview in HRS 1998. Proxy interviews are allowed in HRS when the respondent is unable or unwilling to complete an interview. Proxy interviews were required for 9.6% of respondents in 1998 (HRS, 2011). Available data suggest that proxy interviews are more likely among older participants: For instance, in HRS 1998, 16% of those aged 75 and older required a proxy interview,
compared to just 6% of those aged 51 to 55 (HRS, 2011). Poor cognitive functioning and poor health are two primary reasons underlying the need for proxy interviews in HRS (HRS, 2011; Ofstedal, Fisher, & Herzog, 2005). The majority of couples excluded from the preliminary sample in the present study because of a proxy interview (76%) were comprised of couples in which only the husband required a proxy interview. This potentially limits the ability to generalize study findings to couples in which the husband is of advanced age and suffering from major cognitive and/or health problems.

Second, despite the oversampling of minority populations in HRS, the representation of couples of minority race and/or ethnicity within the initial couples’ sample was sparse. Requiring respondents to report that they were legally married and currently living together in the community may have disproportionately excluded couples of color from the initial selection process. For example, studies have shown that Blacks are less likely to be married than Whites (e.g., McKinnon, 2003), but are more likely to cohabit (Raley, 2000). Additionally, although the actual numbers of potentially eligible Black couples (n = 465) and those of Hispanic descent (n = 377) were sizeable, the distribution of minority racial/ethnic status across other variables of interest (e.g., gender, reports of pain) often resulted in extremely small cell sizes. Although not ideal, the decisions to combine those of “other” race with those reporting Black race into a “non-White” racial category and to exclude Hispanic ethnicity as a predictor in most analyses were necessitated by these small cell sizes. Findings with respect to race (mainly a lack of effects) should therefore be interpreted with caution. It is possible that combining multiple non-White races into a single category may have masked important differences between minority racial groups. Because racial and ethnic disparities in
health are potentially greatest during middle and early old age (House et al., 1994), it will be important in future research to examine the influence of race and ethnicity on reports of pain and the relationship between pain and depressive symptomatology in middle-aged and older couples. Such investigations, however, will likely require larger samples and/or greater representation of those of minority race and/or ethnicity. Future research with minority populations might also benefit from using an expanded definition of marriage that includes other types of co-residential unions.

Third, the current sample was limited to couples in which both spouses were living (together) in the community at the time of study. Thus, couples in which one or both spouses were living—even temporarily—in an institutional setting (e.g., nursing home) at the time of the 1998 HRS survey were excluded. Note that rates of pain have been found to be considerably higher (e.g., prevalence estimates of 50-83%) among institutionalized older adults (e.g., Ferrell et al., 1995; Sengstaken & King, 1993; also see review by Fox, Raina, & Jadad, 1999). Thus, findings from the present study cannot be generalized to couples in which one or both spouses reside in an institutional setting.

**Uniqueness of DP Couples**

The focal sample of DP couples comprised just a small proportion (8%) of the preliminary sample of HRS couples. Thus, the characteristics of DP couples are likely to diverge even further from those of the broader population of aging U.S. couples. Although DP couples shared some similarities with the other couples in the preliminary sample, DP spouses were generally older, less healthy, and had fewer resources than the spouses in couples in which neither partner reported pain. However, as previously discussed, additional study is needed to determine the causal ordering between pain and
differences in these background characteristics.

Differences between DP spouses and their peers of the same gender with pain in husband-only (or wife-only) pain couples were less marked. However, because this study focused on couples in which both spouses reported pain, results should not be generalized to couples in which only one spouse has pain. Within such couples, the relationships between pain and depressive symptomatology could potentially differ at the intra-individual level, the inter-individual level, or both. The husband-wife pain intensity interaction effect observed among DP couples in this study provides some support for this notion. Future research should examine the pain-depressive symptomatology relationship at both the intra-individual and inter-individual levels in couples in which one spouse reports pain, but the other does not. Such studies also might provide useful insights regarding the contribution of gender to cross-spouse effects of pain.

Finally, it must be acknowledged that findings regarding the intra- and the inter-individual relationships between pain and depressive symptomatology may be specific to the cohorts of DP couples who participated in HRS 1998. These couples may be uniquely defined by social and historical influences. In addition, most DP couples were in decades-long marriages. Thus, these husbands and wives may adhere to more traditional social roles and responsibilities. These analyses should be replicated in later cohorts of aging couples who may display different relationship dynamics. Any observed differences would provide fertile ground for future studies.

**Strategic Directions for Research & Theory Building**

Several concrete directions for future research have been identified elsewhere in this chapter and will not be reiterated here. Instead, this section highlights a few strategic
directions for future research and scholarship aimed at advancing our understanding of how pain is experienced within aging couples. Whereas prior suggestions were tied to specific findings and/or limitations of the present study, those outlined below have been derived from consideration of the study and its findings as a whole. Four strategic directions have been identified and are discussed in order of importance: (1) Theory building and research on the cross-spouse effects of pain; (2) Validating and explaining gender differences; (3) Balancing specificity and generality in replicating and extending this study; and (4) Examining the relevance of the chronic versus acute pain distinction in later life.

Confronting the Limitations of Activity Limitation Theory:
The Need for Theory Building and Additional Research on the Cross-spouse Effects of Pain

The absence of any straightforward cross-spouse effects of pain intensity in dual-pain couples suggests the need to develop a more sophisticated conceptual model to guide the study of the cross-spouse effects of pain. The cross-spouse effects observed in the present study were gender-specific and conditioned on the pain intensity levels of both spouses. Several potential explanations were proposed and, although some could reasonably account for selective portions of the findings, none appeared to be able to encompass the entire set of findings. Although some effects were consistent with expectations derived from the proposed conceptual framework, others (including the lack of some expected effects and contradictory effects) are challenging to explain, even drawing on concepts from alternative theoretical perspectives (e.g., protective buffering).

Because this is the first known study to have examined the cross-spouse effects of
pain in couples in which both spouses report problems with pain, it is difficult to evaluate whether some of these findings might be tied to unique features of the sample or to the study methodology. This task is further complicated by several issues. First, no prior research has compared dual-pain couples to couples in which only one partner reports pain. Second, the majority of prior studies that have examined the effects of one spouse’s pain on the well-being of his/her partner have used samples drawn from clinical settings (e.g., chronic pain centers, rheumatology outpatient clinics) and/or populations defined by specific diseases or types of pain (e.g., persons with arthritis, cancer patients, persons with chronic musculoskeletal pain). As the resulting samples tend to be limited with respect to age ranges and distributions and the types/locations of pain, they do not present a realistic picture of aging couples facing pain in the community. In some studies, sample selection may also confound pain status with gender and/or caregiving roles (e.g., if most arthritis patients are female, or if most spouses are providing instrumental support to the patient). Third, most existing research in this area has not been truly dyadic in nature. Although studies may collect data from both spouses, few collect equivalent data on all key study constructs. For example, a study might ask patients to rate their own pain, but spouses are asked only to rate the level of pain of the patient. In addition to limiting the extent to which findings from this study can be compared to those from prior research, these issues also hamper theory building.

Research on the cross-spouse effects of pain is (presumably) predicated on an assumption that the marital relationship constitutes an important context for individuals experiencing pain. Yet, few studies offer any substantive theoretical perspective to guide the study of these effects. Some make passing reference to general stress and coping
paradigms, suggesting that pain in one’s spouse serves as a source of stress for the partner. Others augment such paradigms by invoking notions of increased caregiver burden tied to the spouse’s pain. And, as noted in Chapter 2, a number of chronic pain scholars approach couple-level research from a behavioral perspective—e.g., studying how the reactions of partners reinforce the pain-related behaviors of the chronic pain patient (e.g., Romano et al., 1995; Turk et al., 1992).

Lack of attention to theory is certainly not unique to the study of couples and pain. Outside of the voluminous body of research on caregiving, most studies examining the relationships between the physical and mental health of spouses—especially those in aging couples—are theoretically deficient. In a review of 45 studies of the health and relationship dynamics of aging couples (excluding caregiving studies), Walker and Luszcz (2009) observed that, “much of the research, apart from the concordance literature, was not integrated with relevant theory, nor did it contribute new theoretical perspectives” (p. 477). Their comment requires additional clarification in order to avoid the suggestion that studies of spousal concordance have been strongly theory-driven.

Most of the 13 concordance studies included in the review mentioned multiple theoretical paradigms (e.g., assortative mating, shared living arrangements, contagion); further, most theories had been offered post hoc, in an attempt to account for study findings. A total of seven different theoretical/conceptual frameworks were mentioned across the set of concordance studies. Moreover, aside from a tendency for the same groups of authors to cite the same theoretical framework(s), there appeared little agreement as to which theoretical framework was most relevant. The only exception was affective contagion, which was commonly mentioned among studies that found evidence of spousal
concordance in depression/depressive symptomatology.

Perhaps as a consequence of the limited availability of relevant theoretical frameworks, research on couples in mid and later life remains focused on very basic questions. For example: Is characteristic X of Spouse A related to characteristic X of Spouse B? Or, similarly: Does characteristic X of Spouse A affect characteristic Y of Spouse B? It is critical that future research and theory building attempt to clarify how, and why, these cross-spouse effects occur.

In the absence of any well-articulated theoretical perspectives, this dissertation attempted to extend an individual conceptual model, based largely on the activity limitation/restriction model (e.g., Williamson & Schulz, 1992a), to the study of pain at the inter-individual level. The foundation for this extension centered on (a) some compelling findings from research with chronic pain patients and spouses, (b) an evolving body of scholarly work concerning the experience of aging within intimate relationships, and (c) several basic tenets derived from interdependence theory (e.g., Kelley & Thibaut, 1978) and other theoretical frameworks concerning interpersonal relationships (e.g., Hatfield et al., 2002). Although supportive evidence was found for several proposed relationships at the inter-individual level (e.g., Paths 1, 2, 3), other hypothesized relationships received only partial (e.g., Path 4W) or no support (e.g., Path 5). There was no evidence of a relationship between one spouse’s physical limitations and the depressive symptomatology of the partner. In addition, the hypothesis that physical limitations would mediate the relationship between each spouse’s pain intensity and the depressive symptoms of his/her partner was not supported.

It would certainly be premature—on the basis of the (non-) findings from this
single study—to dismiss completely the activity limitation model as a potential framework for understanding the cross-spouse effects of pain on psychological distress in aging couples. However, it seems appropriate to use the findings from this study to critically evaluate the activity limitation model as applied to the cross-spouse context of pain. This evaluation may suggest strategies to enhance the utility of the model in the study of couples and pain in later life. Alternatively, this evaluation may identify alternative models or constructs to pursue. The three most promising directions for research and theory building are outlined below.

**Refine the Construct of Activity Limitation for Application at the Inter-Individual Level**

In the current study, activity limitation was operationalized specifically as limitations in basic physical abilities (e.g., difficulty walking, climbing stairs, stooping, etc.). Consistent with expectations derived from theory, greater pain intensity was associated at the intra-individual level with increased limitations in these basic physical abilities, which in turn were associated with greater depressive symptomatology. At the couple level, however, neither spouse’s physical limitations were significantly related to the depressive symptomatology of the other partner.

This pattern of findings suggests that these basic physical activities may be consequential for an individual spouse’s own well-being, but not for that of his/her partner. It is possible that individuals perceive and react to these physical limitations differently when they are observed in others, even in a spouse. Perhaps when witnessed in others, these limitations are regarded as an expected part of the aging process aging and are therefore less distressing than when experienced personally. As mentioned
earlier, threshold effects may also exist. For example, one spouse’s physical limitations may need to reach a certain number, level, and/or duration in order for the partner to notice and/or be distressed by them. Or, perhaps these basic physical limitations, by themselves, are not sufficient to influence the psychological well-being of the partner. Partners may not be affected until these limitations progress further along the disablement pathway (Verbrugge & Jette, 1994) and begin to impede the spouse’s performance of daily activities or self-care tasks (i.e., IADLs or ADLs).

It would seem relatively straightforward to construct research questions to address some of the possibilities mentioned above. In fact, data from these DP couples could be used to examine the relationships between the partner’s psychological distress and several alternative measures of the spouse’s activity limitation (e.g., ADLs, IADLs). Potential research questions might include: Which types of limitation are more consequential for the partner’s well-being? Is there a hierarchical ordering across the different types of limitation, and does this ordering have implications with respect to the potential mediation of the cross-spouse effects of pain. Results from such studies could help to further refine the activity limitation construct. These refinements could then inform modifications to activity limitation theory that would enhance its relevance to the study of pain in couples.

Consider Alternative and/or Additional Mediators

It is entirely possible that the mechanism that mediates the cross-spouse effects of pain is fundamentally different from the mechanism(s) presumed to operate at the intra-individual level. Although activity limitation theory (as developed by Williamson & Schulz, 1992a, and sharpened in the conceptual model developed here) appears to
provide a reasonable pathway through which pain affects depressive symptomatology at the intra-individual level, the theory may not be adequate or appropriate for application in an inter-individual context.

*Might Mediation Take Place at a Different Level?*

 Perhaps the effect of one spouse’s pain on the psychological distress of his/her partner is mediated at a different level—one that generally is not accessible to measurement using traditional social science methods. For instance, such mediation may occur solely at a physiological level. Perhaps recognizing that one’s spouse is experiencing pain is a sufficient stimulus for eliciting emotional distress in the partner. Two different lines of research provide evidence in support of this possibility.

First, interest has recently coalesced within the field of cognitive neuroscience at the seemingly unlikely intersection of research on human pain, human emotion, and social interactions in primates (e.g., observational learning, mimicry, attachment). Research teams have begun to investigate the neural bases of human empathy—in particular, the empathic experience of another’s pain and suffering. Using advanced neuroimaging techniques (e.g., fMRI), researchers have found that perceiving pain in others activates several regions in the brain that are also activated when we experience pain ourselves (e.g., Oschsner et al., 2008; Singer et al., 2004)

Second, recent efforts to incorporate physiological measures into the study of caregiving, which heretofore has been studied mainly from a psychosocial perspective, have yielded some interesting findings. In one study, researchers measured the cardiovascular reactivity (blood pressure, heart rate) of the spouses of osteoarthritis patients as they watched their partners/patients and strangers (separately) perform a
painful physical task (Monin et al., 2010). Study findings dovetailed nicely with those from neuroimaging studies of empathy: Simply watching another person perform a painful task increased caregivers’ physiological responses. Together, the findings from these two lines of research suggest that we may be hard-wired to experience emotional distress when others are in pain. Although, technically, these studies do posit mediation via specific neurological and/or physiological pathways in the partner, researchers concerned with self-reported intra- and interpersonal processes might consider these effects unmediated.

*Other Possible Mediators*

Alternatively, activity limitation may not be the most relevant or compelling mechanism that mediates the relationship between pain and psychological distress at the couple level. The existence of an alternative mediating construct/pathway in the cross-spouse effect of pain is supported by another important finding of the Monin et al. (2010) pain observation study: Caregiver reactivity was higher when the person performing the task was a spouse, versus a stranger. Furthermore, this difference was observed *even when* the stranger displayed more overt pain behaviors (e.g., grimacing, massaging a painful joint) than the spouse (as judged by multiple, independent raters). This finding suggests that the distress experienced by one person when witnessing another person in pain may be influenced by the nature of the relationship between the people. The distinction in caregiver reactivity to the perception of pain in a spouse, as compared to a stranger, implies the existence of a mediating mechanism that involves the cognitive and/or perceptual processes of the partner/caregiver. Perhaps witnessing a loved one in pain invokes a feeling of helplessness borne of a desire to alleviate his/her suffering. In
such a scenario, psychological distress—(e.g., depressive symptomatology) might then be a result of extended feelings of helplessness (Taylor, 1984).

Another possibility is that the cross-spouse mediation process is more complicated than that presumed by the activity limitation model. Perhaps multiple mediators operate simultaneously, or perhaps the mediation process involves a sequence of mediating constructs. Consider again the Monin et al. (2010) study: While the task assigned to the patient was designed to elicit pain, it also highlighted the patient’s physical limitations to the caregiver. It is therefore possible that observing the physical limitations of the patient could have contributed to the caregiver’s physiological reactivity. Because the Monin et al. task conflated pain and activity limitation, it is impossible to conclusively pinpoint the underlying mediation process. However, these findings suggest that activity limitation could still be involved in mediating the cross-spouse effect of pain; perhaps it comprises one element in a sequence of mediators.

Although many additional constructs or processes could be involved in a sequence of multiple mediators, potential candidates might be identified by considering some concrete ways in which one spouse’s activity limitation might affect his/her partner and/or their relationship. Perhaps the spouse’s activity limitation alters the distribution of tasks and responsibilities within the couple (e.g., now the husband must do the all the grocery shopping), and the increased and/or unfamiliar responsibilities heighten the partner’s psychological distress. It is also possible that one spouse’s activity limitation reduces the extent to which the couple (or only the partner) is able to engage in shared activities or participate in social events, and the partner is negatively affected by this reduction. Note that the latter possibility hypothesizes the existence of an additional
activity limitation pathway—potentially expanding Path 5 in the proposed conceptual model—involving the partner’s activity limitation and/or activity limitation in the couple. The concept of activity limitation in the partner is consistent with activity limitation/restriction as discussed within the caregiving literature (e.g., Bookwala & Schulz, 2000; Nieboer et al., 1998); here, however, it is embedded within an interpersonal context that does not presume the existence of an explicit caregiving relationship.

Especially for application within a couples’ context, it may be helpful to more fully explicate various elements of the disablement process model, or another suitable model (e.g., Tomey & Sowers, 2009).

Explore (Other) Potential Moderating Constructs

The pattern of findings with respect to the cross-spouse effects of pain intensity—namely, the presence of conditional effects (i.e., the interaction of spouses’ pain intensity), the varied nature of those effects, and the existence of effects in husbands only—strongly suggests that underlying processes are likely to be complex. In addition to exploring alternative and perhaps additional mediating pathways, future research should examine other factors (beyond partner pain intensity and gender) that potentially moderate the cross-spouse effects of pain.

A variety of constructs could be considered as potential moderators of the cross-spouse effect of pain. Two potential moderators—interpersonal closeness and each spouse’s expression of pain—were mentioned earlier in this chapter (under Model Misspecification). These constructs are important additions to consider in the design of future research studies; however, since they have already been discussed in some detail, they will not be discussed again here. Attention will be given instead to a few constructs
that appear to offer considerable promise in studying pain in aging couples.

Two well-developed theoretical frameworks the intra-individual level—Folkman and Lazarus’ (1984) stress and coping model and Pearlin et al.’s (1991) stress process model—are rich with constructs that could potentially moderate the relationship between one spouse’s pain and the psychological distress of the other spouse. In fact, several constructs originating from these models have already been re-conceptualized for application at the couple level (e.g., Berg & Upchurch, 2007; O’Brien & DeLongis, 1997; Revenson, 1994), although they have seen little application in the study of couples and pain. Two of these constructs—appraisal and coping—are considered briefly below. Appraisals and coping are traditionally regarded as constructs or processes that mediate (or potentially moderate) the relationship between stress and well-being at the intra-individual level. However, application within the current context concerns how certain appraisals and/or coping strategies might moderate the inter-individual effects of pain.

*Is it His Pain, Her Pain, or Our Pain?*

Berg and Upchurch (2007) outlined a developmental-contextual approach to couples dealing with chronic illness throughout the lifespan. One major contribution of their model is the attention given to how appraisals can be dyadic and can affect the adjustment of the partners and the couple. Two specific dimensions, or types, of appraisals outlined by Berg and Upchurch have specific relevance to pain in the context of the aging couple: stress appraisal and ownership appraisal. The former concerns whether or not, and the extent to which, a partner’s illness (in this case, pain) is appraised by the spouses as stressor. Some discuss such appraisals more in terms of a perceived threat and/or challenge (e.g., Folkman et al., 1986). For example, the partner might
appraise the spouse’s pain as stressful if he/she believes that it poses a threat or challenge to the relationship, perhaps because it foreshadows a change in the spouse’s behavior or the need for the partner to assume additional responsibilities. Stress appraisals are also likely to influence how the partner responds to the spouse’s pain. An experiment with adult friendship pairs, in which one member was exposed to a painful task, found that, in addition to influencing the coping strategies and pain tolerance of the pain-exposed individuals, threat appraisals (vs. benign or mixed appraisals) also affected the strategies used by their friends to help them cope (Jackson, Huang, Chen, & Phillips, 2009).

Appraisals of ownership concern beliefs or judgments about where the illness is situated or located (Berg & Upchurch, 2007). For example, is the pain seen as belonging to the spouse, the partner, or the couple? Such appraisals also reflect the extent to which the illness (i.e., pain) of the spouse is included in the partner’s future plans and goals? Ownership appraisals have been explored among couples in which one partner suffers from cancer (e.g., Kayser et al., 2011). However, there are no known studies of such appraisals in couples dealing with pain. Findings from the current study suggest that it may be valuable to examine pain-related threat and ownership appraisals in couples and the extent to which such appraisals influence levels of emotional distress regarding the spouse’s pain. Of course, in DP couples, it would be important to examine not only each partner’s appraisal of the other’s pain, but also appraisals of his/her own pain as well.

Do We Cope With Pain as Individuals, as a Couple, or Both?

Most prior empirical and conceptual work has focused on coping and adjustment as an intra-individual process. However, it can occur inter-individually, as well. In fact, there is a long history of interest in coping efforts that are either focused toward the
relationship (e.g., O’Brien & DeLongis, 1997) and/or reflect the engagement of the dyad in joint problem-solving and adaptation (e.g., Coyne & Smith, 1991; Revenson, 1994; Revenson, Kayser, & Bodenman, 2005). With respect to DP couples, it would be valuable to examine the coping mechanisms and processes that are used by each spouse to deal with his/her own pain and the pain of his/her spouse. It would also be important to explore the extent to which DP spouses engage jointly in different coping efforts. Given the importance of coping for individual-level psychological well-being (e.g., Folkman & Lazarus, 1984), it is likely that the well-being of DP spouses will be influenced by individual- and couple-level coping efforts. It is also possible that such efforts could buffer or exacerbate the distress linked to a spouse’s pain.

It is important that the activity limitation model not be cast aside entirely during the investigation of potential moderators. It is conceivable that the spouse’s activity limitation may, in fact, mediate the effect of the spouse’s pain on the partner’s depressive symptomatology but only under certain circumstances. This possibility was alluded to earlier in this chapter. For example, the spouse’s pain may have an effect on the partner’s depressive symptoms through the spouse’s activity limitation, but only when the spouse’s activity limitation is advanced enough to require the partner’s assistance with instrumental tasks (e.g., IADLs). Future research should explore the possibility of moderated mediation and/or mediated moderation involving activity limitation and the cross-spouse effects of pain. It is also possible that some types of activity limitation act as moderators (instead of mediators) of the relationship between one spouse’s pain and the depressive symptomatology of his/her partner.
Do Couple-Level Patterns Matter; and, If so, How?

The idea of dyadic patterning with respect to constructs of interest has been a recurring theme throughout this discussion of potential moderating variables. Patterning is an important concept in dyadic research, but is often overlooked. Underlying the examination of couple-level patterns is the notion that the joint distribution of spouses’ characteristics or statuses represents something unique; moreover, this pattern can have an effect on the well-being of each partner (and the dyad) beyond the effects associated with the characteristic or status of each spouse. It was this possibility that led to the incorporation of a pain intensity interaction effect within the present study. Surprisingly, the pattern of pain intensity in DP couples was found to be related to the depressive symptom levels, but in DP husbands only. Perhaps not so surprising was the difficulty encountered in attempts to generate an explanation that could encompass the different effects associated with the various patterns of pain intensity in DP couples.

Couple-level patterns can be conceptualized and studied from a variety of different perspectives. Although congruence/similarity (or, conversely, incongruence/dissimilarity) is just one possible perspective, it is the perspective used most frequently in research on couples (Berg & Upchurch, 2007). In studying pain in aging couples, it may be valuable to consider the notion of congruence, as it might play an important role in the well-being of spouses, especially in couples in which both spouses have pain. Congruence/similarity in spouses’ ownership appraisals of pain, for example, could moderate the cross-spouse effect of pain on psychological distress. Couple-level congruence in pain-related coping strategies could also feature prominently in the well-being of spouses. Well-being in DP spouses could also be influenced by congruence on a
variety of other constructs and dimensions. One additional avenue to pursue in an attempt to identify potential moderators of the cross-spouse effects of pain is congruence in terms of prior experience(s) of significant pain. Perhaps prior struggles with pain heighten a partner’s empathic response toward the spouse’s pain.

In studying pain in aging couples, it may also be valuable to consider couple-level patterns more broadly. Rather than emphasizing congruence and similarity, it may be helpful to consider: Are there times when couple-level dissimilarity could be more adaptive? Consider the following scenario involving a DP couple:

The wife experiences moderate pain, but it is mainly limited to her hands and shoulders. As a result, she has difficulty performing tasks involving fine motor control and reaching for things overhead. Her husband also has problems with pain, but his pain is mainly in his lower limbs and affects his ability to walk and drive a car.

Is it possible that each spouse could experience fewer IADL and/or ADL limitations because his/her partner is able to compensate for his/her limitations? Perhaps couples adjust by matching the abilities of each spouse to the various tasks that emerge in daily life—for example, the husband above may take responsibility for opening bottles and jars and doing the laundry, whereas the wife may do all of the grocery shopping and serve as the primary driver. In this instance, dissimilarity—or, more specifically, complementarity—may be more valuable for the well-being of these spouses. Consideration of couple-level patterns may make it possible to extend the notion of person-environment fit (e.g., Kahana, 1982; Lawton et al., 1980) to the level of the couple. Along these lines, it may also be possible to develop a model of the disablement process that is specific to couples. In such a model, “disability” would occur only when the demands exceed the joint capabilities of the couple. In this instance, the nature and
patterning of the physical limitations of both spouses shape the outcomes for the couple. Although it would be possible to explore couple-level patterns across a variety of constructs in future research, the creation of such second-order constructs should be driven primarily by theory (Thompson & Walker, 1989). From a methodological perspective, the study of dyadic congruence/complementarity in appraisals, coping, and key constructs within activity limitation and disablement process models will be most fruitful if data are collected from both spouses over a considerable period of time.

Finally, it is worth noting that many of the strategies outlined above could also help to advance the activity limitation model at the intra-individual level. For example, refining the construct of activity limitation may help to clarify some thorny conceptual and methodological issues that plague the application of activity limitation theory at the intra-individual level, for example: Which types of limitation are most consequential for the individual’s well-being? How are different types of activity limitation related? Are these relationships hierarchical; if so, does the hierarchy conform to existing theoretical models (e.g., Verbrugge & Jette, 1994)? What are the implications of combining different types of activity limitation into a single “activity limitation” measure? The activity limitation model could also be significantly enhanced if multiple concurrent or sequential mediating constructs could be identified at the intra-individual level. Finally, numerous potential characteristics other than gender could moderate either the direct or indirect effects of pain on psychological distress at the intra-individual level. In particular, the potential moderating effects of age and race/ethnicity are worth exploring. In addition, efforts to integrate selected elements of stress and coping theory (e.g., appraisal, coping) and activity limitation theory within a model of the disablement
process are likely to generate significant insights into the intra-individual relationship between pain and psychological distress.

**Validating and Explaining Gender Differences in the Relationships Between Pain and Psychological Distress**

Findings for husbands and wives in DP couples did replicate the usual findings that, compared to men, women report higher pain intensity, more physical limitations, and greater depressive symptomatology. Also in keeping with some prior research, the relationship between pain intensity and depressive symptomatology was found to be stronger in wives than in husbands. While the consistency of results is reassuring, it is also notable given the unique nature of this sample. These gender differences were observed within a specific group of marital dyads in which many potential confounding factors (e.g., marital status, SES, reported trouble with pain, general age range) were controlled, or at least attenuated.

In this sample of DP couples, a gender difference was also observed in the extent to which physical limitations mediated the relationship between pain intensity and depressive symptomatology. This intra-individual relationship was fully mediated in husbands, but only partially mediated in wives. Existing evidence regarding gender differences in mediation by activity limitation is sparse and inconclusive.

Another novel finding in the present study involved the inter-individual relationship between each spouse’s pain and his/her partner’s psychological distress. Wives’ pain intensity was related (conditionally) to husbands’ depressive symptomatology, but husbands’ pain intensity was not predictive of wives’ depressive symptoms. This finding is not easily contextualized, as prior research on the cross-
spouse effects of pain intensity is extremely limited and existing studies are generally not comparable. When considered against the backdrop of the growing body of research on aging married dyads, the gender difference in the cross-spouse effects of pain joins a minority of studies that has found the well-being of husbands to be (more) affected by the health status of their wives than vice-versa. Although this study yielded many compelling findings regarding gender differences in aging couples experiencing pain, these findings also leave many questions unanswered.

There is a need to explore the processes that account for such differences. There are still unanswered questions about the precise mechanism underlying the relationship between both spouses’ health and indicators of social well-being. Exploring and validating these gender differences should be an important priority in future research.

One speculation offered regarding the gender difference observed in the cross-spouse effect of pain intensity was that husbands and wives may be sensitive to different features of the pain in the spouse. Two features that can be explored relatively easily in future research are pain presence and pain transitions. It may be, for example, that wives are most affected by the initial onset of pain in their husbands. Or, perhaps wives are more sensitive to a change in their husbands’ pain, such as the transition from occasional to persistent or chronic pain. Husbands, on the other hand, may be more affected by ongoing pain in their wives. Although longitudinal data would be needed to investigate these possibilities, both are relatively approachable next steps with respect to advancing the study of DP couples. A number of experts have identified a pressing need for further investigation of gender (and/or sex) differences in pain. Most relevant to the findings of the present study are directives to clarify the role of psychological factors in, and explore
whether pain chronicity contributes to, observed gender differences (Fishbain et al., 2009; Greenspan et al., 2007). Other potential avenues for further research on gender include attention to pain histories, especially since women are more likely to have a history of pain (cite Greenspan et al., 2007) and the role of pain appraisals and coping, especially catastrophizing, defined as a general cognitive coping style or strategy characterized by feeling overwhelmed (e.g., Keefe et al., 2000 & Sullivan et al., 2000).

Balancing Specificity and Generality in Replicating and Extending This Study

Findings from the current study do not suggest one clear path for future research. As was outlined with respect to the construct of activity limitation above, understanding of the key constructs in this study could be advanced along several dimensions.

The current findings also raise questions about the roles of pain, physical limitations, limitations in other activities, and depressive symptomatology within the broader disablement process. Are IADLS & ADLS both additional mediators, do other factors moderate the effects of these on depressive symptoms? Earlier, in discussing the observed gender difference in activity limitation mediation at the intra-individual level, the possibility was raised that limitations in social activity might further mediate the pain-depressive symptomatology relationship in women. No prior study appears to have tested whether limitations in social functioning might mediate the relationship between physical limitations and depressive symptomatology.

Similar strategies could be used to replicate and extend the current findings with respect to the outcome variable in the present study—depressive symptomatology. Future studies may wish to examine whether relationships between pain intensity and
depressive symptomatology are replicable with respect to different dimensions of depressive symptomatology—for instance, somatic versus affective symptoms. This line of research might be especially warranted, given that the CES-D8 items endorsed most frequently by DP spouses tended to be more “somatic” in nature (i.e., hard to get going, restless sleep). It is also worth considering the impact of pain on specific symptoms of depression. For example, recent research suggests that lack of sleep may play an important role in the development and maintenance of depression. Moreover, pain appears to be a particular barrier to quality sleep. For instance, in a recent survey, 20% of adults reported that “pain or physical discomfort” disrupted their sleep at least a few nights per week (NCHS, 2006). Another potential direction worth exploring is the impact of pain on the constellation of depressive symptoms in older persons. Some researchers have suggested that depression in older adults is more characterized by a lack of positive affect than an elevation in negative affect (e.g., Gatz et al., 2001; Piazza et al., 2007). Perhaps pain contributes to this phenomenon among older persons by reducing their ability to experience positive affect. This is consistent with some work by Zautra and colleagues suggesting that pain might narrow the range of emotional experience and lead to an increasingly inverse relationship between positive and negative affect (e.g., Zautra et al., 2005).

Alternatively, future research could broaden the outcome under study and examine other consequences of pain. Among other potential indicators of psychological distress, anxiety is most well-studied. Several empirical investigations have linked pain to symptoms of anxiety, as well as anxiety disorders such as panic attacks and generalized anxiety disorder (e.g., McWilliams, Goodwin, & Cox, 2004). Additional
insight into gender differences might also be gained from examining more externalizing dimensions of psychological distress (e.g., anger), or behaviors reflective of such distress (e.g., negative social interactions, violence, etc.). Future research should also explore the relationship between pain and positive outcomes, such as life satisfaction (e.g., Kahana et al., 1997; Krueger & Stone, 2008). Self-rated health is another potential outcome of interest. In their mediation study, Bookwala et al. (2003) found that both physical and social functioning mediated the relationship between pain and perceived global health.

More research is needed to explore differences in pain intensity and the effects of pain across groups of persons defined by specific disease conditions (e.g., arthritis, lung disease, etc.). To this end, it would be useful to examine the potential additive and non-additive effects of multiple disease conditions and/or specific patterns of multi-morbidity. One potentially fruitful line of research is based on recent work by Dominick et al. (2012) in a representative sample of community-dwelling adults in Australia. Although they found that several disease conditions (e.g., arthritis, bowel disease) were independently related to reports of chronic pain, they also found that these conditions acted in an additive manner to increase the risk of reporting chronic pain. In addition, they showed that the type of disease condition and additional comorbid load (measured as a count of residual conditions—i.e., those that were not independently associated with pain, such as asthma and thyroid problems) were both independently associated with chronic pain. Specifically, having two or more residual conditions increased the odds of reporting chronic pain, above and beyond those chronic disease conditions that were independently associated with chronic pain, even after controlling for other relevant sociodemographic and background variables. Future research should examine the patterning of disease
conditions in relation to pain intensity. Data from the present study suggest that this could prove very challenging, however, largely because of the level of multi-morbidity observed in DP spouses. Future investigations might also explore whether the relationships between pain and depressive symptomatology differ across different disease conditions and/or combinations of conditions.

As the study was cross-sectional in nature, extensions using prospective, longitudinal data are warranted. Ultimately, we need prospective studies of individuals, and of couples, prior to the onset of significant problems with pain. Only by studying the relationship between pain and psychological distress as they develop and evolve over time can we really hope to firmly establish the causal ordering of pain and psychological distress at both the intra- and inter-individual levels. Prospective studies are also required to enhance support for activity limitation as the mediator of the intra-individual pain-depressive symptomatology relationship. And, of course, prospective studies will also inform ongoing efforts to identify the mechanism (or mechanisms) through which one spouse’s pain intensity influences the depressive symptomatology of his/her partner.

Although this study found evidence supporting one important pathway through which pain intensity influences depressive symptomatology (i.e., physical limitations), it is possible (even likely) that there are subordinate pathways or mediating mechanisms that lie within this mediation pathway. That is, there is likely to be a mediating pathway between each pair of contiguous constructs in the intra-individual path model: (1) between pain intensity and physical limitations (e.g., desire to do, fear of doing); (2) between physical limitations and depressive symptomatology (e.g., decreased ability to do larger-scale purposeful activities; decreased sense of independence or increased sense
of dependence; because cannot do some activities, may not receive rewards or positive feedback that is associated with those activities).

It is worth noting that most of the different directions outlined above could also be pursued at the inter-individual level. For example, perhaps different dimensions of depressive symptomatology are differentially affected by the pain of one’s spouse. There might also be a very specific mediating pathway in the cross-spouse effect of pain—for instance, pain in one spouse specifically disrupts the partner’s sleep, which then has a negative impact on the partner’s psychological well-being; however, it would only be possible to examine such a pathway if the construct of depressive symptomatology was uncontaminated by items related to sleep disturbance. With respect to pain, future studies might examine whether other dimensions or characteristics of one spouse’s pain exhibit similar associations with the partner’s depressive symptomatology. One possibility that was raised earlier in this chapter is that partners may be differentially affected by the onset, as opposed to the intensity, of the spouse’s pain. Or, perhaps the frequency with which the spouse experiences severe or “breakthrough” pain (i.e., a transient increase in pain intensity that “breaks through,” or is not alleviated by, his/her current level of pain medication) is most relevant to the psychological well-being of the partner. Alternatively, perhaps it is being able (or unable) to attribute the spouse’s pain to a specific cause that most negatively influences the partner’s emotional well-being.

The size of the DP sample and the availability of advanced statistical modeling techniques make it possible to explore the effects of any number of different combinations and interactions involving the key constructs in the proposed conceptual model. The interactions tested in the current study were selected on the basis of
theoretical importance, the suggestive findings of prior research, and specific gaps in the existing literature. Although the decision to limit the number of interactions tested in the present study was defended on theoretical, empirical, and practical grounds, the potential moderating effect of numerous other variables can, and should, be explored in future research. Two prime candidates have already been identified—age at the *intra*-individual level, and a measure of marital closeness or interdependence at the *inter*-individual level.

Research is needed to explore differences in pain intensity and the effects of pain across groups of persons defined by various demographic characteristics (e.g., gender, race, ethnicity, socioeconomic status, etc.) Other directions for future research include studying couples in more recent cohorts of middle-aged and older persons, as well as couples in other age groups. Extensions of this research to other types of couples (e.g., unmarried heterosexual and gay or lesbian couples) may also be of particular interest. Increased trends toward cohabitation and legally-recognized civil unions imply that coming generations of older adults will be more diverse, non-traditional couples will be more prevalent (Heaphy, Yip, & Thompson, 2004). Pain may fit nicely as a novel direction in which to move the study of non-traditional relationships in later life.

As the above discussion makes clear, there is no shortage of possible directions that could be pursued in subsequent studies. In attempting to replicate and extend the findings from this study, it will be important to temper the mechanized pursuit of those various possibilities with a commitment to the systematic and reasoned expansion of the underlying knowledge base. Each of the directions outlined above is likely to yield a host of interesting findings. However, additional theoretical and methodological work is needed to adequately understand those findings. Investments in theoretical development
and improved measurement should pay dividends to future researchers in terms of helping them to conceptually disentangle various constructs (e.g., physical limitations, IADL limitations, social disability) and allowing them to specify and evaluate specific mechanisms of effect (e.g., mediation, moderation, moderated-mediation, etc.) within different contexts (i.e., intra-individual, inter-individual).

Finally, relatively large residual variances were observed in the final multi-level models of the pain-physical limitations-depressive symptomatology relationship. This was true at both the intra-individual (model 4) and cross-spouse (model 9) levels, and for both DP husbands and wives. Thus, considerable amounts of variability in the depressive symptomatology of DP spouses remained unexplained. In fact, a fair amount of unexplained variance remained for the other two endogenous variables in the model (i.e., pain intensity and physical limitations), as well. The unexplained variability in each of these measures is likely to be related to a multitude of factors, only a few of which have been considered here. There is little shortage of possible directions for future research.

Evaluating the Relevance of the Chronic Versus Acute Pain Distinction in Mid and Later Life

Several findings from this study suggest that the HRS general pain item may be measuring pain that is perceived by respondents as “persistent” or “chronic.” The overall rates with which husbands and wives in the preliminary sample of HRS couples reported pain are more consistent with studies that have specifically sought to measure chronic pain. Additional evidence is provided by the lack of strong correspondence between DP spouses’ acknowledgement of disease-specific pain and their endorsement of the general pain item (e.g., that 75% of the husbands in the NP and WP couples reported arthritis-
related pain, even though they answered “no” to the “often troubled by pain” item).
Findings with regard to gender and the association between pain and depressive symptomatology (stronger in women) more closely parallel those from studies comprised of individuals with chronic and/or long-lived pain. Finally, the pattern of findings supporting (at least partial) mediation by physical limitations at the intra-individual level is most consistent with the findings of prior mediation studies in which pain was (either explicitly or implicitly) defined as chronic, persistent, and/or recurrent. Together, these data make a compelling case that the general pain question in HRS may be measuring chronic or persistent pain. Findings from the current study also dovetail nicely with those of Crook et al. (1984; see review in Chapter 4, p. 25) regarding the validity of self-reported “troublesome” pain and the tendency of individuals to downplay (rather than exaggerate) the persistence of their pain.

One practical implication of these findings is the need for additional clarification regarding how respondents in HRS interpret the “often troubled with pain” question. Qualitative research techniques, such as focus groups or cognitive interviewing, might help to illuminate the perceptual and cognitive processes that underlie participants’ responses to this item. It would be especially important to identify which (if any) dimensions and/or characteristics of the pain experience are considered by respondents when deciding how to answer. It would also be useful to explore the relationship between this item and alternative measures of pain intensity (along with other dimensions of pain) that have been used in different settings (e.g., the Brief Pain Inventory, visual analogue scales, etc.). Efforts such as these could yield several distinct benefits. First, findings could help to establish standards regarding how the responses of HRS
participants to this item should be conceptualized and characterized in published research. Second, these efforts could inform efforts to improve the measurement of pain in future, large-scale studies of community-dwelling aging adults, including HRS.

If observations about the nature of the pain reported by HRS participants can be confirmed through additional—especially longitudinal—analyses, they could challenge conventional notions of chronic pain (e.g., pain that persists long after damaged tissue has recovered; pain without an organic cause; pain that lasts 3 months, 6 months, or longer). Many types of pain are unlikely to meet the various criteria used to identify chronic pain—both clinically, and in research. For example, pain that lasts for less than 3 or 6 months, but is recurrent, may be a form of chronic pain that does not satisfy the current definition (Von Korff et al., 2000). Unfortunately, much of the pain experienced by older adults may fall into these definitional “grey areas.” This may be due to any number of reasons (e.g., lack of good pain control medications for use in older adults; multi-morbidities in older adults may result in multiple sources of pain that, together, contribute to an experience of chronic pain). One additional possibility—that “chronic” pain may be a fundamentally different phenomenon among aging adults—has been debated periodically by those who study pain in older adults (for examples, see Gagliese, 2009, 2011; Melding, 1991). Especially among aging adults, the distinction between chronic and acute pain may not be that valuable or relevant. As reported in Chapter 2, the American Geriatrics Society has made a very deliberate choice in its publications on pain to eschew the terminology of “chronic” pain. Instead, they advocate the use of “persistent” pain (AGS, 2002, 2009). Regardless of language, additional study of the pain reported by midlife and older adults is clearly warranted.
Conclusion: Study Strengths and Contributions

Pain has been relatively understudied in middle-aged and older persons, despite being common among aging persons. Myriad deleterious effects of pain have been documented, including negative impact on individuals and society. Yet, pain occurs in a social context and is known to affect families; spouses seem especially vulnerable. Most of what is known about couples and pain comes from research in clinical settings (e.g., with chronic pain patients and their spouses). Little is known about the pain experienced by couples living in the community—especially those in mid and later life for whom the prevalence of pain is high. Moreover, existing research has generally ignored the possibility that each spouse could be suffering from pain.

Recognizing that pain occurs in a social context, the present study sought to examine reports of pain among aging couples. One aim of the present study was to examine reports of pain in a nationally-representative sample and to use this sample to identify a subsample of community-dwelling couples in which both partners reported pain. As part of this aim, these “Dual-Pain” Couples were described and compared to other couples in HRS, including those in which one or neither spouse reported pain. This is the first known study to specifically examine reports of pain at the level of married couples.

One notable contribution of the present study is the identification of a sizeable group of couples in which both spouses report problems with pain. DP spouses comprised only eight percent of couples in this wave of HRS; however, when extrapolated to current population data, the number of DP couples can be expected to be sizeable. Moreover, analyses identified several ways in which these couples (and the
husbands and wives in them) differed from those couples in which only one spouse or neither reported problems with pain. Further research will be required to determine whether the observed differences are antecedents, consequences, or incidental to the presence of pain in both spouses. The selection of these dual-pain couples as the focal sample is also significant from both methodological and conceptual standpoints. The demonstrated existence of a sizeable number of midlife and older couples in which both report problems with pain introduces additional complications to the study of pain in aging couples. Researchers would be wise to carefully evaluate the partners of their “index” pain patients/subjects. Researchers might consider screening for pain in partners early in the recruitment process, and then be prepared to administer parallel measures to both spouses. Because both husbands and wives reported problems with pain, these couples provided an unprecedented opportunity to investigate an extensive range of research questions and hypotheses.

Unlike many previous studies that have focused on pain within married couples, DP couples were not selected on the basis of one spouse seeking treatment. In addition, although it is possible that some DP couples were involved in relationships characterized by high levels of unidirectional support and assistance, the couples were not selected based upon some pre-defined “caregiving” relationship. In contrast, the dual-pain (DP) couples in the present study were identified within a large, nationally-representative survey of community-dwelling middle-aged and older adults; most importantly, survey respondents were not selected on the basis of any pre-existing disease condition or the receipt of clinical services. This study also did not limit reports of pain to some arbitrary criterion for chronicity (e.g., 3 months, 6 months). As a result, it is likely these couples
represented a more naturally-occurring continuum of pain intensity, physical health status, and levels of impairment and disability than have been the focus of many prior studies of pain.

This study was strongly grounded in theory. Research questions and hypotheses at the intra-individual level were guided primarily by a conceptual framework that attempted to integrate Williamson’s activity restriction model of depressed affect (a.k.a., activity limitation theory) and Verbrugge & Jette’s (1996) model of the disablement process. Using this framework research questions and hypotheses were developed around the relationship between pain and psychological distress and whether it was mediated by activity limitation (Study Aim 2).

Drawing on established conceptual tenets related to interdependence among intimate partners (e.g., Kelley & Thibault, 1978) the activity limitation framework was expanded to the inter-individual level. This framework was used to examine the cross-spouse relationships between pain and depressive symptomatology. Research questions and hypotheses were developed around the cross-spouse relationship between each spouse’s pain and psychological distress of his/her partner, and whether this relationship it was mediated by the spouse’s activity limitation. Although more conceptual and theoretical work remains to be done in this area, this study provided some insights into the effects of pain on depressive symptoms within aging couples (Study Aim 3).

Even though several of the measures used in this study have some acknowledged limitations, strengths are present as well: Although many large-scale national studies do include one or two questions about pain, such items may only assess certain types or locations of pain (e.g., “joint pain,” “hip or leg pain” in NHANES studies) or may only
be asked of those with specific conditions (e.g., arthritis). The data source for the present study—HRS—did go beyond just assessing the presence of pain to include at least one dimension or facet of pain (i.e., pain intensity). HRS also included some additional disease-specific pain questions; these helped to place the pain reports of respondents into a somewhat broader context. The available measure of activity limitation was also clearly focused on basic physical limitation—a construct more consistent with models of the disablement process, especially within this age group.

The dyadic structure of the data also permitted examination of spousal concordance and differences in key study variables (parts of Study Aims 2, 3, and 4). The dyadic data and the use of multilevel modeling provided an opportunity to examine research questions at both the intra-individual and inter-individual levels. One benefit conveyed by the use of dual-intercept MLM models is that these models adjust for measurement error in the dependent variable. In the present study, both main endogenous variables (physical limitations and CES-D) were examined in mediation models that were adjusted for measurement error. This reduces the likelihood of inflated estimates and effects in mediation models (e.g., Kenny, Kashy, & Bolger, 1998; Kenny, 2009). The dual-intercept multilevel model also has other advantages—for example, the ability to examine and test possible gender differences, not just in the direct effects, but also in mediated effects. In addition, it provided the ability to examine and test possible gender differences in both direct and indirect effects, and to do so in a more efficient (and potentially more powerful) way than having to include all possible interaction terms within a multiple regression model. This permitted examination of gender differences beyond a main effects model tested in most prior studies. This study advances the
existing literature base by characterizing the relationship between pain intensity and depressive symptomatology separately for husbands and wives. The approach adopted in this study is consistent with calls for researchers to routinely assess gender differences in scientific research and also to report results—especially for health-related outcomes—separately for men and women (IOM, 2010).

Study hypotheses, especially those related to the proposed activity limitation mediation pathway, were evaluated in a rigorous manner. Firstly, the size of the indirect effect of pain intensity on depressive symptomatology was evaluated statistically, whereas, with few exceptions (e.g., Bookwala et al., Kahana et al.), prior mediation studies have typically concluded that mediation has occurred when the initial direct effect has been reduced to zero or is no longer statistically significant. Secondly, mediation in the present study was evaluated in the presence of a relatively large number of covariates suggested from theory and/or prior research (except Bookwala et al., 2003; Kahana et al., 1997). In addition, these models incorporated some variables that have not received attention in prior studies (e.g., BMI, wealth). Finally, as noted above, husbands’ and wives’ data were modeled separately, but simultaneously. The present study was able to evaluate whether any of the relationships among pain, activity limitation, and depressive symptomatology varied by gender. Moreover, the use of dual-intercept modeling also allowed gender differences to be evaluated statistically. Other study strengths include the use of multiple imputation as a strategy to address missing data, and sensitivity analyses to examine the effects of several key decisions in the modeling of the data.

This study also made several important contributions to the existing literature with respect to the relationships between pain and psychological distress at the intra-
individual level. First, mediation was demonstrated using a measure of activity limitation comprised solely of physical limitations (vs. IADL and/or ADL limitations). Second, gender was found to moderate the extent to which activity limitation mediated the pain-depressive symptomatology relationship, with more complete mediation observed in husbands as compared to wives. Third, mediation was found in a sample of married couples in which both partners reported problems with pain. And, although this sample could be distinguished from those of prior mediation studies on several levels (e.g., equal gender representation, all respondents were married, and all were married to another person with pain), study findings regarding mediation were still largely consistent with the findings of prior research. Fourth, and finally, mediation was found in a sample of adults of advanced age.

This dissertation is the first known study to identify a large sample of community-dwelling, aging couples in which both spouses have pain, and to use this sample to examine the impact of each spouse’s own pain, as well as the pain experienced by his/her partner, on the depressive symptomatology of both spouses. This study also examined a novel interaction effect—one that heretofore has not been subject to conceptual or empirical consideration—the joint effect of both spouses’ pain intensity ratings on the psychological distress of each spouse. This study adds to the growing body of empirical evidence that the characteristics and experiences of one partner in a close relationship can influence the well-being of the other, above and beyond the effects of his/her own characteristics and experiences.

Findings from this study suggest that pain should be considered from a dyadic perspective in future research and practice. Dual-pain couples, in particular, merit
additional attention and more intensive study. Several directions for future research and theory building were identified, many of which may help to elucidate the mechanisms underlying the gender differences observed in this study. Findings from this study can be used to inform social work practice and advocacy efforts with aging couples affected by pain. Ultimately, it is hoped that these findings can contribute to ongoing efforts to improve the lives of individuals and couples struggling with pain.

Although few people die of pain, millions die in pain, and even more live in pain.

(IASP, 2012, “Global Year Against Pain”)

6-196
Appendix A

*HRS 1998 Questionnaire Items*

**Conventions Used in This Presentation**

Question numbers are provided where possible, and correspond to the section of the HRS 1998 survey in which they were asked (e.g., Section B contained health items). The text presented in each section represents the exact wording of the interview questions and the response options provided to respondents, with three general exceptions:

1. Interviewers did not read question (or subquestion) numbers to respondents, nor did they read any of the codes or values assigned to the response options.

2. Instructions to interviewers and/or coding algorithms used in the interview software (e.g., skip patterns, options in wording, reading “pre-loaded” responses from prior data collection wave) are presented in [brackets].

3. Wording of response options may have varied slightly, depending on whether the options had been introduced in a prior question and/or whether a response card or prompt card was used during the interview. Some response options were only used to code respondents’ answers and were not read aloud by interviewers—for example, DK (Don’t know), and RF (Refused).

Due to the detailed nature of the HRS interview questions used to elicit financial data and some background information (e.g., enumeration of household residents) and the complicated sequencing, response categories, and follow-up prompts associated with those questions, only variable names (from the RAND data set) and general descriptions of the content are provided for some sections. Details about the “Unfolding-Bracketing” strategy used by HRS to collect financial data are presented in Appendix B. Additional
details regarding the construction of variables are available on the HRS website and/or in
the *RAND Version I Codebook* (St. Clair et al., 2009).

For questions with pre-defined response options, the response options and the
original codes or values assigned to each are presented in a table following the question.
Details regarding coding algorithms employed in the interview software, decision rules
used by RAND to assign new codes or values, and any calculations or transformations
applied prior to the release of the data (by RAND or HRS) are presented (as bullet points)
under the “Notes” heading in each section. Other relevant information (e.g., original item
source) is presented there, as well.

**Depressive Symptomatology**

C5.  Now think about the past week and the feelings you have experienced.

   Please tell me if each of the following was true for you much of the time
during the past week:

   C5a.  You felt depressed?
   C5b.  You felt that everything you did was an effort?
   C5c.  Your sleep was restless?
   C5d.  You were happy?*
   C5e.  You felt lonely?
   C5f.  You enjoyed life?*
   C5g.  You felt sad?
   C5h.  You could not get going?

<table>
<thead>
<tr>
<th>Response option</th>
<th>Yes</th>
<th>No</th>
<th>DK</th>
<th>RF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Original code(s)</td>
<td>1</td>
<td>5</td>
<td>8</td>
<td>9</td>
</tr>
</tbody>
</table>
Notes:

- Item source: Center for Epidemiologic Studies-Depression Scale (Radloff, 1977).
- *Denotes items that were reverse-scored, so that 1 = presence of symptom.

Pain

B18. Are you often troubled with pain?

<table>
<thead>
<tr>
<th>Response option</th>
<th>Yes</th>
<th>No</th>
<th>DK</th>
<th>RF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Original code(s)</td>
<td>1</td>
<td>5</td>
<td>8</td>
<td>9</td>
</tr>
</tbody>
</table>

[If 5, 8, or 9, skip to next section]

B18b. How bad is the pain most of the time: mild, moderate, or severe?

<table>
<thead>
<tr>
<th>Response option</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
<th>DK</th>
<th>RF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Original code(s)</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>8</td>
<td>9</td>
</tr>
</tbody>
</table>

In the last two years,…[or if re-interview: “Since your last interview…”]:

B7i. Have you had any angina or chest pains due to your heart? (HD*)

B11c. Do you sometimes have pain, stiffness, or swelling in your joints? (AR*)

<table>
<thead>
<tr>
<th>Response option</th>
<th>Yes</th>
<th>No</th>
<th>DK</th>
<th>RF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Original code(s)</td>
<td>1</td>
<td>5</td>
<td>8</td>
<td>9</td>
</tr>
</tbody>
</table>

[If 5, 8, or 9, skip to next set of questions]

Notes:

- *These questions were disease-specific and were administered only as part of the question sequence for those reporting heart disease (HD) and arthritis (AR).
Activity Limitation: Physical Limitations

E59. We need to understand difficulties people may have with various activities because of a health or physical problem. Please tell me whether you have any difficulty doing each of the everyday activities that I read to you. Exclude any difficulties that you expect to last less than three months. Because of a health problem, do you have any difficulty with…

E60. Walking several blocks?  [If 1, skip to 3; if 6, 7, 8, or 9, skip to 3]
E61. Running or jogging about a mile?  [If 1, 6, 7, 8, or 9, skip to 4]*
E62. Walking one block?
E63. Sitting for about two hours?
E64. Getting up from a chair after sitting for long periods?
E65. Climbing several flights of stairs without resting?  [If 5, skip to 8]
E66. Climbing one flight of stairs without resting?
E67. Stooping, kneeling, or crouching?
E68. Reaching or extending your arms above shoulder level?
E69. Pulling or pushing large objects like a living room chair?
E70. Lifting or carrying weights over 10 pounds, like a heavy bag of groceries?
E71. Picking up a dime from a table?

<table>
<thead>
<tr>
<th>Response option</th>
<th>Yes</th>
<th>No</th>
<th>Can’t Do</th>
<th>Don’t Do</th>
<th>DK</th>
<th>RK</th>
</tr>
</thead>
<tbody>
<tr>
<td>Original code(s)</td>
<td>1</td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
</tr>
</tbody>
</table>

Notes:

- *This item was not used in the current study.
Disease Conditions

[If re-interview and respondent reported the condition in the prior interview, respondent was read the following: “Our records from your last interview show that you have …{condition}.” If not disputed, code of 1 (Yes) was entered.]*

[If re-interview and respondent did not report the condition in the prior interview, respondent was asked: “Since we last talked to you, that is since {month, year of last interview}, has a doctor told you that you have…” ], else:

Has a doctor ever told you that you have…

B3. High blood pressure or hypertension?

B4. Diabetes or high blood sugar?

B5. Cancer or a malignant tumor, excluding minor skin cancers?

B6. Chronic lung disease such as chronic bronchitis or emphysema?

B7. [had a] Heart attack, coronary heart disease, angina, congestive heart failure, or other heart problems?

B9. [had a] Stroke?

B11. Arthritis or rheumatism?

<table>
<thead>
<tr>
<th>Response option</th>
<th>Yes</th>
<th>No</th>
<th>DK</th>
<th>RF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Original code(s)</td>
<td>1</td>
<td>5</td>
<td>8</td>
<td>9</td>
</tr>
</tbody>
</table>

Notes:

- *RAND cleaned these data by referring to prior and future years’ surveys and also to disputes or corrections that respondents made to “pre-loaded” information presented during the interview. RAND staff looked at all prior and future years’ data to determine whether any recorded dispute appeared “valid;” they then would correct the current response, if necessary.
Body Weight

B22. About how much do you weigh?

________ Pounds   DK   RF

B22d. About how tall are you?

________ Feet    ________ Inches   DK   _____RF

Notes:

- RAND transformed values into metric units.
- BMI was then calculated as: (Weight in kilograms) / (Height in meters^2).

Individual-level Background Characteristics

General Note:

- For most individual demographic variables (e.g., race, education, etc.), RAND used the first, non-missing value across all waves of HRS raw data and the Tracker file.

Gender

_______ Female     _____ Male

Notes:

- Gender information was pre-loaded in the interview software on the basis of information from prior screening to identify eligible households. HRS interviewers were instructed to check this information during the interviews and ask the respondent directly if there was a question.
Age

A1. In what month, day and year were you born?

______Month   _____Day _____Year ____DK _____RF

Notes:

• RAND calculated Age (in years) as: Interview End Date (Year and Month) – Respondent Birth Date (Year and Month).

Ethnicity

A7. Do you consider yourself Hispanic or Latino?

<table>
<thead>
<tr>
<th>Response option</th>
<th>Yes</th>
<th>No</th>
<th>DK</th>
<th>RF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Original code(s)*</td>
<td>1</td>
<td>2</td>
<td>8</td>
<td>9</td>
</tr>
</tbody>
</table>

[If 5, 8, or 9 skip to A8]

A7a. Would you say you are Mexican American, Puerto Rican, Cuban American or something else? [choose all that apply]

<table>
<thead>
<tr>
<th>Response option</th>
<th>Mexican American/Chicano</th>
<th>Puerto Rican</th>
<th>Cuban American</th>
<th>Other (specify)</th>
<th>DK</th>
<th>RF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Original code(s)*</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>7 [recorded response]</td>
<td>8</td>
<td>9</td>
</tr>
</tbody>
</table>

Notes:

• *If a respondent reported that he/she was Hispanic, but no race was provided in response to question A8, RAND set race to White/Caucasian.
Race

A8. Do you consider yourself primarily white or Caucasian, Black or African American, American Indian, or Asian, or something else?

<table>
<thead>
<tr>
<th>Response option</th>
<th>White/Caucasian</th>
<th>Black/African American</th>
<th>American Indian/Alaskan Native</th>
<th>Asian/Pacific Islander</th>
<th>Other (specify)</th>
<th>DK</th>
<th>RF</th>
</tr>
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<td>2</td>
<td>3</td>
<td>4</td>
<td>7 [response recorded]</td>
<td>8</td>
<td>9</td>
</tr>
</tbody>
</table>

Notes:

- *Beginning with wave 3 (1996), race data are masked by HRS; no details are available about the race information reported under “Other” race.

SES: Education

A3. What is the highest grade of school or year of college you completed?

<table>
<thead>
<tr>
<th>Response option</th>
<th>No Formal Education</th>
<th>Grades</th>
<th>High School</th>
<th>Some College</th>
<th>College Grad</th>
<th>Post College</th>
<th>Other (specify)</th>
<th>DK</th>
<th>RF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Original code(s) (entered as # years)</td>
<td>0</td>
<td>1-11</td>
<td>12</td>
<td>13-15</td>
<td>16</td>
<td>17</td>
<td>97*</td>
<td>8</td>
<td>9</td>
</tr>
</tbody>
</table>

Notes:

- *HRS typically re-coded “Other” responses into existing categories.
Couple-Level Background Characteristics

Length of Marriage

A40a. In what month and year were you married?

_____Month  _____Year  DK  RF

Notes:

- Marriage history was asked of all respondents at their first interview and updated as necessary in subsequent waves. Information in RAND dataset is for the current marriage.

- Duration of marriage was calculated by RAND as: Interview End Date (Year and Month) – Respondent’s Date of Current Marriage (Year and Month).

Household Composition: Others Living in the Household

HHwHHRES: The number of residents in the household is derived by a computer algorithm that counts the number of people reported on the household roster.* All household residents, including the respondent and his/her spouse, appear as observations on the household roster.

Notes:

- *HH roster is completed at the start of the interview with the designated HRS “Household Respondent.”

- In the current study, household composition was considered as a dichotomous variable that indicated whether or not anyone else lived in the household with the married couple.
Total Household Income

**HwITOT**: Calculated by RAND as income during the last calendar year from all household members,* from all sources:

a. RwIEARN (and SwIEARN, if applicable): Sum of Respondent’s (and Spouse’s, if applicable) wage/salary income, bonuses, overtime pay, commissions/tips, second job earnings, professional practice or trade income.

b. HwICAP: Household capital income, including business and farm income, self-employment income, rent income, dividend/interest income, other asset income.

c. RwIPENA (and SwIPENA): Income from all pensions and annuities.

d. RwISSDI (and SwISSDI): Income from Social Security Disability (SDI) and Supplemental Security Income (SSI), including disability, survivor, or dependent benefits.

e. RwISRET (and SwISRET): Income from Social Security retirement payments.

f. RwIUNNC (and SwIUNNC): Income from unemployment and worker’s compensation.

g. RwIGXFR (and SwIGXFR): Income from other government transfers, e.g., veteran’s benefits, welfare, food stamps.

h. HwIOTHR: All other household income including alimony, lump sum payouts from insurance, pensions, inheritances since prior wave.

**Notes**:

- Items asked only of the designated HRS “Financial Respondent.”
- *Starting with Wave 3, HRS only asked for respondent’s income and that of his/her spouse/partner, if applicable.
Total Household Wealth

HwATOTA: Calculated by RAND as net value of total household wealth: (Sum of all household wealth or assets) – (Sum of all household debts).

a. Sum of all household wealth components:
   i. HwAHOUS: Total value of primary residence.
   ii. HwARLES: Net value of real estate other than primary residence.
   iii. HwATRANS: Net value of vehicles owned.
   iv. HwABSNS: Net value of all or part of owned farm or business.
   v. HwAIRA: Net value of all IRA/Keogh the accounts (only questioned about 3 largest accounts individually).
   vi. HwSTCK: Net value of all stocks and mutual funds.
   vii. HwACHCK: Net value of all checking, savings, and money market accounts.
   viii. HwACD: Net value of all CDs, government savings bonds, T-bills.
   ix. HwABOND: Net value of all bonds or bond funds, including corporate, municipal, or foreign bonds.
   x. HwAOTHER: Net value of all other savings or assets (e.g., jewelry, money owed, collectibles/collections).

b. Sum of all household debts:
   i. HwMORT: Value of all mortgages (1\textsuperscript{st}, 2\textsuperscript{nd}, or land contract).
   ii. HwHMLN: Balance of all other home loans, including home equity lines of credit.
   iii. HwDEBT: Balance of all other debt (e.g., credit cards, medical bills, loans from relatives).

Notes:

- Items asked only of the designated HRS “Financial Respondent.”
Appendix B

Details Regarding the HRS “Unfolding-Bracketing” Strategy and the RAND Imputations for HRS Household Income and Wealth Data*

HRS “Unfolding-Bracketing” Strategy

In order to gather more accurate estimates regarding potentially-sensitive income and wealth data from respondents, HRS investigators designed a questioning strategy that used a series of progressive probes with increasingly-specific ranges of values presented to respondents—a so-called “unfolding and bracketing” strategy (Moon & Juster, 1995). If the household financial respondent indicated that he/she had a particular source of income (or owned a particular asset), the interviewer asked for the current value. If the respondent was unable or unwilling to provide an exact amount, the interviewer asked whether or not the amount was over some value (e.g., “was interest income over $2,500?”). Depending on the respondent’s answer, the interviewer followed-up with a sequence of probes intended to characterize the amount within increasingly narrow ranges (“brackets”). Different categories of income and assets had different brackets. For example, final brackets for some sources of income were $0 to $250, $201 to $500, $501 to $2,500, $2,501 to $10,000, and $10,001 or more. The initial value presented to respondents is an “entry point”—$2,500 for interest income in the example above. In HRS 1998, the entry points for the various income and asset brackets were randomly assigned to avoid biasing respondents (St. Clair et al., 2009, Section 3).

RAND Imputations for HRS Household Income and Wealth Data

Respondents in HRS were permitted to opt-out of the question sequence (i.e., the “unfolding brackets”) for income and wealth at any time. Hence, in addition to valid
“no/no ownership” responses (i.e., respondent does not have that type of income/asset), true income/asset values of zero, and responses indicating an exact value, the raw HRS data for income and wealth components had four “categories” of missing data:

1. The respondent reported having/owning that type of income/asset, but did not know or refused to provide any amount, or answer any bracketing questions;
2. He/she responded to an early bracketing probe, but was unable or unwilling to complete additional (or the final) bracketing probe;
3. The respondent did not know or refused to answer the initial question about whether or not he/she had/owned a particular type of income/asset; and
4. No financial respondent was available for this household at this wave.

When an income or wealth variable was missing or incomplete, RAND imputed the values using a progressive, three-step imputation process (St. Clair et al., 2009). The first step involved imputing income type or asset ownership for cases in which this information was unknown (categories 3 and 4 above). The second step involved imputing a range (i.e., value bracket) for cases in which only ownership or an incomplete range was known (categories 1 and 2 above, as well as imputed data from the first step). The third step involved imputing an exact amount in cases in which only a range was known (special cases of category 2 above, as well as imputed data from the second step).

Different imputation procedures were used for each of the imputation steps. All procedures were single-imputation strategies that involved replacing missing or incomplete values for a given type of income or asset with probable and plausible values derived from statistical models based on cases with complete data for that income or asset type (St. Clair et al., 2009). Logistic regression models (with random selection of a
predicted probability cutoff point) were used to impute ownership (step 1). Ordinal logit models (with random selection of a predicted or conditional probability cutoff point) were used to impute brackets/ranges (step 2).

Imputation of exact values (step 3) was done differently for closed-range brackets (e.g., $1,000 to $5,000) and open-range brackets (e.g., more than $2,500). For closed-range brackets, an OLS regression model was first estimated for households reporting an exact continuous value. This model was then used to calculate predicted values for all households (both those with and without exact values). Then, each household missing an exact value was assigned the exact continuous value that was reported by the household(s) that was closest in predicted value. Because this procedure would have yielded too many implausible values and too little variability for those households with incomplete data in the open-range brackets, a different procedure was used for these data. First, a Tobit model was estimated on the basis of the (non-missing) households in the top 75% of values for a given variable. Predicted values were then estimated for the missing households based on this model. To ensure adequate variability, a random procedure was used to draw values from the residual distribution; these values were then added to the predicted value to generate an imputed exact amount for those in the open-range brackets. The same set of explanatory variables (e.g., education, occupation, gender) was included in all of the asset imputation models, and another consistent set was used in all of the income imputation models (St. Clair et al., 2009, Section 3).

*Note. Information contained in this Appendix was summarized and rephrased from the RAND HRS Version I Codebook (St. Clair et al., 2009, Section 3).
### Appendix C

#### Variables in the Datafile Used in the Multiple Imputation Procedure

<table>
<thead>
<tr>
<th>#</th>
<th>Variable Name</th>
<th>Label and Coding</th>
<th>Model (MOD), Auxiliary (aux), or Other (oth)</th>
<th>Imputation Notes</th>
<th>Special Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Identifying Variables</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>H4HHID</td>
<td>Household ID [HRS]</td>
<td>No</td>
<td>ID var only</td>
<td></td>
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<tr>
<td>2</td>
<td>RAGENDER</td>
<td>Gender of Respondent</td>
<td>No</td>
<td>ID var only</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>h_RAHHIDPN_new</td>
<td>Husband Household ID + Person Number [HRS]</td>
<td>No</td>
<td>ID var only</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>w_RAHHIDPN_new</td>
<td>Wife Household ID + Person Number [HRS]</td>
<td>No</td>
<td>ID var only</td>
<td></td>
</tr>
<tr>
<td><strong>Couple/Household Variables</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>H4MCURLN_rnd</td>
<td>Length of marriage (rounded to 1/2 yrs.; used W value, but subst. H value if W value missing)</td>
<td>MOD</td>
<td></td>
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<tr>
<td>6</td>
<td>H4TOT_lg</td>
<td>HH Income (logged)</td>
<td>MOD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>H4ATOTB_ihs</td>
<td>HH Wealth (ihs, inv hyperbolic sine transformation)</td>
<td>MOD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>H4HRES_d</td>
<td>Others Living in HH (yes/no)</td>
<td>MOD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>H4_FAMR</td>
<td>Wife is Family respondent this wave (y/n)</td>
<td>aux</td>
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<td></td>
</tr>
<tr>
<td>10</td>
<td>H4_FINR</td>
<td>Wife is Financial respondent this wave (y/n)</td>
<td>aux</td>
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<td></td>
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<tr>
<td>11</td>
<td>H4CHILD_tp8</td>
<td># living children total (incl. children of either Respondent or Spouse, or both)</td>
<td>aux</td>
<td>Constraints: [ 0 - 8]</td>
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<tr>
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<td>H4CENDIV</td>
<td>Census Division [original var] [9 cat]</td>
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<td></td>
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<tr>
<td>13</td>
<td>cpl_pn4cat</td>
<td>Couple Pain Category (1-4)</td>
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<td>duplicated info from H and W Pain vars</td>
<td></td>
</tr>
<tr>
<td><strong>Individual Variables (file contains both h_ &amp; w_ versions of each)</strong></td>
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<tr>
<td>14</td>
<td>R4AGEY_E</td>
<td>Age</td>
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<td></td>
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<tr>
<td>15</td>
<td>RAEHYRS</td>
<td>Yrs. of Education (0 - 17+)</td>
<td>MOD</td>
<td>Constraints: H [0 - 17]</td>
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<td>16</td>
<td>RARACEM</td>
<td>Race (RAND var [3 cat]: White, Black, Other)</td>
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<tr>
<td>17</td>
<td>RAHISPAN</td>
<td>Hispanic (y/n)</td>
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<td>18</td>
<td>R4ARTHRE</td>
<td>Arthritis (y/n)</td>
<td>MOD</td>
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<tr>
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<td>R4CANCRE</td>
<td>Cancer (y/n)</td>
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<td>R4LUNGE</td>
<td>Lung Disease (y/n)</td>
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<td>R4DIABE</td>
<td>Diabetes (y/n)</td>
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<tr>
<td>22</td>
<td>R4HEARTE</td>
<td>Heart Condition (y/n)</td>
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<td>23</td>
<td>R4HIBPE</td>
<td>High Blood Pressure (y/n)</td>
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<tr>
<td>24</td>
<td>R4STROKE</td>
<td>Stroke (y/n)</td>
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<tr>
<td>25</td>
<td>R4HEIGHT</td>
<td>Height (in meters)</td>
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<td>Constraints: H [1.37 - 2.11] W [0.94 - 1.91]</td>
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<tr>
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<td>R4WEIGHT</td>
<td>Weight (in kg)</td>
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<td>Constraints: H [45.35 - 172.36] W [36.28 - 149.24]</td>
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<tr>
<td>27</td>
<td>R4PAIN</td>
<td>Often troubled by pain (y/n)</td>
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<td>duplicated info from H and W Pain Intensity vars</td>
<td></td>
</tr>
<tr>
<td>28</td>
<td>R4PNINT_4lev</td>
<td>Pain intensity (4-levels, 0-3; 0 = No Pain to 3 = Severe Pain)</td>
<td>No</td>
<td>Used centered version; see var 95 below</td>
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<tr>
<td>29</td>
<td>R4PNLIM</td>
<td>Pain limits usual activity (recoded var [y/n], with 0 = No Lim or No Pain)</td>
<td>MOD</td>
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<td></td>
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<tr>
<td>30</td>
<td>F1168_d</td>
<td>Chest pain (y/n; no includes those w/no heart problem and also those w/heart problem but no symptoms and no doctor visit or current meds for heart problem)</td>
<td>MOD</td>
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</tr>
<tr>
<td>31</td>
<td>F1197_d</td>
<td>Joint pain (y/n; no also includes those w/out Arthritis)</td>
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<td></td>
</tr>
<tr>
<td>32</td>
<td>R4WALKSA_r</td>
<td>PhysLimits: Walk Blocks (y/n; w/logical imputation of some missing values)</td>
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<tr>
<td>33</td>
<td>R4WALK1A</td>
<td>PhysLimits: Walk 1 Block (y/n; conditional, but was coded OK)</td>
<td>MOD</td>
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<tr>
<td>#</td>
<td>Variable Name</td>
<td>Label and Coding</td>
<td>Model (MOD), Auxiliary (aux), or Other (oth)</td>
<td>Imputation Notes</td>
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<td>---------------------------------------------</td>
<td>------------------</td>
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<tr>
<td>34</td>
<td>R4SITA</td>
<td>PhysLimits: Sit 2 hrs. (y/n)</td>
<td>MOD</td>
<td>Used in Imputation Model (Y)</td>
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<td>35</td>
<td>R4CHAIRA</td>
<td>PhysLimits: Get up from Chair (y/n)</td>
<td>MOD</td>
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<tr>
<td>36</td>
<td>R4CLIMSA_r</td>
<td>PhysLimits: Climb Flights (y/n; w/logical imputation of some missing values)</td>
<td>MOD</td>
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<td></td>
</tr>
<tr>
<td>37</td>
<td>R4CLIM1A</td>
<td>PhysLimits: Climb 1 Flight (y/n; conditional, but was coded OK)</td>
<td>MOD</td>
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<tr>
<td>38</td>
<td>R4STOOPA</td>
<td>PhysLimits: Stoop (y/n)</td>
<td>MOD</td>
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<td></td>
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<tr>
<td>39</td>
<td>R4ARMSA</td>
<td>PhysLimits: Raise Arms (y/n)</td>
<td>MOD</td>
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<tr>
<td>40</td>
<td>R4PUSHHA</td>
<td>PhysLimits: Push/Pull (y/n)</td>
<td>MOD</td>
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<tr>
<td>41</td>
<td>R4LIFTA</td>
<td>PhysLimits: Lift/Carry (y/n)</td>
<td>MOD</td>
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<td>42</td>
<td>R4DIMEA</td>
<td>PhysLimits: Pick up Dime (y/n)</td>
<td>MOD</td>
<td></td>
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<tr>
<td>43</td>
<td>R4DEPRES</td>
<td>CES-D8: Depressed (y/n)</td>
<td>MOD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>44</td>
<td>R4EFFORT</td>
<td>CES-D8: Effort (y/n)</td>
<td>MOD</td>
<td></td>
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</tr>
<tr>
<td>45</td>
<td>R4SLEEPR</td>
<td>CES-D8: Restless Sleep (y/n)</td>
<td>MOD</td>
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<td></td>
</tr>
<tr>
<td>46</td>
<td>R4WHAPPY</td>
<td>CES-D8: Happy (y/n; reverse-coded after imputation)</td>
<td>MOD</td>
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</tr>
<tr>
<td>47</td>
<td>R4FLONE</td>
<td>CES-D8: Lonely (y/n)</td>
<td>MOD</td>
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</tr>
<tr>
<td>48</td>
<td>R4ENLIFE</td>
<td>CES-D8: Enjoy Life (y/n; reverse-coded after imputation)</td>
<td>MOD</td>
<td></td>
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</tr>
<tr>
<td>49</td>
<td>R4SAD</td>
<td>CES-D8: Sad (y/n)</td>
<td>MOD</td>
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<tr>
<td>50</td>
<td>R4GOING</td>
<td>CES-D8: Could Not Get Going (y/n)</td>
<td>MOD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>51</td>
<td>ATTRbyW6_d1</td>
<td>Wave 6 Attrition status (2 dummy-coded vars, reference group is No attrition) Dummy 1 - Death</td>
<td>aux</td>
<td></td>
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</tr>
<tr>
<td>52</td>
<td>ATTRbyW6_d2</td>
<td>Wave 6 Attrition status (2 dummy-coded vars, reference group is No attrition) Dummy 2 - Other</td>
<td>aux</td>
<td></td>
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</tr>
<tr>
<td>53</td>
<td>RACOHBYR_Scat</td>
<td>Birth cohort (recoded var [5 cat]: HRS, AHEAD, CODA, WB, Early BB or No Cohort)</td>
<td>aux</td>
<td></td>
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</tr>
<tr>
<td>54</td>
<td>R4PREVMAR</td>
<td>Married previously (y/n)</td>
<td>aux</td>
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</tr>
<tr>
<td>55</td>
<td>NOTUSBORN</td>
<td>Not born in the US (y/n; note: no could mean foreign born or born in a US territory)</td>
<td>aux</td>
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<tr>
<td>56</td>
<td>RELIMP</td>
<td>Importance of religion (recoded var [3 levels]: Not too, Somewhat, Very)</td>
<td>aux</td>
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<td></td>
</tr>
<tr>
<td>57</td>
<td>R4WALKRA</td>
<td>ADL Difficulty: Walking across room (y/n)</td>
<td>aux</td>
<td></td>
<td></td>
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<tr>
<td>58</td>
<td>R4DRESSA</td>
<td>ADL Difficulty: Dressing (y/n)</td>
<td>aux</td>
<td></td>
<td></td>
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<tr>
<td>59</td>
<td>R4BATHA</td>
<td>ADL Difficulty: Bathing (y/n)</td>
<td>aux</td>
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<td></td>
</tr>
<tr>
<td>60</td>
<td>R4EATA</td>
<td>ADL Difficulty: Eating (y/n)</td>
<td>aux</td>
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</tr>
<tr>
<td>61</td>
<td>R4BEDA</td>
<td>ADL Difficulty: Getting in/out of bed (y/n)</td>
<td>aux</td>
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<tr>
<td>62</td>
<td>R4TOILTA</td>
<td>ADL Difficulty: Using toilet (y/n)</td>
<td>aux</td>
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<td></td>
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<tr>
<td>63</td>
<td>R4PHONEA</td>
<td>IADL Difficulty: Making phone calls (y/n)</td>
<td>aux</td>
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<td></td>
</tr>
<tr>
<td>64</td>
<td>R4MONEYA</td>
<td>IADL Difficulty: Managing money (y/n)</td>
<td>aux</td>
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<td></td>
</tr>
<tr>
<td>65</td>
<td>R4MEDSA</td>
<td>IADL Difficulty: Taking medications (y/n)</td>
<td>aux</td>
<td></td>
<td></td>
</tr>
<tr>
<td>66</td>
<td>R4SHOPA</td>
<td>IADL Difficulty: Grocery shopping (y/n)</td>
<td>aux</td>
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</tr>
<tr>
<td>67</td>
<td>R4MEALSA</td>
<td>IADL Difficulty: Preparing hot meals (y/n)</td>
<td>aux</td>
<td></td>
<td></td>
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<tr>
<td>68</td>
<td>R4MAPA</td>
<td>IADL Difficulty: Using a map (y/n)</td>
<td>aux</td>
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<td></td>
</tr>
<tr>
<td>69</td>
<td>F2617_d</td>
<td>IADL Difficulty: Help with house/yard (y/n) [note: not part of traditional IADL scales]</td>
<td>aux</td>
<td></td>
<td></td>
</tr>
<tr>
<td>70</td>
<td>R4SHTL_n</td>
<td>Self-rated health (1-5, low-hi)</td>
<td>aux</td>
<td></td>
<td></td>
</tr>
<tr>
<td>71</td>
<td>R4ANYDISAB</td>
<td>Health causes any disability? (y/n)</td>
<td>aux</td>
<td></td>
<td></td>
</tr>
<tr>
<td>72</td>
<td>F1501_d</td>
<td>Had a lot of energy (y/n)</td>
<td>aux</td>
<td></td>
<td></td>
</tr>
<tr>
<td>73</td>
<td>R4_FALL</td>
<td>Fallen in past 2 yrs. (y/n; coded no if &lt; age 64 b/c not asked of those &lt; 65 [sometimes 64])</td>
<td>aux</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Notes:**
- Used in Imputation Model (Y): Y
- Special Notes: Y
- Constraints: W [1 - 5]
<table>
<thead>
<tr>
<th>#</th>
<th>Variable Name</th>
<th>Label and Coding</th>
<th>Model (MOD), Auxiliary (aux), or Other (oth)</th>
<th>Imputation Notes</th>
<th>Used in Imputation Model (Y)</th>
<th>Special Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>74</td>
<td>F1220_d</td>
<td>Urinary incontinence in past 12 mos. (y/n)</td>
<td>aux</td>
<td>Y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>75</td>
<td>F1236_n</td>
<td>Rating of hearing (w/aid, if use; 1 = poor to 5 = excellent)</td>
<td>aux</td>
<td>Y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>76</td>
<td>F1228_n</td>
<td>Rating of eyesight (bottom coded, 1 = poor or blind to 5 = excellent)</td>
<td>aux</td>
<td>Y</td>
<td>Constraints: H [1 - 5]</td>
<td></td>
</tr>
<tr>
<td>77</td>
<td>R4BEDDAY</td>
<td>Any bed days in past month? (y/n)</td>
<td>aux</td>
<td>Y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>78</td>
<td>R4cSHLT_d</td>
<td>Health in childhood was poor or fair (y/n)</td>
<td>aux</td>
<td>Y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>79</td>
<td>R4SMOKEV</td>
<td>Ever smoke? (y/n)</td>
<td>aux</td>
<td>Y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>80</td>
<td>R4SMOKEN</td>
<td>Currently smoke? (y/n)</td>
<td>aux</td>
<td>Y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>81</td>
<td>R4DRINKD_n</td>
<td># days drink/week (0 = does NOT drink; 1 = 1 day or less per week to 7 = all 7 days)</td>
<td>aux</td>
<td>Y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>82</td>
<td>R4DRINKN_n</td>
<td># drinks on days when drink? (0 = does NOT drink)</td>
<td>aux</td>
<td>Y</td>
<td>Constraints: H [0 - 12]</td>
<td></td>
</tr>
<tr>
<td>83</td>
<td>R4VIGACT</td>
<td>Engage in regular vigorous exercise or activity? (y/n)</td>
<td>aux</td>
<td>Y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>84</td>
<td>R4DOCTIM_6</td>
<td># doctor visits in past 2 yrs. (recoded into 6 levels, 0-5; 0 = None, 1 = 1-6 visits, 5 = 25 or more)</td>
<td>aux</td>
<td>Y</td>
<td>Constraints: H &amp; W [0 - 5]</td>
<td></td>
</tr>
<tr>
<td>85</td>
<td>R4HOSP</td>
<td>Overnight hospital stay in past 2 yrs. (y/n)</td>
<td>aux</td>
<td>Y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>86</td>
<td>R4HOMCAR</td>
<td>Home Care Use in past 2 yrs. (y/n)</td>
<td>aux</td>
<td>Y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>87</td>
<td>R4DRUGS</td>
<td>Regularly take prescription drugs (y/n)</td>
<td>aux</td>
<td>Y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>88</td>
<td>R4OUTPT</td>
<td>Outpatient surgery in past 2 yrs. (yes/no)</td>
<td>aux</td>
<td>Y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>89</td>
<td>R4PCFAC</td>
<td>Use other special facilities/svcs in past 2 yrs. (y/n; instructions specifically mention: outPT rehab, adult day care, social work, and meals/transportation/other svcs for elderly/disabled)</td>
<td>aux</td>
<td>Y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>90</td>
<td>R4PSYCHE</td>
<td>Psychiatric/Mental problems (y/n; ever diagnosed)</td>
<td>aux</td>
<td>Y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>91</td>
<td>R4HIGOV</td>
<td>Health Insurance (recoded into 2 categorical vars; not necessarily mutually exclusive, but combinations represent all categories) Var1: Govt/public insurance (y/n)</td>
<td>aux</td>
<td>Y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>92</td>
<td>R4PRVINS</td>
<td>Health Insurance (recoded into 2 categorical vars, not necessarily mutually exclusive, but combinations represent all categories) Var2: Private insurance (y/n; from own or spouse employer or other)</td>
<td>aux</td>
<td>Y</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Interaction Terms & Components (hXw = Couple variable, others are Individual variables--i.e., h_ and w_):**

<table>
<thead>
<tr>
<th>#</th>
<th>Variable Name</th>
<th>Description</th>
<th>Model (MOD), Auxiliary (aux), or Other (oth)</th>
<th>Imputation Notes</th>
<th>Used in Imputation Model (Y)</th>
<th>Special Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>93</td>
<td>R4PHLIMav_c</td>
<td>Physical Limitations-Total (11 PhysLims items, scored y/n; calculated as average of available items X 11 items and mean centered by gender)</td>
<td>oth</td>
<td>Y</td>
<td>needed to compute interaction terms; calculated using guidelines by</td>
<td></td>
</tr>
<tr>
<td>94</td>
<td>hXwPHLIMav</td>
<td>HUSB X WIFE Physical Lims Total Interaction Term (calculated using both spouse's R4PHLIMav_c [avg of avail PhysLims items X 11 &amp; mean centered by gender])</td>
<td>oth</td>
<td>Y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>95</td>
<td>R4PNINT_4lev_c</td>
<td>Pain Intensity (orig scored 0 [none] to 3 [severe]; mean centered by gender)</td>
<td>MOD</td>
<td>Y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>#</td>
<td>Variable Name</td>
<td>Label and Coding</td>
<td>Model (MOD), Auxiliary (aux), or Other (oth)</td>
<td>Imputation Notes</td>
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<td>------------------</td>
<td>---------------------------------------------</td>
<td>-----------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>96</td>
<td>hXwPNINT</td>
<td>HUSB X WIFE Pain Intensity Interaction Term (calculated using the centered pain intensity ratings of both spouses [R4PNINT_4lev_c])</td>
<td>oth</td>
<td>Y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>97</td>
<td>hXwPNLIM</td>
<td>HUSB X WIFE Pain Limitation Interaction Term (calculated using the pain limitation var of both spouses [R4PNLIM])</td>
<td>oth</td>
<td>Y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>98</td>
<td>PNINTxPHLIMav</td>
<td>Pain Intensity X Physical Limitations-Total Interaction Term (calculated using each spouse’s R4PNINT_4lev_c [centered pain intensity rating] and R4PHLIMav_c [his/her avg of avail PhysLims items X 11 and centered])</td>
<td>oth</td>
<td>Y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>99</td>
<td>PNINTxPNLIM</td>
<td>Pain Intensity X Pain Limitation Interaction Term (calculated using each spouse’s R4PNINT_4lev_c [centered pain intensity rating] and R4PNLIM [his/her pain limitation])</td>
<td>oth</td>
<td>Y</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

# vars listed above used in imputation model: 92

# duplicate indiv-level vars (i.e., for the spouse): 81

**TOTAL # VARS USED IN IMPUTATION: 173**

**TOTAL # VARS IN FILE: 182**

Note. ID = identification; HRS = Health & Retirement Study; H = Husband; W = Wife; y/n = a dichotomous variable coded yes or no; var(s) = variable(s); PhysLims = Physical Limitations [difficulty with [task]]; CES-D8 = 8-item version of the Center for Epidemiologic Studies-Depression Scale; IADL = Instrumental Activity of Daily Living; ADL = Activity of Daily Living.

1Notations indicate that a variable was part of the study’s conceptual/analytical model (MOD), an auxiliary variable drawn from the larger dataset to provide additional information to help impute missing values (aux), or a specially-derived variable used to incorporate interaction terms of interest into the imputation model (oth).

2Contains information about why a variable was or was not included in the imputation model and/or any constraints that were specified for the imputation of a variable (e.g., a specified range for imputed values – H or W indicates if the range was applied to both, or only one, spouse).

3Even though pain limitation was later dropped from the present study, it was included in all missing data analyses and in the imputation process.
Appendix D

Procedures for Fixing the Initial Error Variances

in the Dual-Intercept HLM Models

Cross-sectional, dyadic data are comprised of only two data points (e.g., one for the husband, one for the wife), which limits the degrees of freedom available for model estimation. In order to estimate the two random effects specified in a dual-intercept multilevel linear model, it is necessary to “fix” the initial error variances (i.e., the initial values of $u_{ij}$ and $u_{wj}$ in the Level-2 equation that is outlined in Appendix E). This strategy has been recommended by several experts in order to permit model estimation with limited dyadic data (e.g., Kenny, Kashy, & Cook, 2006; Lyons & Sayer, 2005).

In the software package utilized in the present study (HLM 6) the initial error variances can be fixed by directing the program to construct a “precision weight” based on a variable in the input dataset(s) that represents the “known” error variance of the dependent variable. The error variance of a measure is commonly derived from an estimate of the measure’s reliability. In the case of multi-item scales, Sayer and colleagues (e.g., Sayer & Klute, 2005; Sayer, 2007) recommend that the Cronbach’s alpha estimate of the internal consistency reliability of the scale be used to calculate the initial error variance estimate.

General Formula for Estimated Error Variance

The general formula for calculating the estimated error variance ($EV$) for a dependent variable measured using a multi-item scale is as follows:

$$EV_Y = (1 - \alpha_Y) \times Var_Y,$$

where: 

...
\[ Y = \text{the dependent variable (measured as a multi-item scale)}, \]
\[ \alpha_Y = \text{the Cronbach’s alpha internal consistency reliability estimate of } Y, \]
and
\[ Var_Y = \text{the observed variance of } Y. \]

**Formula for Estimated Error Variances in Distinguishable Dyads**

In dual-intercept models involving distinguishable dyads, Sayer and colleagues (e.g., Sayer & Klute, 2005) further recommend that error variance estimates be calculated separately for members of the two distinguishable groups. In the present study, dyad members were distinguished by gender. The above formula was thus extended to the current dyadic case as follows:

\[ EV_{Yh} = (1 - \alpha_{Yh}) \times Var_{Yh}, \]
\[ EV_{Yw} = (1 - \alpha_{Yw}) \times Var_{Yw}, \]

where all terms are as before, except \(Y\) is now further distinguished as:

\(Y_h = \text{the dependent variable measured in husbands, and}\)
\(Y_w = \text{the dependent variable measured in wives.}\)

**Sample Calculation of \(EV_{CESD8}\)**

Below is a sample calculation of the initial error variance estimates for the CES-D8 scores for DP husbands and wives. Note that the possible range of scores on the scale was 0 to 8. Known quantities derived from the observed data were as follows:

\[ Var_{CESD8h} = 4.29, \alpha_{CESD8h} = .75 \quad \text{(husbands)}, \]
\[ Var_{CESD8w} = 5.50, \alpha_{CESD8w} = .80 \quad \text{(wives)}. \]

Therefore:

\[ EV_{CESD8h} = (1 - .75) \times 4.29 = 1.06 \]
\[ EV_{\text{CESD8w}} = (1 - .80) \times 4.29 = 1.10 \]

The above quantities were then assigned (by gender) to each individual under a newly-created variable (e.g., “ErrVar_CES-D8”) in the Level 1 HLM6 dataset.

**Special Adjustments**

As described below, two adjustments to the above strategy were required in the present study.

*Multiply-Imputed Datasets*

The first adjustment was necessary because multiple imputation (MI) was used to address missing data in this study. As discussed in Chapter 4, the MI process generated eight separate imputations (or imputed datasets) that were used in subsequent analyses. The multi-item scales that served as dependent variables in the present study all had some missing values. Thus, for any given scale, the observed variance of scores and the calculated Cronbach’s alpha internal consistency estimate differed slightly from imputation to imputation. Because both quantities are used in the above formula, it was necessary to calculate the initial error variance estimates for each scale separately within each imputed dataset; in addition, these values were all calculated separately by gender (within each dataset).

*Single-Item Measure of Pain Intensity*

The use of a single-item measure of pain intensity necessitated a second adjustment. Given the lack of a Cronbach’s alpha reliability estimate (as well as data for other types of estimable reliability), an alternate strategy was needed to estimate the “reliability” of this measure. One alternative strategy that was considered, but rejected, included adopting a reliability estimate (or, an error variance estimate) from a prior study.
that used a multi-item measure of pain intensity. Aside from concerns about the comparability of data derived from the traditional clinical samples that have been used in prior research to the data that might be expected from using such a measure with the current sample of DP couples, this option was also rejected because of the lack of existing multi-item scales specific to the construct of pain intensity.

An alternative strategy for estimating the reliability of the pain intensity measure was developed that made use of other relevant, sample-specific data. This strategy was devised in consultation with leading experts in dyadic data analysis (J. Z. Smith & A. G. Sayer, personal communication, February, 24, 2008). An imputation-specific “average wife reliability estimate” was derived from the Cronbach’s alpha for wives’ scores on the two multi-item scales used in the present study—the 8-item CES-D and the 11-item physical limitations scale. This average was then used as the imputation-specific reliability estimate for wives’ pain intensity ratings; estimates ranged from .80 to .81.

In the current sample, husbands’ scale scores were consistently slightly less reliable than those of their wives. Thus, an imputation-specific “average husband reliability estimate” was derived by first calculating the differences between the Cronbach’s alpha estimates for the scores of wives and husbands on these two scales (CES-D8 and physical limitations). Next, these differences were averaged. Finally, this average reliability difference was subtracted from the imputation-specific reliability estimate of wives’ pain intensity. The resulting value was used as the imputation-specific reliability estimate for husbands’ pain intensity ratings; estimates ranged from .77 to .78.

These values were then used to calculate imputation- and gender-specific initial error variance estimates for pain intensity using the earlier formula (see p. D-2).
Appendix E

General Multilevel Model for the Dual-Intercept Model

The general multilevel equation for the dual-intercept model (as applied in the present study using HLM6) consisted of two levels. The equation, as specified at each level (and using depressive symptomatology as the dependent variable), was as follows:

Level-1 Equation

In the Level 1 model, the depressive symptomatology score (CES-D8) for a DP spouse was modeled as the sum of his/her latent true score, plus some amount of measurement error. The model was written as:

\[ Y_{ij} = H_{ij} (B_{hj} + r_{nj}) + W_{ij} (B_{wj} + r_{wj}), \]

where:

\[ Y_i = \text{the CES-D8 response for individual } i \text{ in couple } j \text{ (where } i = 1, 2; \text{ and } j = 1 \ldots N \text{ couples}), \]

\[ H_{ij} = \text{an indicator variable for individual } i \text{ in couple } j; H \text{ has a value of 1 if the response was obtained from the husband, 0 if the response was obtained from the wife}, \]

\[ B_{hj} = \text{the husband’s latent CES-D8 score in couple } j, \]

\[ r_{nj} = \text{the measurement error associated with the husband’s latent CES-D8 score in couple } j, \]

\[ W_{ij} = \text{an indicator variable for individual } i \text{ in couple } j; W \text{ has a value of 1 if the response was obtained from the wife, 0 if the response was obtained from the husband}, \]

\[ B_{wj} = \text{the wife’s latent CES-D8 score in couple } j, \]
\[ r_{wj} = \text{the measurement error associated with the wife’s latent CES-D8 score in couple } j. \]

As noted in the description of the multivariate analyses in Chapter 4 (and detailed in Appendix D), \( r_{hj} \) and \( r_{wj} \) (the measurement errors for husbands and wives) were estimated from the internal consistency reliability estimates and observed variances of the depressive symptomatology scale in husbands and in wives. These values were then fixed within the HLM6 program.

**Level-2 Equation (baseline model only)**

Each of the parameters from the Level-1 model (e.g., \( B_h, B_w \)) then became an outcome or dependent variable in the Level-2 analysis. The baseline Level-2 model (i.e., with no predictors) was written as:

\[
B_{hj} = \gamma_{10} + u_{hj},
\]

\[
B_{wj} = \gamma_{20} + u_{wj},
\]

where:

\( \gamma_{10} \) and \( \gamma_{20} \) (Gammas) are the Level-2 intercepts—interpreted as the mean or average latent CES-D8 score—for husbands and wives, respectively, and

\( u_{hj} \) and \( u_{wj} \) are the residuals—interpreted as the individual deviations from the mean or average latent score—for husbands and wives, respectively.

Level-2 was thus comprised of two simultaneous regression equations—one for husbands, and one for wives. In multilevel modeling terms, this Level-2 model has two fixed effects (i.e., \( \gamma_{10} \) and \( \gamma_{20} \), which are assumed to be uniform across husbands and across wives), and two random effects (i.e., \( u_{hj} \) and \( u_{wj} \), or residuals), which are allowed
to vary across husbands and across wives.

The Level-2 residuals were assumed to have variances $\tau_{hh}$ and $\tau_{ww}$ and a covariance $\tau_{hw}$. The variance components $\tau_{hh}$ and $\tau_{ww}$ provided estimates of the variability around the mean latent score for husbands and wives, respectively. HLM6 generated a chi-square based hypothesis test for each variance component. If the null hypothesis (that the variance component was zero) was rejected, then this indicated that the scores of individual husbands and wives differed significantly from their group’s respective mean scores. Models were then built that attempted to predict this variability—so called “conditional” models (see Appendix F). In some circumstances, this Level-2 model also generated an estimate of the amount of shared variance (a.k.a., dependency or covariation) in husbands’ and wives’ latent depressive symptomatology scores ($\tau_{hw}$).

Because of the limited amount of data available in dyadic models, additional predictors added in later conditional models were assumed to have an average (i.e., fixed) effect on all husbands (or wives). Most research questions and hypotheses were evaluated in terms of the statistical significance, direction, and strength of these estimated Level-2 fixed effects coefficients (i.e., $\gamma$ coefficients for specific predictors). The tau (i.e., variance) components estimated by the HLM6 program were used to calculate the amount of unexplained variance remaining for each group (i.e., husbands or wives), after each model. These values were also used to calculate PREs (Proportion Reduction in Error) estimates—a pseudo-$R^2$ type of measure (see Chapter 4 for details).
Research Question 7 asked: Is there a cross-spouse relationship between each spouse’s pain intensity and his/her partner’s depressive symptomatology? The corresponding hypothesis (H7) predicted that each spouse’s pain intensity would be related to greater depressive symptomatology in his/her partner.

**Level-1 Equation**

Recall from Appendix E, the Level-1 equation was:

$$Y_{ij} = H_{ij} (B_{hj} + r_{hj}) + W_{ij} (B_{wj} + r_{wj}),$$

where:

$B_{hj}$ and $B_{wj}$ represented the latent CES-D8 scores for husbands and wives, respectively.

**Level-2 Equation**

As shown in Appendix E, the latent CES-D8 scores for husbands and wives were then modeled as outcome variables at Level 2. Once a baseline Level-2 model was established, predictor variables were then incorporated into the Level-2 model (creating conditional models) in an attempt to explain the variance in husbands’ and wives’ depressive symptomatology.

In order to address Hypothesis 7, a series of nested, conditional HLM6 models was developed. These models built on the final *intra*-individual model, Model 4, which included relevant individual- and couple-level background characteristics and the respondent’s own health-related characteristics, pain intensity, and physical limitations. Hypothesis 7 was tested through the introduction of two subsequent models: Model 5
added the **spouse’s** health-related characteristics (i.e., number of disease conditions and body weight); and Model 6 added the **spouse’s** pain intensity. The illustration below begins with Model 4 and shows only the Level-2 nested, conditional models:

**Model 4** included the following covariates: the respondent’s background characteristics (e.g., age, race/ethnicity); the couple-level background characteristics (e.g., household income, duration of marriage); the respondent’s health-related characteristics (e.g., disease conditions, body weight), and the respondent’s pain intensity:

\[
B_{\text{Hij}} = \gamma_{10} + \gamma_{11} (H_{\text{Background}}) + \gamma_{12} (C_{\text{Background}}) + \gamma_{13} (H_{\text{Healthchars}}) + \gamma_{14} (H_{\text{PainInt}}) + \gamma_{15} (H_{\text{PhysLims}}) + u_{\text{Hij}},
\]

\[
B_{\text{Wij}} = \gamma_{20} + \gamma_{21} (W_{\text{Background}}) + \gamma_{22} (C_{\text{Background}}) + \gamma_{23} (W_{\text{Healthchars}}) + \gamma_{24} (W_{\text{PainInt}}) + \gamma_{25} (W_{\text{PhysLims}}) + u_{\text{Wij}},
\]

where:

- **H Background** was a vector containing the background characteristics of the husband,
- **W Background** was a vector containing the background characteristics of the wife,
- **C Background** was a vector containing the background characteristics of the couple,
- **H Healthchars** was a vector containing the health-related characteristics of the husband,
- **W Healthchars** was a vector containing the health-related characteristics of the wife,
HPainInt was the pain intensity of the husband,

WPainInt was the pain intensity of the wife,

HPPhysLims was the physical limitations of the husband,

WPPhysLims was the physical limitations of the wife,

and

\( \gamma_{10} = \) intercept for husbands—interpreted as the average CES-D8 across husbands,

\( \gamma_{11} = \) a vector of the effects of the husband’s background characteristics,

\( \gamma_{12} = \) a vector of the effects of the couple-level background characteristics,

\( \gamma_{13} = \) a vector of the effects of the health-related characteristics of the husband,

\( \gamma_{14} = \) the effect of the husband’s pain intensity,

\( \gamma_{14} = \) the effect of the husband’s physical limitations,

and

\( \gamma_{20} = \) intercept for wives—interpreted as the average CES-D8 across wives,

\( \gamma_{21} = \) a vector of the effects of the wife’s background characteristics,

\( \gamma_{22} = \) a vector of the effects of the couple-level background characteristics,

\( \gamma_{23} = \) a vector of the effects of the health-related characteristics of the wife,

\( \gamma_{24} = \) the effect of the wife’s pain intensity

\( \gamma_{25} = \) the effect of the wife’s physical limitations

and

\( u_{ij} \) and \( u_{wj} \) are the residuals for husbands and wives, respectively.
**Model 5 added** the spouse’s health-related characteristics (e.g., number of disease conditions, body weight):

\[
B_{ij} = \gamma_{10} + \gamma_{11} (H_{\text{Background}}) + \gamma_{12} (C_{\text{Background}}) + \gamma_{13} (H_{\text{Healthchars}}) + \\
\gamma_{14} (H_{\text{PainInt}}) + \gamma_{15} (H_{\text{PhysLims}}) + \gamma_{16} (W_{\text{Healthchars}}) + u_{bij},
\]

\[
B_{wj} = \gamma_{20} + \gamma_{21} (W_{\text{Background}}) + \gamma_{22} (C_{\text{Background}}) + \gamma_{23} (W_{\text{Healthchars}}) + \\
\gamma_{24} (W_{\text{PainInt}}) + \gamma_{25} (W_{\text{PhysLims}}) + \gamma_{16} (H_{\text{Healthchars}}) + u_{wj},
\]

where terms **added** include:

- **W_{Healthchars}**, the vector containing the health-related characteristics of the wife (added to the husband’s equation), and
- **H_{Healthchars}**, the vector containing the health-related characteristics of the husband (added to the wife’s equation),

and

\[
\gamma_{16} = \text{a vector of the effects of the health-related characteristics of the wife (on the husband’s CES-D8), and}
\]

\[
\gamma_{26} = \text{a vector of the effects of the health-related characteristics of the husband (on the wife’s CES-D8).}
\]

[continued on next page]
**Model 6 added** the spouse's pain intensity:

\[
B_{ij} = \gamma_{10} + \gamma_{11} (H_{\text{Background}}) + \gamma_{12} (C_{\text{Background}}) + \gamma_{13} (H_{\text{Healthchars}}) + \\
\gamma_{14} (H_{\text{PainInt}}) + \gamma_{15} (H_{\text{PhysLims}}) + \gamma_{16} (W_{\text{Healthchars}}) + \\
\gamma_{17} (W_{\text{PainInt}}) + u_{ij},
\]

\[
B_{wj} = \gamma_{20} + \gamma_{21} (W_{\text{Background}}) + \gamma_{22} (C_{\text{Background}}) + \gamma_{23} (W_{\text{Healthchars}}) + \\
\gamma_{24} (W_{\text{PainInt}}) + \gamma_{25} (W_{\text{PhysLims}}) + \gamma_{16} (H_{\text{Healthchars}}) + \\
\gamma_{27} (H_{\text{PainInt}}) + u_{wj},
\]

where terms **added** include:

- \( W_{\text{PainInt}} \), the wife’s pain intensity (added to the husband’s equation), and
- \( H_{\text{PainInt}} \), the husband’s pain intensity (added to the wife’s equation)

and

\[
\gamma_{17} = \text{the effect of the wife’s pain intensity (on the husband’s CES-D8)}, \text{ and} \\
\gamma_{27} = \text{the effect of the husband’s pain intensity (on the wife’s CES-D8)}.
\]

Taken together, the equations in Model 6 attempted to explain husbands’ and wives’ depressive symptomatology as a function of *intra*-individual predictors (e.g., the respondent’s own age and other background characteristics, number of disease conditions, pain intensity, and physical limitations), couple-level predictors (e.g., household income and wealth), and *inter*-individual or cross-spouse predictors (e.g., his/her spouse’s disease conditions and, most importantly, pain intensity). The overall model was constructed in a way that isolated the both the *intra*-individual and the cross-spouse effects of pain intensity.

Hypothesis 7 specifically concerned the cross-spouse effect of each spouse’s pain
intensity on the depressive symptomatology of his/her partner. The hypothesis was
evaluated in terms of the statistical significance, direction, and strength of the estimated
fixed effect (i.e., \(\gamma\) [Gammas]) for each spouse’s pain intensity. Specifically, \(\gamma_{17}\)
represented the effect of the wife’s pain intensity on the husband’s depressive
symptomatology, and \(\gamma_{27}\) represented the effect of the husband’s pain intensity on the
wife’s depressive symptomatology. Stated in terms of the conceptual model presented in
Figure 5 (Chapter 3): \(\gamma_{17}\) represented Path 4W, and \(\gamma_{27}\) represented Path 4H.
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R-27


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R-67


R-87


