THE LONGITUDINAL ASSOCIATION BETWEEN DEPRESSIVE SYMPTOMS
AND ALCOHO USE IN MIDDLE-AGED AND OLDER ADULTS:
COMPARISON BY RETIREMENT STATUS

by

OK MI BAIK

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Dissertation Advisor: Dr. Kathleen J. Farkas

Mandel School of Applied Social Sciences
CASE WESTERN RESERVE UNIVERSITY

January, 2011
We hereby approve the thesis/dissertation of

Ok Mi Baik

candidate for the _______Doctor of Philosophy _______degree *.

(signed) _____ Kathleen Farkas, Ph.D

(chair of the committee)

_________ Kathryn Adams, Ph. D

_________ Meeyoung Oh Min, Ph. D

_________ Diana Morris, Ph. D

(date) _____ 11- 09- 2010_____

*We also certify that written approval has been obtained for any proprietary material contained therein.
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The purpose of this dissertation was two fold: 1) to examine the relationships between depressive symptoms and alcohol use among middle-aged and early older adults and 2) to examine whether retirement status moderates the differences in the relationships. For examining these aims, this study utilized a multi-group cross-lagged structural equation modeling using a pane data of the Wisconsin longitudinal study (N=3,204).

No support for a mutually reinforcing relation between depressive symptoms and alcohol consumption was found in the current study. Higher levels of depressive symptoms did not lead to higher alcohol consumption among early older adults, and alcohol consumption was not associated with later depressive symptoms by analyzing the cross-lagged SEM. Therefore, the nature of the causal processes contributing to the obtained pattern of relationships between depressive symptoms and alcohol consumption cannot be determined by this study. These results may reflect that the mechanisms responsible for co-morbidity of depressive symptoms and alcohol-related problems could vary across individuals and represent etiologically distinct subgroups. There was no
significant moderating effect of retirement status in the relationship between depressive symptoms and alcohol use by analyzing the multi-group SEM.

Several caveats should be taken into consideration when interpreting the findings of this study. Although this study is a population-based study, the sample was limited to a certain population who graduated from Wisconsin high schools in 1957, and thus, the results have a limitation in terms of generalization. Despite several limitations, the study makes contributions in several areas. Given the fact that a longitudinal design is the optimal research endeavor when examining the relationships between depressive symptoms and alcohol use, this study will provide invaluable information for understanding the relationships. This study has also provided a rigorous test of the cross-lagged relationships using SEM between depressive symptoms and alcohol use among middle-aged and early older adult samples. Overall, the results of this study will add to the existing literature by suggesting that individuals with depressive symptoms and alcohol use represent a complex group with significant psychosocial consequences and treatment needs.
CHAPTER 1. Introduction

1-1. Background and Significance

The purpose of this dissertation is to examine the relationship between depressive symptoms and alcohol use during the transition of retirement in a sample of community-dwelling adults.

The investigation of the relationship between depressive symptoms and alcohol use in mid- and later life is important for several reasons. First, demographic change has great importance for America’s health care system because older persons make considerably greater use of health care services than do younger populations and further, older adults have health care needs that are often more complex. From now to 2030, the number of adults aged 65 and older in the United States will almost double, from around 37 million to more than 70 million, an increase from 12% of the U.S. population to almost 20% (U.S. Census Bureau, 2008). While this is partly due to increasing longevity and partly to a declining birth rate, the increase is mainly due to the maturing of the “baby boom generations” born in the U.S. between 1946 and 1964 (Siegel, 1996). During the Baby Boomer years, approximately 76 million American were born and today, this represents 28 percent of the American population. In addition to the existing 13% of older adult population, upon the entrance of the large baby boom generation into mid- and later life now, there has been an increasing demand for information about this transitional life stage, promoting research on this topic (Wethington, Cooper, & Holmes, 1997).

Furthermore, from several theoretical perspectives, expanded from the life course perspective, the transition from the midlife to the later life period has significant
implications for health and mental health research. While the prevalence of chronic medical conditions and functional limitations is much higher for the elderly, their prevalence rises during midlife, and increases steadily for the remainder of a person’s life (Merrill & Verbrugge, 1999). Ryff and Heidrich (1997) reported that normative events or transitions were significant predictors of multiple aspects of present and future well-being for different age groups such as young, middle-age, and old age. Transition from midlife to later life is often associated with increased stress, defined as any event that causes pressure to change (Kramer, 1993; McCubbin, 1995). The most obvious of these changes is the transformation of retirement (Hyde & Higgs, 2004). Retirement is a significant event for most people, health changes observed after retirement are likely to be attributed to retirement. A number of studies reported that retirees showed more symptoms of depression than the working peers (Alpas et al, 2000; Bosse et al, 1991). Therefore, examining changing psychological and physiological factors and their associations during this life stage has great importance.

Third, both heavy alcohol use and depressive symptoms are widespread among the general older adult population (Grant, 1995; Kessler et al., 1996) and are associated with substantial societal and personal costs (Roy-Byrne et al., 2000). An increasingly strong association between aging and depression is emerging, with several clinical studies showing that the prevalence of depression increases in old age. Depression is three times as frequent in the elderly as it is in the rest of the population, and affects about 30% of people aged over 65 (Roberts et al., 1997; Blazer, 2002).

While the literature generally indicates that older adults drink less and report fewer drinking problems as they age, recent studies reported total alcohol consumption and
drinking frequency may actually remain stable or increase among older adults (Benzies et al., 2008; Moos et al., 2004). Recent national epidemiologic surveys of psychopathology in the United States have demonstrated that while approximately half of the general population experiences a major psychiatric illness at some point over their lifetime, the majority of affected individuals simultaneously meet diagnostic criteria for depression and alcohol use disorder (Kessler et al., 1994).

A large recent survey reported that abuse of alcohol and prescription drugs among adults 60 and older as one of the fastest growing health problems facing the nation and those problems remain underestimated, under-identified, under-diagnosed, and under-treated (SAMHSA, 1998, Oslin, 2004). A number of factors contribute to this under-identified and under-diagnosed situation for this age group. First, many symptoms of alcohol problems are similar to those of other disorders common among the aged group, such as dementia. Second, alcohol use problems are sometimes overlooked because of biases held by health practitioners. That is, clinicians are less likely to screen for alcoholism among older individuals, women, and the educated. The third reason is the tendency for both patients and clinicians to avoid the topic of alcohol because they feel it is uncomfortable to discuss. In addition, some elderly patients and their families tend to think that drinking is a kind of compensation for older retirees (Sorocco & Ferrell, 2006).

Given the rapidly entering into older adulthood of baby boom cohort, the prevalence of alcohol use disorder may be even greater in this population in near the future. According to a report, rates of heavy alcohol use have been shown to be higher among baby boomers than in earlier cohorts (DHHS, 2000). Also, 49% of the baby-boom cohort had ever used illicit drugs in their lifetime, compared to only 11% of adults aged 50 and
older (SAMHSA, 1996). In addition to being more likely to be illicit drug and heavy alcohol users than previous cohorts, the baby-boom cohort is larger than earlier cohorts (Koenig et al., 1994). Taken together, these data suggest that the prevalence of problematic substance use among older adults may increase as the baby-boom cohort ages.

Fourth, clinically, depressive symptoms complicated by alcohol use is more complicated to treat and thus, is associated with high rates of chronicity, suicide, disability and treatment resistance (Helzer & Pryzbeck, 1988). Individuals with comorbid conditions follow a more chronic and treatment-resistant course than those with only one disorder, and they are more likely to experience severe psychosocial impairment (Hagnell & Grasbeck, 1990; Kessler, 1995; Murphy, 1990). Thus, the combined presence of comorbid conditions negatively affects the clinical course and optimal treatment for both disorders and financial burdens society. Further, depressive symptoms and alcohol use are strongly linked across the lifespan, and this comorbid association persists into later life (Grant & Harford, 1995; Kessler et al., 1994, 1997).

This dissertation examines the relationship between alcohol use and depressive symptoms during the retirement transition in middle and later life through secondary data analysis. The following section provides an overview about the relationship between alcohol use and depressive symptoms.

1-2. Alcohol Use in Middle and Older Adults

*Definition of alcohol use*
There have been varied alcohol-related terms based on different dimension used in the previous literature: ‘alcohol use’ focused on the level of alcohol consumption, ‘alcohol use disorder’ based on the diagnostic criteria of alcohol-related disorder and ‘alcohol problems (or drinking problems)’ focused on the consequence by the level of alcohol consumption. Oslin (2004) suggested that older adult can be placed in one of the following three general categories of drinking behavior: (a) abstinence or low-risk drinking, (b) problem drinking, and (c) heavy drinking. Abstinence or low-risk drinkers are those who choose not to drink or follow standard drinking guidelines while heavy drinkers are those who drink more than the standard drinking guideline. Several guidelines exist, but as frequently referred standard drinking guidelines, National Institute on Alcohol Abuse and Alcoholism (NIAAA) and American Geriatrics Society (AGS) clinical guidelines currently define risky drinking amounts for people aged 65 and older as more than seven drinks per week or more than three drinks on any single day while the NIAAA defines risky drinking a maximum of four or three (four for men, three for women) drinks per week for young and middle adults (NIAAA, 2005; Moore, 2006).

While clinic-based studies frequently used the diagnostic criteria of alcohol use disorder, the community-based studies tended to use the level of alcohol use.

Definitions of alcohol use disorder vary markedly between studies. The most widely used definitions for alcohol use disorders are found in two major classification systems of disease: the Diagnostic and Statistical Manuals of Mental Disorders (DSM) of the American Psychiatric Association (APA), and the International Classification of Diseases (ICD) of the World Health Organization (WHO). The DSM-IV describes problem-related alcohol use as ‘alcohol abuse’ or ‘alcohol dependence’. Two diagnoses
are specified: alcohol abuse requires a pattern of pathological (excessive or uncontrolled) alcohol use plus impairment in social or occupational functioning due to alcohol use. Alcohol dependence diagnosis requires either a pattern of pathological use or impairment in social or occupational functioning plus evidence of tolerance or withdrawal (physical dependence).

In addition to these diagnostic categories, many researchers also use a variety of terms and definitions interchangeably including alcohol use, alcohol misuse, alcohol abuse, alcohol problems, problem-drinking or alcohol-related problems. ‘Alcohol use’, ‘alcohol misuse’ or ‘alcohol abuse’ in community studies broadly includes guideline-defined excessive alcohol consumption with or without alcohol dependence (Watts, 2008). ‘Alcohol problems’ or ‘alcohol-related problems’ denote a more inclusive category that also counts specific or focal social, behavioral or health problems caused by excessive or inappropriate drinking that may not meet criteria for a formal diagnosis (Atkinson, 1999). This study uses a term of alcohol use that will be described fully in chapter 3.

*Prevalence of Alcohol use*

The reported prevalence of alcohol use-related disorder varies depending on the restrictiveness of diagnostic criteria used, with higher rates for “excessive alcohol consumption” and “alcohol abuse” than “alcohol dependence.” For example, community based studies have estimated the prevalence of alcohol misuse or dependence as 2-4% (Adams & Cox, 1995) with much higher rates of 17% of men and 7% of women when looser criteria such as excessive alcohol consumption are used (Blow et al., 1998).
Also, prevalence of alcohol use-related disorder is often related to the type of setting. In community-based samples, the prevalence of alcohol use disorder in older adults ranges between 1 and 22% in population based studies (Dufour & Fuller, 1995). Community-based samples are those that include people living independently in the community. Some community-based samples are randomly selected and others are convenience samples based on housing type or geography. Up to 15% of older adults living in the community have a drinking problem and the number of older adults in need of alcohol misuse treatment and rehabilitation will increase from 1.7 million in 2000 to 4.4 million by the year 2020 (Gfroerer et al., 2003). The National Institute of Alcohol Abuse and Alcoholism (1992) reported prevalence for alcohol abuse and dependence of 7% in 1992 in older adults. Another community sample showed that among ages 60 to 84 years, 52.8% of men and 37.2% of women were current drinkers (Breslow, Faden, & Smothers, 2003) while one other survey reported that 6.9% of adults aged 65 or older reported binge drinking and 1.8% reported heavy drinking (SAMHSA, 2004).

In general, the prevalence for aged inpatients is higher than for elderly people in the community, with estimates of 14% for patients in emergency departments, 18% for medical inpatients, and 23-44% for psychiatric inpatients (Goldstein et al., 1996). A prevalence rate of 8.6% for alcohol dependence according to the DSM-IV criteria was observed among a geriatric mental health outpatient clinic (Holroyd & Duryee, 1997) and 23% among the substance abused patients in VA hospitals (Moos, Mertens, & Brennan, 1993). Callahan and Tierney (1995) reported that based on the CAGE questionnaire, 10.5% of adults 60 years and over in a primary practice screened positive for alcohol-related problems. Adams et al. (1996) also reported 9% men and 3% women aged 60
years and over screen positive for alcohol-related problems based on the CAGE. Among the inpatient clinics with 55 years and over, 9.9% alcohol use disorder was found (Johnson, 2000) and in an emergency department sample, 11% of people aged 60 and older screened positive for alcohol-related problems based on CAGE (Friedmann et al., 1998). A nursing home-based survey reported higher prevalence of alcohol-related problems: 8% of the residents met on the DSM-III-R criteria for current alcohol abuse and 53% met criteria for lifetime alcohol abuse (Herrmann & Eryavec, 1996). Another study reported 18% of the nursing home patients older than age 50 met DSM-III-R criteria for alcohol abuse or dependence (Joseph et al., 1995).

Effects of Alcohol use in mid- and older adults

Primary risk factors in the elderly alcoholic include major life changes and losses. Major life changes may precipitate drinking or an increase in drinking. Alcohol may be used to relieve the boredom or depression stemming from unfulfilled expectations. Substantial losses, such as a decline in economic status, the death of a spouse or close friends, and deterioration of health with worsening medical problems, are all risk factors for drinking in the elderly. Alcohol may be used to reduce the psychological, emotional, or physical stress. Other risk factors include substance abuse earlier in life, comorbid psychiatric disorders, family history of alcoholism, and concomitant substance abuse of nicotine and psychoactive prescription medicines.

Compared to younger populations, the presentation of alcohol-related disorder in elderly people tend to be atypical such as falls, confusion, depression or masked by comorbid physical or psychiatric illness, which makes detection all the more difficult (O’Connell et al., 2003).
Alcohol use disorders in elderly people are associated with widespread impairments in physical, psychological, social, and cognitive health. Also, alcoholism is known to be associated with poorer health outcomes, including worse outcomes following illness and increased mortality (Blazer, 2003) and especially, suicide in late middle and old age (Osgood & Manetta, 1998).

Alcohol may accelerate aging-associated changes in physiological functioning, and thus, it is associated with increased morbidity and mortality from alcohol-induced diseases, including cardiovascular and liver diseases (Oslin, 2004). Research reported that rates of physical illness among elderly alcoholics are higher than would be expected in a non-drinking population of similar age (Hurt et al., 1988).

Alcohol use also can result in falls leading to hip fracture, a leading cause of death in this group (Saitz, 2003). This may in part be due to increased biological sensitivity to alcohol: with increasing age there is a decrease in lean body mass and total body water which leads to higher peak blood alcohol concentrations for a given dose (Johnson, 2000). Furthermore, older adults are also uniquely vulnerable to the effects of alcohol because of their high risk for drug or alcohol interactions. Older adults generally take more prescription and over-the-counter medications than do younger populations. The aging body is more susceptible to adverse drug and alcohol interactions; slower metabolic and clearance mechanisms delay their resolution (Sorocco & Ferrell, 2006). Thus, alcohol can exacerbate medical disorders that are common in elderly people, including congestive heart failure and hypertension (Saitz, 2003). Alcohol use is also associated with negative health and mental health problems, especially for elderly population.
On the other hand, some epidemiologic studies suggest the benefits of little amounts of alcohol use. Low-volume alcohol use is associated with positive health outcomes such as decreased rates of cardiovascular disorders, including coronary artery disease and increased longevity (Dawson, 2000).

1-3. Depressive Symptoms in Middle and Older Adults

**Definition of depressive symptoms**

The definition of depressive symptoms has been widely used in the literature with the definitions of depression which mainly focuses on as diagnostic criteria. Two main classification systems are used for diagnostic criteria of mental disorders: the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychological Association, 2000) and the International Classification of Diseases (ICD-10; World Health Organization, 2009). The American Psychiatric Association (APA, 2004) uses the DSM-IV to describe the symptoms and diagnostic criteria for identifying a major depressive episode as follows,

“a period of at least two weeks during which there is either depressed mood or the loss of interest or pleasure in nearly all activities. The individual must also experience at least four additional symptoms drawn from a list that includes change in appetite or weight; sleep; psychomotor activity; decreased energy; feelings of worthlessness or guilt; difficulty thinking, concentrating or making decisions; or recurrent thoughts of death or suicidal ideation, plans, or attempts. The symptoms must persist for most of the day, nearly every day, for at least two consecutive weeks. The
episode must be accompanied by clinically significant distress or impairment in social, occupational, or other important areas of functioning (APA, 2004, pp 356).

Using the DSM-IV and the ICD-10, there are two main instruments that have been developed for use in surveys: the Composite International Diagnostic Interview (CIDI) and the Diagnostic Interview Schedule (DIS). While the CIDI and DIS is to measure diagnostic measure of depression, the Center for Epidemiological Studies Depression Scale (CES-D) is to measure the symptoms of depression. The CES-D is based on a self-report from the respondent to measure the symptoms of depression. This study uses the definition of depressive symptoms based on the CES-D, and it will be fully described in the chapter 3.

**Prevalence of depressive symptoms in middle and older adults**

The prevalence rates vary depending on the type of setting, patient population, conceptual and operational definition, and type of depression. The exact prevalence of depression including depressive symptoms in middle-aged adults is unknown because most national surveys usually include adults aged 18 or more. Major depression affects approximately 9.9 million Americans (Narrow, 1998) or roughly 5% of the population, and it is the leading cause of disability in the United States (Murray & Lopez, 1996). In the general adult population aged 18 or more, lifetime prevalence of depression is 20.8% (Kessler et al., 2005).

Also, the prevalence of depression in older adults varies according to settings examined. Djernes (2006) reported the prevalence of depression in elderly by a comprehensive review of the published papers after 1993. The prevalence of major
depression ranges from 0.9% to 9.4% in private households, from 14% to 42% in institutional living, and from 1% to 16% among elderly living in private households or in institutions; and clinically relevant depressive symptoms in similar settings vary between 7.2% and 49%. It is important to note that several studies identified that the prevalence of depression in the older adult group is underidentified, under-diagnosed, or misdiagnosed and thus, under-treated (Department of Health and Human Services [DHHS] 1999). It is estimated that clinically significant depression goes untreated in 60% of cases (Steffens et al., 2000).

*Effects of depressive symptoms in middle and older adults*

Depressive symptom is one of the most prevalent mental problems and it is associated with poor health outcomes and suicide (Bartels et al., 2002; Conwell et al., 2000). Biological, psychological and social factors influence the development of depressive symptoms in later life (Blazer & Hybels 2005). Life stressors of older adulthood are linked to depressive symptoms and include medical co-morbidity, disability and decreasing social network (Blazer & Hybels, 2005).

For the older adult population, milder forms of depression are much more common, specifically minor depression, subsyndromal or subthreshold depression, dysthymic disorder or depressive symptoms. Even though the symptoms of those mild depressions are not as severe as major depression, aged populations are often associated with significant functional disability (Beekman et al., 1997; Koenig & Blazer, 1996). Also, mild and major forms of depression are associated with physical, mental, social, and functional impairment. It is also associated with all-cause mortality, increases in healthcare costs and utilization, physical health conditions, long-term care placement, as
well as increased risk for suicide (Friedman et al., 2005; Cully et al., 2008). Many older adults do not receive adequate mental health treatment because of the severity of disability associated with depression. Disability in depression involves impairment in mobility, activities of daily living (ADLs) and instrumental activities of daily living (IADLs).

Treatment for geriatric depressive symptoms can be effective and available, yet older patients tend not to take these mental health services due to some biases about mental health services (Van Citters & Bartels, 2004). Geriatric depressive symptoms are usually associated with diseases linked to physical and mental aging, or by difficult social or financial situations, through both direct and reactive mechanisms. Although the symptoms that characterize a state of depression in the elderly overlap those observed in younger people, it is not always easy to identify depression in later life (Beekman et al., 2000; Cole & Dendukuri, 2003).

Multiple studies have demonstrated that older adults with depressive symptoms are more likely to be and become disabled (Bruce, 2001; Alexopoulos et al., 1996; Lenze et al., 2001). Similarly, studies in middle-aged populations show strong associations between depressive symptoms and health-related quality of life and work-related disability (Spitzer et al., 1995).

1-4. Depressive Symptoms and Alcohol Use in Middle and Older Adults

Prevalence of depressive symptoms with alcohol use in middle and older adults
Relatively little attention has been directed to characterizing co-occurring disorders and models of treatment in the rapidly growing population of older adults with co-occurring disorder.

Estimates of depressive symptom prevalence accompanied by alcohol use vary because of variations in where the samples were located, in the use of inclusive versus narrow diagnostic criteria, and in assessment protocols.

In the community-based studies, nearly half of community-dwelling older adults with a history of alcohol abuse have co-occurring depressive symptoms (Blazer, 2003). Among a frail population of older adults receiving in-home services, 9.6% had an alcohol abuse problem and two-thirds of those individuals (6% of the overall sample) had a comorbid psychiatric illness such as depression or dementia (Jinks & Raschko, 1990).

The prevalence of comorbid depressive symptoms with alcohol use among older adults also varies by population, and ranges from 7% to 38% of those with a psychiatric illness and from 21% to 66% of those with substance abuse (Bartels et al., 2006). In particular, high rates of co-occurring depression and alcohol use disorder among older adults are found in psychiatric clinical populations. An investigation by Olfson et al. (2000) in an urban primary care sample found that of the 7.9% of patients diagnosed with a substance use disorder, most frequently alcohol use disorder, 36% met the criteria for co-occurring major depressive disorder. Similarly, the WHO Collaborative Study (1994) found that 43% of individuals with alcohol dependence met the criteria for at least one other psychiatric disorder.

Blow and colleagues examined the presence of psychiatric diagnoses in approximately 4,000 alcoholic Veterans Affairs patients between age 60 and 69 (Blow et
In their study, affective disorders were the most common psychiatric diagnosis found in 21% of these individuals and approximately 43% of these individuals had major depression. In clinical samples, the lifetime rates of co-occurrence are greater still, ranging from 50% to 70% (Cornelius et al., 1995; Curran & Booth, 1999). In a study of the prevalence of dual disorders in older psychiatric inpatients aged 60 or more, 37.6% had co-occurring depression and substance use disorders, consisting of 71% with alcohol abuse and 29% abusing both alcohol and other substances (Blixen, 1997). Oslin et al. (2000) reported that in a series of 2,666 patients admitted to geriatric psychiatry treatment programs in a health care system, 11.1% of depressed patients were drinking preadmission and 3.5% of these depressed patients were drinking daily preadmission. In conclusion, although the prevalence rate of co-occurring depressive symptoms and alcohol use disorder is diverse, considerable research documents the higher prevalence of co-occurring disorder in elderly population than common expectation.

Impact of depressive symptoms with alcohol use in middle and older adults

Co-occurring depressive symptoms with alcohol use among older adults are associated with poor health outcomes, higher health care utilization, more complications, and higher rates of active suicidal ideation and social dysfunction relative to individuals with either disorder alone (Bartels et al., 2002). In a ten-year study of psychiatric hospitalizations of older veterans aged 55 or older, 75% of patients with a co-occurring psychiatric disorder were admitted to a psychiatric inpatient unit, compared to 62% among those with an alcohol or drug abuse/dependence alone (Brennan et al., 2002). In addition, co-occurring depressive symptoms and alcohol use disorder has also been
associated with more frequent inpatient re-admission and higher costs (Moos & Moos, 2006).

The depressive symptoms with alcohol use among older adults are associated with higher rates of suicidal ideation and suicide attempts in older adults. Suicidal ideation was present in 3% of persons with at-risk alcohol use alone and was present in 12% of older adults with major depression alone while 15% of older adult primary care patients with both major depression and at-risk alcohol use had suicidal ideation (Bartels et al., 2002).

1-5. Retirement transition, depressive symptoms and alcohol use

Retirement transition is a period of change, when the roles and relationships associated with individuals’ previous labor market positions are transformed. It is not simply an objective life course transition, but is also a subjective developmental and social-psychological well-being (Moen, 2001). Although retirement still largely viewed as a highly desirable life transition now, there seems to be a popular perception that retirement closely precedes ill health and death. On the other hand, retirement may promote well-being because retirees move out from demanding and stressful jobs.

Definitions of retirement

Retirement is a loosely defined construct in the previous and current literature. While the official retirement age in most western countries is still 65 years, the actual pattern of retirement often does not conform to this definition (Leibfritz, 2003). For many individuals, retirement is more accurately viewed as the time when one’s main job ceases and receipt of pension commences, irrespective of age (O’Rand, 1990). Given these
definitional complexities, retirement is often operationalised as absence from the labor force beyond a certain age (typically 45 years).

**Effects of retirement**

The transition to retirement is accompanied by shifts in income (Palmore, Fillenbaum, & George, 1984), and social and physical activity (Wells & Kendig, 1999). With the loss of paid work, existing roles and identities come under increased scrutiny and pressure, and are likely to undergo substantial change in response to new resources, such as time, money, personal space, health status and social networks.

In particular, stress associated with retirement could be a mechanism by which retirement influences health. Some studies suggested that retirement-related stress can lead to increased risk of illness and disease (Kiecolt-Glaser, Marucha, Malarky, Mercado, & Glaser, 1995; Alpass, Neville, & Flett, 2000). However, another study reported the improvements in health after retirement (Midanik et al, 1995).

Transition to retirement also changes psychological well-being (Mein, Martikainen, Hemingway, Stansfeld, & Marmot, 2003). Retirement itself may lead to diminished well-being, as individuals lose their occupational attachments, their social network of co-workers, and a major identities. Others found no effects on psychological well-being associated with retirement (Gall, Evans, & Howard, 1997). Thus, the relationship between retirement and health outcomes bears further exploration.

1-6. **Knowledge Gaps**

*Complexity of the association*
Researchers have attempted to recognize the cause of co-occurrence of depression and alcohol use disorder in a variety of ways, but findings have been inconclusive. Basically, in addition to psychopathology, this situation has been linked to complex interactions of genetic, biochemical, cognitive-behavioral, and environmental influences, mental disorders can often be heterogeneous in etiology while appearing homogeneous in clinical expression (Swendsen & Merikangas, 2000).

One of the explanations of the co-occurrence is that pharmacologic effects of alcohol produce symptoms of depression directly during periods of intoxication and/or withdrawal (Brown et al., 1995). Other studies suggest that individuals with depressive symptoms are motivated to drink alcohol in an effort to cope with negative affect, a potential mechanism for development of depression (Cooper, Frone, Russell, & Mudar, 1995; Schuckit, Smith, & Chacko, 2006). Others also suggest that substance use disorder and mental disorders share a common etiology. That is, the comorbidity between mental disorder and substance use disorder is due to a third, unspecified common factor—a common biologic, environment or social factor – which contributes to the development of both comorbid conditions.

Further, the association between alcohol use disorder and depressive symptoms is a complex for several reasons. While one study reported depressed patients engaged in more drinking (Holahan et al., 2004), longitudinal studies based on community samples failed to find the association between alcohol use and depressive symptoms in mixed age group (Moscato et al., 1997; Lipton, 1994). Measurement issues regarding alcohol use, alcohol problems and depressive symptoms also complicate studies of the relationship between alcohol consumption and depression (Graham et al., 2007).
Second, it is unclear whether the relationship of alcohol use disorder and depressive symptoms is causal, meaning that one causes the other (Dixit & Crum, 2000), and even the direction of a possible causal relationship is also not clear (Steunenberg et al., 2008). Does alcohol use cause depressive symptoms, or do depressive symptoms foster alcohol use, or are both caused by a third factor? A number of studies have found a reciprocal association: depressive symptoms predict alcohol problems and alcohol problems also predict depressive symptoms (Hettema et al., 2003; Compton et al., 2000; Paljarvi et al., 2009; Agosti & Levin, 2006), but some studies found that one problem does not predict the other over time (Wu & Anthony, 1999; Vaillant, 1993). The mechanisms of the relationship between depressive symptoms and alcohol use are not well understood.

*The need to include contextual factors fitted in mid- and later life*

While the relationships between depressive symptoms and alcohol use have been widely examined for the younger adults and elderly, the period of midlife and later life has received little scholarly attention. Thus, little information in these groups can be achieved. Also, when specific populations were examined, there are few studies about middle-aged populations, while a few studies were examined for elderly groups with different definitions of elderly with 50 or older, 60 or older, or 65 or older. As there are different issues associated with midlife than with later stages in the life course, it is not appropriate to assume that research on the elderly applies to middle-aged adults. Thus, separate analysis which detects the change of the relationships in the same cohort over time is needed.

Further, much of the prior research regarding this relationship addresses the development of psychopathology from a single perspective, oftentimes using other
disorders as predictors of a mental health problem. That is, the previous studies have not adequately considered a number of potential confounding factors or contextual factors affecting the relationship between depressive symptoms and alcohol use, such as employment status, health status, etc. Such approaches inadvertently bias results by assuming an inherent temporal progression of associated symptoms. Although comorbidity is mainly viewed as an etiological perspective, comorbidity may be better understood in the life context of conditions in which symptoms frequently co-occur.

To date, no studies have examined the association of alcohol use and depressive symptoms within the context of a major life course transition, retirement. Retirement is the one of the most important later life status transitions (Barnes & Parry, 2004). It is not simply an objective life course transition, but is also a subjective developmental and social-psychological transformation that may be related to physical and psychological well-being (Moen, 2001). Retirement transitions may have negative consequences for mental health, such as increased symptoms of depression (Turner, Killian, & Cain, 2004; Pahkala, 1990) and the highest rates of alcohol abuse are found in the transition of retirement (Catalano, Dooley, Wilson, & Hough, 1993). However, empirical evidence is inconsistent. Cross-sectional studies have shown retirees are more likely to report poor psychological well-being compared with their employed peers (Bosse et al., 1987). Also, middle-aged retirees were associated with a common mental disorder (Butterworth, 2006) while other studies reported that there is no association between retirement and measures of mental health or psychological distress (Ross & Drentea, 1998). The relationship between retirement and alcohol use is also inconclusive. While Hammer (1992) reported that unemployment led to a decreased level of alcohol use, Kessler et al. (1987) reported
unemployment led to an increased alcohol use. Some argue that retirement can relieve a person of the stressors of the working environment, and increase their sense of personal control (Drentea, 2002; Kim & Moen, 2002). Conversely, retirement may have an adverse effect through the absence of positive benefits derived from employment, including: financial stability, a sense of purpose, or social activity (Moen, 1996).

This dissertation addresses these gaps in the previous literature. This study will examine the relationship of alcohol use and depressive symptoms using a longitudinal design in a cohort of middle-aged and early older adults. Retirement will be used to examine the difference of the change in the relationship of alcohol use and depressive symptoms over about a 10 year period.

This dissertation examines the longitudinal relationship between depressive symptoms and alcohol use to explore the nature of relationship in retirement transition from middle-aged to older adults. This study uses two waves of the Wisconsin Longitudinal Study (WLS): the 1993 wave when most participants were 52-56 years old and the 2004 wave when the participants were 63-67 years old. Using a large birth cohort dataset from the United States, this study specifically considers causal relationship in the association between depressive symptoms and alcohol use and further, the relationship will be examined by their retirement status. The data for present study are investigated by multi-group Structural Equation modeling in a cross-lagged panel designs for exploring the direction of association over time during retirement transition from middle to older adulthood.

Exploring the relationship between depressive symptoms and alcohol use among the middle-aged and older population is of critical importance today because about one-
third of the U.S. population currently belongs to baby boom generation and the oldest members of this cohort are on the brink of transitioning from midlife to late life; thus, in addition to aging society with 13% of all population in U.S., the findings revealed among members of the WLS cohort (born around 1939) may offer an early glimpse into the potential challenges facing Baby Boomers as they parent adult children.
CHAPTER 2. Review of Literature

The key relationship examined in this dissertation is the one between depressive symptoms and alcohol use in middle and later life. This study will also compare the relationship between alcohol use and depressive symptoms by retirement status. This chapter includes three main parts: 1) a review of the literature on the relationship between depressive symptoms and alcohol use in middle and later life, 2) on retirement transition and its association with depressive symptoms and alcohol use, and 3) on the other risk factors for depressive symptoms and alcohol use in middle-aged and older adults.

2-1. A Review of the Literature on the Relationship between Depressive Symptoms and Alcohol Use in Middle and Later life

The causes of depressive symptoms and alcohol use comorbidity have been extensively studied in clinical and epidemiologic research over the past three decades (Swedsen & Merikangas, 2000). The most notably cited explanations for co-occurring alcohol use and depressive symptoms are that of causal relationship, the existence of risk factors for either the alcohol use or depressive symptoms, or that factors may mediate or moderate the association between co-occurring or comorbid alcohol use and depressive symptoms (Degenhardt, Hall & Lynskey, 2001). Various studies have suggested that the co-occurrence of alcohol use and depressive symptoms can develop via a variety of pathways.

The methodological approaches to explore the association

The evidence for a statistical association between alcohol use and depressive symptoms would imply a dynamic link of the prevailing conditions. That is, one condition might have caused the other, triggered an existing predisposition for the other,
increased the risk of developing the other condition, or changed the course of the other condition.

Previous studies have examined the association by several ways. First, a number of studies compared age of onset of two disorders. That is, a number of surveys have collected retrospective information about age of onset of depressive symptoms and alcohol use, and then, compared the age of onset of the two disorders (Kessler et al., 1994; Merikangas & Stevens, 1998). Kaplan-Meier Age of Onset curves were frequently used to examine visual age of onset distributions among people with comorbidity who reported an earlier age of onset of their depressive symptoms and alcohol use. Regarding this method, Kessler (2004) suggests that simple comparisons of temporal priorities are inadequate to document predictive associations. This documentation need to analyze reciprocal series of survival analyses in which each of the two sets of disorders is treated as a series of time-varying covariates that predict first onset of the disorders in the other set (Kessler, 2004). However, the information with retrospective reports could be involved with recall biases, thus, longitudinal studies with prospective data are needed to confirm the association.

The most common ways to numerically conceptualize the relationship is the use of the odds ratio (Swendsen & Merikangas, 2000). This method compares the prevalence of co-occurrence of two disorders with the statistically expected co-occurrence given their individual prevalence. Thus, if the odds ratio for the comorbidity of alcohol dependence and depression is 2.0, that means an individual diagnosed with alcohol dependence is twice as likely to be depressed as an individual without a diagnosis of depression.
Most importantly, the causal relationship could be found with the advance of statistical technique such as Structural equation modeling (SEM) in longitudinal analyses methods. SEM has the advantage of determining causal priority and causal predominance when reciprocal relationships are found (Klein, 1998), but to date a few studies have analyzed it due to a lack of available datasets.

**Etiological models of substance use disorder and psychiatric disorder**

Regarding the explanation of the relationship between depression and alcohol use disorder, several competing hypotheses have been proposed. Merikangas (1990) proposed that the comorbidity of substance use disorder and psychiatric disorder is likely to fall into two basic classes: causal explanations and shared etiology. The class of causal hypothesis includes the primary psychiatric disorder-secondary substance use disorder model, the primary substance use disorder-secondary psychiatric disorder model, and the bi-directional model whereas the other class includes shared etiology or the common factor model. The models are mutually exclusive, but each different model accounts for different segments of the population of those with comorbid disorders.

_Causal hypothesis_

Causal hypothesis poses that depression is often comorbid with substance abuse, especially alcohol abuse, because the abuse of alcohol either directly or indirectly causes major depression or, in the same manner, depression causes alcohol use disorder directly or indirectly. In this causal hypothesis, there are several directional causal relationship models.
First, the primary substance use disorder-secondary psychiatric disorder model posits that substance abuse directly, “pharmacologically induces major depression and its symptoms” (Hasin & Grant, 2002, p. 794). Also, substance abuse causes some factors, and in turn, those factors lead to depression. Such factors might include unemployment, relational conflicts, and financial hardship. As both direct and indirect causal hypothesis consistently views depression as the product of substance abuse (Swendsen & Merikangas, 2000), the principal focus of treatment in this case is on treating the substance use disorder.

Second, the primary psychiatric disorder-secondary substance use disorder models posit that severe mental illness increases patients’ vulnerability to developing substance use disorder. For the individuals with mental disorder, relatively small amounts of substances may result in psychological impacts, and furthermore, this may evolve into a clear use disorder because of their vulnerability to the effects of psychoactive substances (Drake, Osher, & Bartels, 1996).

The tension reduction hypothesis and the self-medication hypothesis are popular explanations among the several causal hypotheses. According to the tension reduction theory, alcohol serves to reduce tension or anxiety, possibly because of the depressing or tranquilizing effects of alcohol on the nervous system and drinking is thus reinforced by the tension reduction effects obtained (Conger, 1956, as cited in Kushner, Sher, Wood, & Wood, 2006). Therefore, this hypothesis conceptualizes abusive alcohol use as a motivated response to the presumably unpleasant experience of tension.

Khantzian’s (1997) self-medication hypothesis posits that alcohol or other substances may be used by people with a negative state of some variety to help them cope
as a “self-medication”, and substance use may be especially reinforcing for depressed people because it can decrease negative feelings. A broader definition might identify self-medication as any substance use that is engaged primarily in response to feelings of pain or distress, whether consciously or unconsciously. Also, a negative state can be thought of as the experience of unpleasant or painful physical or psychological states. Thus, self-medication is a reaction or a purposive coping behavior to a stressor, in this case psychological pain or distress. Therefore, self-medication conceptualizations would predict that an individual who is suffering from a diagnosable negative affect state, such as depression or anxiety, may drink in an effort to mentally escape.

Last, bidirectional models explain ongoing, interactional effects between psychiatric illness and substance use disorder. Substance use disorder and psychiatric illness develop independently at different times, but interact so that either disorder can initiate or influence the other, and become mutually maintaining. Substance use disorder could trigger mental illness in a biologically vulnerable individual, which is subsequently maintained by continued substance use disorder due to socially learned cognitive factors, such as beliefs, expectancies, and motives for substance use (Graham, 1998). In this model, the patients with two comorbid disorders need treatment for both psychiatric and substance use disorders. Despite the evidence that substance use disorder worsens the course of mental illness, this model remains largely theoretical and untested (Mueser et al., 1998).

*Shared etiology hypothesis*

This hypothesis suggests that substance use disorder and psychiatric disorder share a common etiology (Maier & Merikangas, 1996; Cadoret et al., 1996). The shared etiologic
factors include common exposure to prenatal environmental factors (e.g., maternal alcohol use or other toxin), biologic environmental risk factors (e.g., lead poisoning), or nonbiologic environmental risk factors, such as disruptive family environment (Merikangas, Risch, & Weissman, 1994). Thus, the increased rate of comorbidity between mental illness and substance use disorder is due to a third common factors such as genetic, psychosocial and environmental factors (Hasin & Grant, 2002). This hypothesis often has been addressed by examining the familial patterns of alcohol use and depressive symptoms. For example, if these two disorders share the same set of genes or familial environmental factors, the risk of alcohol use in relatives of depressive symptoms probands would be increased, as would the risk of depressive symptoms in relatives of alcohol use probands.

While this hypothesis has not often been replicated, some evidence exists to continue entertaining the shared etiology hypothesis as a possible explanation (Steunenberg et al., 2008). Thus, several competing explanations have been proposed until now. Although each model may account for only certain portion of the populations with comorbidity, the mechanisms discussed offer possible causal connections between alcohol use and depressive symptoms.

**Empirical findings regarding the association of alcohol use and depressive symptoms**

It is important to note that according to the study population examined, such as community-based study or clinic-based study, the approach used was different. First, community-based studies conducted structured interviews to assess the prevalence of psychiatric and mental disorders, and then, they analyzed the comorbidity or association of the two disorders. The cross-sectional studies in community-based studies examined
alcohol use’s effects on depressive symptoms or vice versa or correlation between two disorders. A few large community-based studies have examined the prevalence of the comorbidity of alcohol use and depressive symptoms with mixed age groups both the United States (Grant & Harford, 1995; Kessler et al., 1996, 1997; Regier et al., 1990; Warner et al., 1995) and other countries (Kessler et al., 2001; Merikangas & Stevens, 1998; Jacobi et al., 2004). The majority of those studies consistently found that depressive symptoms and alcohol use co-occur at much higher than chance levels. The Epidemiologic Catchment Area (ECA) Survey in the early 1980s (Robins et al., 1991) and the National Comorbidity Survey (NCS) in 1990s (Kessler et al., 1997) are the large national community surveys that examined the causal association between two factors. These surveys are relatively free from selection biases and thus provide the most generalizable statistics for the population as a whole.

On the other hand, the clinic-based studies are more popular in this area. While community surveys are population-based and give estimates of incidence and prevalence, clinic-based surveys can provide more in depth examinations in populations that have high incidence/prevalence of a problem. In the clinic studies, researchers collect data in treatment facilities with disorder-focused programs whether the disorder is depression-related disorder or alcoholism-related disorder. They are instantly selecting samples with one of the two disorders in which they are interested, and then, they measure the incidence of the second disorder to assess the comorbidity. Thus, some studies examine comorbidity among people with depression/depressive symptoms and alcohol use disorder by selecting for the depression first, and also conversely, some studies examine the association with depression/depressive symptoms among patients with alcohol use
disorder. The latter case, the alcohol use disorder-first approach may be more prevalent than depression-first approach (Brown et al., 1995). The results from the clinic studies may not be externally valid for a broader, non-treatment-seeking population even though the importance of these research findings should not to be underestimated. Followings are the findings from the cross-sectional and longitudinal approaches examining the association.

The results from the cross-sectional studies

1) Alcohol use effect in patients with depressive symptoms

Mueller et al. (1994) examined lifetime alcohol effect on the course of depression among 588 inpatients and outpatients with major depression. They found the patients who have lifetime alcohol use disorder decreased likelihood of recovery from major depression over 10 years (1.2% per week vs. 2.3% per week). By examining lifetime alcohol use disorder impact on depression with primary care inpatients, Cook et al. (1991) also reported that increased proportion describing a lack of overall improvement in major depression at follow-up. However, Melartin et al. (2002) reported no difference of current alcohol abuse and dependence on depression in psychiatric inpatients with depression.

Sullivan et al. (2005) reviewed the literature that examined the impact of alcohol problems in patients with major depression in clinical-based settings. The results based on 35 published papers examining the alcohol problem effect on major depression with mixed age groups showed that alcohol problems were associated with worse outcomes with respect to the course of patients’ depression (Sullivan et al., 2005).
2) Depressive symptoms’ effect in patients with alcohol use

In the studies examined with mixed aged group with 18 years or more in the clinic sample, Holahan et al. (2003) reported that depressed patients engaged in more drinking and problem drinking to cope than did community control sample.

Compton et al. (2000) explored the relationship between alcohol use disorder and comorbid depression among 425 persons in drug treatment who met DSM-III-R criteria for drug dependence. They found alcohol dependence and depression was divided nearly evenly between earlier (primary disorder) and later (secondary disorder).

3) Findings from community-based studies

Gratzer et al. (2004) examined the effects of alcoholism on depression in community-sample aged 15-64, in Ontario, Canada (n = 7195). The study found there were significantly higher rates of alcohol use in people who are depressed relative to control groups.

Bolton et al. (2009) examined the association between alcohol use and depressive symptoms based on the NESARC data, a large (n = 43,093, age 18 years and older) nationally representative survey of mental illness in community-dwelling adults. In their study, the use of alcohol and drugs to relieve affective symptoms was common among individuals with mood disorders in the general population, and it is related to substantial psychiatric comorbidity.

On the other hand, Merikangas et al. (1998) uncovered the association in their analysis of the World Health Organization International Consortium in Psychiatric Epidemiology (ICPE) database, a series of community epidemiologic surveys in six
countries. The study reported ORs of lifetime mental disorder with alcohol dependence was 4.7.

The paper that reviews the comorbidity between mental disorder and substance use disorder published up to January 2006, reported that people with a substance use disorder had higher comorbid rates of mental disorders than vice versa (Jane-Llopis & Matytsina, 2006). In particular, the review reported that while causal pathways differ across substances and disorders, there is evidence that alcohol is a casual factor for depression (Jane-Llopis & Matytsina, 2006).

In summary, some cross-sectional studies report self-medicating effect of depressive symptoms on alcohol use, but others rejected the hypothesis or reported that alcohol use is a causal factor of depressive symptoms. However, most of cross-sectional studies examined the one disorder effect on the course of the other disorder, these studies cannot adequately determine causal effect, and thus, longitudinal studies are need. Even though cross-sectional studies cannot determine temporal priority in this progression and those are unable to adequately explain causal relationship, these studies are important to understand co-occurrence of depressive symptoms and alcohol use.

The results from the longitudinal studies

Comparatively few longitudinal studies examining the association between depressive symptoms and alcohol use have been conducting in middle and older adult groups. There are three different kinds of findings when causal relationships were examined in longitudinal studies: the findings supported by only the primary substance use disorder-secondary psychiatric disorder model, the findings supported by only the
primary psychiatric-secondary substance use disorder model, and the findings supported by bi-directional model.

1) Studies supported by the primary substance use disorder-secondary psychiatric disorder model

The study based on the National Comorbidity Survey (NCS), a general population survey of persons aged 15-54 years in the non-institutionalized civilian population shows that most cases of lifetime major depressive disorder (MDD) are secondary. That is, they occur in people with a prior history of another DSM-III-R disorder, such as alcohol use disorder (AUD). Secondary MDD is, in general, more persistent and severe than pure or primary MDD (Kessler et al., 1996). Paljarvi et al. (2009) conducted the prospective population-based two-wave cohort study (aged 20-54 years at baseline) with 5 years interval. The study found a positive association between baseline binge drinking and depressive symptoms 5 years later. Agosti and Levin (2006) also analyzed the causal relationship from the NCS. In their study, current substance use disorder including alcohol use disorder increased the risk of past year depression.

In the Epidemiological Catchment Area (ECA) Survey, alcohol and drug use disorders were strongly associated with lifetime mood disorders (Regier et al., 1990). This result is the same as the studies based on the NCS data (Kessler et al., 1996; Agosti & Levin, 2006). Similarly, in a general community survey, the National Longitudinal Alcohol Epidemiological Survey (NLAES) found that lifetime alcohol dependence significantly increased the risk of past year MDD (Grant & Harford, 1995; Hasin &
Grant, 2002). Wu and Anthony (1999) reported that substance use predicts increased depression, but depression does not predict increased substance use.

2) Studies supported by the primary psychiatric-secondary substance use disorder model

The findings supported by only primary psychiatric-secondary substance use disorder model are relatively few. In the data came from the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC), a large (n=43,093, age 18 years and older) nationally representative survey of mental illness in community-dwelling adults, almost one-quarter (24%) of individuals with a mood disorder use alcohol, illicit drugs, or misuse prescription medications in an effort to relieve their mood symptoms. This suggests that self-medication is common among individuals with mood disorders in the general population (Bolton et al., 2009).

Holahan et al. (2004) also examined the underlying causes of the co-occurrence between depressive symptoms and alcohol use. The results showed that depressed patients engaged in more drinking, to cope with the depressive symptoms as supported by self-medication hypothesis.

Crum, Storr, and Chan (2005) assessed whether depression is associated a risk for alcohol dependence in community-based middle-aged adults. The study reported that the odds of lifetime alcohol dependence among those who have depression were significantly elevated, but no association was found for depression with new onset of alcohol dependence.
Kuo et al. (2006) used a genetically informative population-based twin adult sample to examine the temporal relationship of alcohol dependence and major depression. In this study, prior major depression significantly affected risk for developing alcohol dependence, but by contrast, preceding alcohol dependence didn’t affect the risk for major depression.

3) Studies supported by bi-directional model

A few studies found the reciprocal relationship between depressive symptoms and alcohol use.

Compton et al. (2000) examined the relationship between substance use disorders and comorbid psychiatric conditions with 425 persons (mean age = 32.5) in drug treatment who met DSM-III-R criteria for drug dependence. In this study, alcohol dependence and depression were divided nearly evenly between earlier (primary disorder) and later (secondary disorder). Depression commonly preceded as well as followed drug dependence, making treatment and evaluation quite complicated.

The study examining the Epidemiologic Catchment Area survey indicates that baseline symptoms of depression or alcohol dependence increase the risk of developing alcohol dependence or depression, respectively, at 1 year (Gilman & Abraham, 2001). In particular, this study found that the association was stronger in individuals with alcohol dependence than one in the individuals with depression.

Paljarvi et al. (2009) also examined whether the relationship between alcohol consumption and depressive symptoms operates both ways. They found that alcohol
consumption measures were related strongly to measures of depressive symptoms and
conversely, prior depressive symptoms predicted later alcohol consumption over time.

4) Studies that reported no relationship

The Canadian National Population Health Survey (NPHS) with 12,290 adults in
community showed that drinking level did not have an elevated risk of major depression
(Wang et al., 2001). Some studies reported that frequency of drinking showed no
relationship with either depressive symptoms (Graham & Schmidt, 1999) or clinical
depression (Patten & Charney, 1998).

In summary, the clinic-based and community-based studies converge in several
respects concerning the comorbidity of alcohol use and depressive symptoms. The well-
received conclusion is that these disorders are risk factors for each other, and that this
relationship was evidenced by both clinic and community-drawn samples. In particular,
when considering causal association between these disorders, there is slightly more
support for the model that alcohol use increases the risk or severity of depressive
symptoms than the reverse. However, no consistent pattern emerges from these
paradigms to fully rule out any of the etiologic models reviewed earlier, and furthermore,
the temporal relationship or causal relationship between these two disorders is still
controversial.

Gender difference in the association

The literature demonstrates that gender differences pose an additional challenge to
establishing temporality. Several studies reported that the trajectories and types of
comorbid condition among adult samples differ by gender (Moscato et al., 1997; Schutte
et al., 1995, 1997). The studies suggested that although links between depressive symptoms and alcohol use have been proposed for men as well as women, depressive symptoms has long been hypothesized to have a more prominent role in the alcohol use of women (Turnbull & Gornberg, 1988). Also, depressive symptoms more frequently is an antecedent of drinking problems among women than among men while men may more often experience depressive symptoms as a consequence of drinking behavior than as an antecedent (Brennan et al., 1993).

Moscato et al. (1997) examined the relationships between depressive symptoms and alcohol problems among the household adults (aged 19 years or more) from Erie County, New York, assessed in 1986, 1989, and 1993 (n = 1306). In this study, for females, depressive symptoms predicted subsequent alcohol problems over 3 years and 4 years, but not for 7 years while for males, no association was found. Schutte et al. (1995) analyzed the relationship between depressive symptoms and drinking behavior separately for 1572 late-middle-aged community residents (55 to 65 years old) with 3-wave longitudinal data. The study found among women that heavier alcohol consumption predicted less depressive symptomatology 1 and 3 years later, whereas among men, having more depressive symptoms predicted less alcohol consumption later on.

In summary, by examining the previous studies, the association of depressive symptoms and alcohol use was differed by gender. The association of alcohol use with depressive symptoms was stronger among women than men, or depressive symptoms were a predictor of alcohol use only for women.
Literature gaps in terms of methodological aspects

The previous literature examining the relationship between depressive symptoms and alcohol use has limitations in terms of methodological approaches. First, most studies have been cross-sectional, making it difficult to conclude causal relationships and unable to take into account the cohort effects. Unraveling the nature of the relation between depressive symptoms and drinking behavior to determine the circumstance under which depressive symptoms acts as an antecedent or as a consequence of drinking behavior requires a longitudinal research design that controls for the effects of previous levels of functioning and uses time intervals sufficiently long to rule out the possibility that physiological effects of alcohol on the central nervous system can account for findings. In addition, cross-sectional research does not capture the variability in alcohol consumption characteristics of middle and older adults. Even though some studies used longitudinal data, they used retrospective longitudinal designs rather than prospective designs. Retrospective longitudinal studies are limited by recall bias of drinking patterns during the past few years. Therefore, failure to recall exactly may result in biased or underestimated findings. Birth cohort data are the ideal means of studying the accumulation over the life course of hazards of various kinds (Gunnell et al., 1998; Davey et al., 1998).

Second, many of the cross-sectional databases have been derived from clinic populations, raising issues of representativeness and generalizability. Community-based data has the advantage of generalizability to a large population.

Third, most longitudinal data sets include a wide age range and include a greater population of younger rather than older people. Thus, there is a possibility of under-represented drinking problems in older age groups. Many longitudinal and cross-sectional
data sets are not as useful as they appear to be. The numbers of older people identified as alcohol abusers are small and the variables chosen tend to be more relevant to younger drinkers than to older drinkers. There is relatively little knowledge of the sociodemographic and health characteristics and comorbidity patterns among individuals who have co-occurring depressive symptoms and alcohol consumption in the transition of retirement in middle and later life. Other important factors closely related to middle-age and older adults are rarely controlled for and it increases the chance of bias.

Fourth, in a longitudinal design, although it has been suggested that researchers should study pathways rather than stages, the majority of studies on midlife development have concentrated on this single life stage without considering the life event of individuals in terms of experiences and transitions through late adulthood, all of which might shape the quality of present and future existence.

Fifth, many of the larger studies took place in the 1970s to 1980s, and these cohorts may not represent current middle-aged and older adults.

Sixth, few studies include a wide range of predictors, such that relationships among different factors can be examined. The relationship could be better understood in the context of conditions in which symptoms co-occur.

Last, comprehensive empirical tests of all possible relationships using sophisticated statistical techniques, such as structural equation modeling are also rare. Thus, findings of the causal relationship between two disorders remain inconclusive. Also, confounding factors, especially closely related to the older adult population such as health conditions and social support are not considered in the analysis. Therefore, the results should be cautiously interpreted.
Conclusion

Although several mechanisms of comorbidity have been investigated, no single definitive causal or shared etiological risk factor has been found to underlie both disorders (Schuckit, 2006). Also, no sufficient data were available for testing whether alcohol use at the first time of measurement would predict change in depressive symptoms over time or vice versa (Conner et al., 2008). In particular, there are few studies focused on middle-aged and older adult samples.

By reviewing the previous findings, it is found that the temporality of conditions is a common characteristic of comorbidity studies because a key component of identifying the possible causal connection between two disorders is establishing their relationship to each other in time. However, differences in sample selection, data collection methods, and periods of observation might lead researchers to approach different hypotheses about the association between depressive symptoms and alcohol use (Angold, Costello, & Erkanli, 1999). For example, current comorbidity implies a common phenomenology, while successive or lifetime conditions imply a causal relationship between the two disorders. Thus, the previous findings obscure the potentially mutual and reciprocal processes that generate the associations between these disorders (Angold et al., 1999). While this perspective may be useful from a descriptive point of view, it limits explanation of the association, as the consideration of one disorder as antecedent or consequent to another obscures the hypothesis that an underlying predisposition interacts with psychosocial risk factors such as gender and age to determine the expression of a disorder (SAMHSA, 1999).
Despite the increasing focus on the high co-occurrence of depressive symptoms and alcohol use, relatively little research has been undertaken to examine whether an association between depressive symptoms and alcohol use is also present in middle and later life (Speer & Bates, 1992; Schutte et al., 1998; Atkinson, 1999).

2-2. Retirement, Depressive Symptoms, and Alcohol Use

Retirement is most commonly described in the research literature as an adjustment process spanning years rather than a discreet life event (Atchley, 1974; Marshall, Clarke & Ballantyne, 2001). The process of planning and adjusting to retirement can produce significant stress. Also, the stress might increase depressive symptoms and thus, the depressive symptoms might increase alcohol consumption during or after retirement event. The following section provides theoretical frameworks and empirical findings about retirement and its association with depressive symptoms and alcohol use.

Theoretical perspectives about retirement and mental health

Several theoretical perspectives were proposed to explain the association between retirement and mental health/alcohol use. Among them, three theoretical perspectives - role theory, continuity theory, and the life course perspective - have been frequently used as frameworks in retirement transition and retirement satisfaction studies (Auick & Moen, 1998; Wang et al., 2008).

Role theory.

Role theory (George, 1993), in conjunction with a life course perspective (Elder, 1995), provides the most commonly used explanations of adjustment to retirement.
According to role theory, social roles which define the social position of an individual within a given social system are based on enduring relations with other people and provide both a sense of identity and behavioural guidance (Thoits, 1992). Holding a variety of social roles, such as partnership, parenthood or paid labor, may affect the health of the individual since these provide the ‘‘object’’ and ‘‘meaning’’ to her or his life (Durkheim, 1951 as cited in Pescosolido & Georgianna, 1989). Therefore, movements into retirement are frequently experienced in terms of tension, loss, reluctance and failure, especially where individuals are strongly attached to occupations (Phillipson, 1993). Thus, the individuals who retire from their career jobs are vulnerable to feelings of role loss, which can lead to psychological distress.

Several studies have shown that social roles are related to depressive symptoms (Brown, 2002), psychological distress (Wheaton, 1990), and subjective well-being (Burton, Armstrong, & Rushing, 1993), and substance use (Burton, Johnson, Ritter, & Clayton, 1996). Whereas retirement has previously been conceptualized in terms of the loss of a social role, it is now more widely recognized that it entails the opportunity to acquire new roles (such as volunteer or part-time worker), and to continue in other roles (such as parent or friend) (Reitzes et al., 1998). Also, retirees with other role involvements or those who are retiring from an unpleasant job may be less troubled by and even pleased with the loss of those work roles (Adams, Prescher, Beehr, & Lepisto, 2002). For individuals who find their job stressful or burdensome, retiring could be a very positive experience—a relief from ongoing strains and conflicts.

Continuity theory
In this theory, continuity refers to a consistency of patterns over time, the accommodation of change without the experience of a stressful disruption. According to Atchley (1999), there is considerable continuity in identity and self-concept over the retirement transition, and this continuity contributes to the retirement adjustment process. He reported that “middle-aged and older adults attempt to preserve and maintain existing structures . . . and prefer to accomplish this objective by using continuity, i.e., applying familiar strategies in familiar arenas of life” (Atchley, 1989, p.183). Therefore, retirement is not seen as a stressful disruption but rather as an opportunity to maintain a certain lifestyle and social contacts (Quick & Moen, 1998). The theory also suggests that individuals who have been deeply involved in their work will try to sustain their daily routines by participating in activities which they value highly (Atchley, 1999).

Continuity theory does not preclude the existence of psychological stress led by role exit and role transitions. Instead, it emphasizes that maintaining continuity is critical for retirees to keep their psychological well-being. That is, retirees who maintain their lifestyle or activities through retirement or who view retirement as a fulfillment of a prior goal should not experience significant decline of psychological well-being during the retirement transition (Wang, 2007).

A life-course perspective

The life-course approach (Elder, 1995) highlights the dynamic processes of development and change over the life span. According to the life course theory, agegraded trajectories are subject to changing conditions and to short-term transitions such as entry into retirement (Elder, 1995). Normative later life stages may be characterized by movement to activities and roles that involve less responsibility to others (e.g., leisure
activities and retirement roles), and thereby, individuals should enjoy their postretirement life more and more over time and approach a stabilized psychological well-being state.

The life course perspective suggests that the experience of life transitions and developmental trajectories is contingent on the specific circumstances under which the transition occurs. The perspective also emphasizes the influence of individual attributes, job related psychological variables, and family-related variables in retirement-related decision-making (Wang et al., 2008).

In terms of the shape of the general postretirement psychological well-being trajectory after transition, the life course perspective suggests that the overall retirement adjustment direction should point to a stabilized and comfortable well-being state for retirees (Levinson & Levinson, 1996; Super, 1990). Therefore, after adjusting to retirement, retirees should enjoy their postretirement life more and more over time and approach stabilized psychological well-being states. As such, although some retirees may experience negative changes in their psychological well-being during the retirement transition, their psychological well-being may eventually recover and approach a more positive state (Gall et al., 1997).

Evidence of the association between retirement and mental health

The research regarding the effects of retirement on mental health reports inconsistent results. Previous studies reported that retirement may have an adverse effect through the absence of positive benefits derived from employment, including financial stability, a sense of purpose, or social activity (Moen, 1996). Conversely, some argue that retirement can relieve a person of the stressors of the working environment, and increase their sense of personal control (Drentea, 2002). Cross-sectional studies have shown both
that retirees are more likely to report poor psychological well-being compared with their employed peers (Kim & Moen, 2002) or that there is no association between retirement and measures of mental health or psychological distress (Herzog et al., 1991; Ross & Drentea, 1998). These discrepancies have not been resolved by longitudinal investigations. While some studies have reported increased depression and/or anxiety symptoms (Richardson & Kilty, 1991), others have found improved mental health following retirement (Mein et al., 2003). Also, many large-scale studies show no significant overall influence of retirement on wellbeing (Calasanti, 1996; Charles, 2002; Midanik et al., 1995; Reitzes et al., 1996).

Theoretical perspectives about retirement and alcohol use

From the previous literature, retirement may have a dual effect on retirees’ drinking behavior depending on its social framing as either a loss or relief (Goffman, 1974).

*Loss perspective*

According to Bacharach et al. (2004), retirement is associated with the exacerbation of drinking problems with two key assumptions: (a) retirement is a highly stressful and dysphoric experience because it is accompanied by a distancing from coworkers providing a critical basis of support, and (b) increased alcohol consumption may serve as a means by which to self-medicate the strain and feelings of despair resulting from such a loss of support. Recent research suggests that retirees may experience status loss (Bacharach et al., 2004), or a reduction in income or perceived economic security (Bosse, Aldwin, Levenson, & Workman-Daniels, 1991).
Especially for those already manifesting a drinking problem, the literature on stress and alcohol suggests that there is a heightened risk that the stress and despair potentially associated with such a qualitative loss of tangible support will be associated with a subsequent exacerbation of problem drinking (Bacharach et al., 2007).

**Relief perspective**

Conversely, some researchers view retirement as a stress reducing experience or as a “relief,” because retirees are able to disengage not only from the stresses and frustrations of work (Bosse et al., 1991), but also from those social networks and subcultures that may have encouraged drinking as one means by which to cope with such work-based stressors and frustrations (Ekerdt et al., 1989). In this perspective, Retirement may be beneficial because, by effectively removing the individual from potentially risky work-related social contexts, it also likely removes from the individual’s social network those peripheral support providers potentially subscribing to permissive drinking norms (Roman & Johnson, 1996). A number of studies support this perspective, demonstrating that support can be associated with heavier or more problematic consumption, particularly in terms of those spending much of their time in social contexts characterized by more permissive drinking norms (Sonnenstuhl, 1996).

**Evidence of the relationship between retirement and alcohol use**

Recent studies have focused increasingly on the role of retirement in the onset and exacerbation of alcohol problems among older Americans (Bacharach et al., 2004; 2007). Most of these studies have been grounded on the assumption that retirement is a stressful life event and generally stressful experience which serves as "an invitation to increased alcohol consumption or abuse" (Ekerdt et al., 1989, p. 347). Thus, researchers conjecture
that retirees turn to alcohol to help them cope with this stress (Ekerdt et al. 1989; Brennan & Moos, 1996; Glass, Prigerson, Kasl, & de Leon, 1995).

In addition to increased stress, the researchers also conjecture that retirees have more leisure time to consume alcohol. With few role constraints or social obligations, the retiree may consume alcohol with greatly reduced risks of adverse social consequences (Perreira & Sloan, 2001).

However, the results from the related previous studies have been inconclusive, with some studies finding a positive association between retirement and drinking problems among older individuals (Perreira & Sloan, 2001; Catalano, 1993), others finding that retirement is associated with diminished drinking problems and lower levels of alcohol use (Gallo et al., 2001; Neve, Lemmens, & Drop, 2000). Also, some studies report no direct impact on problem drinking (Bacharach et al., 2004; 2007).

Age and cohort effects on mental health

It is important to note that the previous literature suggested that both age and cohort may confound the interpretation of research in this area. Cohort analysis distinguishes age and birth cohort as two types of time-related variations. This distinction is important “for attributions of etiology or social causation in that ‘age effects’ largely represent internal developmental changes of individuals, and ‘cohort effects’ are conceptualized as the essence of social change that reflects the imprint of exogenous social environmental factors rather than developmental or aging phenomena” (Yang, 2007). This distinction also relates to the generalizability of research findings. In the absence of cohort effects, age changes in depressive symptoms are broadly applicable across individuals of different cohorts. On the other hand, differences in cohorts suggest
effects of differential exposure to social risk factors for depressive symptoms that are
cohort-specific (George, 2004).

Advancing age is often reported as a risk factor for depressive symptoms, perhaps
in part because of age-related differences in lifestyle characteristics (Forman-Hoffman et
al., 2007). Recently, an increasingly strong association between aging and depressive
symptoms has been reported from several clinical studies (Blazer, 2002).

Age associated with retirement is also a measure of biological and psychological
functioning, a determinant of social roles and norms, and an indicator of birth-cohort
membership (Moen, 1996). Therefore, age-related change effects on mental health can
confound the relationship. A number of studies have demonstrated that older adults
experience a lower prevalence of common mental disorders and psychological symptoms
than middle-aged population (Andrews, Henderson, & Hall, 2001; Henderson et al.,
1998; Melzer, Buxton, & Villamil, 2004). Recent epidemiologic studies have found an
increase in rates of major depressive disorder among U.S. birth cohorts from 1905 to
1975 (Kessler & Walters, 1998; Klerman et al., 1996). Therefore, better mental health
observed at retirement may reflect age or cohort differences rather than the effect of
retirement (Butterworth et al., 2006). The psychological effects of retirement may be
also influenced by the social norms surrounding employment status at different ages
(Herzog et al., 1991). Retirement is the norm for men aged 65 and older in most western
nations, with most men below this age still in paid employment. Herzog et al. (1991)
proposed that the adverse psychological effects of retirement might be greater for
younger retirees relative to those at or above the official retirement age.
Life course theories suggest that depressive symptoms rise with older age and varies by cohort; adjusting for major life course changes such as marital, economic, and employment statuses explains some of these age and cohort patterns of depressive symptoms (Yang, 2007).

2-3. The Other Risk Factors Affecting Depressive Symptoms and Alcohol Use

Understanding the processes that generate the interrelationships between depressive symptoms and alcohol use is facilitated by considering several key risk factors that have been implicated in the development of these conditions during middle and later life. There are a number of important predictors to be considered in this relationship. In a comprehensive review about the risk factors associated with depressive symptoms in the elderly, Vink et al. (2008) report that substantial evidence is found for the following risk factors for both prevalence and incidence of depression: chronic diseases, poor self-perceived health, functional disability, previous psychopathology, smaller social network size, being unmarried, qualitative aspects of social network, stressful life events and female gender. Also, the risk factors found to be associated with a higher likelihood of alcohol use in older adults include male sex, younger age, living alone, being divorced, more-active lifestyle, better health and functional status, and smoking (Hajat et al., 2004; Ganry et al., 2002; Brennan & Moos, 1996).

Sociodemographic factors

Several studies have sought to document a relationship among sociodemographic factors, depressive symptoms and alcohol use. Females are more likely to exhibit depressive symptoms than males (Strawbridge et al., 2002; Blazer et al., 2002) and,
conversely, males are more likely than females to have alcohol use disorder. The findings from the NCS with adults aged 18 or older found that 18.6% of females versus 11% of males reported a lifetime prevalence of depression, and 11% of females versus of 6% of males reported experiencing major depressive disorder in the past 12 months (Kessler et al., 1996). In contrast, lifetime alcohol abuse was found by 12.5% of males versus 6.4% of females and lifetime alcohol dependence was reported by 20.1% of males and 8.2% of females. Gender was also treated as a potential moderator in the relationship because alcohol tends to have stronger effects on women (Davies & Bowen, 1999), and women who experience more health problems may be more likely to reduce their alcohol consumption (Brennan et al., 1999).

Socioeconomic status (SES) is well documented in the literature as the risk factors for depressive symptoms and alcohol use (Kessler et al., 2001). Low SES status is consistently associated with increased levels of depressive symptoms. Lower income is associated with higher depressive symptoms (Siegel et al., 2004), but some studies report no significant association (Horowitz et al., 2005; Strawbridge et al., 2002). Lower level of education was associated with depressive symptoms (Blazer et al., 2002) or depressive disorders (Beekman et al., 2001; Geerlings et al., 2000). However, some studies reported no significant difference in the association of education with depressive symptoms (Horowitz et al., 2005; Bisschop et al., 2004).

The previous research on marital status and mental health has consistently shown that married people have better mental health than those who are nonmarried (Leino et al., 1995; Power et al., 1999). Married individuals have repeatedly been shown to have a lower prevalence of major depressive disorder than those who are divorced, widowed, or
separated (Wade & Cairney, 2000). Some studies suggest that depressive symptoms may cause individuals to be “selected” out of marriage; alternatively marital disruption may “cause” depressive symptoms (Wade & Pevalin, 2004; Bulloch et al., 2009).

**Health**

Health condition, measured by number of diseases/symptoms or functional status, is a factor to be related the association with depressive symptoms and alcohol use in the middle and older adult groups. The number of chronic illnesses and alcohol use is negatively associated in later life (Steunenberg et al., 2008). The reason of this association is that having a chronic illness is protective for alcohol consumption in this population. That is, elderly with one or more chronic illnesses use more medication and therefore less alcohol, knowing that alcohol consumption is not allowed in combination with most of the medicines taken. Chronic illnesses and difficulties in performing daily activities are also associated with both emergence and persistence of depressive disorders (Cole & Dendukurt, 2003).

In summary, even though the previous studies suggest possible confounding effects of several factors, such as socioeconomic factors, education, and health status on the relationship between depressive symptoms and alcohol use, few studies have adequately included those factors in analyses. This study includes these factors as time varying covariates when examining the relationship between depressive symptoms and alcohol use. In addition to using a longitudinal data which can be adequately explored the causal relationship, conceptualizing with various possible confounding factors could be contributed to uncover the relationship between two disorders.
Conclusion

The previous literatures have suggested varied theoretical perspectives and conflicting empirical findings regarding the relationship among retirement and depressive symptoms/alcohol use. Through a careful review of the evidence, this study conceptualizes that retirement may have the effect of increased depressive symptoms based on the role theory, and thus, increased depressive symptoms in retirees may affect increased alcohol use based on the loss perspective of retirement. Thus, this study considers that retirement will moderate the relationship between depressive symptoms and alcohol use.

2-4. Conceptual Framework and Research Questions

In order to answer the research questions, the conceptual framework depicted in Figure 2-1 is used. The conceptual model includes the variables of depressive symptoms and alcohol use with two time points in addition to retirement and several controlled variables. As Figure 2-1 shows, there are several paths. First, the correlation between depressive symptoms and alcohol use at time 1 will be analyzed (hypothesis 1) and also, the correlation between depressive symptoms and alcohol use at time 2 will be analyzed (hypothesis 2). Next, separate auto-lagged relationship of depressive symptoms and alcohol use between time 1 and time 2 will be analyzed (hypothesis 3 & 4). Next, cross-lagged relationship between depressive symptoms at time 1 and alcohol use at time 2 will be analyzed (hypothesis 5-1), and cross-lagged relationship between alcohol use at time 1 and depressive symptoms at time 2 will be analyzed (hypothesis 5-2). Finally, group comparison by retirement status in the relationship will be analyzed (hypothesis 6-1 & 6-
2). As controls, gender and education at baseline and marital status, household income
and a number of illness at time 2 is included in the analyses.
Figure 2-1. Conceptual model

Covariates:
- Gender
- Education
- Income
- No. of illness

Group comparison:
- Retired vs. Non-retired

Depressive symptoms

Alcohol use - 1994

Alcohol use - 2005
Based on the literature review, the following research questions and hypotheses were developed:

Research question 1.
What is the relationship between depressive symptoms and alcohol use at midlife?

Hypothesis 1. Depressive symptoms and alcohol use will be correlated with each other at midlife.

Research question 2.
What is the relationship between depressive symptoms and alcohol use at early later life?

Hypothesis 2. Depressive symptoms and alcohol use will be correlated with each other at early later life.

Research question 3.
What is the relationship of alcohol use at midlife on alcohol use at early later life?

Hypothesis 3. Alcohol use at midlife will be positively related to alcohol use at early later life.

Research question 4.
What is the relationship of depressive symptoms at midlife on depressive symptoms at early later life?

Hypothesis 4. Depressive symptoms at midlife will be positively related to depressive symptoms at early later life.

Research question 5.
What is the direction of the relationship between depressive symptoms and alcohol use from middle to early later life?
Hypothesis 5-1: Alcohol use at midlife will be positively related to depressive symptoms at early later life.

Hypothesis 5-2: Depressive symptoms at midlife will be positively related to alcohol use at early later life.

Research question 6-1.
Does retirement status moderate the relationship between depressive symptoms at midlife and alcohol use at early later life?

Hypothesis 6-1: Depressive symptoms at midlife and alcohol use at early later life will be significantly increased only for the employed peers.

Research question 6-2.
Does retirement status moderate the relationship between alcohol use at midlife and depressive symptoms at early later life?

Hypothesis 6-2: Alcohol use at midlife and depressive symptoms at early later life will be significantly increased only for the retired peers.
CHAPTER 3. Method

This study employs a longitudinal panel design using two waves of the Wisconsin Longitudinal Study (WLS). This chapter discusses the design of the study, study sample and data source, measures of major variables used in this study, and data analysis.

3-1. Study Design

This study utilized a non-experimental panel research design using the Wisconsin longitudinal study (WLS). A panel study or cohort study is a form of longitudinal study. A panel study measure the same sample of respondents at different points in time. Unlike trend studies, panel studies can reveal both net change and gross change in the dependent variable (Kazdin, 1998). WLS followed the samples in 1957, 1964, 1975, 1993-1994, and 2004-2005 (Sewell, Hauser, Springer, & Hauser, 2004). This study used the samples of the two waves of 1993-1994 and 2004-2005 from the WLS.

3-2. Sample

WLS includes a one-third random sample of 10,317 men and women who graduated from Wisconsin high schools in 1957 and of their randomly selected brothers and sisters. Survey data were collected by phone and mail from the original respondents or their parents in 1957, 1964, 1975, 1993, and 2004. The WLS includes a full record of social background, schooling, family formation, labor market experiences, social participation, psychological characteristics, health behaviors, measure of health and mental health.

The two most recently released data sets collected in 1993-1994 and 2003-2005 were used for this study because depressive symptoms and alcohol use-related questions were not asked in the earlier waves of the WLS. The sample included interviews with
8,493 of the surviving 9,741 members in 1993 and with 7,265 members in 2003–2005. The sample was aged 53-56 during 1993 data collection and aged 64–67 during the 2003–2005 period. At each year, all surviving WLS graduates were fielded for contact via telephone and consented for research. Phone interviews were conducted for a one-hour and audio-recorded using computer-assisted techniques, WLS graduates were also mailed a 55-page paper mail-back survey.

A 79% random sample was selected to be asked depression, depression history and drinking problems/alcohol symptoms questions in the 1992-3 respondent telephone interviews (N = 6,636 in 1993-1994 and 5,665 in 2003-2005 among the original respondents in 1957). The alcohol behaviors items were also randomly subsampled at just under 80%, and participants who completed this section constituted the baseline sample of 6,489. In the 2004 follow-up, 5,283 of the baseline respondents completed the telephone interview (81% of baseline participants). Both time points also included a supplemental mail interview with similarly high response rates. The final sample used for this study is restricted to those who completed both categories on depression and alcohol behaviors at each time point. Also, to examine the moderating effect of retirement status, this study is restricted to those who were employed at baseline. Thus, study sample used in this study is those who respond each time point and non-retired at time 1 (N = 3,204).

3-3. Measures

Depressive symptoms.

Depressive symptoms are defined as having depressed mood or a loss of interest or pleasure in daily activities based on the DSM-IV. According to the DSM-IV, a person who suffers from major depressive disorder must either have a depressed mood or a loss
of interest or pleasure in daily activities consistently for at least a two week period. This mood must represent a change from the person’s normal mood; social, occupational, educational or other important functioning must also be negatively impaired by the change in mood. For this study, depressive symptoms were assessed by the 20 items of Center for Epidemiological Studies’ Depression Scale (CES-D; Redloff, 1977). The CES-D scale is a widely used and valid measure of depressive symptoms. Respondents were asked to report how many days they experienced each of the following symptoms within the past week (0 = zero days, 1 = one day, 2 = two days, 3 = three days, 4 = four days, 5 = five days, 6 = six days, 7 = seven days): (1) feeling blue; (2) feeling bothered by things; (3) feeling a failure; (4) feeling happy; (5) feeling unfriendly from others; (6) feeling lonely; (7) enjoy life; (8) crying spells; (9) feeling disliked by others; (10) feeling sad; (11) feeling depressed; (12) having trouble focusing; (13) poor appetite; (14) feeling just as good as other people; (15) feeling did everything an effort; (16) feeling hopeful about the future; (17) feeling fearful; (18) sleep restlessly; (19) talk less than usual; and (20) feeling could not get going. Items (4), (7), (14), and (16) are reverse coded so that high values on the scale indicate greater psychological distress. The highest possible score was thus 20 * 7 = 140.

For analyzing by SEM, this study used the four factors derived from the CES-D 20-items. The four factors include depressive affect-, well-being-, somatic-, and interpersonal symptoms. Deriving the four factors was based on previous literature (Knight et al., 1997). Also, this study confirmed that the four factors fit well for the study sample by exploratory factor analysis. The depressive affect symptoms include (1) feeling blue, (2) feeling bothered by things, (3) feeling a failure, (6) feeling lonely, (8)
crying spells, (10) feeling sad, (11) feeling depressed, and (17) feeling fearful. Well-being symptoms include (14) feeling just as good as other people, (16) feeling hopeful about the future, (4) feeling happy, and (7) enjoy life. Somatic symptoms include (12) having trouble focusing, (13) poor appetite; (15) feeling did everything an effort, (18) sleep restlessly, (19) talk less than usual, and (20) feeling could not get going, and interpersonal symptoms include (5) feeling unfriendly from others and (9) feeling disliked by others.

The CES-D was designed for use in general population surveys, and is therefore a short, structured self-report measure (Radloff, 1977). The scale has been used in numerous studies, and it has been reported by many clinicians and researchers as a reliable and valid instrument. The previous studies indicate that the CES-D exhibits good internal consistency reliability with an alpha level of .85 in the general population and .90 in a patient population, with test-retest correlations ranging from .45 to .70, which is in the moderate range (Radloff, 1977; Orme, Reis, & Herz, 1986). Validity has also been reported to high, meaning that the scale is able to differentiate between a clinical and general population (Radloff, 1977). Also, the CES-D correlates well with other scales designed to measure depression (Radloff, 1977; Orme, Reis, & Herz, 1986).

Alcohol use

Alcohol use was defined as prolonged and excessive intake of alcoholic drinks, leading to a breakdown in health and an addiction to alcohol such that abrupt deprivation leads to severe withdrawal symptoms (Heuberger, 2009). This study uses the two distinct measures to assess alcohol use: frequency and quantity.
Frequency was measured by the question that during the last month on how many
days did you drink any alcoholic beverages such as beer, wine, liquor, or mixed alcoholic
drinks. Quantity was measured by the question that number of alcoholic drinks
respondent had in the last month.

**Retirement status**

Respondents were asked their current retirement status at each wave. They were
separated into two groups: those in retired group between wave 1 and wave 2 versus
those in non-retired group at wave 2 (retired=1, non-retired = 0).

**Control variables.**

Control variables include gender, level of education, total household income, and
number of illness.

Gender at baseline was dichotomized (males = 0, females = 1).

Education at baseline (1993) was measured through a series of questions
regarding the highest level or degree of schooling completed. These were transformed
into equivalent years of education. Completion of high school received a value of 12,
while completion of college received a value of 16, etc. Household income was
ascertained at baseline and follow-up, in thousands of dollars.

A number of illnesses were measured at each wave using a modified version of
the Duke Older Adults Research Survey (Duke University Center for the Study of Aging
and Human Development, 1978). Respondents reported whether a medical professional
had ever diagnosed them with any of the following 17 medical conditions (allergies,
anemia, arthritis/rheumatism, asthma, serious back trouble, bronchitis/emphysema,
cancer, circulation problems, colitis, diabetes, heart trouble, high blood pressure, high
cholesterol, kidney/bladder problems, chronic liver trouble, multiple sclerosis, and ulcer). Total number of medical diagnosis was coded ranged from 0 to 17.

3-4. Data Analysis

The hypothesized relationships are examined by multi-group cross-lagged Structural equation model (SEM) to simultaneously address the direction of relationship between depressive symptoms and alcohol use and the difference by retirement status.

SEM was chosen as the best method of data analysis because it allows complete and simultaneous tests of all the relationships between one or more independent and dependent variables and has an advantage over a series of regression equations (Ullman, 2001). Currently, all other multivariate techniques can examine only one relationship at a time. SEM can examine a dependent variable that is composed of more than one outcome, such as the present model. It can examine a series of dependence relationships simultaneously, as depicted in the hypothesized path model described above, where an independent variable becomes a dependent variable in subsequent temporal levels (Hair, Anderson, Tatham, & Black, 1998). The advantage of this simultaneous estimation is the ability to test lagged and contemporaneous reciprocal effects, i.e., to empirically test for hypothesized causal ordering. SEM also gives an overall indication of the fit between the proposed theory and data. In addition, the relationships that are tested by SEM are free of measurement error, since measurement error is estimated and removed (Ullman, 2001).

This dissertation uses the SPSS version 17 (SPSS Inc., 2008) for data preparation and descriptive analyses. Amos version 17.0 (2008) is used for the multi-group cross-lagged analyses using SEM.
3-4-1. Overview of SEM

SEM consists of a measurement model to define hypothetical latent constructs in terms of measured variables, and a structural model to depict relationships among latent constructs. SEM is a multivariate method combining aspects of factor analysis and multiple regression in analyzing a set of interrelated relationships among manifest and latent variables simultaneously.

SEM is distinguished for several reasons from conventional analysis. First, the basic statistic in SEM is the covariance. While conventional analysis such as ordinary least squares regression attempts to minimize differences between observed and expected individual cases, SEM aims to minimize differences between observed and expected covariance matrices. Based on the covariance statistic, SEM attempts “to understand patterns of correlations among a set of variables and to explain as much of their variances” (Kline, 1998, pp. 10-11).

Second, SEM allows researchers to assess measurement error in the model estimation process. In this method, a regression coefficient is composed of two elements: structural coefficient between the independent and dependent variable and the reliability of the predictor variable. By distinguishing a structural model from a measurement model, SEM can examine the relationship among constructs that are not influenced by measurement errors (Newcomb, 1994).

Third, SEM allows incorporation of latent variables into the analyses unlike conventional analysis, which focuses solely on observed variables. Because SEM is not limited to relations among observed variables, it gives researchers more flexibility to
study any combination of relations (path analysis [only observed variables], CFA [only latent variables], and hybrid models [some observed and some latent variables]).

Finally, SEM allows researchers to estimate very complicated multivariate relationships, while the conventional analysis cannot accommodate multiple indicators of the same construct due to potential problems such as multicollinearity. Also, conventional multivariate techniques which even allow multiple dependent variables (e.g., multivariate analysis of variance and canonical analysis) can examine only a single relationship at a time. SEM examines a series of dependent relationships simultaneously (Kline, 1998). Thus, SEM has the advantage of determining causal priority and causal predominance when reciprocal relationships are found (De Lange et al., 2004).

Analytic procedures of SEM are conducted by following steps.

In SEM, it is important to follow the four steps: (1) determine input matrix and estimation method, (2) assess the identification of the model, (3) evaluate the model fit, and (4) respecify the model and evaluate the fit of the revised model.

First, a covariance matrix is used as an input data form as most estimation methods in SEM presume the analysis of unstandardized variables. For a method of model estimation, the Maximum Likelihood method (ML) is utilized for the proposed model. Compared to other estimation procedures (e.g., multiple regression), ML estimation can calculate estimates of all model parameters all at once. The parameters of the model are: (1) variances and covariances of exogenous variables, (2) direct effects (path coefficients) on the endogenous variables, and (3) variances of the disturbances (residual errors). The advantages of ML estimation include the automatic derivation of indirect and total effects and the availability of
several indexes of overall model fit (Kline, 1998). In addition, robust statistics (corrected for nonnormality in large samples) are applied in light of the several problems related to analysis of nonnormal multivariate data.

Second, the ability of the proposed model to generate unique solutions is assessed. In order for the model to be identified, the model should be able to calculate a unique estimate for every one of the parameters in a model and the model as a whole. A proposed model should be over-identified to meet basic requirement for model identification. In other words, the number of parameters must be less than the number of observations. The over-identified model results in positive degrees of freedom, which allows for rejection of the model.

Third, overall model fit (the goodness of fit between the hypothesized model and the sample data) is assessed with several goodness-of-fit indexes because a single index reflects only a particular aspect of fit. Chi-square statistics is one of the most commonly used techniques to examine overall model fit. A nonsignificant goodness-of-fit $\chi^2$ statistic is favored because it indicates that the implied covariance matrix is nearly identical to the observed data. However, $\chi^2$ statistics is too sensitive with a sample size. With large samples, very minor differences can yield a significant $\chi^2$, indicating the rejection of a good model (Keith & Witta, 1997). Therefore, considering the relatively large sample size of this study sample (N=3,204), the results of the chi-square test were not considered to be critical in evaluating model fit for this study.

For the present study, additional fix indexes have also been utilized: Comparative Fit Index (CFI; Bentler, 1990), the Tucker-Lewis Index (TLI; Tucker & Lewis, 1973) and Root Mean Square Error of Approximation (RMSEA; Browne & Cudeck, 1993). These
indexes compare the proposed model to the null model. CFI calculates the proportion in
the improvement of the overall fit of the proposed model relative to the null model.
Compared to other similar indexes which are affected by sample size (e.g. NFI), CFI is
utilized to take sample size into account. CFI provide a measure of covariation in the data
and with values greater than .90 or above indicative of an acceptable fit to the data.
Finally, RMSEA is a measure of the discrepancy between the observed and model-
reproduced population covariance matrices, averaged over the model degrees of freedom.
It adjusts for model complexity by distributing the overall discrepancy over all possible
areas of disagreement, as well as removes the influence of sample size on the estimated
discrepancy. It has been recommended that RMSEA values of .05 or lower should be
deemed to reflect good fit. Later, both Steiger (1990) and Brown and Cudeck (1989)
recommended that .05 be used in conjunction with an upper bound (on a 90% confidence
interval) no higher than .08 be used conjointly. Hu and Bentler (1999), based on their
power analyses of RMSEA with MLE extraction, recommended an RMSEA of .06 or
lower. This study follows the Steiger (1990) and Brown and Cudeck (1989)’s
recommendation.

The last step is modification of the hypothesized model by examining the
measurement model and goodness-of-fit indexes to improve the model based on
theoretical justification. Even though SEM is a “priori,” many applications of SEM are a
combination of confirmatory and exploratory analyses (Kline, 1998). Ideally, the causal
model should be estimated once and then a decision made to accept or reject the model
based on its correspondence to the data. However, there is a practical advantage to
utilizing the “model-generating application of SEM” (Jöreskog, 1993). First, this
approach allows the researcher to identify data related problems and potential sources of poor fit. Second, the modification process can help researchers to acquire new insights into the relationship among variables. Values of the Lagrange Multiplier (LM) test and the Wald test are used as a guide for the hypothesized model modification. The LM test assesses “the statistical viability of specified restriction in the model” (Bryne, 1994, p. 11). In other words, the value of LM test approximates the extent to which the model’s overall $\chi^2$ would decrease (i.e. significantly better fitting model) if a given parameter were freely estimated. A large value of an LM test indicates the improvement of the overall fit of the hypothesized model. Also, the Wald test estimates whether the model’s overall $X^2$ increases if a particular parameters is set to zero. A nonsignificant value of the Wald test indicates that the elimination of a particular parameter does not bring a significant degradation in model fit.

3-4-2. Preliminary data analyses

Preliminary analyses consist of power analysis, assumption check and data preparation for analyzing SEM.

First, the statistical power for the study sample will be examined. The statistical power involved in SEM is quite complicated compared to that of regression modeling (Bentler & Chou, 1987). Several factors needed to be considered in computing the power of goodness-of-fit statistics to evaluate the appropriateness of a SEM model. These factors were: the sample size (N), the selected significance level ($\alpha$), the size of the model as determined by the degree of freedom (df), test statistics of the null hypothesis (F0), and test statistic of the alternative hypothesis (Fa). The detailed procedure will be mentioned in chapter 4.
Next, SEM requires certain assumptions to be met (e.g., continuous variables, normality, linearity of all relationship), it is important to examine whether the assumptions of SEM have been met. Variables used in SEM are typically continuous (Kline, 1998). Another requirement of SEM is the linearity of relationship between exogenous and endogenous variables. In order to examine the linearity, residual plots against independent variables are closely examined. In case of violation of the linearity assumption, the use of transformed independent variables is considered to uphold these requirements. For each variable, the present study evaluates the linearity assumption by plotting the studentized residuals against the predicted values. The data show a linear relationship between the residuals and the predicted values.

Multicollinearity can be a cause of a non-positive covariance matrix in SEM (Kline, 1998). In order to inspect multicollinearity between variables, correlation matrix was used. The values over .85 are used as an indicator of potential problems of multicollinearity based on Kline’s (1998) suggestion.

Normality tests are conducted by examining skewness and kurtosis of the data, and normal probability plots. Skewness refers to the level of symmetry of the data distribution and kurtosis indicates the peakedness or flatness of a distribution. Kline (1998) suggested that data sets with absolute values of univariate skew indexes greater than 3.0 be described as “extremely” skewed while the absolute value of the kurtosis index that is greater than 10.0 indicates a problem with the data. Because outliers can contribute to both univariate and multivariate non-normality, outliers are modified for scores so that they have less influence in the analysis. If the variables are not normally distributed, some techniques such as a log transformation will be used to get normalize
skewed distributions. In addition, a covariance matrix analyzed with ML (Maximum Likelihood method) is applied for the present study because ML has been found to be fairly robust with the data that violate multivariate normality (Bollen & Long, 1993).

Consistency of measurements is tested by examining the reliability. A general method of testing reliability is to calculate Cronbach’s alpha. The value of reliability needs to be .70 or above for further data analysis (Nunnally, 1978).

Finally, for dealing with missing data, several methods are used. Traditional methods for fixing problems of missing data are “listwise” deletion (i.e., deleting cases which are not complete and using only complete in analysis) or “pairwise” deletion (i.e., deleting cases in forming statistics to be used in analysis). These methods are simple and easy to understand, but deleting some cases in the data set has critical limitations such as loss of subjects, increased standard errors, and bias if the data is not MCR (Missing Completely at Random) (Rubin, 1987). Multiple Imputation methods fills in missing values by a method that randomly selects values from the scores that other cases like this case have (Loehlin, 2004). SEM software such as AMOS (Arbucke, 1997), allows researchers to use the advanced method such as Full Information Maximum Likelihood Method (FIML; Arbuckle, 1996) dealing with missing information. FIML can handle missing values with no loss of information contained in the available dataset. FIML estimates missing information by using the raw data rather than to the covariance matrix, using maximum likelihood function (Loehlin, 2004). In order to use the FIML method, the data must be (1) missing at random and (2) multivariate normal (Raykov, 2005).

Regarding the normality, the skewness and kurtosis of each variable are examined.

3-4-3. Descriptive statistics and correlations
The means and standard deviations of the study variables as well as bivariate correlations between them are calculated using the version 19 of SPSS package. These descriptive statistics allow researchers not only to better understand phenomena of interest, but also to replicate the SEM analysis.

3-4-4. Confirmatory factor analysis

Data analyses were conducted using SEM with a two step approach as suggested by Anderson and Gerbing (1988). According to Anderson and Gerbing (1988), measurement model should be assessed before and independent of the structural (i.e. hypothetical) model by a confirmatory factor analysis (CFA). The CFA ensures that the hypothesized latent constructs fit into the data. The measurement model for this study was comprised of four latent variables measured by two to four indicators each. The efficacy of the overall measurement model will be tested based on popular and conventional fit indices such as $\chi^2$ statistic, Comparative Fit Index (CFI), and Root Mean Squared Error of Approximation (RMSEA).

3-4-5. Examining study hypotheses

This dissertation uses a cross-lagged multi-group SEM to examine the reciprocal relationships between depression and alcoholism.

*Cross-lagged analysis*

The cross-lagged analysis is technique is particularly designed to test causal structures where measurements of the same variables have been made at two different times in the same sample (Edwards, Guppy & Cockerton, 2007). Cross-lagged panel designs are not intended to establish the causality of such relationships in the traditional framework of using an experimental design, controlling for extraneous variables, and so
on. They can, however, provide information about the strength of the temporal relationship among the variables, which is necessary in establishing causality (Menard, 1991).

Figure 3-1 is an analytic model that involves a cross-lagged panel design. There are two main types of such paths: the paths within each latent variable and the paths between the latent variables. The autoregressive paths (e.g., the path between alcoholism at time 1 and time 2 & the path between depression at time 1 and time 2), or paths that link a latent variable measured later with the same variable measured earlier (e.g., the path between depression at time 1 and alcoholism at time 2 & the path between alcoholism at time 1 and depression at time 2), provide information about the relative stability of the construct, with higher values indicating greater stability. The paths measured across latent variables (e.g., the path between depression at time 1 and alcoholism at time 2) provide information about the degree to which one variable is a stronger temporal predictor of the other (e.g., Does a stronger relationship exist between baseline depression and follow-up alcoholism, or vice versa?) and are used to draw conclusions about potential causality.

The baseline latent variables (depression at time 1 and alcoholism at time 1) are generally conceptualized as correlated with each other, as are the disturbance (or error) terms associated with the latent variables at later time points. These disturbance terms, such as all error terms, indicate the amount of variability in the endogenous variables associated with unknown factors. Error terms of the same measured variable assessed on different occasions are also conceptualized as correlated with each other because of the assumption that factors contributing to measurement error in any specific variable will be
consistent across measured occasions. For example, in Figure 3-1, the latent variables that represent the error associated with the depression at baseline and follow-up, respectively, are modeled as being correlated with each other.

Figure 3-1. Multi-group Cross-lagged model

Multi-group analyses

The multi-group SEM analyses models allow this study to test for the differences in retirement status in the estimated path coefficients between depression and alcoholism variables. By running a multi-group model simultaneously for the retired and non-retired models, this dissertation tests whether path coefficients differ across the two groups. A significant difference in the path coefficients suggests a significant difference between two groups. This way is equivalent to testing if there is an interaction between retirement status and each predictor variable in each of the cross-lagged models.
As suggested in the literature, models are assessed using several goodness-of-fit criteria. As the chi-square is highly sensitive to sample size and distributional assumptions (Hu & Bentler, 1995), three other measures of the overall goodness-of-fit are used. The root mean square error of approximation (RMSEA), the comparative fit index (CFI) and the Tucker–Lewis index (TLI). The CFI and TLI range in value from 0 to 1, with a value of greater than 0.9 indicating a good fit, RMSEA values range upwards from 0, a perfect fit, through to 0.05, a good fit, up to 0.08, a fair fit, and >0.1, a not acceptable fit (Browne & Cudeck, 1993). Correlations of some errors between outcomes at the same time point are freely estimated to reduce biases arising from unmeasured third variables.

The analyses procedure of multi-group cross-lagged analyses consists of two steps. In step one, metric invariance tests over time are conducted by equality constraints of the same factor loading over time, and then, the differences between the two retirement-related groups are compared using a series of nested path models. The chi-square difference tests show differences in model fit when parameters are estimated freely. A significant chi-square difference between two models suggests a model with less constrained parameters fits the data better than the alternative. All models control for gender and education at baseline, and household income and health condition at all time points.

In step two, this study tests for differences between retired and non-retired groups in all paths in our model by freeing paths one at a time, from depression to alcohol use and from alcohol use to depressive symptoms and comparing model fit with a fully constrained model. In the final model, paths that are significantly different between the two groups are estimated freely.
CHAPTER IV. Results

This chapter includes the results derived from the empirical analysis and hypothesis testing of the study’s theoretical model. The first section discusses the results related to preliminary data analysis, including issues of normality and power, along with a presentation of general descriptive statistics for the sample. Following this, the next sections deal specifically with the testing of both the proposed measurement and structural models.

4-1. Preliminary Analyses

*Power considerations*

Because of its influence on estimation methods, statistical significance, statistical power and empirical validity, assessing the appropriateness of sample size is a prudent and often necessary initial step in data analysis (Cohen, 1992). General rules for assessing minimum sample size have previously been proffered and referenced by various authors. For example, the 10:1 ratio of subjects to variables suggested by Nunnally (1978) has become a relative standard in determining sample size for factor analysis. In terms of path analysis and linear regression, Green (1991) proposes the 50+8m rule, where m represents the number of independent variables in the regression equation. In the case of this study’s analysis, the sample (N=3,204) conforms and exceeds the minimum requirements of Nunnally (1978) and Green (1991) for path and regression-type equations.

For Confirmatory factor analysis (CFA), while sample size is important, the ability of the measurement model to be identified is critical (Kline, 2005). According to Kline (2005), SEM models need to be over-identified in order to allow for scientific
interpretation. Over-identified models occur when the number of estimable parameters does not exceed the number of data points. Estimable parameters include factor loadings, error variances of both the observed variables and latent constructs and the covariates among the latent constructs. The number of estimable parameters of the analysis in the present study is equal to 43 (note that for each factor one observed variable is fixed and therefore not counted). The number of data points can be calculated based on the formula \( p(p + 1)/2 \), where \( p \) is the number of observed variables. In the CFA model there are 12 observed variables, thus the number of data points is 12(12+1)/2 or 78. Therefore, this number exceeds 43 so the CFA can be said to be over-identified, and thus, it allows for model fit and path coefficient estimations.

*Assumption consideration for SEM*

As mentioned in chapter III, preliminary analyses include assumption check for SEM and missing data analyses. Because the current study restricted analyses of the sample to the non-missing cases of major variables, alcoholism and depression, at time 1 and 2, there are only a few item-level missing cases. Those incomplete data were treated by a Full Information Maximum Likelihood (FIML; Arbuckle, 1996) method in Amos19 to incorporate the incompleteness of data in estimating parameters. FIML does not impute data but breaks the likelihood function down into components based on patterns of missing data, allowing estimation to proceed using all available data. To implement FIML, intercepts of observed variable and means of latent factors are estimated in the model. FIML estimation of SEM parameters requires data with normal distribution.

Next, for checking normality assumption, skewness and kurtosis were examined for the study variables. The variable of depressive affect, somatic, interpersonal and
quantity variables were not normally distributed. For treating a non-normality problem, square root transformation methods were used. In the results, all study variables met the criteria for normal distributions (Table 4-1).

4-2. Sample Characteristics

Table 4-1 presents sample size, means, medians and standard deviations for all observed variables. The sample consists of 3,204 middle-aged adults in 1993-1994 that were resurveyed in 2003-2005: 1,558 male (48.6%) and 1,646 female (51.4%). The mean age of respondents was 55.89 years old at time 1, and 66.89 years old at time 2. The total depressive symptoms measured by the CES-D were decreased over time. This change was statistically significant by analyzing the paired t-test \[ t(3,203) = 9.335, \ p<.001 \]. Except somatic symptoms, three factors of depressive symptoms tended to decrease with age. However, alcohol use was increased with age (from 6.5 to 7.6 days for the last month) and quantity of alcohol use increased from 12.94 to 14.61 drinks for a previous month \[ t(3,198) = -8.463, \ p<.001 \] for alcohol frequency, \& \[ t(3,192) = -3.257, \ p<.01 \] for alcohol quantity]. These results are consistent with the results from recent studies that total alcohol consumption and drinking frequency remain stable or increase among older adults (Benzies et al., 2008; Moos et al., 2004). The median scores of total household income were decreased with age from 58,000 to 51,000, and the respondents who have two or more illnesses were increased from 25.1% to 48.2%.
Table 4-1. Sample characteristics

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD/%)</td>
<td>Skewness</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mean (SD/%)</td>
</tr>
<tr>
<td>Age</td>
<td>55.89(54-57)</td>
<td>1,646(51.4%)</td>
</tr>
<tr>
<td>Gender (female)</td>
<td>1,646(51.4%)</td>
<td>13.9 (2.4)</td>
</tr>
<tr>
<td>Education</td>
<td>13.9 (2.4)</td>
<td>0.90</td>
</tr>
<tr>
<td>Income</td>
<td>58,000</td>
<td>1.67(65)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>51,000</td>
</tr>
<tr>
<td>&lt;20,000</td>
<td>341(10.6%)</td>
<td>530 (16.5%)</td>
</tr>
<tr>
<td>20,001-40,000</td>
<td>523 (16.3%)</td>
<td>714 (22.3%)</td>
</tr>
<tr>
<td>40,001-60,000</td>
<td>817 (25.5%)</td>
<td>651 (20.3%)</td>
</tr>
<tr>
<td>60,001-80,000</td>
<td>607 (18.9%)</td>
<td>466 (14.5%)</td>
</tr>
<tr>
<td>80,001-100,000</td>
<td>341 (10.6%)</td>
<td>261 (8.1%)</td>
</tr>
<tr>
<td>&gt;100,000</td>
<td>573 (17.9%)</td>
<td>582 (18.2%)</td>
</tr>
<tr>
<td>No. of illness</td>
<td>1.0 (0-11)*</td>
<td>0.27 (T)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1.0 (0-14)*</td>
</tr>
<tr>
<td>0</td>
<td>1400 (43.7%)</td>
<td>1000 (31.2%)</td>
</tr>
<tr>
<td>1</td>
<td>804 (25.1%)</td>
<td>1547 (48.2%)</td>
</tr>
<tr>
<td>≥2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol</td>
<td>6.51 (8.7)</td>
<td>1.66</td>
</tr>
<tr>
<td>frequency</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol</td>
<td>12.94 (20.8)</td>
<td>4.0 (0-240)*</td>
</tr>
<tr>
<td>Quantity</td>
<td></td>
<td>4.0 (0-248)*</td>
</tr>
<tr>
<td>CES-D</td>
<td>14.9 (14.2)</td>
<td>12 (0-126)*</td>
</tr>
<tr>
<td>Affect</td>
<td>3.85 (5.9)</td>
<td>0.86 (T)</td>
</tr>
<tr>
<td>Wellbeing</td>
<td>5.66 (5.9)</td>
<td>1.16</td>
</tr>
<tr>
<td>Somatic</td>
<td>9.72 (3.7)</td>
<td>0.07 (T)</td>
</tr>
<tr>
<td>Interpersonal</td>
<td>1.06 (1.75)</td>
<td>0.90 (T)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. (T) = transformed score, * = median values (range)

Table 4-2 shows the results of sample characteristics according to retirement status. Alcohol frequency and quantity in the retired group were significantly increased
over time from 6.7 to 7.9 \([t (2,135) = -7.396, p<.001\) for alcohol frequency, \(t (2,128) = -4.465, p<.001\) for alcohol quantity]. Among the employed group, alcohol frequency was significantly increased over time from 6.0 to 7.0 \([t (934) = -4.265, p<.001\]. Depressive symptoms for both groups were decreased over time \([t (2,128) = 9.009, p<.001\) for retired group, and \(t (934) = 3.558, p<.001\) for employed group]. Alcohol frequency and quantity at time 2 between retired group and employed group were significantly different \([t (3,024) = -2.390, p<.05\) and \(t (3,024) = -3.354, p<.001\] but depressive symptoms was not different between groups \((p>.05)\).

Table 4-2. Comparison of the sample characteristics by retirement status

<table>
<thead>
<tr>
<th></th>
<th>Retired((N = 2,135))</th>
<th>Employed((N = 934))</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1994 Mean (SD)</td>
<td>2005 Mean (SD)</td>
</tr>
<tr>
<td>Age</td>
<td>55.95</td>
<td>66.95</td>
</tr>
<tr>
<td>Gender (female)</td>
<td>1,023 (47.9%)</td>
<td>529 (56.6%)</td>
</tr>
<tr>
<td>Education(years)</td>
<td>13.9 (2.4)</td>
<td>14.0 (2.4)</td>
</tr>
<tr>
<td>Income (median)</td>
<td>61,000</td>
<td>50,000</td>
</tr>
<tr>
<td>No. of illness</td>
<td>936 (43.8%)</td>
<td>398 (18.6%)</td>
</tr>
<tr>
<td></td>
<td>666 (31.2%)</td>
<td>673 (31.5%)</td>
</tr>
<tr>
<td></td>
<td>533 (25.0%)</td>
<td>1056 (49.4%)</td>
</tr>
<tr>
<td>Frequency of alcohol</td>
<td>6.7 (8.8)</td>
<td>7.9 (9.8)</td>
</tr>
<tr>
<td>Quantity of alcohol</td>
<td>13.5 (20.4)</td>
<td>15.5 (25.0)</td>
</tr>
<tr>
<td>Depressive symptoms</td>
<td>15.2 (14.4)</td>
<td>12.6 (12.9)</td>
</tr>
</tbody>
</table>

Table 4-3 and 4-4 show the results according to usual volume of alcohol and depressive symptoms at time 1 and time 2. Individuals were classified using the National Institute of Alcoholism and Alcohol Abuse’s (NIAAA) criteria for older adults (NIAAA, 1995). Those who reported alcohol consumption of less than or equal to two drinks per day \((\leq 14 \text{ drinks per week})\) were classified as moderate drinkers, and those who reported
consumption of more than two drinks per day (more than 14 drinks per week) were classified as heavy drinkers. The people who reported heavy drinking and depressive symptoms was 3.8% at time 1 and 0.9% at time 2. The prevalence of co-occurring symptoms of depression and heavy alcohol use were lower than those of previous studies. The prevalence of comorbid depressive symptoms with alcohol use among older adults varied from 7% to 38% of those with a psychiatric illness and from 21% to 66% of those with substance abuse (Bartels et al., 2006). The association between level of alcohol volume and depressive symptoms was not significant at time 1 ($\chi^2=2.94, p>0.05$), but at time 2, it was significant ($\chi^2=15.53, p<0.001$).

Table 4-3. Results of usual volume of alcohol and depressive symptoms in 1994

<table>
<thead>
<tr>
<th></th>
<th>Non-drinkers(0)</th>
<th>Moderate drinkers (1-14drinks/wk)</th>
<th>Heavy drinkers (≥15drinks/wk)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CES-D ≤ 21</td>
<td>642 (20.0%)</td>
<td>1649 (51.4%)</td>
<td>90 (2.8%)</td>
</tr>
<tr>
<td>CES-D &gt; 21</td>
<td>244 (7.6%)</td>
<td>537 (16.7%)</td>
<td>31 (0.9%)</td>
</tr>
</tbody>
</table>

Table 4-4. Results of usual volume of alcohol and depressive symptoms in 2005

<table>
<thead>
<tr>
<th></th>
<th>Non-drinkers(0)</th>
<th>Moderate drinkers (1-14drinks/wk)</th>
<th>Heavy drinkers (≥15drinks/wk)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CES-D ≤ 21</td>
<td>690 (21.5%)</td>
<td>1,729 (54%)</td>
<td>121 (3.8%)</td>
</tr>
<tr>
<td>CES-D &gt; 21</td>
<td>232 (7.2%)</td>
<td>403 (12.6%)</td>
<td>29 (0.9%)</td>
</tr>
</tbody>
</table>

Table 4-5 shows the results of the relationship between dichotomized depressive symptoms and usual volume of alcohol according to retirement status. The prevalence of co-occurring symptoms of depression and heavy alcohol use in the employed group was lower (3.3%) than that of the retired group (4.8%). The association between usual volume of alcohol and depressive symptoms was not significant for the employed group ($\chi^2=5.53, p>0.05$), but it was significant for the retired group ($\chi^2=10.63, p<0.01$).
Table 4-5. Comparison of the relationship according to retirement status

<table>
<thead>
<tr>
<th></th>
<th>CES-D ≤ 21</th>
<th>CES-D &gt; 21</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-drinkers (0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Employed</td>
<td>203 (21.7%)</td>
<td>78 (8.3%)</td>
</tr>
<tr>
<td>Retired</td>
<td>450 (21.1%)</td>
<td>144 (4.3%)</td>
</tr>
<tr>
<td>Moderate drinkers (1-14 drinks/wk)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Employed</td>
<td>495 (52.9%)</td>
<td>129 (13.8%)</td>
</tr>
<tr>
<td>Retired</td>
<td>1,172 (54.9%)</td>
<td>257 (12.0%)</td>
</tr>
<tr>
<td>Heavy drinkers (≥15 drinks/wk)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Employed</td>
<td>22 (2.3%)</td>
<td>7 (0.7%)</td>
</tr>
<tr>
<td>Retired</td>
<td>92 (4.3%)</td>
<td>20 (0.9%)</td>
</tr>
</tbody>
</table>

Bivariate correlations

Table 4-6 presents the correlation coefficients of the study variables. Overall there is consistency in the relationships among measures of depression and measures of alcohol use at both time 1 and time 2. The measures of depression, affective, well-being, somatic, and interpersonal factors, at time 1 were significantly correlated depression measures at time 2 (0.14 to 0.48, p<0.1). Frequency and quantity of alcohol use at time 1 also were significantly correlated to frequency and quantity of alcohol use at time 2 (0.61 to 0.70, p< 0.01). The correlations between the alcohol use variables and the depression variables were not strong either at time1 or time 2. A closer examination reveals that individual indicators of theoretical constructs have very weak correlations. Specifically, affective symptoms and interpersonal symptoms at time 1 show weak correlations with the alcohol frequency and quantity at time 2, and in the same manner, frequency and quantity of alcohol use at time 1 also show weak correlations with the affective, well-being, and interpersonal factors of depressive symptoms at time 2. However, these indicators were incorporated in further analysis because of the strong theoretical support for the association. There were statistically significant relationships between gender and alcohol use and depressive symptoms (e.g., female gender & affective at time 1 [r = .13, p<.01] and time 2 [r = .15, p<.01], female gender & frequency of alcohol at time 1 [r = -.19,
and time 2 \( r = -0.18, p < 0.01 \), gender & quantity at time 1 \( r = -0.24, p < 0.01 \) and time 2 \( r = -0.25, p < 0.01 \). There were also statistically significant relationships between covariates, such as sociodemographic and health variables and alcohol use and depressive symptoms. Education level and wellbeing symptoms at time 1 was correlated at time 1 \( r = -0.09, p < 0.01 \), and there were correlated between education level and alcohol frequency at time 1 \( r = 0.08, p < 0.01 \) and time 2 \( r = 0.14, p < 0.01 \). Also, there were correlation between education and alcohol quantity at time 1 \( r = 0.04, p < 0.05 \) and time 2 \( r = 0.09, p < 0.01 \), income and alcohol frequency at time 1 \( r = 0.14, p < 0.01 \) and time 2 \( r = 0.19, p < 0.01 \), income and alcohol quantity at time 1 \( r = 0.14, p < 0.01 \) and time 2 \( r = 0.18, p < 0.01 \). A number of illness at time 1 and 2 were positively correlated with all factors of depressive symptoms at time 1 and 2 and negatively correlated with alcohol frequency and quantity each time point (e.g., illness & affective symptoms at time 1 & 2 \( r = 0.18, p < 0.01 \), illness & alcohol frequency at time 1 \( r = -0.05, p < 0.01 \) & time 2 \( r = -0.09, p < 0.01 \), illness & alcohol quantity at time 1 \( r = -0.05, p < 0.01 \) & time 2 \( r = -0.07, p < 0.01 \)). In addition, income level at time 1 was highly correlated with at time 2 \( r = 0.53, p < 0.01 \) and a number of illness was highly correlated over time, too \( r = 0.39, p < 0.01 \). Taken together, the strength and direction of the relationships among variables found in the correlation analysis has partially supported that guarantee a more formal test of the theoretical model.
Table 4-6. Correlation coefficients among observed variables

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
<th>13</th>
<th>14</th>
<th>15</th>
<th>16</th>
<th>17</th>
<th>18</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Gender (female)</td>
<td>1</td>
<td>.13**</td>
<td>-.15</td>
<td>.12</td>
<td>.13</td>
<td>.01</td>
<td>.08</td>
<td>-.01</td>
<td>-.19**</td>
<td>-.24**</td>
<td>-.16**</td>
<td>.10**</td>
<td>.15</td>
<td>.03</td>
<td>.12</td>
<td>-.01</td>
<td>-.18**</td>
<td>-.25**</td>
</tr>
<tr>
<td>2. Education</td>
<td>1</td>
<td>.37**</td>
<td>-.07</td>
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<td>-.08**</td>
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<td>-.09**</td>
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</tr>
<tr>
<td>6. Wellbeing</td>
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<td>-.05**</td>
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<td>-.04</td>
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<td>10. Quantity</td>
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<td>-.10**</td>
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<td>.14**</td>
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<tr>
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<tr>
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</tr>
<tr>
<td>15. Somatic</td>
<td>1</td>
<td>.23</td>
<td>-.02</td>
<td>-.02</td>
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<tr>
<td>16. Interpersonal</td>
<td>1</td>
<td>-.05**</td>
<td>-.03</td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<td>17. Frequency</td>
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<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>18. Quantity</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>
The correlations among constructs provide some preliminary evidence for the hypothesized model (see Table 4-7). Depressive symptoms at time 1 was significantly correlated with alcohol use at time 2 ($r = -.037, p < .01$), and alcohol use at time 1 was significantly correlated with depressive symptoms at time 2 ($r = -.041, p < .01$). These negative correlations indicate an inverse relationship between the two constructs.

Table 4-7. Correlation coefficients among constructs

<table>
<thead>
<tr>
<th></th>
<th>Alcohol use94</th>
<th>Alcohol use05</th>
<th>Depressive symptoms94</th>
<th>Depressive symptoms05</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcohol use94</td>
<td>1</td>
<td>.651**</td>
<td>-.029</td>
<td>-.041*</td>
</tr>
<tr>
<td>Alcohol use05</td>
<td>1</td>
<td></td>
<td>-.037*</td>
<td>-.070**</td>
</tr>
<tr>
<td>Depressive symptoms94</td>
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<td>1</td>
<td></td>
<td>.515**</td>
</tr>
<tr>
<td>Depressive symptoms05</td>
<td></td>
<td></td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

Table 4-8 and table 4-9 show the separate correlation results of the employed and the retired respondents in 2005. The correlation between depressive symptoms and alcohol frequency in the retired group was stronger ($r = -.107, p < .01$) than that of the employed group ($r = -.075, p < .05$). Also, the correlation between depressive symptoms and alcohol quantity in the retired group was significant ($r = -.061, p < .01$) while the correlation between depressive symptoms and alcohol quantity in the employed group was not significant ($r = -.043, p > .05$).
Table 4-8. Correlation coefficients of the employed respondents in 2005

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender(F)</td>
<td>1</td>
<td>- .207**</td>
<td>- .294**</td>
<td>.100**</td>
<td>.136**</td>
<td>- .223**</td>
<td>- .268**</td>
<td>- .143**</td>
</tr>
<tr>
<td>Education</td>
<td>1</td>
<td>.317**</td>
<td>- .046</td>
<td>- .051</td>
<td>.152**</td>
<td>.059</td>
<td>.075*</td>
<td></td>
</tr>
<tr>
<td>Income</td>
<td>1</td>
<td>- .077*</td>
<td>- .123**</td>
<td>.208**</td>
<td>.159**</td>
<td>.088**</td>
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<td></td>
</tr>
<tr>
<td>Illness</td>
<td>1</td>
<td>.249**</td>
<td>- .065*</td>
<td>- .053</td>
<td>.062</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CES-D</td>
<td>1</td>
<td>- .075*</td>
<td>- .043</td>
<td>.073*</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Frequency</td>
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<td>.046</td>
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<tr>
<td>Quantity</td>
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<td></td>
<td></td>
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</tr>
</tbody>
</table>

Table 4-9. Correlation coefficients of the retired respondents in 2005

<table>
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<tr>
<th></th>
<th>1</th>
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<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender(F)</td>
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<td>- .090**</td>
<td>- .162**</td>
<td>.101**</td>
<td>.084**</td>
<td>- .162**</td>
<td>- .233**</td>
<td>- .192**</td>
</tr>
<tr>
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<td>- .025</td>
<td>- .082**</td>
<td>.145**</td>
<td>.082**</td>
<td>- .024</td>
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</tr>
<tr>
<td>Income</td>
<td>1</td>
<td>- .023</td>
<td>- .119**</td>
<td>.125**</td>
<td>.087**</td>
<td>.047*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Illness</td>
<td>1</td>
<td>.198**</td>
<td>- .063**</td>
<td>- .035</td>
<td>.059**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CES-D</td>
<td>1</td>
<td>- .107**</td>
<td>- .061**</td>
<td>.074**</td>
<td></td>
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<td></td>
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<tr>
<td>MAST</td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>
4-3. Measurement Model: Confirmatory Factor Analysis (CFA)

The present study utilized SEM to test the hypothesized models about relationships among observed and latent variables for depressive symptoms and alcohol use. Data analyses were conducted using SEM with a two step approach as suggested by Anderson and Gerbing (1988). The first step was to test the measurement model to ensure that the hypothesized latent constructs fit into the data via a confirmatory factor analysis. The measurement model was comprised of four latent variables measured by two to four indicators each. This measurement model with a total of 12 indicators (i.e., observed variables) plus error terms for each variable represents the measurement model in this study. Testing the measurement model revealed that the model’s fit is acceptable ($\chi^2 = 983$, df = 47, CFI = .940, RMSEA = .073). Table 4-10 shows the standardized factor loadings for depressive symptoms and alcohol use at time 1 and time 2. The critical ratios of all unstandardized $\lambda$ values were more than 2.58 which mean all are significant at a 1% significance level. Therefore, null hypothesis was rejected. The items loaded on depressive symptoms at time 1 with loadings ranging from 0.44 to 0.84. Depressive symptoms at time 2 produced loadings ranging from 0.44 to 0.80 at the 0.001 level of statistical significance. Also, the items of alcohol use at time 1 and time 2 loaded 0.79 to 0.98. Bagozzi and Yi (1988) suggest that standardized $\lambda$ values with 0.5 or more to 0.95 or less mean construct reliability and convergent validity. With the exception of one variable, the interpersonal, the factor loadings of all variables were over 0.5. Also, the composite reliability values are also all above 0.70, a threshold normally used to assess reliability efficacy (Koufteros, 1999). The constructs showed reliability because the Cronbach’s alpha scores of the depressive symptoms at time 1 and time 2 were .85 and
.84 each while the Cronbach’s alpha scores of alcohol use at time 1 and time 2 were .88 and .89 each. There were no significant cross loadings for any of the items included in the measurement model. Thus, these statistics provide substantial evidence that the constructs included in the measurement model demonstrate acceptable levels of convergent validity.

Table 4-10. The results of Confirmatory factor analysis

<table>
<thead>
<tr>
<th>Affect1994 ← Depression1</th>
<th>Estimate</th>
<th>S.E</th>
<th>C.R</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>.843</td>
<td>.093</td>
<td>25.800</td>
<td>***</td>
<td></td>
</tr>
<tr>
<td>Wellbeing1994 ← Depression1</td>
<td>.548</td>
<td>.069</td>
<td>22.603</td>
<td>***</td>
</tr>
<tr>
<td>Somatic1994 ← Depression1</td>
<td>.553</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>interpersonal1994 ← Depression1</td>
<td>.441</td>
<td>.019</td>
<td>19.398</td>
<td>***</td>
</tr>
<tr>
<td>Affect2005 ← Depression2</td>
<td>.801</td>
<td>.066</td>
<td>27.491</td>
<td>***</td>
</tr>
<tr>
<td>wellbeing2005 ← Depression2</td>
<td>.532</td>
<td>.055</td>
<td>23.134</td>
<td>***</td>
</tr>
<tr>
<td>Somatic2005 ← Depression2</td>
<td>.605</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Interpersonal2005 ← Depression2</td>
<td>.437</td>
<td>.014</td>
<td>19.830</td>
<td>***</td>
</tr>
<tr>
<td>Frequency1994 ← Alcoholism1</td>
<td>.946</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quantity1994 ← Alcoholism2</td>
<td>.831</td>
<td>.040</td>
<td>52.520</td>
<td>***</td>
</tr>
<tr>
<td>Frequency2005 ← Alcoholism1</td>
<td>.938</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quantity2005 ← Alcoholism2</td>
<td>.871</td>
<td>.039</td>
<td>58.863</td>
<td>***</td>
</tr>
</tbody>
</table>

Note. The variance of the somatic1994, somatic2005, frequency1994 and frequency2005 were constrained to 1. *** p<.001
4.4. Structural Model: Cross-lagged Modeling

Since the measurement model was appropriate, proceeding with the second step of testing the structural models was conducted. The basic idea of a cross-lagged modeling is to identify the causal priority of the variables by comparing the crossed and lagged paths between constructs. The crossed path coefficient represents the longitudinal
prediction of one construct by the other, above and beyond the autoregressive prediction of that construct from the earlier measure of itself (Curran & Bollen, 2001).

For model estimation as a first step, SEM with a full sample was conducted. This model yields an acceptable fit with the data ($\chi^2/df = 944.489/47$, CFI = .952, RMSEA = .077). Figure 4-2 shows a final model for hypotheses testing. Following the examination of the overall model fit indices, individual parameter estimates were assessed in order to examine feasibility of their estimated values. There were no individual parameters with unreasonable estimates “falling outside of acceptable range” (i.e., standard errors that extremely large or small, correlation greater than 1, and negative variance) (Byrne, 1994). Like most applications of significance tests, parameter estimates are divided by standard error (similar to z-statistics) in order to test whether parameter estimates in SEM are statistically different from zero. Based on an alpha level of .05 two tail standard, if test statistics are greater than ±1.96, it can be certain that the data are in the critical region and that the value of a sample statistics differs significantly from zero. Within the measurement model, all of the measured variables were significant indicators of their respective latent constructs (See table 4-10). Overall, it appeared as well-defined constructs.

In order to analyze the cross-lagged model, the model requires to be confirmed the metric invariance, path invariance and error covariance invariance over time. Factor loadings, coefficients for the stability and lagged effects were, respectively, constrained to equality across waves, making these parameters equivalent to average effects over the duration of the panel. The empirical adequacy of such equality constraints can be assessed via the likelihood ratio test, against the unconstrained model, as these are nested
one within the other. Where no loss of fit is incurred by fixing these parameters to equality over time, the constrained model should generally be preferred.

Table 4-11 presents the results of a series of invariance testing. In the table, model 1 represents the basic model without any constraint. Model 2 represents that all factor loadings between depressive symptoms constructs and their observed variables were constrained to be equal over time for examining metric invariance of depressive symptoms while model 3 shows that factor loadings between alcohol use and their observed variables were constrained to be equal over time for examining metric invariance of alcohol use. Model 4 represents the auto regressive coefficients and cross regressive coefficients were constrained to be equal for examining path invariance of both constructs. Finally, model 5 shows that the error covariance between two constructs was constrained to be equal to examine error covariance invariance.

Table 4-11. The results of invariance testing of the study model

<table>
<thead>
<tr>
<th>Model fit indices</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
<th>Model 5</th>
</tr>
</thead>
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<tr>
<td>X²</td>
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<td>962.506</td>
<td>963.299</td>
<td>963.299</td>
<td>963.299</td>
</tr>
<tr>
<td>Df</td>
<td>47</td>
<td>50</td>
<td>51</td>
<td>51</td>
<td>51</td>
</tr>
<tr>
<td>P</td>
<td>.000</td>
<td>.000</td>
<td>.000</td>
<td>.000</td>
<td>.000</td>
</tr>
<tr>
<td>CFI</td>
<td>.952</td>
<td>.952</td>
<td>.952</td>
<td>.952</td>
<td>.952</td>
</tr>
<tr>
<td>TLI</td>
<td>.921</td>
<td>.924</td>
<td>.926</td>
<td>.926</td>
<td>.926</td>
</tr>
<tr>
<td>RMSEA (90%CI)</td>
<td>.077 (.073-.082)</td>
<td>.075 (.071-.080)</td>
<td>.075 (.071-.079)</td>
<td>.075 (.071-.079)</td>
<td>.075 (.071-.079)</td>
</tr>
</tbody>
</table>

As mentioned in the chapter 3, the χ² test is not considered to be critical in evaluating model fit for this study since the χ² test is not a good indicator of model fit when the sample size is large (Marsh, Balla, & McDonald, 1988). As shown the Table 4-
11, there was no difference among the models in terms of CFI, TLI and RMSEA indices, and thus, it can be assumed that the model has the invariance of metric, path, and error covariance over time.

The final model is presented as the Figure 4-2.

Figure 4-2. A final model

4.5 Hypotheses Testing

Since the model fit was satisfactory, regression coefficients of this model were examined to test the study hypotheses 1, 2, 3, 4, 5-1 and 5-2. Researchers often revise structural equation models by omitting non-significant paths to obtain a more parsimonious model. However, this study used the current model as a final model to examine the hypothesized relationships because the aim of this study was not building a
best model of depressive symptoms and alcohol use rather examining the hypothesized relationships. Table 4-12 reports standardized path coefficients of the relationships among major variables without covariates.

Table 4-12. Standardized path coefficients of relationship

<table>
<thead>
<tr>
<th>Standardized regression weights</th>
<th>Unstandardized estimates (SE)</th>
<th>Standardized estimates</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression ↔ alcohol use - T1</td>
<td>-.417 (.192)*</td>
<td>-.045 (Correlation)</td>
</tr>
<tr>
<td>Depression ↔ alcohol use - T2</td>
<td>-.408 (.124)***</td>
<td>-.086 (Correlation)</td>
</tr>
<tr>
<td>Depression T1→ depression T2</td>
<td>.651 (.023)***</td>
<td>.703</td>
</tr>
<tr>
<td>Alcohol use T1→ alcohol use T2</td>
<td>.793 (.017)***</td>
<td>.711</td>
</tr>
<tr>
<td>Depression T1→ alcohol use T2</td>
<td>-.172 (.128)</td>
<td>-.020</td>
</tr>
<tr>
<td>Alcohol use T1→ depression T2</td>
<td>-.004 (.002)</td>
<td>-.035</td>
</tr>
</tbody>
</table>

* p<.05, ** p<.01, *** p<.001

Specifically, depressive symptoms was significantly related to alcohol use at time 1 (φ = -0.417, p<.05) (Hypothesis 1). Also, at time 2, depressive symptoms was significantly related to alcohol use (ρ = -.408, p<.001) (Hypothesis 2). The correlations between latent variables within each time period were negatively correlated (-.045 at time 1 & -.086 at time 2). However, with correlations in this range, depressive symptoms and alcohol use must be considered separate and distinct factors because the correlation at time 2 was measured by covariance of disturbance terms at time 2. Disturbance terms represent the residual variances of endogenous variables in the model and there are often good reasons to expect positive correlations between them (Anderson & Williams, 1992). The correlation between the disturbance terms represent all factors affecting depressive
symptoms and alcohol use at time 2 exclusive of the time 1 factors; the magnitude of this correlation suggests that some of these intervening factors influence both depressive symptoms and alcohol use.

Next, depressive symptoms at time 1 was positively associated with depressive symptoms at time 2 ($\beta = .651, p < .001$) (Hypothesis 3). Also, alcohol use at time 1 was positively associated with alcohol use at time 2 ($\beta = .793, p < .001$) (Hypothesis 4).

Regarding the hypothesis 5-1 and 5-2, no significant relationships were found in the cross-lagged regression. Depressive symptoms at baseline was associated with decreased alcohol use at follow-up, but it was not statistically significant ($-.172, p > .05$). Alcohol use at baseline was related to decreased depressive symptoms at follow-up, but it was not statistically significant, too ($-.004, p > .05$).

Figure 4-3. Results of cross-lagged SEM
Next, four covariates were entered in this cross-lagged relationships. The covariates included gender and education, and also total household income and a number of illnesses as time-varying covariates. This model yields an acceptable fit with the data ($\chi^2/df = 1389.081/103$, CFI = .941, RMSEA = .062). Table 4-13 shows the results of cross-lagged SEM with controlling of covariates.

Table 4-13. Standardized path coefficients of major relationships with controlling of covariates

<table>
<thead>
<tr>
<th>Standardized regression weights</th>
<th>Estimates (SE)</th>
<th>Standardized estimates</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression ↔ alcohol use - T1</td>
<td>-.034 (.181)</td>
<td>-.004 (correlation)</td>
</tr>
<tr>
<td>Depression ↔ alcohol use - T2</td>
<td>-.294 (.119)</td>
<td>-.065 (correlation)</td>
</tr>
<tr>
<td>Depression T1→ depression T2</td>
<td>.618 (.023)***</td>
<td>.677</td>
</tr>
<tr>
<td>Alcohol use T1→ alcohol use T2</td>
<td>.774 (.017)***</td>
<td>.693</td>
</tr>
<tr>
<td>Depression T1→ alcohol use T2</td>
<td>-.081 (.127)</td>
<td>-.009</td>
</tr>
<tr>
<td>Alcohol use T1→ depression T2</td>
<td>-.002 (.002)</td>
<td>-.015</td>
</tr>
</tbody>
</table>

*p<.05, ***p<.001

After entering covariates, depressive symptoms was not significantly related to alcohol use at time 1 ($\rho = -.034$, $p>.05$) (Hypothesis 1). Also, at time 2, depressive symptoms was significantly related to alcohol use ($\rho = -.294$, $p<.05$) (Hypothesis 2). These two relationships were measured by covariance of disturbance terms. That is, the results show that by controlling several related factors, the correlated relationships between depressive symptoms and alcohol use at each time point were eliminated. These results reflect that covariates affected the relationship between depressive symptoms and alcohol use.
Depressive symptoms was positively associated with depressive symptoms over time ($\beta = .618, p < .001$) (Hypothesis 3). Also, alcohol use was positively associated with alcohol use over time ($\beta = .774, p < .001$) (Hypothesis 4).

Regarding the hypothesis 5-1 and 5-2, no significant relationships were found in the cross-lagged regression. Depressive symptoms at time 1 was negatively associated with alcohol use at time 2, but it was not significant ($\beta = -.081, p > .05$) (Hypothesis 5-1). Alcohol use at time 1 was negatively related to depressive symptoms at time 2, but it was not significant, too ($\beta = -.002, p > .05$) (Hypothesis 5-2).

*Multi-group analysis*

In order to explore the hypothesis 6-1 and 6-2 (to examine the differences of the relationships by retirement status), multi-group SEM was conducted. Since a reasonable fit was established for the whole data set, an analysis of the separate groups was performed. Analyzing the group types involves a cross validation. Cross validation refers to the model’s ability to be invariant across groups.

By analyzing multi-group SEM, model 5 was fitted across retired and employed group differences. The model fit was acceptable ($\chi^2/df = 1411.258/206, CFI = .943, RMSEA = .044$). The results of the standardized path coefficients of model 5 are given in Figure 4-4 and 4-5. All relationships between study variables were similar in the two groups. In order to test whether this difference between two groups was statistically significant, confirmation of the structural model invariance across the groups is needed. Therefore, the process of metric invariance constraints and cross-group equality constraints must be realized. At first step, metric invariance for depressive symptoms over time was conducted by equality constraints across groups, and then, in the same
manner, metric invariance for alcohol use over time was conducted across groups. Next, the model was first estimated for retired group and employed group with all of the relevant paths freely estimated, and then re-estimated with each path in interest constrained to equality across groups. Considering the relatively large sample size of this study, the results of the chi-square test were not considered to be critical in evaluating model fit for this study. CFI and RMSEA were considered to evaluate model fit. In results, this study found metric invariance and cross-group invariance between two groups (Table 4-14). Compared to the feely estimated model, the two constrained models were the same in terms of the model fit indices.

Table 4-14. Tests of invariance between groups

<table>
<thead>
<tr>
<th>Model fit indices</th>
<th>Freely estimated model</th>
<th>Metric invariance constrained model</th>
<th>Cross-group invariance constrained model</th>
</tr>
</thead>
<tbody>
<tr>
<td>X²</td>
<td>1411.258</td>
<td>1443.549</td>
<td>1443.549</td>
</tr>
<tr>
<td>Df</td>
<td>206</td>
<td>218</td>
<td>218</td>
</tr>
<tr>
<td>CFI</td>
<td>.943</td>
<td>.942</td>
<td>.942</td>
</tr>
<tr>
<td>RMSEA</td>
<td>.044 (.042-.046)</td>
<td>.043 (.041-.045)</td>
<td>.043 (.040-.044)</td>
</tr>
</tbody>
</table>

Table 4-15 shows the chi-squared difference tests for nested models of all paths. The difference in the chi-square statistics between nested models was not statistically significant for the path ($\Delta \chi^2 = 3.978, p=.409$). It suggested that retirement status has not significantly moderated the longitudinal relationship between depressive symptoms and alcohol use (Hypothesis 6-1 and 6-2).
Table 4-15. The results of Chi-square difference tests

<table>
<thead>
<tr>
<th>Path</th>
<th>$\Delta \chi^2$</th>
<th>P-value of $\Delta \chi^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression T1 $\rightarrow$ depression T2</td>
<td>2.091</td>
<td>.148</td>
</tr>
<tr>
<td>Alcohol use T1 $\rightarrow$ alcohol use T2</td>
<td>.116</td>
<td>.733</td>
</tr>
<tr>
<td>Depression T1 $\rightarrow$ alcohol use T2</td>
<td>.808</td>
<td>.369</td>
</tr>
<tr>
<td>Alcohol use T1 $\rightarrow$ depression T2</td>
<td>1.119</td>
<td>.290</td>
</tr>
</tbody>
</table>

Note. $\Delta \chi^2 = \text{difference of } \chi^2$

For further understanding the relationship, the standardized coefficients for the paths were examined across the retired and employed groups. The standardized coefficients for the paths between depressive symptoms at time 1 and alcohol use at time 2 were .006 ($p > .05$) for the retired group and -.025 ($p > .05$) for the employed group. The standardized coefficients for the paths between alcohol use at time 1 and depressive symptoms at time 2 were -.025 ($p > .05$) for the retired group and -.020 ($p > .05$) for the employed group.

Figure 4-4. The retired group
Figure 4-5. The employed group

![Diagram of variables: Depressive (1), Depressive (2), Alcohol use (1), Alcohol use (2). Arrows indicate correlations: Depressive (1) → Depressive (2) = .704***, Depressive (2) → Alcohol use (1) = .023, Alcohol use (1) → Depressive (1) = .003, Alcohol use (1) → Alcohol use (2) = .723***, Alcohol use (2) → Alcohol use (1) = .020, Alcohol use (2) → Depressive (2) = -.042.]

Table 4-16. Comparison of Standardized path coefficients according to retirement status

<table>
<thead>
<tr>
<th></th>
<th>All sample</th>
<th>Retired</th>
<th>Employed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression1-Degression2</td>
<td>.693***</td>
<td>.692***</td>
<td>.704***</td>
</tr>
<tr>
<td>Alcohol1-Alcohol2</td>
<td>.706***</td>
<td>.696***</td>
<td>.723***</td>
</tr>
<tr>
<td>Depression1-Alcohol2</td>
<td>-.002</td>
<td>.006</td>
<td>-.023</td>
</tr>
<tr>
<td>Alcohol1-Depression2</td>
<td>-.001</td>
<td>-.025</td>
<td>.020</td>
</tr>
</tbody>
</table>

*** \( P < .001 \)

4.6. Effects of Sociodemographic Variables and Health Status

Based on the past empirical studies and theories, four covariates were entered for analysis. Table 4-17 shows the results of fit indices about the differences between freeing paths to differ between groups and constraining paths to be equal across groups. The results show path invariance testing across groups.
Table 4-17. Fit indices of the differences between freeing paths to differ between groups and constraining paths to be equal across groups

<table>
<thead>
<tr>
<th></th>
<th>$\chi^2$/df</th>
<th>CFI</th>
<th>TLI</th>
<th>RMSEA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender – depression1</td>
<td>1445.045/219</td>
<td>.942</td>
<td>.909</td>
<td>.043(.041-.045)</td>
</tr>
<tr>
<td>Gender – alcohol use1</td>
<td>1443.552/219</td>
<td>.942</td>
<td>.909</td>
<td>.043(.041-.045)</td>
</tr>
<tr>
<td>Gender – depression2</td>
<td>1443.683/219</td>
<td>.942</td>
<td>.909</td>
<td>.043(.041-.045)</td>
</tr>
<tr>
<td>Gender – alcohol use2</td>
<td>1444.087/219</td>
<td>.942</td>
<td>.909</td>
<td>.043(.041-.045)</td>
</tr>
<tr>
<td>Education – depression1</td>
<td>1443.651/219</td>
<td>.942</td>
<td>.909</td>
<td>.043(.041-.045)</td>
</tr>
<tr>
<td>Education – alcohol use1</td>
<td>1443.718/219</td>
<td>.942</td>
<td>.909</td>
<td>.043(.041-.045)</td>
</tr>
<tr>
<td>Education – depression2</td>
<td>1444.241/219</td>
<td>.942</td>
<td>.909</td>
<td>.043(.041-.045)</td>
</tr>
<tr>
<td>Education – alcohol use2</td>
<td>1446.515/219</td>
<td>.941</td>
<td>.909</td>
<td>.043(.041-.045)</td>
</tr>
<tr>
<td>Income1 – depression1</td>
<td>1445.745/219</td>
<td>.942</td>
<td>.909</td>
<td>.043(.041-.045)</td>
</tr>
<tr>
<td>Income1 – alcohol use1</td>
<td>1443.879/219</td>
<td>.942</td>
<td>.909</td>
<td>.043(.041-.045)</td>
</tr>
<tr>
<td>Income2 – depression2</td>
<td>1443.829/219</td>
<td>.942</td>
<td>.909</td>
<td>.043(.041-.045)</td>
</tr>
<tr>
<td>Income2 – alcohol use2</td>
<td>1447.810/219</td>
<td>.941</td>
<td>.908</td>
<td>.043(.041-.045)</td>
</tr>
<tr>
<td>Illness1–depression1</td>
<td>1443.689/219</td>
<td>.942</td>
<td>.909</td>
<td>.043(.041-.045)</td>
</tr>
<tr>
<td>Illness1–alcohol use1</td>
<td>1444.676/219</td>
<td>.942</td>
<td>.909</td>
<td>.043(.041-.045)</td>
</tr>
<tr>
<td>Illness2–depression2</td>
<td>1444.229/219</td>
<td>.942</td>
<td>.909</td>
<td>.043(.041-.045)</td>
</tr>
<tr>
<td>Illness2–alcohol use2</td>
<td>1445.159/219</td>
<td>.942</td>
<td>.909</td>
<td>.043(.041-.045)</td>
</tr>
</tbody>
</table>

In the analyzing with the total sample, female gender was positively related to depressive symptoms at measurement points and negatively related to alcohol use at both times. Female gender has consistently been reported as a risk factor for depressive symptoms across the life span (Barefoot et al., 2001; Dendukuri, 2003; & Djernes, 2006).

This study found that higher education is positively related to alcohol use at time 2 in the total sample (Table 4-18). This result is consistent with the previous study (Moore et al., 2005) but it is inconsistent with the previous studies that lower education level was associated with greater quantity of alcohol (Casswell et al., 2003).
Higher income was positively associated with alcohol consumption in the total sample at times 1 and 2. These results are consistent with the previous literature. High average alcohol consumption has been linked to higher income (Moore et al., 2005; Casswell et al., 2003). There is a possible pathway underlying observed patterns of alcohol consumption by income: increases in income may be associated with increased demand for alcohol (Cook & Moore, 2000). However, income showed a significant, yet weak negative relationship with depression at time 1 only. A number of Illnesses showed an positive association with depression at both time1 and 2, but a number of illnesses were not significantly associated with alcohol use at either measurement point.

In terms of retirement status, for employed persons at time 2, female gender was negatively related alcohol use, and higher income was positively related to alcohol use. Gender and alcohol use showed a negative association yet income and alcohol use showed a positive relationship. Illness was positively related to depression among employed people at time2. For retired persons at time 2, gender, education, and illness were negatively related to alcohol use and illness were positively related to depressive symptoms while income was negatively related to depressive symptoms. These results may indicate that different factors might affect the change in depressive symptoms and alcohol use between the retired group and the employed group during mid life to later life.
Table 4-18. Standardized path coefficients of covariates according to samples

<table>
<thead>
<tr>
<th></th>
<th>Total sample-T1</th>
<th>Total sample-T2</th>
<th>Retired T2</th>
<th>Employed-T2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender - depression</td>
<td>.089***</td>
<td>.053**</td>
<td>.085</td>
<td>.106</td>
</tr>
<tr>
<td>- alcohol use</td>
<td>-.222***</td>
<td>-.061***</td>
<td>-.972**</td>
<td>-1.098**</td>
</tr>
<tr>
<td>Education - depression</td>
<td>.035</td>
<td>-.019</td>
<td>-.028</td>
<td>.000</td>
</tr>
<tr>
<td>- alcohol use</td>
<td>-.018</td>
<td>.039**</td>
<td>.059***</td>
<td>.005</td>
</tr>
<tr>
<td>Income†- depression</td>
<td>-.102***</td>
<td>-.037</td>
<td>-.050*</td>
<td>-.048</td>
</tr>
<tr>
<td>- alcohol use</td>
<td>.122***</td>
<td>.036**</td>
<td>.018</td>
<td>.084***</td>
</tr>
<tr>
<td>Illness† - depression</td>
<td>.208***</td>
<td>.129***</td>
<td>.129***</td>
<td>.150***</td>
</tr>
<tr>
<td>- alcohol use</td>
<td>-.017</td>
<td>-.023</td>
<td>-.037*</td>
<td>-.003</td>
</tr>
</tbody>
</table>

† = time-varying covariates, *p<.05, **p<.01, ***p<.001
Table 4-19. Summary of findings

<table>
<thead>
<tr>
<th>No</th>
<th>Hypotheses</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Depressive symptoms and alcohol use will be correlated with each other at midlife.</td>
<td>Not supported. Depressive symptoms was not significantly related to alcohol use at time 1 ($\rho = -0.034$, $p &gt; .05$)</td>
</tr>
<tr>
<td>2</td>
<td>Depressive symptoms and alcohol use will be correlated with each other at early later life.</td>
<td>Not supported. Depressive symptoms was not significantly related to alcohol use ($\rho = -0.294$, $p &gt; .05$)</td>
</tr>
<tr>
<td>3</td>
<td>Alcohol use at midlife will be positively related to alcohol use at early later life.</td>
<td>Supported. Alcohol use was positively associated with alcohol use over time ($\beta = .774$, $p &lt; .001$)</td>
</tr>
<tr>
<td>4</td>
<td>Depressive symptoms at midlife will be positively related to depressive symptoms at early later life.</td>
<td>Supported. Depressive symptoms was positively associated with depressive symptoms over time ($\beta = .618$, $p &lt; .001$)</td>
</tr>
<tr>
<td>5-1</td>
<td>Alcohol use at midlife will be positively related to depressive symptoms at early later life.</td>
<td>Not supported. Depressive symptoms at time 1 was negatively associated with alcohol use at time 2, but it was not significant ($-0.002$, $p &gt; .05$)</td>
</tr>
<tr>
<td>5-2</td>
<td>Depressive symptoms at midlife will be positively related to alcohol use at early later life.</td>
<td>Not supported. Alcohol use at time 1 was negatively related to depressive symptoms at time 2, but it was not significant, too ($-0.081$, $p &gt; .05$)</td>
</tr>
<tr>
<td>6-1</td>
<td>Depressive symptoms at midlife and alcohol use at early later life will be significantly increased only for the employed peers.</td>
<td>Not supported. Retirement status was not significantly moderated the longitudinal relationship between depressive symptoms and alcohol use (P-value of $D\chi^2 &gt; .05$)</td>
</tr>
<tr>
<td>6-2</td>
<td>Alcohol use at midlife and depressive symptoms at early later life will be significantly increased only for the retired peers.</td>
<td>Not supported. Retirement status was not significantly moderated the longitudinal relationship between alcohol use and depressive symptoms (P-value of $D\chi^2 &gt; .05$)</td>
</tr>
</tbody>
</table>
V. Discussion

The purpose of the present study was two fold: 1) to examine the relationships between depressive symptoms and alcohol use among middle-aged and early older adults and 2) to examine whether retirement status moderates the differences in the relationships. For examining these aims, this study utilized a cross-lagged panel design using a longitudinal panel data. The following sections provide major findings and related consideration.

5-1. Summary of Findings

5-1-1. Major descriptive findings

The most interesting result from this study was that there was a decline in depressive symptoms while alcohol consumption was increased from middle-aged to early older adulthood. These results are inconsistent with several previous studies. Previous studies reported that in general, alcohol use tends to decrease in older adulthood while depressive symptoms tend to increase in their later life. Advancing age is often reported as a risk factor for depressive symptoms, perhaps in part because of age-related differences in lifestyle characteristics (Forman-Hoffman, Yankey, Hillis, Wallace, & Wolinsky, 2007) even though comparing results of studies of the association between age and depressive symptoms is difficult because of methodological differences among studies, including differences in age categories used.

The possible reasons of the decreasing alcohol consumption in older adults are that several inherent factors contribute to an overall decrease in alcohol consumption among this population. First, adults age 60 or older are unlikely to be heavy drinkers, simply because the heaviest drinkers tend to succumb at an earlier age from physical
complications, accidents, or other injuries related to excessive alcohol consumption (Mirand & Welte 1996; Mertens et al. 1996). Also, the aging process may result in decreased physical tolerance for alcohol's effects during and after drinking episodes, leading to reduced consumption.

One of the possible reasons of increased drinking in this study may result from the sample selection. The study sample included an early older adult population at follow-up with an age range of 65 to 68 years old. Most previous studies included broader age range of 18 years or older or only older adults of over 65 years. Since the study sample was not very old, differences between this sample and previous studies may reflect the age cut-points and cohort influences on this group.

Furthermore, the mean age of the study sample is related to the usual retirement age, and, thus, during this transitional period changes could occur in their drinking behavior. These results are consistent with the previous studies. Some studies argue that retirement is likely for several reasons to play a central role in the exacerbation of heavy alcohol consumption and drinking problems (Ekerdt et al., 1989; Perreira & Sloan, 2001). First, retirement may be accompanied by the loss of status and feelings of social marginalization that results in psychological stressors with which retirees cope by drinking. Second, retirement may provide drinkers, particularly those who already drink heavily and have drinking problems, with greater opportunities for alcohol consumption because they are subject to fewer role restrictions and thus experience fewer adverse social consequences when they drink. Third, retirees may become involved in subcultures, such as retirement communities, in which highly permissive drinking norms encourage the development of a pattern of increased consumption. The result from this
study may support the possibility of second or third hypotheses because the retirees showed increased drinking while they reported decreased depressive symptoms. As shown in Table 4-2 in chapter 4, retirees’ depressive symptoms decreased from 15.2 to 12.6 while their employed peers showed a change from 15.2 to 13.6 and alcohol frequency and quantity in the retired group at time 2 showed significantly increased patterns \( t (3,024) = -2.390, p<.05 \) and \( t (3,024) = -3.354, p<.001 \) than those of the employed group.

In addition, the sample criteria in this study limited time1 eligibility by employment. It is possible that the people who depressed at time 1 were excluded from the analysis sample because they were already retired or out of the workforce due to their depression at time 1. In the same manner, it is possible that most of the heavy drinkers at time 1 were excluded from the analysis because they were already retired or out of the workforce due to their alcohol problems at time 1. In general, unemployment is positively related to increased or problematic alcohol use (Catalano et al., 1993) among all age groups. The subgroup analysis with the people who were excluded in the analysis due to retirement at time 1 (N = 605) revealed that the retired group at time 1 showed more depression (mean score of CES-D = 16.9) compared to that of the study sample (mean score of CES-D=14.9), and the greater quantity of alcohol (13.9) compared to that of the study sample (12.9). Even though the difference between the study sample and the excluded cases were not big, it is possible that the overall scores of depressive symptoms and alcohol frequency and quantity in the study sample could be under-represented with this restriction of the study sample.
In particular, it is important to note that this study sample seems to have less alcohol consumption compared to those of the previous studies. The study sample showed heavy drinking rates with 14 more drinks per week of 3.7% in additional analysis while the previous community-based studies have reported the prevalence of alcohol misuse or heavy alcohol use with much higher rates of 17% of men and 7% of women when looser criteria such as excessive alcohol consumption are used (Blow et al., 1998). The prevalence of clinically relevant depressive symptoms varied between 7.2% and 49% in previous studies (Djernes, 2006) while this study showed 25.3% at time 1 and 20.7% at time 2. Healthy habits regarding alcohol consumption in the study sample could affect the relationship between depressive symptoms and alcohol use.

Also, the study sample included a highly educated and healthier group than those of other studies. The study sample included the individuals who graduated from Wisconsin high schools in 1957. Thus, the lowest level of education in the study sample was high school graduates. Also, the rates of persons without any illness were 41.6% at time 1 and 20.0% at time 2. These healthy characteristics of the study sample could affect the results.

The correlation between depressive symptoms and alcohol use over time was weak in a negative way. That is, more alcohol use was related to less depressive symptoms over time ($r = -.041, p<.05$) and similarly, more depressive symptoms were related to less alcohol use over time ($r = -.037, p<.05$). This study performed a series stratified analyses to evaluate the combined prevalence of the level of alcohol consumption (i.e., non-drinkers, moderate drinkers, and heavy drinkers) and dichotomized depressive symptoms. A few people (0.9%) were found to have both
depressive symptoms and heavy alcohol use despite alternative classification of the alcohol use and depressive symptoms.

5-1-2. Cross-sectional relationships between depressive symptoms and alcohol use among middle-aged and older adults

Without covariates, depressive symptoms and alcohol use were negatively related at time 1 and time 2 in cross-lagged SEM analysis. However, after controlling the several covariates, no significant relationship was found at time 1 (Hypothesis 1). Also, in their later life, depressive symptoms and alcohol use were related, but after entering covariates, the association was eliminated, too (Hypothesis 2). These were unexpected results in that this study hypothesized positive association between two factors at both time points. The previous cross-sectional studies have reported that alcohol use and depression are associated with each other in adult community samples (Grant, 1995; Grant & Harford, 1995; Kessler et al., 1997; Merikangas et al., 1996; Rohde et al., 1996). Even though some study reported no significant association between depression and alcohol use (Steunenberg et al., 2008), these results were unexpected one.

5-1-3. Longitudinal relationships between depressive symptoms and alcohol use among middle-aged and older adults

The presence of notable stability in depressive symptoms and alcohol use over time was found in the present study, suggesting that alcohol consumption remains largely stable over the period of time in which individuals become eligible to retire. Depressive symptoms at time 1 were positively related to depressive symptoms at time 2 (Hypothesis 3) and, alcohol use at time 1 was positively related to alcohol use at time 2 (Hypothesis
These results reflect that previous level of depressive symptoms/alcohol use may predict depressive symptoms/alcohol use even in their later life.

In addition, in the correlation results, heavier alcohol consumption predicted less depressive symptoms over the course of about ten years in the present study. This may suggest that alcohol consumption is part of a process related to the successful alleviation of depressive symptoms. That is, one possible explanation is that middle-aged and older persons increase their alcohol use while socializing, and that the social contact and interaction accompanying alcohol use has important bearing on their emotional well-being. Further studies are needed to examine this possibility, as well as to explore other variables that may help explain how alcohol use is related to an alleviation of depressive symptoms among middle-aged and early older adults.

Unexpectedly, no significant relationships between depressive symptoms and alcohol use were found in the analysis of the cross-lagged SEM. Depressive symptoms at mid-life did not significantly associated with early later life alcohol use (Hypothesis 5-1), and alcohol use at mid-life did not predict later life depressive symptoms (Hypothesis 5-2). These results are in contrast to other studies which found depressive symptoms to be related to later life alcoholism (Gilman & Abraham, 2001) and vice versa (Mueller et al., 1994). However, these results are also consistent with a number of previous studies that have reported no relationship between the two factors (Wang & Pattern, 2001; Moscato et al., 1997; & Lipton, 1994). For example, Perreira & Sloan (2002) reported that high levels of alcohol consumption at baseline had no statistically significant associations with the onset of either depression or the more general psychiatric problem measure, and Crum et al. (2005) also found that depressive symptoms did not appear to increase risk
for the new onset of alcohol dependence. In addition, some studies reported that
depression may be associated with better outcomes on drinking measures (Kranzler, Del
Boca, & Rounsaville, 1996) such as level of alcohol use, problem drinking, and heavy or
binge drinking (Windle & Miller, 1990; Holahan et al., 2003; Dixit & Crum, 2000).
Overall, the previous evidences have reported conflict results. In particular, in the
longitudinal, lagged designs that appear especially important in understanding the
relationship between depressive symptoms and alcohol use reported inconsistencies in the
relationships (Schutte et al., 1995).

A possible explanation about the nonsignificant association is that alcohol
consumption at these exposure levels may not directly cause depressive symptoms,
although it may affect the prognosis of depressive symptoms.

There are several explanations to interpret these unexpected results.

First, the reason in contrast to other studies which did find a significant
association is the study population this study used. The majority of the previous studies
were based on treatment-seeking patients. Saunders et al. (1991) included only the men
who had a history of alcohol problems in the past, and Graham and Schmidt (1999)
included elderly patients discharged from a psychiatric hospital. The study sample was
derived from general community-dwelling population aged 53-67. Thus, the relationships
could be different between clinic-based studies and community-based studies. The
somewhat weaker effects observed in these community samples compared to clinical
studies that confirm Berkson’s bias, and indicate that substance users with depression
may indeed be more likely to enter treatment (Swedsen & Merikangas, 2000).
Also, as mentioned before, this study restricted the sample with only employed respondents at baseline with excluding retired persons at baseline. Thus, it is possible that seriously depressed and alcoholic persons were excluded in the analyses, and thus, the associations could be weaker than in clinical samples or in samples that don’t restrict by employment status.

Another reason for the unexpected findings could be found in the methodological characteristics the study employed. This study used measures of quantity and frequency to measure alcohol use. The data set did not capture any information regarding problems related to drinking which may further limit our understanding of the relationships between alcohol use and depressive symptoms in this age group while most clinic-based studies, which found a positive relationship, tended to use some clinical criteria of alcohol abuse or dependence based on the DSM-IV. The previous studies have reported that alcohol abuse or dependence tended to have a relationship with depression or depressive symptoms while the level of alcohol consumption or larger amounts per occasion have reported conflicting results (Graham et al., 2007). In addition, the previous studies indicated that results varied according to the methodological characteristics of studies and thus, they argued strongly for the importance of developing equivalent measurement strategies and longitudinal research procedure in alcohol-related studies. Therefore, comparison of the results in this domain has often been limited by an inability to assess the contribution of confounding factors, especially methodological differences, to the variation in results between studies (Fillmore, 1988; Helzer & Canino, 1992).
Another possible explanation is that this study used the longer time lag with 10 years than those in previous studies. It is possible that shorter time lags might reveal more cross-lagged effects.

In addition, one important point for the unexpected results is the healthy characteristics of the study sample. As mentioned, in the previous section, the study sample showed healthy habits in alcohol consumption and further, the sample was highly educated. These sample characteristics could affect the relationship between alcohol use and depressive symptoms.

This study found a positive association between a number of illness and depressive symptoms in middle-aged and early later life. The total household income was also positively related to alcohol use, and female gender was negatively related to alcohol use while it was positively related to depressive symptoms. These effects of covariates on depressive symptoms and alcohol use are consistent with most of previous studies.

In summary, no support for a mutually reinforcing relation between depressive symptoms and alcohol consumption was found in the current study. Higher levels of depressive symptoms did not lead to higher alcohol consumption among early older adults, and alcohol consumption was not associated with later depressive symptoms. Therefore, the nature of the causal processes contributing to the obtained pattern of relationships between depressive symptoms and alcohol consumption cannot be determined by this study.

5-1-3. Group differences in the relationship between depressive symptoms and alcohol use
One of the main questions of the present study was to examine whether retirement status is a moderator in the association between depressive symptoms and alcohol use. In the results, there was no significantly different relationship between the retired group and the employed group. This result is consistent with some studies, and at the same time, it is conflict one with other studies. As mentioned in chapter 2, a number of studies reported that retirement increases the likelihood of increased alcohol consumption and the incidence of drinking problems. Using 6 years of panel data, Perreira and Sloan (2001) found retirement to be associated with increased drinking, particularly among those with a preretirement history of problem drinking. Also, retirees were more likely than their employed age peers to report the onset of periodic heavier drinking and drinking problems.

In terms of the relationship with well-being of retirement, some studies reported that retirees (ranging from 10% to over 30% in specific studies) seem to experience problems or decreased well-being after retirement (Hardy & Quadagno, 1995; Kim & Moen, 2002; Richardson & Kilty, 1991, 1995). The reason is that retirement process can be stressful or depressing, and thus, retirees tend to increase alcohol use while others suggested that retirement gives more time to enjoy drinking, and thus they tend to drink more after retirement.

On the other hand, some studies reported that there is no association between retirement and measures of mental health or psychological distress (Herzog, House, & Morgan, 1991; Ross & Drentea, 1998; Calasanti, 1996; Charles, 2002; Midanik et al., 1995; Reitzes, Mutran, & Fernandez, 1996).
That retirement captured by the WLS in this study did not moderate the relationship between alcohol use and depressive symptom may seem surprising, at least at first glance. But mechanisms underlying the relationships between specific life events, such as retirement occurring to older individuals are complex. For example, persons experiencing adverse health events tend to retire early, and it may be also advised to reduce alcohol consumption or stop it entirely. In this study, it is found that a number of illnesses were negatively related to alcohol use for only retired persons at time 2 while a number of illnesses were positively related to depressive symptoms. It seems to be that having a chronic illness may be related to the decision to retire and to be linked to the decision to decrease alcohol use.

A number of methodological factors may underlie the inconsistency in these research findings. First, inconsistencies may result from the way in which researchers conceptualize retirement and, in particular, from the way they handle the increasing proportion of workers who fail to completely disengage from the labor force while officially retiring and taking retirement benefits. It is possible that according to the environment related to retirement, the results could be different. That is, some retirees are voluntary retired with enough retirement benefits and others are not, thus, their retirement’s effect on their wellbeing could be different from each other. Also, it is important to note that most of the drinking and retirement research conducted to date is based on the operationalization of retirement as a dichotomous phenomenon (i.e., one either is or is not retired) rather than as a process. Herzog et al. (1991) indicate that this binary operationalization does not reflect workers’ actual disengagement experiences.
This study used a binary measure to capture retirement status and may well miss many of the nuances of attitudes and behaviors associated with leaving the work force.

Another possible reason of the unexpected result is that this study used the post-retirement data with unknown date of retirement. The WLS asked the respondents “after the last survey, did you retire?” Considering that the duration between the two surveys was about 10 years, the actual date of their retirement could be ranged from a few months ago to 10 years ago. Thus, the effects of retirement on drinking/depression were highly bounded, and this can limit the association. Future studies examining the effects of retirement on drinking or well-being should ideally control for length of time since retirement.

5-2. Limitation and Strength

Several caveats should be taken into consideration when interpreting the findings of this study. First, it is unclear how well these findings can be generalized to the all middle-aged and early older adult population. Although this study is a population-based study, the sample was limited to a certain population who graduated from Wisconsin high schools in 1957. The majority of the sample were white (75%) and thus, minorities are not well-represented; there is only a handful of African American, Hispanic, or Asian persons in the sample. Future studies are needed to include a nationally representative sample with diverse ethnic groups for understanding the relationships between depressive symptoms and alcohol use.

Second, some methodological limitations remain when interpreting the findings. This study attempted to elaborate the progression of each class of symptoms over time, however, only two time points were available. In order to explore the development of
symptoms over time, multiple time points are more appropriate. Also, the two wave longitudinal panel design with about 10-year interval might be too long for examining the relationships. Future research needs to examine the relationships between depressive symptoms and alcohol use using shorter time lags such as 1 to 3 year.

Another potential problem is that this study relied mostly on information obtained by interview and the reliability of gathering information may underestimate the prevalence of excessive alcohol use or because of the voluntary nature of inclusion of respondent probably some problem drinkers did not wish to participate.

Also, this study used simple measures of alcohol use. Using the measure of frequency and quantity for assessing alcohol consumption, it is limited to confirm the relationship between alcohol use and depressive symptoms. More complex measures such as the alcohol use disorders identification test (World Health Organization, 2001) are needed to include for future study.

Finally, although the time-varying covariates in the SEM analysis were used in the analysis, this study cannot rule out the possibility that the baseline values and the pathways of alcohol-use/depressive symptom rely on some unmeasured variables that accounts for the observed relationships.

Despite those limitations, the study makes contributions in several areas. This study used a comparatively large longitudinal data set. Given the fact that a longitudinal design is the optimal research endeavor when examining the relationship between depressive symptoms and alcohol use, additional follow-ups and waves on these subjects will provide invaluable information.
Second, the sample consisted of cohort data which is the one of the best methods for examining the nature of relationships. The cohort data, which include the persons who graduated Wisconsin high schools in 1957, is one of the best types of samples to use when the study aims are to uncover the relationship between two factors over time. In addition, the WLS obtained data on a number of potentially important correlates of alcohol use and depressive symptoms, not just information on demographic characteristics, but also on health status that may be systematically related to alcohol consumption and depressive symptoms.

Third, this study provided a rigorous test of the cross-lagged relationships using SEM between depressive symptoms and alcohol use among middle-aged and early older adult samples. The majority of the literature heavily relied on cross-sectional study as mentioned in the chapter 2. Even when they used longitudinal data, they tended to use simple methods that are hard to confirm the nature of associations between the two factors. Cross-lagged structural equation model is a best technique to examine the inherent time ordered nature of panel data to address such questions of causal ordering models, and thus, longitudinal, cross-lagged SEM appear especially important in understanding the relationship between depressive symptoms and alcohol use (Finkel, 1995; Campbell & Kenny, 1999).

Fourth, it is important to note that there are few studies examining the relationship between depressive symptoms and alcohol use according to the change from middle-aged to early older adult population. The majority of the previous studies tended to use wide age range with 18 years or older, or they focused on the only older adult populations with
65 years or older and thus, the results may not generalize to the middle-aged and older population.

Finally, this study extended the knowledge about the relationship between depressive symptoms and alcohol use by examining the difference of the relationship between the two factors according to retirement status. The previous studies tended to focus on the etiological relationship between the two factors. Even though there is a need to examine the difference of the relationships by the possibly related contextual factors, there have been few studies until now.

5-3. Implications

Theoretical and Methodological Implications

The current findings have important theoretical and methodological implications. With respect to theory, as a theory of the relationship between depressive symptoms and alcohol use, this study considered the causal hypothesis. Regarding moderating effects of the retirement status, this study also considered a life course perspective and role theory. However, in contrast to the study hypotheses, no significant associations were found between depressive symptoms and alcohol use and also, no significant moderating effects of retirement were found with this sample.

From an etiologic perspective, this study does not resolve questions regarding the casual mechanisms underlying the relationship between depressive symptoms and alcohol use disorders. Thus, the fact that no significant relationships between depressive symptoms and alcohol use were found and the covariates were related to the relationship in analyses may point to the etiological theories which espouse a third factor relationship between the two. There may be common underlying factors, which predispose to an
elevated risk for both alcohol use or abuse and depressive symptoms or depression. These may include genetic factors as well as social or environmental characteristics (Nurnberger et al., 2002; Prescott et al., 2000; Kendler et al., 1993; Dohrenwend et al., 1992; Maier and Merikangas, 1996). Thus, the shared etiology theory may be the most helpful in developing conceptualizations of the relationship in future cohorts of middle-aged and early older adults. The results are inconsistent with several studies, but it is also consistent with some studies. In some population studies, comorbid presentations were heterogeneous (Lynskey, 1998). For example, the study with twin pairs reported that the correlations between substance use problems and depression could be wholly explained by shared genetic factors and individual environmental factors (Tambs et al., 1997). While these findings clearly need to be replicated, they suggest that the association between alcohol dependence and affective disorders may be wholly explained by third factors antecedent. One more reason that is needed to reexamine the relationship between the two factors is that the previous literature reported that while clinic-based investigations indicate high comorbidity for alcohol use disorder and depression, no single model of comorbidity was supported to the exclusion of the others (Steunenberg et al., 2008). Future studies need to include various environmental factors that are closely related to this population either as a mediator or a moderator in order to confirm the relationship between depressive symptoms and alcohol use.

It is important to note that most of the previous studies focused on clinic-based samples with younger age range when they were theorizing the relationship. Thus, selection biases in clinic samples may lead to overestimates of the prevalence of true comorbidity between two factors. This study included only middle-aged and early older
adult population in non-treatment seeking individuals. Thus, it is possible to show no causal relationships due to the difference of sample characteristics. Theoretical models tested with middle-aged and older populations in community are needed to confirm the relationship in future studies.

In addition, the results from this study suggest that when theorizing the relationship between the two factors, standardized and accepted measures of alcohol consumption are needed to confirm the relationship. The previous literature used several different measures of alcohol use. It is possible that the difference of the measure of alcohol use made the different results. For example, this study used frequency and quantity of alcohol use in order to measure the alcohol consumption and depressive symptoms measured by CES-D. Even though these measures are widely used measures when examining the relationship, it is possible that the use of alcohol quantity and frequency items and the CES-D may result in different findings than other standard assessments of alcohol use and depressive symptoms. It is also possible that measures of the problems associated with alcohol use would provide different results. Overall, there is a considerable need for well-designed research that studies the comorbid relations between mental and substance use disorders.

With respect to retirement-related conceptualization, this study considered that retirement status could be a moderator in the relationship between depressive symptoms and alcohol use in middle-aged and early older adults. This hypothesis was derived from several theoretical models that explain the post-retirement process and its association with well-being and alcohol use. The life course perspective was considered as a mean of examining the developmental trend from middle-aged to older adulthood. Even though
the life course perspective offers few concrete hypotheses for variables other than the timing of retirement and level of education, this study suggests that it may serve as a general framework for studying retirement effect by directing researchers’ attention to variables that may be of interest, whereas the concrete hypotheses on mechanisms regarding these variables may be drawn from other theories, such as role theory or continuity theory.

In the results, the retired people in this sample showed decreased depressive symptoms and increased alcohol use over time. Since the trend of alcohol use and depressive symptom patterns showed stability over time, the life course perspective was supported, too. According to the life course perspective, the experience of previous stage in life course affects health or behavior in later life stage. When these results are taken as a whole, it seems that a more integrated theory is needed to account for the current findings and to guide future research in the field of adjustment to aging.

Implications for Practice

The study results reflecting the effects of common risk factors which increase the risks of both alcohol use and depressive symptoms may have implications in practice. If the comorbidity between disorders is explained by common or shared risk factors, then the treatment of either condition will not necessarily have any effect on the other, and thus, treatment for each disorder needs to consider environmental factors which are closely related to either depressive symptoms or alcohol use/abuse. The previous studies suggested that a disruptive family environment is one of the factors related to depressive symptoms and alcohol use, but those factors haven’t been considered for the researches focused on the older adult population. Thus, several issues which are possibly related to
treatment outcomes should be considered when planning treatment of the patients who have depression, alcoholism or depressed alcoholics.

However, even if there is no causal relationship between the onset of alcohol use disorder and depressive disorders, it is still possible that having one disorder may exacerbate symptoms of the other disorder. For example, experiencing depressive symptoms may act to increase both alcohol consumption and alcohol related harm amongst those individuals who have a preexisting vulnerability to alcohol disorder. It may also impair compliance with treatment of alcohol dependence. Even though the causal association was not found, the study sample still included the persons about 1% of respondents with co-occurring two disorders. Thus, treatment services and treatment protocols need to address issues of comorbid psychiatric disorder, and further, all individuals seeking treatment from a given agency should be fully assessed for the presence or absence of a range of psychiatric disorders.

This study found that alcohol consumption increased between the two time points; the sample reported a higher level of alcohol use later in life than earlier in life. These results supported the observations that alcohol consumption patterns remain stable even in later life. The increases in affluence, education, and life expectancy achieved in the 20th century have produced an aging population that has not only increased in numbers but also has more leisure time and disposable income, a more positive attitude toward alcohol, and higher rates of alcohol consumption than in previous generations (Masters, 2003). Therefore, the results suggest the importance of conducting a routine assessment of alcohol problems, and developing and implementing a treatment model targeted to this populations.
This study also found that after retirement, retirees drink more than their employed peers. This result reflects that the experience of retirement may bring other issues that increase the need of alcohol consumption. Older adults may have different biological, psychological, social, emotional and economic needs depending on their work and retirement status. In this context, there is a need for social workers to broaden the way that they define retirement for assessment and advocacy purposes. Providing quality psychiatric care to retirement communities is of major importance to the general public health.

VI. Conclusion and Recommendations for Future Study

This study did not confirm an association between depression and alcohol use in a community-dwelling middle-aged and early older adult population. Furthermore, this study did not find the difference in the relationship between depressive symptoms and alcohol use according to retirement status. These results may reflect that the mechanisms responsible for co-morbidity of depressive symptoms and alcohol-related problems could vary across individuals and represent etiologically distinct subgroups.

Further studies are warranted in older persons to better delineate the relationships among depression, alcohol consumption and alcohol use disorders. Subsequent investigations may evaluate other potential etiologic variables (varied loss events such as loss of spouse or family members, role strain, physical or functional health status, and social support), as well as evaluate the impact of specific socio-demographic variables, including potential age, race or poverty effect. In addition, the processes through which depressive symptoms and drinking behavior are related is likely connected to other
factors not examined in this study, such as life stressors, social resources, alcohol expectancies, coping strategies and the relations among these variables. Replication of these findings in other community samples is imperative.

In order to uncover the difference of the relationship between the two factors by retirement status, it is needed to include more detailed information about retirement process. For example, the effects of voluntary retirement and forced retirement could be different. Future study can examine it according to retirees’ occupation, and it is possible to explore the moderating effect on the relationship with the variables according to the retirement-related decision.

Overall, the results of this study add to the existing literature by suggesting that individuals with depressive symptoms and alcohol use represent a complex group with significant psychosocial consequences and treatment needs.
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