STRESS, SOCIAL SUPPORT, AND SKIN BARRIER RECOVERY

DISSERTATION

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By

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ABSTRACT

How do social relationships get "under our skin" and affect health? This study tested whether the beneficial effects of social support on physiological reactivity could be replicated and extended to a clinically relevant health outcome: recovery of the skin's barrier function after minor disruption. The specific aims of this study were to: 1) Replicate previous research on acute stress-induced delays in skin barrier recovery; 2) Determine whether a social support manipulation before acute psychological stress would speed skin barrier recovery; 3) Investigate the effects of the social support manipulation on cardiovascular and cortisol reactivity; 4) Link stress-related cardiovascular and cortisol responses to skin barrier recovery; and 5) Characterize the time course of cortisol reactivity and recovery in response to acute stress.

Prior to randomization, 85 healthy participants underwent "tape-stripping," a procedure that disrupts normal skin barrier function. Participants were then randomly assigned to a No Stress condition (reading alone), a Stress condition (public speaking), or a Stress + Social Support condition, involving support and encouragement from a laboratory confederate prior to the Stress task. Cardiovascular and cortisol responses were measured before and following the task. Skin barrier recovery was assessed by measuring transepidermal water loss from disrupted skin up to 2 h after tape-stripping.

The acute stressor delayed skin barrier recovery after 2 h. Support provided by a confederate before the acute stressor did not reduce physiological reactivity or speed skin

barrier recovery. While acute stress delayed skin barrier recovery, autonomic and cortisol reactivity were not related to skin barrier recovery. In addition, while cortisol reactivity and recovery are reliable patterns of change, they may not be truly distinct. These findings suggest that acute stress delays skin barrier recovery, and that the physiological mechanisms that explain stress-related delays in skin barrier recovery need additional study in humans.

Dedicated to my parents,

Angel, a nurse and mother,

Teodoro, an engineer and teacher,

for their example, patience, support, and love.

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1. Robles, T. F., Shaffer, V. A., Malarkey, W. B., & Kiecolt-Glaser, J. K. (2006). Positive behaviors during marital conflict: Influences on stress hormones. *Journal of Social and Personal Relationships, 23,* 305-325.

2. Robles, T. F., Glaser, R., & Kiecolt-Glaser, J. K. (2005). Out of balance: A new look at chronic stress, depression, and immunity. *Current Directions in Psychological Science, 14,* 111-115.

3. Glaser, R., Robles, T. F., Sheridan, J., Malarkey, W. B., & Kiecolt-Glaser, J. K. (2003). Mild depressive symptoms are associated with amplified and prolonged inflammatory responses following influenza vaccination in older adults. *Archives of General Psychiatry,* 60, 1009-1014.

4. Robles, T. F., & Kiecolt-Glaser, J. K. (2003). The physiology of marriage: Pathways to health. *Physiology and Behavior, 79,* 409-416.

5. Heffner, K. L., Loving, T. J., Robles, T. F., & Kiecolt-Glaser, J. K. (2003). Examining psychosocial factors related to cancer incidence and progression: In search of the silver lining. *Brain, Behavior, and Immunity, 17(Supplement),* 109-111.

6. Stowell, J. R., McGuire, L., Robles, T. F., & Kiecolt-Glaser, J. K. (2003). Psychoneuroimmunology. In I. B. Weiner (Series Ed.) & A. M. Nezu, C. M. Nezu, &

P. A. Geller (Vol. Eds.), *Comprehensive handbook of psychology: Vol. 9. Health psychology* (p. 75-95). New York: Wiley.

7. Kiecolt-Glaser, J. K., Robles, T. F., Heffner, K. L., Loving, T. J., Glaser, R. (2002). Psycho-oncology and cancer: Psychoneuroimmunology and cancer. *Annals of Oncology, 13,* 165-169.

8. Kiecolt-Glaser, J. K., McGuire, L., Robles, T. F., & Glaser, R. (2002). Emotions, morbidity, and mortality: New perspectives from psychoneuroimmunology. *Annual Review of Psychology, 53,* 83-107.

9. Kiecolt-Glaser, J. K., McGuire, L., Robles, T. F., & Glaser, R. (2002). Psychoneuroimmunology and Psychosomatic Medicine: Back to the future. *Psychosomatic Medicine, 64,* 15-28.

10. Kiecolt-Glaser, J. K., McGuire, L., Robles, T. F., & Glaser, R. (2002). Psychoneuroimmunology: Psychological influences on immune function and health. *Journal of Consulting and Clinical Psychology, 70,* 537-547.

11. Robles, T. (2001). Commentary. In C. D. Ryff & B. H. Singer (Eds.), *Emotion, Social relationships, and health* (pp. 233-242). New York: Oxford University Press.

FIELDS OF STUDY

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CHAPTER 1

INTRODUCTION

General overview

Social relationships have health consequences for humans and a wide variety of animal species (Cacioppo, Berntson, Sheridan, & McClintock, 2000; Uchino, Cacioppo, & Kiecolt-Glaser, 1996). Epidemiological studies suggest that social isolation is a major risk factor for morbidity and mortality, comparable to well-established health risk factors such as cigarette smoking, blood pressure, blood lipids, obesity, and physical activity (House, Landis, & Umberson, 1988). In addition to the mere presence of social relationships, qualitative aspects of social relationships contribute to health, including social influences on behavior, tangible assistance or information and advice during times of need, and emotionally supportive behaviors (Cohen, Underwood, & Gottlieb, 2000). These qualitative aspects are generally referred to as social support.

Evidence for the role of cardiovascular, neuroendocrine, and immune function in mediating the relationship between social relationships and health comes from studies of stress, social contexts, and physiological pathways (Uchino, Cacioppo, & Kiecolt-Glaser, 1996). The common elements in this research are brief and stressful laboratory tasks that induce physiological stress responses, with the presence or absence of a supportive individual as the experimental manipulation (Kamarck, Peterman, & Raynor, 1998). In general, social support provided during laboratory stressors lowers physiological

responses to stress (Thorsteinsson & James, 1999; Uchino, Cacioppo, & Kiecolt-Glaser, 1996); although as discussed in the *Acute stress, social support, and physiology* section, this is a very broad statement that masks the complexity in the research literature.

Acute stress and social support research (frequently referred in this paper as acute stress/social support research) contends that social relationships, through their mere presence or through supportive behaviors, can modify health-relevant physiological responses. The two types of physiological responses studied in this literature are cardiovascular and cortisol reactivity. Cardiovascular reactivity has received most of the attention in acute stress/social support research, which makes sense given its prominent role in studies of stress and risk for cardiovascular disease (T. W. Smith & Ruiz, 2002). A literature search by Linden and colleagues yielded 2381 references for cardiovascular reactivity (Linden, Gerin, & Davidson, 2003). Within the domain of acute stress/social support research, around 20-25 studies have examined cardiovascular reactivity, usually heart rate (HR) changes, and systolic or diastolic blood pressure changes (SBP or DBP). Cortisol reactivity to acute stressors has received increasing attention over the past three decades, with almost 200 studies to date (Dickerson & Kemeny, 2004). Only two are in the acute stress/social support domain (Kirschbaum, Klauer, Filipp, & Hellhammer, 1995; Thorsteinsson, James, & Gregg, 1998). I will review the acute stress/social support research in the *Acute stress, social support, and physiology* section.

Studying mechanisms <u>and</u> linking them back to health

Many researchers propose that stress-related cardiovascular and cortisol responses are health-relevant changes (e.g., Linden et al., 2003, McEwen, 1998). However, at this point there is little direct evidence tying stress-related physiological reactivity to health

outcomes, although evidence is beginning to accumulate for cardiovascular disease-related outcomes (Smith & Ruiz, 2002, Trieber et al., 2003). There are a number of reasons why the evidence base is small for health outcomes, including limited generalizability of laboratory tasks to real-life situations, insufficient attention to interactions between individual predispositions and specific situations, and inadequate conceptualization and measurement of "responses" (Schwartz et al., 2003). The latter point is one focus of this study, discussed in the *Reactivity and recovery* section.

In addition, no studies in this area have directly evaluated a measurable health outcome. This is primarily due to the difficulty in finding outcome variables that change within hours, which is the time frame of most acute laboratory stressor studies. In addition, such transient outcomes must be clinically relevant. For instance, although changes in immune function during acute stress are reliable across a number of studies, the clinical relevance of these short-term changes is still unclear (Kiecolt-Glaser, Cacioppo, Malarkey, & Glaser, 1992).

The dynamics of skin repair and wound healing offer a promising model for studying clinically relevant health outcomes which can change within a limited amount of time. As discussed in the *Wound healing and stress* section, one measure of skin repair – recovery of skin barrier function after barrier disruption, has been successfully used in two studies of acute stress and wound healing by independent laboratory groups (Altemus, Rao, Dhabhar, Ding, & Granstein, 2001; Garg et al., 2001). The major goals of this study are to replicate prior work and translate the moderate to large effects of social support on physiological reactivity to skin barrier recovery. Thus, this study extends previous work on stress and wound healing by including a social support manipulation.

The current study

Following the literature reviews of acute stress/social support research and skin barrier recovery research, I outline the design of the study, drawing on the literature reviews for empirical and theoretical rationale. The specific aims of this study are:

1) To replicate previous research on psychological stressors and delayed skin barrier recovery.
2) To extend previous research on acute psychological stressors and skin barrier recovery by including a social support manipulation.
3) To investigate the effects of social support provided before an acute psychological stressor on cardiovascular and cortisol reactivity and recovery.
4) To link stress-related cardiovascular and cortisol responses to skin barrier recovery.
5) To characterize the time course of cortisol reactivity and recovery in response to acute psychological stress.

Acute stress, social support, and physiology

Acute stress and social support studies take place in a laboratory setting, using acute psychological stressors (e.g., public speaking) which reliably increase cardiovascular and cortisol reactivity (Dickerson & Kemeny, 2004, Krantz & McCeney, 2002). The experimental manipulation in these studies is presence or absence of a supportive individual, before or during the laboratory task (Kamarck et al., 1998). This literature has traditionally focused on three domains: cardiovascular reactivity, cortisol reactivity, and self-reported affect or stress. Rather than providing an exhaustive review of the results from each particular study within each domain, I begin by summarizing extant results based on two meta-analytic reviews. Based on the literature, I then discuss important aspects of the social support manipulations and stress-inducing tasks used in these studies.

Meta-analytic reviews

Results from two meta-analytic reviews suggest that social support manipulations decrease cardiovascular and cortisol reactivity to acute stressors (Thorsteinsson & James, 1999; Uchino et al., 1996). Uchino and colleagues found that studies involving social support manipulations in the laboratory (Gerin, Pieper, Levy, & Pickering, 1992; Kiecolt-Glaser & Greenberg, 1984; Lepore, 1995; Lepore, Mata Allen, & Evans, 1993) showed moderate reductions in cardiovascular reactivity (r = .28). The comparisons in these studies were support vs. non-supportive or alone conditions. In non-supportive conditions, participants performed the stressor task in the presence of a companion who did not provide any explicit support; in alone conditions, participants performed alone. Importantly, these were studies using either supportive experimenters or confederates;

studies involving support provided by familiar others, such as friends or pets, were not included.

A more recent and inclusive meta-analysis found similar results across 22 published studies of acute stress and social support (Thorsteinsson & James, 1999). The authors computed effect sizes by comparing social support conditions (e.g., partner, friend, or confederate) with a control condition (e.g., alone or non-supportive confederate). The control condition in 17 out of 22 studies was an alone condition rather than a non-supportive confederate condition. Social support manipulations showed significant reductions in cardiovascular reactivity with medium to large effect sizes (HR, SBP, DPB; *d*'s from .50 - .60). In addition, two studies (Kirschbaum et al., 1995; Thorsteinsson et al., 1998) found that social support had a large effect in reducing cortisol reactivity ($d = .83$; Thorsteinsson & James, 1999).

In summary, these meta-analytic reviews suggest that manipulating social support in the laboratory is an effective method of reducing cardiovascular and cortisol reactivity during acute stress. As with all meta-analyses, there are limitations to the interpretations of these results. Neither review was all-inclusive; the Uchino review due to the lack of available literature at the time, and the Thorsteinsson review due to practical difficulties (e.g., some reviews did not report appropriate statistics, and one study was excluded because of effect size outliers). In addition, although the review addressed several potential moderators of social support effects, including degree of evaluation potential and type of support expression (verbal vs. silent), the review did not include other important moderating variables, such as friend vs. stranger support, or the psychological parameters of the stressors.

A central role for social-evaluative threat?

Throughout the following sections on social support manipulations and acute stressor properties, I refer to "social-evaluative threat." This term, and variants of it, such as evaluation potential or evaluation apprehension, refers to the degree to which the participant perceives they will be judged by others, particularly in a negative manner (Dickerson & Kemeny, 2004; Kamarck et al., 1998; Lepore, 1998). Many laboratory stressors used to evoke physiological responses contain an element of social-evaluative threat. Moreover, Dickerson and Kemeny propose that social-evaluative threat is a specific situational context which is a potent activator of the hypothalamic-pituitary-adrenal (HPA) axis in humans (Dickerson & Kemeny, 2004). Social-evaluative threat has a role in both the social support and acute stress components of this literature, which will be discussed in each section in greater detail.

Social support manipulations

The social support manipulations used in studies of social support and acute stress responses vary considerably. Such heterogeneity is problematic because comparing findings between studies becomes difficult. For instance, is a study where support is provided by a stranger during a stressor comparable to a study where support is provided by a friend before a stressor? While comparisons between studies are difficult, the heterogeneity in acute stress/social support studies also provides important information on different parameters of social support (source of support, types of behaviors) and their differential influences on physiological reactivity. Major sources of heterogeneity are reviewed in this section.

Friends vs. strangers: The source of support

One of the major sources of heterogeneity between studies is the supportive companion. Most research used supportive confederates or experimenters only (Anthony & O'Brien, 1999; Gallo, Smith, & Kircher, 2000; Gerin, Pieper, Levy, & Pickering, 1992; Glynn, Christenfeld, & Gerin, 1999; Hilmert, Kulik, & Christenfeld, 2002; Kiecolt-Glaser & Greenberg, 1984; Lepore, 1995; Lepore, Mata Allen, & Evans, 1993; Sheffield & Carroll, 1996; Thorsteinsson, James, & Gregg, 1998; Uchino & Garvey, 1997), while others used a participant's close friends only (Gerin, Milner, Chawla, & Pickering, 1995; Kamarck, Annunziato, & Amateau, 1995; Kamarck, Manuck, & Jennings, 1990; Kors, Linden, & Gerin, 1997; Uno, Uchino, & Smith, 2002), random assignment of participants to either a stranger or friend condition (Edens, Larkin, & Abel, 1992; Fontana, Diegnan, Villeneuve, & Lepore, 1999; Kirschbaum, Klauer, Filipp, & Hellhammer, 1995; Sheffield & Carroll, 1994; Snydersmith & Cacioppo, 1992), or random assignment to a pet or familiar human companions condition (K. Allen, Blascovich, & Mendes, 2002; K. M. Allen, Blascovich, Tomaka, & Kelsey, 1991). Most studies found that social support manipulations reduced cardiovascular reactivity. The only types of studies where support was unequivocally related to reducing reactivity were studies involving close friends as supportive companions. Two of the 11 studies involving confederates or strangers had null findings, and two of the five studies involving both confederates and friends had null findings. The remainder of this section reviews studies comparing confederates to friends.

Performing mental arithmetic and/or other cognitive tasks in the presence of a friend lowered reactivity compared to in the presence of a stranger (Edens et al., 1992;

Snydersmith & Cacioppo, 1992). Sheffield and Carroll (1994) found no significant differences between participants performing mental arithmetic alone, in the presence of a stranger, or in the presence of a friend. However, the mental arithmetic stressor involved in this study involved written rather than spoken responses, which may have diminished the threatening and challenging aspects of the task.

More recent work by Fontana and colleagues (1999) found that SBP and HR reactivity was greater in participants performing mental arithmetic and a speech alone compared to participants performing in the presence of a nonevaluative stranger or friend. Cardiovascular reactivity did not differ between participants performing in the presence of a stranger or friend. The results and methodology of this study differ from earlier work (Edens et al., 1992), which was attributed to the less threatening social climate in the Edens study. In addition, the companion spent more time (about 3 min longer) with the participant prior to the stressful task in the Fontana study compared to the Snydersmith study.

The final study that included support from either a friend or stranger compared cortisol responses among participants who received support from an opposite-sex significant other (partner condition), an opposite-sex stranger confederate (stranger condition), or no support (no support condition) 10 min prior to the Trier Social Stress Test (TSST, a speech and math stressor; Kirschbaum et al., 1995). Men showed elevated cortisol reactivity in the no support and stranger support conditions, and lower cortisol reactivity in the partner support condition. In contrast, women showed a trend for elevated cortisol reactivity in the partner condition compared to the stranger condition.

In summary, early work found that performing in the presence of a friend decreased reactivity compared to performing in the presence of a stranger. In contrast, more recent work suggested significantly reduced reactivity by social support regardless of the companion. The difference is likely due to methodological differences described above, including decreased threat and evaluation potential in the Edens et al. (1992) and Sheffield and Carroll (1994) studies. Thus, it is unclear whether friends lower cardiovascular reactivity compared to strangers. Finally, the Kirschbaum study suggests that gender interacted with the type of supportive companion (stranger vs. partner) in reducing cortisol reactivity.

Active vs. passive support: Supportive behaviors

Lepore differentiated acute stress/social support studies by the degree of active vs. passive support (Lepore, 1998). Active support consists of the supportive friend or confederate making supportive comments or gestures. Passive support consists of the supportive friend or confederate providing support through their mere presence. In these studies, companions are typically prevented from communicating with the participant. In general, the passive support studies are equivocal compared to the active support studies. With the exception of two studies (Anthony & O'Brien, 1999; Sheffield & Carroll, 1996), the vast majority of studies using active support found effects in the direction of social support lowering cardiovascular reactivity (Christenfeld et al., 1997; Gerin et al., 1992; Glynn et al., 1999; Hilmert et al., 2002; Kiecolt-Glaser & Greenberg, 1984; Lepore, 1995; Thorsteinsson et al., 1998) and cortisol reactivity (Kirschbaum et al., 1995; Thorsteinsson et al., 1998). By contrast, four studies using passive support (K. Allen et al., 2002; K. M. Allen et al., 1991; Edens et al., 1992; Sheffield & Carroll, 1994) found

that social support did not reduce reactivity, compared with seven studies finding significant reductions in cardiovascular reactivity (Fontana et al., 1999; Gerin et al., 1995; Kamarck et al., 1995; Kamarck et al., 1990; Kors et al., 1997; Snydersmith & Cacioppo, 1992; Uchino & Garvey, 1997).

Another way of viewing active vs. passive support is in terms of verbal vs. silent support. Verbal support produced larger effect sizes (*d*'s from .55 - .64) in lowering cardiovascular reactivity compared to silent support (*d*'s from .44 - .60) for all measures of cardiovascular reactivity. Thus, verbal or active support generally had larger and more consistent effects on cardiovascular and cortisol reactivity. At the same time, some researchers have preferred the use of passive support over active support, citing the cardiovascular response-enhancing effect of active support reported in some comparisons. A meta-analysis of Kamarck and colleagues' published and unpublished studies from their laboratory found that active support enhanced cardiovascular responding (*d*'s from .21 - .27; Kamarck et al., 1998). Anthony and O'Brien (Study 1, 1999) found lower DBP reactivity in participants giving a speech alone compared to participants giving a speech in the presence of a supportive confederate. Moreover, in Study 2, increased verbal support was related to increased SBP reactivity. Thus, although active support generally reduced cardiovascular and cortisol reactivity, there may be boundary conditions on its effects. In addition, other important factors may play a role in these effects, particularly social-evaluative threat.

Social-evaluative threat related to support

Another factor that influences support provided by companions is social-evaluative threat. Most studies manipulated social-evaluative threat using the social

support manipulation itself (Anthony & O'Brien, 1999; Christenfeld et al., 1997; Fontana et al., 1999; Glynn et al., 1999; Hilmert et al., 2002; Lepore, 1995; Lepore et al., 1993; Sheffield & Carroll, 1994; Snydersmith & Cacioppo, 1992; Thorsteinsson et al., 1998). These studies assumed that non-supportive or unfamiliar (stranger) companions have more evaluation potential than supportive companions. Only one study explicitly tested this assumption by measuring perceived evaluation in participants, and found no differences in participants' ratings of perceived evaluation between friend and stranger companions (Snydersmith & Cacioppo, 1992).

Other studies limited evaluation potential in passive social support studies by having supportive companions wear headphones playing white noise and read magazines (Edens et al., 1992; Fontana et al., 1999; Kamarck et al., 1995; Kamarck et al., 1990), or physically preventing supportive others from seeing the participants' performance (Kors et al., 1997). Most of these studies compared a support condition to an alone condition, a design that did not allow for contrasting the reduced evaluation potential conditions to an enhanced evaluation potential condition. Thus, these studies cannot examine the effects of social-evaluative threat when a companion is present.

The one exception is a study in which participants brought a same-sex friend to provide support during a 5 min mental arithmetic task (Kors et al., 1997). Friends could either see the participant's questions and responses (evaluative condition) or were physically prevented from doing so (non-evaluative condition). Non-evaluated participants showed less SBP reactivity compared to participants performing the task alone. Moreover, evaluated participants reported feeling more evaluated and less comfortable compared to non-evaluated participants. Although these findings suggest that

evaluation potential drove differences in SBP reactivity, perceptions of support and evaluation were not significantly correlated with cardiovascular measurements. At the same time, these results generally suggested that minimizing a supportive companion's evaluation potential reduces cardiovascular reactivity.

For the most part, the only evidence that social-evaluative threat was actually being manipulated by inclusion of a supportive vs. nonsupportive companion or a friend vs. a stranger comes from cardiovascular reactivity measures. Indeed, the Thorsteinsson meta-analysis found larger effects for social support reducing cardiovascular reactivity when support was characterized by low evaluation potential (*d*'s from .45 to .82) compared to high evaluation potential (*d*'s from .52 to .59; Thorsteinsson & James, 1999). Self-reported perceived evaluation did not differ between friends or strangers or evaluative vs. non-evaluative conditions in two studies. Therefore, when social-evaluative threat was operationalized by cardiovascular reactivity, reducing a supportive companion's evaluation potential reduced social-evaluative threat. However, when social-evaluative threat was operationalized by self-report measures, the effect of reducing a supportive companion's evaluation potential on social-evaluative threat was not consistent across studies.

Acute stressors

Laboratory stressors reliably increase cardiovascular and cortisol reactivity (Dickerson & Kemeny, 2004; Krantz & McCeney, 2002). Cardiovascular responses to acute laboratory stressors are observed soon after onset of the stressor, whereas cortisol responses peak at 21 – 40 minutes after onset. One meta-analytic review suggests that the time course of cortisol responses to acute laboratory stressors is homogeneous across

studies (Dickerson & Kemeny, 2004). This section reviews the characteristics of different types of stressors and the role of social-evaluative threat.

Type and duration

In general, three types of laboratory stressors have been used in acute stress/social support studies, with considerable variation within each category. Studies using public speaking have included speeches that vary in length (1 min to 6 min), composition of audience (supportive companion, experimenter, video recorder), and speech content (role play, supporting or defending a position; Anthony & O'Brien, 1999; Christenfeld et al., 1997; Gallo et al., 2000; Gerin et al., 1992; Glynn et al., 1999; Hilmert et al., 2002; Lepore, 1995; Lepore et al., 1993; Sheffield & Carroll, 1996; Uchino & Garvey, 1997; Uno et al., 2002). Studies using cognitive tasks vary in the type of task, such as mental arithmetic and visual-motor tasks like videogames or mirror tracing, and in the number of tasks used (usually one or two; K. M. Allen et al., 1991; Edens et al., 1992; Gerin et al., 1995; Kamarck et al., 1995; Kamarck et al., 1990; Kiecolt-Glaser & Greenberg, 1984; Kors et al., 1997; Sheffield & Carroll, 1994; Snydersmith & Cacioppo, 1992; Thorsteinsson et al., 1998). One study used a cold pressor task and a mental arithmetic task (K. Allen et al., 2002). Finally, several studies combined a public speaking task and a cognitive task (mental arithmetic), such as the Trier Social Stress Test (TSST; Fontana et al., 1999; Kirschbaum et al., 1995).

Additional factors that should be noted are stressor duration, difficulty, and performance elements. Duration of these tasks ranged from less than 5 min to 10-15 min. Although not much is known about the duration of a task and its relationship to cardiovascular reactivity, it appears that length of tasks is not related to cortisol response

magnitude (Dickerson & Kemeny, 2004). With respect to difficulty, Thorsteinsson and colleagues found that studies that used speech delivery tasks were related to higher average effect sizes of social support lowering cardiovascular reactivity compared to studies using mental arithmetic (Thorsteinsson & James, 1999). As described below, the larger effects of public speaking tasks may also be related to increased social-evaluative threat. Direct manipulations of task difficulty are related to social support effects, with social support reducing cardiovascular reactivity under conditions of high task load (Kiecolt-Glaser & Greenberg, 1984) and high stress (Gerin et al., 1995).

Finally, most studies involved some element of verbal and/or public performance; that is, the participants produced verbal responses and were viewed by others or were told they were recorded. The importance of this element is illustrated by a negative finding from Sheffield and Carroll (1994). In this study, participants performed mental arithmetic and a vocabulary task in the presence or absence of a supportive friend or stranger. The social support manipulation had no effect on cardiovascular reactivity. However, participants in the study did not produce verbal responses. Instead, they filled in their responses on an answer sheet. The non-significant findings in this study suggest that the tasks were not sufficiently difficult or did not involve evaluative threat. Specifically, supportive companions were not able to observe the participant's actual performance, unlike other studies involving verbally produced responses to the mental arithmetic task.

Social-evaluative threat related to the stressor

Almost every study had some element of social-evaluative threat through the presence of an audience, experimenter, supportive person, or video- or audio-recording device during the acute stressor. In some studies, the evaluative aspect of the stressor was

fixed and did not vary across participants, including studies that used audiences, harassing audiences, or recording equipment (Gerin et al., 1992; Kirschbaum et al., 1995; Uchino & Garvey, 1997). Beyond the general conclusion that stressors containing an element of social-evaluative threat increase cardiovascular and cortisol reactivity, little can be said about how social threat interacts with social support without the manipulation of threat.

Several studies directly manipulated the level of social-evaluative threat in the experiment by manipulating aspects of the experimenter. For instance, social support was related to lower blood pressure reactivity compared to no support, but only when the experimenter was actually present; no effects of support were observed when the experimenter was absent (Hilmert et al., 2002). Other work manipulated experimenter behavior to be more or less evaluative (Gerin et al., 1995; Kamarck et al., 1995). Kamarck and colleagues manipulated social threat through two conditions involving the experimenter (Kamarck et al., 1995). The high threat condition consisted of elevated experimenter status (i.e., lab coat, formal attire), loud and impatient tone of voice, prompts to increase speed and accuracy of performance, and constant reminders of the experimenter's presence during tasks. The low threat condition consisted of informal experimenter status (i.e., casual dress, using first name), steady tone of voice, and no reminders of the experimenter's presence during tasks. Participants accompanied by a friend showed lower cardiovascular reactivity compared to participants who were alone; this effect was only significant in the high threat condition.

Similar results were found in another study, where evaluative threat was manipulated by experimenter harassment (Gerin et al., 1995). These results suggested

that social support may be more effective in lowering cardiovascular reactivity when the stressor involves high levels of social-evaluative threat. These results are qualified by two important points. First, the Kamarck meta-analysis of published and unpublished studies from their laboratory found that social support had large effects on reducing cardiovascular reactivity when the experimenter was a male professor (*d*'s from -.53 to -.60). However, in studies that used female research assistants with high threat (lab coats, formal titles, etc.), the effects of social support on cardiovascular reactivity were small and non-significant (*d*'s from .10 to .16), suggesting important gender differences in the source of threat. Finally, it is unclear if experimenter harassment in the Gerin study included an element of social-evaluative threat. Harassment included prompts to perform faster, but did not include statements explicitly suggesting the participant was being evaluated.

Studies manipulating social-evaluative threat suggest that including social threat in laboratory stress protocols and acute stress/social stress studies is informative. These results and the data from studies of evaluation potential in supportive companions generally support the use of social and interpersonal stressors in physiological reactivity research (Linden, Rutledge, & Con, 1998). Importantly, including social threat increases generalizability of laboratory scenarios to real-life situations. It is more likely that a participant will eventually encounter a real-life situation involving public speaking than a cold pressor or Stroop task.

Summary

In general, social support manipulations reduce cardiovascular and cortisol reactivity, although it is unclear whether using friends or strangers is more effective in

producing these effects. Extant research suggests that trained confederates can provide similar support as friends, as rated by participants and objective observers. Active support is more consistently related to reducing reactivity, although one advantage of passive support is that it reduces social-evaluative threat from the supportive companion. While minimizing evaluation potential from the companion is important, maximizing threat from the stressful task accentuates the effects of social support. Thus, tasks that involve verbal performance and social-evaluative threat will be especially fruitful in new research.

Reactivity and recovery

Most acute stress studies measure immediate physiological responses to stress (reactivity) and neglect recovery to baseline levels (Linden, Earle, Gerin, & Christenfeld, 1997). In their review of theoretical assumptions and empirical research on reactivity and recovery, Linden and colleagues (1997) defined reactivity as simultaneous physiological measurement of stress responses, and recovery as measurement during a post-stress rest period. Reactivity informs us as to the threshold, rise-time, and peak responses, whereas recovery informs us as to the peak responses, the degree to which these elevations persist after the stressor has ended, and the return to baseline after the stressor has ended (Linden, Earle, Gerin, & Christenfeld, 1997).

Theoretical and psychometric issues

Linden and colleagues (1997) noted several reliability and construct validity issues in studying recovery. Psychophysiological measurements should be psychometrically similar to psychological measurements, demonstrating good consistency and stability across time and settings (Kamarck & Lovallo, 2003). Recent

research examining cardiovascular recovery suggests that measures of recovery are internally consistent across different tasks (Christenfeld, Glynn, & Gerin, 2000; Rutledge, Linden, & Paul, 2000) and moderately stable across tasks and measurement occasions, even across several years (Rutledge et al., 2000).

The low generalizability of laboratory stressors is a major limitation to studying recovery. Ambulatory measures provide insight into physiological processes during real-life stressors, and recent work found that cardiovascular recovery after an acute laboratory stressor predicted daily ambulatory blood pressure readings above both baseline and cardiovascular reactivity (Rutledge et al., 2000). Thus, physiological recovery assessed in the lab may generalize to ambulatory measures. Moreover, in the same study, only cardiovascular baseline and recovery measures significantly predicted daily ambulatory blood pressure readings when entered together with cardiovascular reactivity (Rutledge et al., 2000). Recovery predicted an additional 3% of the variance in ambulatory cardiovascular measures above both baseline and reactivity levels. This suggests that recovery measures may have incremental validity over reactivity measures.

Design and statistical issues

Several considerations must be made when designing a study to examine physiological recovery. Stressors that allow for immediate recovery may not provide sufficient variability to examine individual differences in recovery. In addition, researchers must be familiar with the typical response curve and half-life of physiological measures. For instance, heart rate and blood pressure changes can occur in less than one minute, whereas cortisol responses take 20-30 min to peak following stressor onset and have a half-life of over an hour (Dickerson & Kemeny, 2004, Kirschbaum &

Hellhammer, 1994). By comparison, when looking at recovery, the Dickerson meta-analysis found that cortisol levels were twice as high at 0-20 min after stressor termination compared to 21-40 min after termination, returning to baseline around 41-60 minutes (Dickerson & Kemeny, 2004). Thus, the nature of the variable will determine the appropriate sampling interval. In addition, "baseline" must be explicitly defined, and this can vary significantly across studies. For instance, one can obtain a resting baseline the same day as the stressor or the day before the stressor.

Recovery also presents issues for data reduction and statistical analysis (Linden et al., 1997). Change scores often have low reliability, though there are appropriate measures which can be taken to increase reliability, such as multiple measurement occasions (Kamarck, 1992). Repeated measures, analysis of covariance, and residualized change scores are another possibility (Llabre, Spitzer, Saab, Ironson, & Schneiderman, 1991). Other options include time to recovery (e.g., participant X took 30 sec to return to baseline levels), although this will often involve defining an arbitrary recovery criterion, and ignores slope of the recovery curve. Area under the curve controls for steepness of the decline, but requires a frequent sampling interval and is likely influenced by initial levels of reactivity. The most widely used method is assessing post-stress levels at arbitrary intervals, but the number and timing of intervals varies. Other measures of recovery include change from post-stress levels at arbitrary intervals to baseline, or change from stress levels to post-stress levels at arbitrary intervals, although both suffer from low reliability, and the latter is influenced by reactivity.

Curve-fitting approaches use all available data and allow for estimating independent parameters of change. Two studies in the literature have used different

methods of curve estimation. Christenfeld and colleagues (Christenfeld, Glynn, & Gerin, 2000) found that applying a three parameter model (level of drop from stressor to post-task, time to drop from stressor to post-task levels, actual post-task recovery level) provided reliable indices of recovery that also showed incremental validity over cardiovascular reactivity. These parameters also exhibited better reliability and incremental validity compared to traditional measures, including time to recovery, post-stress levels at an arbitrary interval, and post-stress levels at an arbitrary level minus baseline change scores. Llabre and colleagues used piecewise latent growth curve modeling to estimate baseline, reactivity, and recovery patterns of SBP (Llabre, Spitzer, Saab, & Schneiderman, 2001). This method controls for measurement error by estimating latent parameters of change, and allows reactivity and recovery to operate as predictors and outcomes of other variables.

Summary

The distinction between reactivity and recovery has important theoretical significance, but several reviews suggest these distinctions have not received sufficient empirical attention (Dickerson & Kemeny, 2004; Linden et al., 1997; Linden et al., 2003). The lack of empirical attention to physiological recovery is unfortunate, as the inability to physiologically "unwind" after exposure to stress is a point of strong emphasis in theoretical formulations of the deleterious consequences of chronic stress (Frankenhaeuser, 1986); McEwen, 1998). For instance, McEwen cited the failure to shut off physiological responses following termination of a stressor as a potential contributor to the long-term effects of stress responses (McEwen, 1998). Indeed, cardiovascular

recovery, notably diastolic recovery may be related to coronary risk factors and increased prevalence of hypertension (Hocking Schuler & O'Brien, 1997).

Methods are now available to reliably measure and statistically capture reactivity and recovery, and some work suggests these components have generalizability beyond the laboratory. While these separate components of physiological change pose important design and statistical issues for researchers, statistical techniques exist for addressing these issues. Thus, more study is needed in the acute stress arena, and certainly in acute stress/social support research.

Some evidence suggests that social support influences cardiovascular and cortisol recovery. For example, Allen and colleagues conceptualized cardiovascular recovery as reaching or falling below a threshold of half the participant's change from baseline to the first minute of the task (K. Allen et al., 2002). Presence of spouses slowed cardiovascular recovery compared to other conditions, and pet owners showed faster recovery in the presence or absence of their pets. Both the Kirschbaum and Thorsteinsson social support studies sampled cortisol up to 50 min after the acute stressor, thus providing the basis for speculating reactivity and recovery differences related to social support (Kirschbaum et al., 1995; Thorsteinsson et al., 1998). Visual inspection of the cortisol levels reported in the Kirschbaum study suggested that for men, social support was related to reduced reactivity, with unclear effects on recovery (Kirschbaum et al., 1995). For women, peak cortisol levels appeared later (approximately 40 min after stress) compared to men, and the reduced reactivity effects of stranger support were more consistently observed in the recovery period. The Thorsteinsson and James study did not find significant differences

between the support and no-support groups in relation to recovery levels of cortisol at 50 min (Thorsteinsson & James, 1999).

Wound healing and stress

Establishing that physiological alterations mediate the relationship between psychosocial factors (i.e., stress and social contexts) and health outcomes ultimately requires measuring objective health outcomes. Beyond physiological measures, assessing objective health outcomes has not been incorporated in acute stress and social support research. This study addressed this shortcoming by including a health outcome that is clinically relevant to a number of skin disorders (e.g., allergic contact dermatitis and psoriasis) and wound healing: skin barrier recovery.

The primary function of the skin is to act as a physical barrier, protecting the body from the external environment. External damage through dry skin or physical wounding disrupts skin barrier function. This section reviews the biology of the skin and skin barrier recovery after disruption, followed by research on stress and skin barrier recovery, and the role of glucocorticoids.

Biology of the skin barrier

Skin structure and layers

The skin is composed of two major layers, the inner dermis layer and the outer epidermis layer (Champion, Rook, Wilkinson, & Ebling, 1998). The dermis layer contains connective tissue, blood vessels, nerves, lymphatics, the hair follicle bulb and hair-associated smooth muscle, sebaceous glands, sweat glands, and Pacinian and Meissner's corpuscles. The epidermis is 95% keratinocytes (skin cells composed of the

protein keratin), which begin attached to the basement membrane, a protein and filament complex that joins the dermis and epidermis, and gradually migrate to the skin surface across four distinct layers, in order of decreasing distance from the outer environment: the stratum basale, stratum spinosum, stratum granulosum, and stratum corneum.

The stratum corneum is the primary protective barrier that prevents evaporative water loss and serves as a barrier against pathogens (particularly the lower portion of the layer). This layer is composed of flattened and tightly-packed cells called corneocytes, which have lost nuclei and cytoplasmic organelles, which are fused together with other corneocytes (Champion, Rook, Wilkinson, & Ebling, 1998). These cells are filled with keratin filaments and contain little water content. The outermost cells (squamous cells) are continuously sloughed off the skin surface.

The stratum corneum is composed of two major components in a "bricks and mortar" fashion (Elias, 2005). The "bricks" are the corneocytes, which are embedded in a lipid-enriched extracellular matrix described as the "mortar." Stratum corneum tissue is now viewed as an active and dynamic tissue layer, in contrast to more traditional views of the layer as a static end-product of epidermal differentiation. Metabolic activity in this tissue layer promotes the formation of the lipid matrix that forms the "mortar," also known as the lamellar membranes. In general, the epidermis is an active site of lipid synthesis, particularly in the stratum granulosum. The skin generates lipids at a rate of 100 mg/day (Champion, Rook, Wilkinson, & Ebling, 1998).

Stratum corneum barrier function is dependent on the number of cell layers, and the amount and type of intercellular lipids (Harvell & Maibach, 1994). For instance, sun-exposed regions of the skin have thicker stratum corneum tissue compared to sun-

protected regions (Huzaira, Ruis, Rajadhyaksha, Anderson, & Gonzalez, 2001). Lipid content in the wrist is much larger compared to the forearm region (Norlen, Nicander, Rozell, Ollmar, & Forslind, 1999), and higher in the forehead compared to the chest and upper back (Yoshikawa et al., 1994). Lipid content decreases with age (Rogers, Harding, Mayo, Banks, & Rawlings, 1996), and corneocyte surface area increases with age (Fenske & Lober, 1986). Moreover, there are seasonal variations in skin barrier function and lipid composition, with declines during winter compared to summer (Rogers et al., 1996). Stratum corneum in Black individuals shows more cell layers, intracellular cohesion, and higher lipid content compared to White individuals, though stratum corneum thickness is similar between Black and White persons (Champion et al., 1998). In general, darker skin is more resistant to barrier disruption and recovers more quickly compared to lighter skin (Reed, Ghadially, & Elias, 1995).

Skin barrier recovery

Any damage to the skin will perturb skin barrier function, reducing the ability of the skin to serve as a barrier against water loss and pathogens. In addition to dry skin and physical wounds, solvents (e.g., acetone) or detergents (e.g., sodium lauryl sulfate) will result in shedding of the stratum corneum and subsequent skin barrier disruption. Moreover, removal of the stratum corneum will disrupt skin barrier function.

Tape-stripping. Tape-stripping is a common method used to disrupt the skin barrier in dermatological research (Bashir, Chew, Anigbogu, Dreher, & Maibach, 2001). The procedure, described in further detail in the *Methods* section, involves removal of corneocytes in the stratum corneum through repeated application and removal of cellophane tape. This method of disrupting the skin barrier is less invasive compared to

other methods used in wound healing research, such as skin punch biopsy (Kiecolt-Glaser, Marucha, Malarkey, Mercado, & Glaser, 1995), mucosal punch biopsy (Marucha, Kiecolt-Glaser, & Favagehi, 1998), or suction blister methods (Glaser et al., 1999).

Following barrier disruption, measures of transepidermal water loss (TEWL) assess skin barrier function. TEWL indicates the skin's ability to prevent water loss from the interior layers. Thus, increased TEWL reflects decreased barrier function. Decreasing TEWL following perturbation indicates barrier recovery. TEWL measures reflect the following: endogenous skin barrier function, hydration state of the stratum corneum, and sweating (Harvell & Maibach, 1994). Total recovery of the skin barrier after disruption takes place over several days. However, estimates of recovery vary across studies, in part because studies vary in the degree of stratum corneum removal (see *Methods: Physiological measures: Tape-stripping*).

Factors affecting skin barrier recovery. A number of factors affect recovery of the skin barrier following disruption. Given the differences in stratum corneum structure (lipid composition, thickness, etc.) among different anatomical locations, it follows that recovery after disruption also differs by anatomical location. For instance, although the forehead was most susceptible to disruption, it showed the fastest recovery compared to the back, abdomen, lower leg, and ventral forearm (which showed similar recovery rates; Fluhr et al., 2002). Older persons showed increased susceptibility to disruption, with fewer strippings needed to achieve the same TEWL compared to younger adults, and slower barrier recovery compared to younger adults (Ghadially, Brown, Sequeira-Martin, Feingold, & Elias, 1995). Individuals with darker skin showed increased resistance to barrier disruption compared to individuals with lighter skin, as mentioned above (Reed et

al., 1995). Lipid-rich areas, such as the forehead and back, appear to show quicker recovery compared to areas with relatively lower lipid levels (Fluhr et al., 2002).

Barrier recovery is lower during the late evening (20:00 – 23:00 h; Denda & Tsuchiya, 2000). This is not due to diurnal variations in skin temperature, but rather, diurnal variations in basal TEWL, with elevated forearm TEWL during the late evening hours (after 20:00 h; Yosipovitch et al., 1998). Recovery of the skin barrier also varies by type of skin disorder. For instance, patients with psoriasis are more susceptible to barrier disruption and show slower recovery compared to normal controls (Tagami & Yoshikuni, 1985), while patients with atopic dermatitis show no differences in susceptibility to disruption and barrier recovery speed compared to normal controls (Tanaka, Zhen, & Tagami, 1997).

Specific mechanisms. Recovery of the skin barrier involves restoring extracellular lipids to the stratum corneum and organization of new lipids into lamellar unit membrane structures (Elias, 2005). This is a biphasic response, with an early increase in cholesterol and fatty acid synthesis within the first 2 - 4 h after disruption of the barrier, and ceramide (a type of lipid) synthesis which peaks later. Lipid synthesis increases because of increased enzyme activity and transcription of enzyme messenger RNA (mRNA) following damage. In addition, preformed pools of lamellar tissues in the stratum granulosum are secreted into the stratum corneum immediately during perturbation. Synthesis and secretion of lamellar tissue continues for several hours. Thus, in the acute phase of barrier recovery, cholesterol and fatty acids are synthesized, and lamellar bodies are formed and secreted into the stratum corneum. In the chronic phase of barrier

recovery, ceramides, epidermal DNA, and a lipid-processing enzyme are synthesized (Proksch, Feingold, Mao-Qiang, & Elias, 1991).

A wide variety of signaling molecules are present in the epidermis, including ions (e.g., calcium or potassium), cytokines, and growth factors. All three are important in regulating lipid synthesis, while cytokines and growth factors promote inflammation, which is a key component of wound healing. This section discusses the role of cytokine signaling. Under normal conditions, interleukin-1α (IL-1α) and the IL-1 receptor antagonist (IL-1ra) are expressed in large amounts in the epidermis (Elias, Wood, & Feingold, 1997). Following barrier disruption, tumor necrosis factor - α (TNF-α) and other growth factor mRNA transcription increases in keratinocytes. This is followed by delayed release of preformed pools of IL-1α and subsequent IL-1α increases (Reilly & Green, 1999), and increased synthesis of other proinflammatory cytokine mRNA, including IL-1β, IL-8, IL-10, and interferon gamma. Secretion of these cytokines and growth factors also promote the expression of adhesion molecules, further promoting the process of inflammation (Elias, 2005; Nickoloff & Naidu, 1994).

Overall, barrier disruption results in processes that promote lamellar body secretion, lipid synthesis, and DNA synthesis, which promotes barrier repair. In addition, barrier damage results in cytokine synthesis and release, which stimulate inflammation (Lowry, 1993). Inflammatory processes promote clearance of pathogens in the wound site and cleansing the site of foreign particles, eventually promoting new tissue formation (A. J. Singer & Clark, 1999). Disruption of inflammatory processes generally results in increased risk for infection, chronic wounds, and development of scar tissue and keloids

(Elias, Wood, & Feingold, 1997; Witte & Barbul, 1997). Individuals with skin disorders such as psoriasis or contact dermatitis experience pathological inflammation following barrier disruption, which can result in altered barrier restoration (Elias et al., 1997). Indeed, cytokine abnormalities can slow recovery of the skin barrier. Older, but not younger IL-1α knockout mice showed slower skin barrier recovery compared to wild-type mice (Ye et al., 2002). This may be due to the role of proinflammatory cytokines in inflammation and in promoting lipid and protein synthesis (Elias, 2005).

Stress and skin barrier recovery

Across several studies, psychological stress delays wound healing (Glaser et al., 1999; Kiecolt-Glaser et al., 1995; Marucha et al., 1998). For instance, dental students took an average of 3 days longer to heal a standardized oral mucosa wound during academic examinations compared to during summer vacation (Marucha et al., 1998). Moreover, stress may exacerbate certain dermatological conditions (O'Sullivan, Lipper, & Lerner, 1998). As previously mentioned, wound healing in the skin is mediated by the endocrine and immune systems (A. J. Singer & Clark, 1999; Slominski & Wortsman, 2000). Thus, these results suggest that psychological stress, through alterations of endocrine and immune function, may slow recovery of the skin barrier following damage.

Studies in humans

Two tape-stripping studies in humans suggest that psychological stress slows skin barrier recovery. The first study used involved assessment of 27 medical students during a high stress period (academic examinations) and two lower stress periods (the end of winter and spring vacation; Garg et al., 2001). Baseline TEWL and the number of tape-strippings required to disrupt the skin barrier to a TEWL value of 20 $g/m^2/h$ did not

differ as a function of stress. During exams, approximately 30% skin barrier recovery was achieved at 3 h following tape-stripping, compared to 45% barrier recovery during the lower stress periods. These stress-related differences in skin barrier recovery persisted for 24 h after skin perturbation. Moreover, students who reported the largest changes in perceived stress (Perceived Stress Scale; Cohen, Kamarck, & Mermelstein, 1983) and mood disturbance (Profile of Mood States; McNair, Lorr, & Dropelman, 1981) showed the slowest recovery relative to lower stress periods (r = -.42 for POMS, r = -.33 for PSS).

In related work relevant to this study, skin barrier recovery 3 h following tape-stripping was slowed by several stressors compared to baseline skin barrier recovery (Altemus et al., 2001). Participants were exposed to an interview stressor almost identical to the TSST, and skin barrier recovery was assessed during a non-stressful period (the day before) and after the interview stressor. Skin barrier recovery 3 h following the interview stressor was delayed (approximately 55%) compared to the non-stressful baseline period (approx. 69%). TEWL measured at the cheek, but not the flexor surface of the forearm, increased after the interview stressor. Moreover, water content of the stratum corneum (skin conductance) measured at the cheek did not change after the interview. This suggests that differences in skin barrier recovery between the baseline and stress days were not related to changes in baseline TEWL or changes in skin water content related to the stressors. Two other stressors were included in this study – sleep deprivation and exercise (50% of maximal heart rate). Sleep deprivation, but not exercise, was associated with delays in skin barrier recovery, and neither stressor was related to changes in basal forearm TEWL.

Potential mediators

These preliminary studies suggest that various forms of acute stressors can delay the restoration of skin barrier function. Both studies found significant effects of stress at 3 h after barrier disruption. As previously mentioned, the initial phases of the response to skin barrier perturbation involve lipid synthesis and cytokine expression (Ghadially et al., 1995; Nickoloff & Naidu, 1994). Thus, any stress-related biological changes should directly or indirectly impact either or both processes. Garg and colleagues speculated that three potential mechanisms may explain stress-related alterations in skin barrier recovery: Stress-related activation of immune and inflammatory processes in deeper skin layers, neuropeptide release from afferent nerves in the peripheral nervous system, and systemic glucocorticoid levels (Garg et al., 2001). Several lines of evidence support the role of glucocorticoid levels in delaying skin barrier recovery.

For glucocorticoids to have a direct effect on cells in the epidermal layers, those cells must contain glucocorticoid receptors. Glucocorticoid and mineralocorticoid receptors are expressed in the skin layers, including immune cells in the epidermis and keratinocytes (Slominski & Wortsman, 2000). The glucocorticoid receptor is primarily expressed in keratinocytes in the lower, basal layers of the epidermis, with little expression in the outer layers, including the stratum corneum (Serres, Viac, & Schmitt, 1996). Mineralocorticoid receptors are also expressed in the epidermis (Kenouch et al., 1994).

Topical corticosteroids are widely used in a number of inflammatory and hyperproliferative skin conditions, and application of corticosteroids delays the proliferation of keratinocytes that normally follows tape-stripping (Bauer, Boezeman,

Rijzewijk, & de Grood, 1989) and slows barrier recovery in mice (Denda, Tsuchiya, Elias, & Feingold, 2000). Moreover, in mice systemic administration of corticosterone resulted in marked delays in barrier recovery, with increased dosage related to slower recovery at 2.5 h (Denda et al., 2000). In the same study, blocking the glucocorticoid receptor with RU-486 impeded the delay in barrier recovery produced by systemic corticosterone administration and stress (cage rotation or restraint).

Only one study in humans examined glucocorticoids as a mediator of the stress – skin barrier recovery relationship (Altemus et al., 2001). Cortisol significantly increased at 20 min after the onset of interview stress, and showed a small decline at 50 min after the onset of interview stress. Cortisol changes were negatively correlated with skin barrier recovery, suggesting that increases in cortisol were associated with slower barrier recovery (r = -.22), but this was not significant. The only other endocrinological or immunological measures significantly correlated with skin barrier recovery were changes in IL-1β and IL-10, with increases in these cytokines related to faster recovery.

The extant research on stress and skin barrier recovery focused on glucocorticoids as a mediator. Another potential, yet unexamined mediator is catecholaminergic pathways. Human keratinocytes express substantial β-adrenergic receptor densities (Steinkraus, Steinfath, Korner, & Mensing, 1992). Such expression may be related to the regulation of keratinocyte differentiation and calcium ion homeostasis (Schallreuter et al., 1995). Overall, the presence of hormone receptors on epidermal cells suggests that psychological stimuli may result in epidermal alterations via neuroendocrine pathways.

Summary

Studying skin barrier recovery provides a promising model for examining stress and objective health outcomes. Recovery of the barrier involves lipid synthesis and inflammatory processes, which happen immediately following disruption. Two studies in humans provide evidence that acute stress can alter skin barrier recovery within the first 3 h following disruption. Research in mice models suggests that systemic glucocorticoid levels may mediate the relationship between stress and delayed skin barrier recovery. Thus, this study offers an opportunity to replicate prior work and examine the role of glucocorticoids through frequent cortisol sampling.

Study overview

Based on the insights gained from the literature, I now review design considerations derived from the review and how they impact the selection of stressor task, the social support manipulation, and measurement of skin barrier recovery in this study. A brief outline of the study design follows, and this section concludes with an outline of the primary study hypotheses.

Design considerations

Stressor task

The literature review suggests that the brief stressful task should have an element of social-evaluative threat, such as an audience. Moreover, the stressor should involve performance in some form with verbal responses, such as a speech. This is also in accordance with recommendations by Dickerson and Kemeny (2004) based on their meta-analysis of acute stressors and cortisol responses. In particular, they recommended using brief stressors with a combination of public speaking and cognitive tasks. This

combination yielded the largest effect sizes of stress on cortisol reactivity (d = .63) compared to public speaking (d = .31), cognitive tasks (d = .34), emotion induction (d = .13), and noise exposure (d = .37). The large effect of public speaking and cognitive combinations was primarily due to the element of social-evaluative threat. Thus, in this study the acute stressor was the Trier Social Stress Test (TSST), a widely-used, well-validated 10 min combination public speaking and mental arithmetic task which reliably enhances cardiovascular and cortisol reactivity (Kirschbaum & Hellhammer, 1994; Kirschbaum, Klauer, Filipp, & Hellhammer, 1995; Kirschbaum, Pirke, & Hellhammer, 1993)

Physiological changes

Psychometric considerations (Kamarck & Lovallo, 2003), and the reactivity and recovery issues described previously suggest that frequent sampling of cardiovascular and cortisol measures is warranted. Only a handful of studies have explicitly studied cortisol recovery (Earle, Linden, & Weinberg, 1999; Matthews, Gump, & Owens, 2001; Roy, Kirschbaum, & Steptoe, 2001). Moreover, cortisol measures were obtained within the 21-40 min post-stressor onset window to capture peak responses. In addition, this study used frequent and extended sampling of cardiovascular and cortisol measures, allowing for a more sophisticated approach to physiological changes and their relationship to wound healing. For instance, this study examined the separate contributions of cortisol reactivity and recovery to skin barrier recovery. Cortisol measures were extended beyond 60 min post-stressor onset, providing a key contribution to acute stress and physiology research through novel data on cortisol recovery.

Social support manipulation

This study used a supportive confederate to provide social support rather than a person familiar to the participant. Confederates afford control over variables that may influence supportive behaviors, such as length of the friendship or perceived closeness (Thorsteinsson & James, 1999). In addition, confederates allow for thorough training in carefully controlled and rehearsed support behaviors. Using confederates is also justified empirically; in meta-analytic comparisons of supportive confederates vs. alone conditions, support provided by a confederate reduced heart rate and blood pressure reactivity to speech stressors in four out of five studies (Thorsteinsson & James, 1999). Men showed lower cortisol reactivity after support provided by a confederate compared to no support (Kirschbaum et al., 1995), and women showed lower cortisol reactivity after confederate support compared to support from an opposite-sex partner. Support provided by a confederate on a video monitor reduced cortisol reactivity in both men and women (Thorsteinsson et al., 1998).

As described earlier, although passive support manipulations are somewhat successful in reducing evaluative threat and reducing cardiovascular reactivity (Thorsteinsson & James, 1999), they also decrease support potential and ecological validity (Lepore, 1998). At the same time, active support manipulations increase the potential for evaluative threat. Thus, combining both active and passive support may maximize effects on physiological reactivity while counterbalancing their respective effects on evaluative threat. The social support manipulation in this study included both passive and active social support prior to the stressful tasks. In addition, manipulating

social support prior to the stressors minimizes the physiological response-enhancing effect of support provided during the stressful tasks (Kamarck et al., 1998).

This study also included an alone condition, which addressed concerns voiced by several researchers when comparisons are conducted between supportive and non-supportive conditions. Specifically, Lepore (1988) pointed out that without an alone condition, it is unclear whether social support effects are due to decreased reactivity related to the supportive companion, increased reactivity related to the non-supportive companion, or a combination of the two effects. Thorsteinsson and James (1999) also agreed omitting an alone condition creates interpretation problems.

Skin barrier recovery

This study assessed skin barrier recovery in a similar manner as Altemus and colleagues (Altemus et al., 2001). However, instead of a within-subjects design where participants serve as their own control, the effects of stress and social support were examined using a between-subjects design, which is a novel contribution to the literature. In addition, TEWL measurements of skin barrier recovery vary for a number of reasons, including individual differences an ambient room conditions. These sources of variance were controlled as much as possible.

Despite these controls, the rate of skin barrier recovery differs across a number of studies. For instance, while the Altemus study found 50-70% recovery after 3 h, the Garg study found 30-45% recovery after 3 h. Other studies have found slower rates of recovery (Fluhr et al., 1999; Ghadially, Brown, Sequeira-Martin, Feingold, & Elias, 1995; Nickoloff & Naidu, 1994). For instance, one study showed 30-40% recovery 12 h after barrier disruption in participants of similar age (Ghadially et al., 1995). However, these

studies vary by the criteria for stopping skin stripping (ranging from 15 g/m^2h to over 50 g/m^2h), instruments used to measure TEWL, and the degree of trauma or irritation produced by the different types of tape (Altemus et al., 2001). The lack of standardized criteria, settings, and equipment makes comparisons among different studies difficult. This study used methods similar to the previous stress and skin barrier recovery studies (Altemus et al., 2001; Garg et al., 2001).

Study outline

The study consisted of one 3.5-h appointment where skin barrier recovery, cardiovascular measures, and salivary cortisol following tape-stripping were measured. Participants were randomly assigned to either a no-stress group (No Stress), acute stress with no social support (Stress group), or acute stress with social support provided by a same-sex confederate for 10 min prior to the stressor (Stress + Support group). Self-report measures assessed changes in subjective affect and anxiety throughout the session, individual difference and social network variables that may influence psychological and physiological responses, and perceptions of the confederate.

Study hypotheses

In this study, I tested the following hypotheses:

1) Participants exposed to an acute psychological stressor will show delayed skin barrier recovery relative compared to participants exposed to no acute psychological stress.
2) Participants receiving social support from a confederate prior to the acute psychological stressor will show faster skin barrier recovery compared to participants receiving no social support before the stressor.

3) Participants receiving social support prior to the acute stressor will show reduced cardiovascular and cortisol reactivity compared to participants receiving no social support before the stressor.

4) Across all participants, increased cardiovascular and cortisol reactivity to acute stress will be related to delayed skin barrier recovery.

5) Across participants exposed to the acute stressor, stress-induced cortisol responses will show distinct and reliable patterns of reactivity from and recovery to baseline.

CHAPTER 2

METHODS

Power analyses

All between-subject effect size estimates and power analyses were conducted using G*Power (Dusseldorf, Germany; Erdfelder, Faul, & Buchner, 1996) and within-subjects effect size estimates and power analyses were conducted using Power Analysis and Sample Size software (PASS) from NCSS (Kaysville, UT; Hintze, 2001). All sample size estimates described below assume $\alpha = .05$, and power = .80 in a two-tailed test.

Cardiovascular and cortisol reactivity

The meta-analytic reviews of acute stress/social support research report effect sizes from between-subjects designs, with comparisons made between groups receiving support or no support. Thorsteinsson reported that the effect size of social support manipulations on cortisol was $d = .83$ (Thorsteinsson & James, 1999), though this was based on only two studies reported in the literature (Kirschbaum et al., 1995; Thorsteinsson et al., 1998). With a $d = .83$, the total sample size estimate is $N = 48$. The authors reported that the effect size of social support manipulations on heart rate was $d = .61$, systolic blood pressure $d = .61$, and diastolic blood pressure $d = .51$. With an average $d = .58$, the total sample size estimate is $N = 96$.

Skin barrier recovery

Both studies assessing stress-related delays in skin barrier recovery used within-subject designs, with participants serving as their own controls (Altemus et al., 2001; Garg et al., 2001). Neither study reported descriptive statistics, although both reported within-subject statistical tests (*t*'s and *F*'s). Estimates of within-subjects effect sizes were derived from estimates of the means and *SD*s from figures in each article.[1] For the purposes of this study, only the Altemus study was used to determine sample size, as it is most similar in design to this study, and has the more conservative effect size estimate (Altemus $d = .53$, Garg $d = 1.58$). Assuming the between-subjects effect size would be the same as the within-subjects effect size of $d = .53$, in a between-subjects comparison the total sample size estimate is $N = 134$.

Target sample size

The target sample size for this study was 100 participants, 30 in the No Stress group, 35 in the Stress group, and 35 in the Stress + Support group. Roughly equal numbers of men and women were included in each group. All power estimates were based on two-tailed, between-subjects comparisons with $\alpha = .05$. This sample size provides sufficient power for testing the large effects of stress on cardiovascular reactivity ($d = 1.36$, power = 1.0; Benschop et al., 1998), and social support

[1] To enhance precision in estimating means and *SD*s, the figures from both studies were scanned into JPEG format. Using Deneba Canvas 8.0 software, the distance (cm) between marked units on the y-axis was estimated by drawing lines between the y-axis reference points (e.g., the distance between 10% and 20%). These measurements yielded the scale of the plot. For example, in the Altemus figure, 1 cm was equivalent to a 25% increment. The distance from 0 on the y-axis to the plotted points was also estimated, and using the scale, the means were estimated. Similarly, *SD*s were estimated by measuring the length of the *SEM* bars, estimating the actual *SEM* values based on scaling, and estimating the *SD*s using the *SEM* estimates and sample size.

manipulations on reducing cardiovascular reactivity (d = .58, power = .78; Thorsteinsson & James, 1999)[2]. The proposed sample size also provided sufficient power for testing the large effects of social-evaluative stress (d = .61, power = .79; Dickerson & Kemeny, 2004), and social support manipulations on cortisol reactivity (d = .83, power = .93). The target sample size is much larger than the samples in both tape-stripping studies. Although prior tape-stripping studies used a within-subjects design, with participants serving as their own controls, the target sample size achieves moderate power to test the effect of stress on skin barrier recovery (power = .59) assuming the between-subjects effect size is the same as the within-subjects effect size (d = .53). The effects of social support on skin barrier recovery are unknown. The sample size provides moderate power to test the effects of social support on skin barrier recovery, assuming the effect is moderate (d = .50, power = .54). The proposed sample size was insufficient for testing small effects (d = .20, power = .13).

Participants

Healthy individuals ages 18 – 44 were recruited from the local community, using fliers posted in university buildings and on public transportation. Potential participants contacted our lab through phone, email, or our website, and were directed to complete an initial screening questionnaire online, which assessed whether individuals met criteria for participation.

Individuals were excluded from the study if they were pregnant, took medications with obvious immunological or endocrinological consequences, or had illnesses with

[2] Effect size estimates were derived by averaging the HR, DBP, and SBP effect sizes reported in both the Benschop et al. (1998) and Thorsteinsson et al. (1999) meta-analyses. It should be noted that the Benschop et al. (1998) estimates are from eight studies of younger and older female adults.

immunological or endocrinological components including preexisting primary skin disease. Individuals with allergies to tape or other adhesives were not included in this study. In addition, disqualifying health problems included smoking, drinking more than 14 alcoholic drinks per week for women or 21 for men, and excessive caffeine use. Use of hormonal-based contraception was an exclusion criterion because it attenuates cortisol reactivity (Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999). Participants were not scheduled based on menstrual cycle stage given the logistical difficulties in attempting to do so. However, because menstrual cycle phase influences stress-related cardiovascular (Girdler, Pedersen, Stern, & Light, 1993) and cortisol reactivity (Kirschbaum et al., 1999), menstrual phase was assessed by self-report (Polefrone & Manuck, 1988).

A flowchart of participant flow in the study is shown in Figure 1. A total of 403 individuals completed the online screening form, and 244 individuals (60%) were determined eligible for the study. Of those 245 individuals, 144 were scheduled for the study, and 45 did not come for their scheduled appointment or canceled and failed to reschedule. Therefore, 99 individuals participated in the study, of which 12 women did not report taking hormonal-based contraception on the online screening form, but reported it during the experimental session. Two individuals were unable to complete the entire protocol. Therefore, data from those 14 individuals were not included in the data analyses that follow, leaving a sample of 85 individuals for the data analyses.[3]

[3] While the total sample for analysis is 85 participants, due to logistical and other reasons (e.g., undetectable cortisol in a saliva sample) there is missing data scattered throughout the various questionnaire, cortisol, cardiovascular, and skin barrier recovery measures. Therefore, the actual *N* in each analysis may be less than 85, and will be reported.

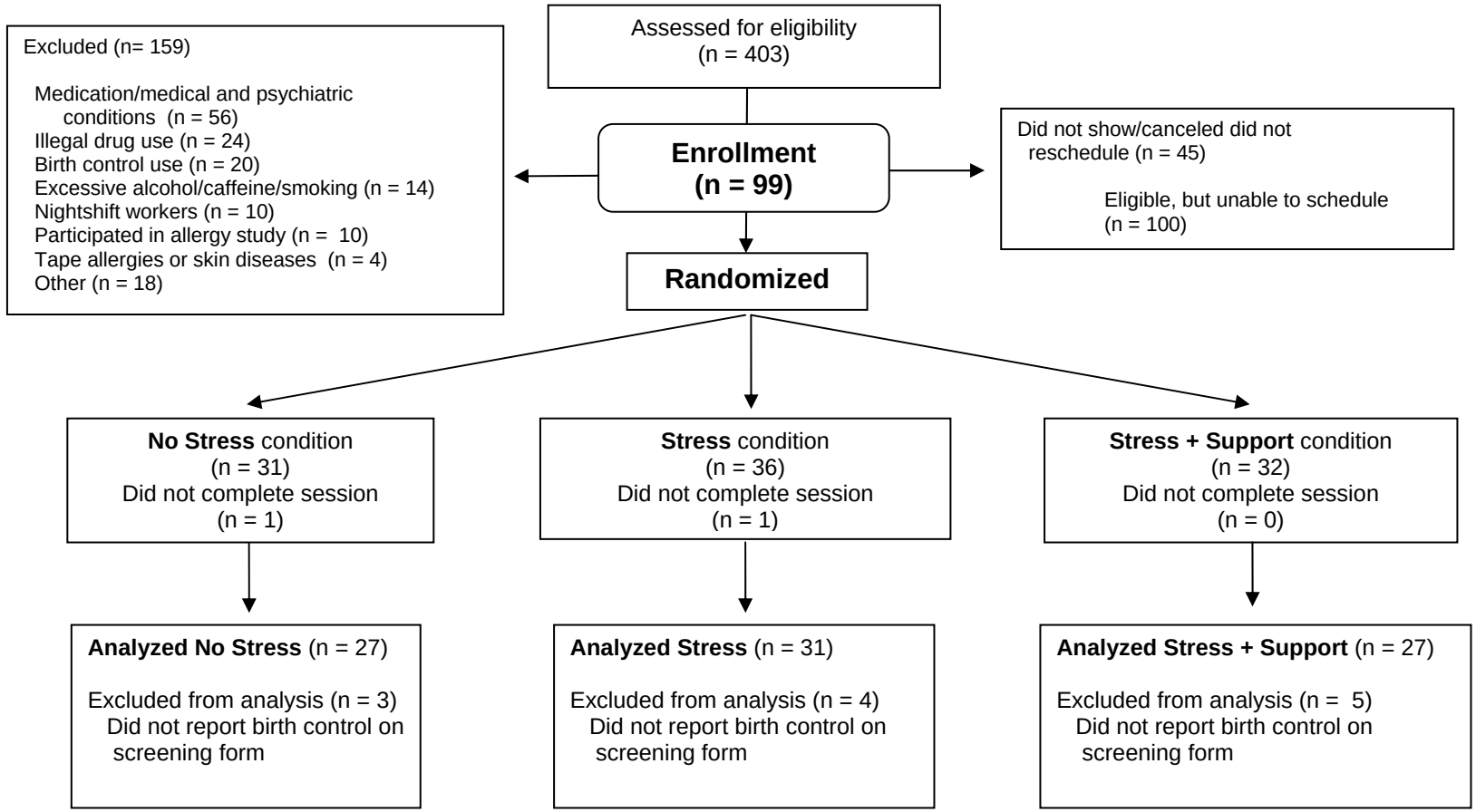

Figure 1. Participant flow through the study, from recruitment to data analyses.

Procedure

A timeline for the study is shown in Figure 1. All testing sessions were scheduled at The Ohio State University General Clinical Research Center (CRC) in the afternoon to maximize the effects of acute psychological stress on cortisol levels (Dickerson & Kemeny, 2004); beginning at 1:30 PM. Participants were asked to refrain from eating, vigorous exercise, and smoking for 1 h prior to the appointment, and drinking alcohol for 24 h prior to the appointment. Informed consent was obtained from participants when they arrived for the appointment. All procedures were approved by The Ohio State University Biomedical Sciences Institutional Review Board.

After arrival and providing informed consent, the participant was taken to the experiment room and fitted with a blood pressure cuff. The participant was asked to sit quietly for 40 min and fill out several questionnaires (Packet 1). After the 40 min adaptation period, a baseline salivary cortisol sample (Cortisol 1) was collected, and baseline heart rate and blood pressure was collected for 10 min (Cardio 1). Next, baseline TEWL was measured, and the skin barrier was disrupted with tape-stripping (see *Tape-stripping*). Tape-stripping was followed by administration of the first thought-listing task to assess psychological responses to tape-stripping (see *Psychological measures*). After the thought-listing task and additional questionnaires (Packet 2), another cardiovascular and salivary cortisol measurement assessed cardiovascular responses to the tape-stripping procedure (Cardio 2 and Cortisol 2).

After these assessments, the experimenter opened an envelope containing the participant's group assignment. Experimenters were blind to group assignments up to the

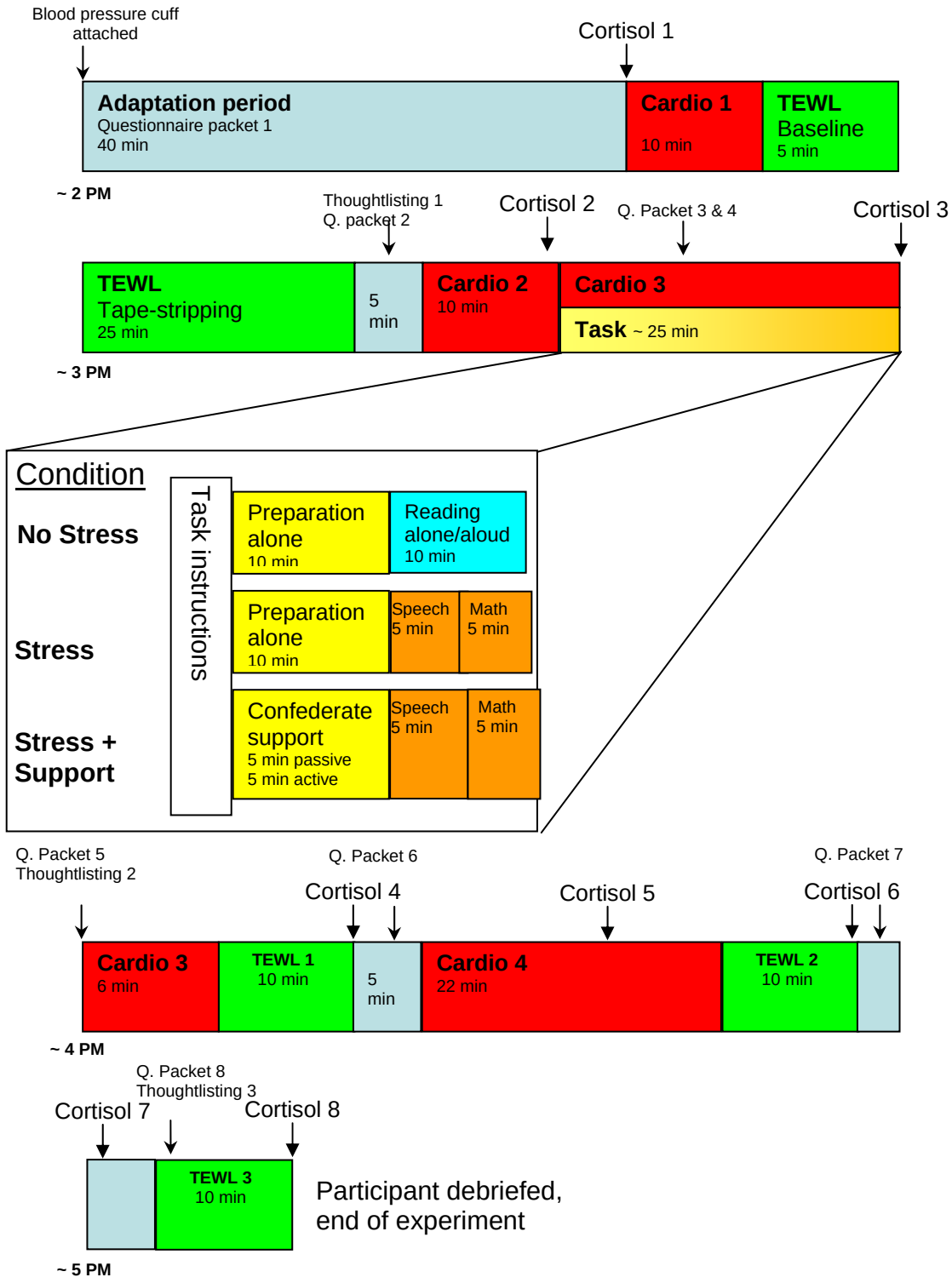

Figure 2. Timeline of the experiment.

completion of tape-stripping. Participants were randomly assigned to one of three groups: No Stress, Stress, or Stress + Support. Participants were then provided instructions for the experimental task. Participants in the No Stress group were provided instructions for the reading task (see below), and participants in the Stress groups were provided instructions for the Trier Social Stress Test (TSST; see below). Task instructions were followed by administration of several brief questionnaires (Packet 3). After the instructions, participants prepared for the tasks for 10 minutes. Participants in the Stress + Support group were greeted by the supportive confederate in Room #1 (see *Social support manipulation* below). Participants in the Stress group and the No Stress group prepared for their tasks alone. Heart rate and blood pressure were measured throughout the preparation period (Cardio 3). At the end of the preparation period, several questionnaires were administered (Packet 4). Following this, participants in the No Stress group began the reading task. Participants in the Stress and Stress + Support group began the TSST.

Participants were seated during the tasks, and cardiovascular measures were recorded every 2 min (Cardio 3 continued). After completion of the tasks, another salivary cortisol sample and a 10 min cardiovascular measurement was collected (Cortisol 3), followed by a second thought-listing task to assess psychological responses to the tasks. After several questionnaires were administered (Packet 5), the first TEWL measurement of the tape-stripping sites was conducted (1 h after tape-stripping, 35 min after the tasks). Additional cortisol samples were obtained at 30, 45, 60, 75, and 90 min after the tasks (Cortisol 4 – 8). Additional cardiovascular measures were taken for a 22 min period approximately 20 min after the tasks (Cardio 4). Questionnaires were administered throughout the period after the tasks (Packets 6 – 8), and a thought-listing

task was administered at 60 min post-task. Additional TEWL measures of the tape-stripped sites were conducted at 90 min and 2 h after tape-stripping (60 and 95 min after the tasks). After the last TEWL measurement, participants were debriefed by the experimenter and provided with a debriefing form with further information.

Experimental manipulations

Trier Social Stress Test

The TSST was used for the Stress and Stress + Support groups. Dr. Kirschbaum has provided a detailed TSST manual to be used by experimenters in implementing this task (Kirschbaum, Pirke, & Hellhammer, 1993). Two confederates, described as the "committee" were escorted into the room and introduced to the participant by the experimenter. A video camera and tape recorder were present in the room to record the task. For the speech, the participant was told to imagine that s/he has applied for a position at a law firm and was invited to an interview by the selection committee. After this the participant was told that s/he would be given 10 min to prepare a speech about why s/he would be best for the job, and 5 min to talk with the committee, followed by a second experimental task. The participant was told that at least one member of the committee was trained in behavioral observation, and thus his/her behavior would be rated accordingly, and that members of the committee would take notes regarding the content and style. In addition, the participant was told that the speech would be video- and audio-recorded, for the purpose of rating "paraverbal signs of stress." The committee was then escorted out of the room, and the participant was given 10 min to prepare for the speech. Participants were allowed to make written notes during this preparatory phase, but that notes could not be used during the speech.

After the 10 min preparatory period, the committee was escorted back into the room and the participant delivered his/her speech. After the speech, the participant was asked to perform mental arithmetic, a common experimental stressor used extensively in cardiovascular research (Cacioppo et al., 1998). Following completion of the TSST, the committee left the room. Participants were told during the debriefing at the end of the experiment that they were not actually being evaluated by the committee, the committee was actually composed of research assistants on the project, and that no one was an expert in behavioral observation.

Social support manipulation

A same-sex supportive confederate trained in providing supportive behaviors in a standardized manner was used in the Stress + Support group. The experimenter introduced the confederate as a person whose primary job would be accurately timing the 10 min preparation period, but would be available to assist the participant if they would like help preparing their speech.

Confederates provided both passive and active support to the participant during the 10 min preparation period. The rationale for passive support comes from work by Uchino (Uchino & Garvey, 1997), in which the mere availability of support during preparation for a speech stressor reduced subsequent cardiovascular reactivity during the speech. Mere availability in the Uchino study was manipulated through the experimenter telling the participant that he/she would be outside the room during preparation and the speech, and that he/she would be available if the participant had any questions or needed help. In this study, confederates introduced themselves to the participant, indicated that they needed to finish something up for a couple of minutes, although they would be

available if the participant needed help. The confederate sat quietly, appearing to read and write at a table for 5 minutes, constituting the passive support portion of the manipulation. In the Uchino study and other work (Sarason & Sarason, 1986), participants solicited help from the available experimenter on rare occasions (e.g., 5 participants out of 49 in the Uchino study). However, the experimenter was out of sight in those studies.

At the end of 5 minutes, or once support was solicited from the confederate, the passive portion of the support manipulation ended and the confederate provided the active component of support. The active support manipulation was patterned after prior work by Lepore (Lepore et al., 1993) and Kirschbaum (Kirschbaum et al., 1995). The manipulation included verbal comments consisting of emotional, instrumental, informational, and validation support (see Appendix A; Nagurney, 2001; Wills & Shinar, 2000). The first unsolicited comments that confederates made to participants were the same as the Lepore study, "Remember, it will all be over in a few minutes" (Lepore et al., 1993). In addition to verbal comments, confederates asked participants questions of both an instrumental and emotional nature, followed by empathic follow-ups to the participants' responses. Confederates were trained to use specific supportive comments, questions, and responses from a "menu" of supportive comments (see Appendix A) from which they could chose from, and each comment was only used once. At the end of the 10 min, the confederate enthusiastically wished the participant luck and provided additional encouragement, and left the room.

Reading task

Just as in the Stress and Stress + Support groups, a video camera and tape recorder was present in the room, but the video camera was turned off and pointed towards a wall. The participant was provided with a newspaper article and was asked to tape record themselves while reading the article aloud. During these instructions, the experimenter emphasized that the participant was not being evaluated, and indicated that the purpose of tape-recording was to ensure that the participant was actually reading aloud. After this the participant was given 10 min to review the article silently. After recording the first article (5 min) the participant was asked to record a second article.

Psychological measures

Self-report measures

Computer-assisted questionnaire administration was used for all self-report data. Most participants are comfortable responding to sensitive questions when they are using a computer, and this appears to be particularly true for health-related topics (Feigelson & Dwight, 2000). A copy of all self-report measures, organized by order of administration, is included in Appendix B.

The background questionnaire collected a variety of sociodemographic and health information including age, weight, height, and ethnicity. Medical history recorded for female participants included the date that their last menstrual period began, the length and regularity of their menstrual cycle over the prior three months, typical length of the menstrual period (number of days of actual bleeding), and any menstrual or gynecological problems.

To crudely determine menstrual phase, I used the self-report data to ascertain length of menses (number of days bleeding) and total length of the cycle. I then assumed the luteal phase was 14 days across all participants and in doing so could ascertain the length of the follicular phase. I then determined the day of the cycle on the day of the CRC admission based on the participant's reporting of the start of their last menstrual period. If the day of the cycle was less than or equal to the menses + follicular period, I determined the participant was in the follicular phase. If the day of the cycle was greater than or equal to the menses + follicular period, I determined the participant was in the luteal phase. Several participants did not correctly fill out the form, indicating that they had had their last period between 47 – 58 days ago ($N = 3$), or indicating an extremely short menstrual cycle (11 days, $N = 1$). The latter participant did not indicate any problems with her menstruation, so I assumed she was on a 28 day cycle. For the participants who reported extremely long latencies between the CRC admission and their last menstrual period, none reported any problems with their menstruation, though 1 reported missing 1 period in the past year. Therefore, I assumed they filled the form out incorrectly, and computed their menstrual phase on the basis of the latency divided by 2, placing them in the luteal phase. 3 participants did not fill out the menstrual cycle questionnaire completely; 2 provided sufficient data (date of last menstrual cycle, number of days bleeding), and I assumed a 28 day cycle for those participants, and 1 participant provided no data. Therefore, data on menstrual phase was missing for 1 participant and estimated based on incomplete data for 5 participants.

Health conditions

Questions from the OARS Multidimensional Functional Assessment Questionnaire assessed problems with lungs, kidneys, liver, digestive system, heart, high blood pressure, migraines, hormonal conditions, thyroid, cancer, cataracts, teeth, hernia, gout, hardening of the arteries, circulatory system, prostate, ovarian or uterine, and muscle-related disorders, as well as any medication used for each condition (Fillenbaum & Smyer, 1981). The format is similar to those used in epidemiological studies (Berkman & Breslow, 1983), and provides a simple way to look at frequency of chronic conditions and medications.

Anxiety, depressive symptoms, and affect

Anxiety and affect levels were assessed at multiple points during the session. The Spielberger State-Trait Anxiety Inventory (STAI) is a 40-item scale assessing subjective anxiety symptoms. State anxiety was measured using the 20-item component scale (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983) across six occasions during the study: During the adaptation period, after tape-stripping, immediately after tasks, and three more times spaced across the final 1.5 h of the study. In this sample, internal consistency ranged from .91 - .94 across the six measurements, and the intraclass correlation (two-way mixed model *ICC*) for a single measurement, which indicates test-retest consistency was .74. The STAI has been used in a large number of studies and shows good excellent validity; for example, individuals reporting higher state anxiety when viewing unpleasant pictures showed elevated corrugator electromyographic activity and skin conductance responses (J. C. Smith, Bradley, & Lang, 2005).

The Mood and Anxiety Symptoms Questionnaire (MASQ) is a 62-item scale assessing general negative affect, anxious arousal, anhedonic depression, anxiety symptoms, and positive affect (Watson, Clark et al., 1995; Watson, Weber et al., 1995). Internal consistency in the sample across the five scales ranged from .75 - .92. The factor structure of the MASQ has been replicated in student, adult, and patient samples, shows good validity in differentiating anxiety and depression, and good convergent validity with other measures of depressive and anxiety symptoms (Watson, Clark et al., 1995; Watson, Weber et al., 1995).

Concern with social-evaluative threat was assessed using the 12-item Brief Fear of Negative Evaluation Scale (BFNE, Leary, 1983). Recent work suggests that the factor structure of the BFNE is composed of two factors: straightforward and reverse scored items (Rodebaugh et al., 2004). Moreover, the straightforward items exhibit greater reliability and validity compared to the reverse scored items in patient samples with social anxiety, including greater internal consistency, unique variance in predicting social anxiety measures, and sensitivity to treatment effects (Weeks et al., 2005). Thus, in this study I report values from the straightforward items on the BFNE, which showed internal consistency of $\alpha = .92$ in this sample.

The 10-item Self-Statements during Public Speaking (SSPS) scale assessed positive and negative cognitions immediately prior to the speech stressor (Hofmann & DiBartolo, 2000). Internal consistency was $\alpha = .59$ for positive cognitions and $\alpha = .79$ for negative cognitions. This scale shows modest correlations wit established measures of social anxiety, strong correlations with instruments specific to public speaking anxiety,

and higher scores on the SSPS-N scale are related to higher subjective units of distress ratings during public speaking in social anxiety patients (Hofmann & DiBartolo, 2000).

The Positive and Negative Affect Schedule includes two 10-item mood scales assessing state and trait positive and negative affect (Watson, Clark, & Tellegen, 1988). The validity of the PANAS as a measure of both positive and negative affect has been demonstrated in both student and population-based samples (Crawford & Henry, 2004; Watson, Clark, & Tellegen, 1988). Mood throughout the session was assessed using the PANAS on 6 occasions: During the adaptation period, after tape-stripping, immediately after tasks, and three more times spaced across the final 1.5 h of the study. Internal consistency for state positive affect ranged from .91 - .94, and for state negative affect from .63 - .92. Test-retest reliability was *ICC* = .87 for state positive affect and *ICC* = .42 for state negative affect.

The trait PANAS assessing trait positive and negative affect was included halfway through study data collection. Internal consistency for trait positive affect was α = .91 and for trait negative affect was α = .86. The two scales are generally uncorrelated, and show good convergent and discriminant validity when related to state mood scales and other variables (Watson et al., 1988). In this sample, state positive and negative affect were not significantly correlated (*r*'s from -.11 - .00), and trait positive and negative affect were not significantly correlated (*r* = .06). Trait positive affect was significantly related to state positive affect (*r*'s from .54 - .78) but not state negative affect, and trait negative affect was significantly related to state negative affect (*r*'s from .44 - .64) but not state positive affect.

Social relationships and loneliness

The 48-item college student version of the Interpersonal Support Evaluation List assessed multiple dimensions of perceived social support, including emotional support, instrumental support, companionship support, and self-esteem maintenance through social comparisons (Cohen, Mermelstein, Kamarck, & Hoberman, 1985). Internal consistency for the four subscales in this sample ranged from .77 - .88. The ISEL is moderately correlated with other social support measures (Cohen, Mermelstein, Kamarck, & Hoberman, 1985), and the factor structure of the ISEL has been confirmed (Brookings & Bolton, 1988). Subjective feelings of loneliness were measured using the widely used Revised UCLA Loneliness Scale (Russell, Peplau, & Cutrona, 1980). Internal consistency for this scale was $\alpha = .89$. Scores on this scale were moderately correlated with daily-diary measures of loneliness collected over 4 months in college students (Pressman et al., 2005).

Subjective perceptions of social status were measured using the MacArthur Scale of Subjective Social Status, a visual scale incorporating two ladders. Participants indicated which "rung" of the ladder they would put themselves on relative to other people in the United States, and relative to other people in their community. In this population, the community was defined as "other people in your social group" (Adler, Epel, Castellazzo, & Ickovics, 2000). Participants also identified their social status relative to other members of their immediate social group (e.g., friends, other graduate students) on a third ladder. Higher ratings of subjective social status are moderately correlated with objective indicators, such as income and education (Adler, Epel, Castellazzo, & Ickovics, 2000), and higher perceptions of social status in a student

sample were significantly related to higher elevations in cortisol during the TSST (Gruenewald, Kemeny, & Aziz, in press).

Perceived stress and stressful life events

General stress appraisals over the past week were assessed using the 10-item Perceived Stress Scale (Cohen, Kamarck, & Mermelstein, 1983). Internal consistency in this sample was $\alpha = .75$. Higher scores on the PSS were related to a number of disease outcomes, including slower wound healing following punch biopsy (Ebrecht et al., 2004) and lower proinflammatory cytokines in blister wounds (Glaser et al., 1999). Stressful life events over the past year were assessed using the Psychiatric Epidemiological Research Inventory Life Events Scale, which contains a list of 102 possible life events (Dohrenwend, Krasnofff, Askenasy, & Dohrenwend, 1978).

Dispositional characteristics

Dispositional optimism and pessimism were assessed with the 10-item Life Orientation Test-Revised (Scheier, Carver, & Bridges, 1994). Internal consistency was $\alpha = .79$ for the optimism subscale and $\alpha = .85$ for the pessimism subscale. Higher pessimism ratings are related to more negative and less positive mood and higher ambulatory blood pressure in daily diary studies, while higher optimism ratings are related to less negative and more positive mood and generally lower ambulatory blood pressure (Raikkonen, Matthews, Flory, Owens, & Gump, 1999). Trait anger expression was assessed with the 24-item Anger Expression Scale (Spielberger, Krasner, & Solomon, 1988), which has a factor structure consisting of outward anger expression, inward anger suppression, and anger control which has been replicated in other studies (Deffenbacher, Oetting, Lynch, & Morris, 1996). For the total anger expression score in

this sample internal consistency was α = .69. The widely used 50-item Cook-Medley Hostility Scale assessed trait hostility (Cook & Medley, 1954). Internal consistency in the sample was α = .86. High scores on the Cook-Medley Hostility Scale are related to increased anger and cynical appraisals of others during competitive tasks, and increased tendency to attribute hostile intent to others' displays of negative social behaviors (Pope, Smith, & Rhodewalt, 1990).

Health-related behaviors

Health-related behaviors that can influence physiological measures were assessed, including medication use, caffeine and alcohol intake (Kiecolt-Glaser & Glaser, 1988). Participants were asked to describe current status and any recent changes in amount of sleep in the last three days, weight changes in the last two weeks. Questions from Baecke et al. (Baecke, Burema, & Frijters, 1982) provided a method to quantify recent physical activity.

Overall sleep quality was assessed using the 9-item Pittsburgh Sleep Quality Index (Buysse, Reynolds III, Monk, Berman, & Kupfer, 1989), a self-rated questionnaire which assesses sleep quality and disturbances over a one-month interval, and has good diagnostic sensitivity and specificity in distinguishing good and poor sleepers. The scale yields a total score as well as 7 subscales which include subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medications, and daytime dysfunction. Internal consistency for the total scale reflecting subjective sleep complaints was α = .63. Scores above 5 for total subjective sleep complaints show good specificity and sensitivity in differentiating "good" from "poor"

sleepers based on scores in sleep disordered patients and polysomnography results (Buysse, Reynolds III, Monk, Berman, & Kupfer, 1989).

Subjective ratings of the confederate

In addition to these questionnaires, participants' appraisals of the confederate were measured using the 64-item Interpersonal Adjective Scales – Revised (Wiggins, Trapnell, & Phillips, 1988), similar to previous work where the participant used the scale to rate another person rather than themselves (Gallo, Smith, & Kircher, 2000). This scale is based on a circumplex framework of personality characteristics, with 8 poles organized in a clockwise fashion: Assured-dominant, gregarious-extraverted, warm-agreeable, unassuming-ingenuous, unassured-submissive, aloof-introverted, cold-hearted, and arrogant-calculating. Internal consistency among the 8 poles/subscales ranged from .64 - .97. Scores were derived by averaging item scores for each subscale, then converting the average scale scores to *t*-scores based on the mean and *SD*s obtained in the original validation study (Wiggins, Trapnell, & Phillips, 1988), with a mean of 40 and an *SD* of 10. This procedure allowed for comparisons with other studies (Gallo et al., 2000). Several studies have demonstrated the validity of using the IAS-R to rate other individuals (T. W. Smith, Gallo, Goble, Ngu, & Stark, 1998; T. W. Smith, Limon, Gallo, & Ngu, 1996).

The degree to which the participant viewed the confederate's behavior as unsupportive was measured using the 24-item Unsupportive Social Interactions Inventory (Ingram, Betz, Mindes, Schmitt, & Smith, 2001). The scale is composed of four factors representing different facets of unsupportive behavior, labeled distancing, bumbling, minimizing, and blaming. Internal consistency across the four subscales ranged from .41

- .83. Ratings of unsupportive social interactions on the USII in the context of specific stressors uniquely predict psychological and physical symptoms over and above measures of general social support and negative affect (Ingram, Betz, Mindes, Schmitt, & Smith, 2001).

Threat, control, and helplessness appraisals.

Cognitive appraisals related to the tasks were assessed using several single-item questions. Appraisals included perceptions of threat and coping (Tomaka, Blascovich, Kelsey, & Leitten, 1993), and feelings of control and helplessness experienced during the stressor (Breier et al., 1987). Perceptions of threat and coping were assessed before and after the preparation period and showed high test-retest correlations (threat r = .91, coping r = .87). Cognitive appraisals of threat and coping are significantly related to patterns of cardiovascular reactivity; specifically, cardiac reactivity is related to low threat appraisals and high coping appraisals, and vascular reactivity is related to high threat appraisals and low coping appraisals (Tomaka, Blascovich, Kelsey, & Leitten, 1993).

Thought-listing

Thought-listing is a production method of cognitive assessment that is particularly useful when there are no predetermined ideas about relevant cognitive dimensions. Cognitions have been assessed in a variety of research settings using thought-listing (Cacioppo, von Hippel, & Ernst, 1997). This study employed a variation of the thought-listing procedure described by Cacioppo and Petty (Cacioppo & Petty, 1981; Cacioppo, von Hippel, & Ernst, 1997) to collect participants' cognitions after tape-stripping, 10 min after the tasks, and 60 after the tasks. Participants were given 2 min to speak into a tape

recorder and describe their thoughts and feelings during the previous 10 min. At the 60 min post-task assessment, participants were asked to describe the thoughts they have been having about the tasks. Two judges blind to participants and hypotheses coded specific dimensions of valence (positive/negative) and reference (self, experimenter, etc).

Physiological measures

Salivary cortisol

Salivary cortisol was collected during both sessions. Saliva was collected using a Salivette (Sarstedt 1534, Sarstedt Inc., Newton, NC), consisting of a sterilized cotton swab, which the participant chews in their mouth for 2 min and places in a small beaker contained in a plastic tube. The assays were performed in the Ohio State University Clinical Research Center laboratory, using chemiluminescent techniques (Glaser et al., 1999). A total of 8 cortisol samples were obtained (Figure 1): prior to and after tape-stripping, and 10, 30, 45, 60, 75, and 90 min after the beginning of the tasks.

Cardiovascular measures

Heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressures were assessed with an automated Dinamap Critikon 1846SX/P monitor. The Dinamap employs the oscillometric method and derives systolic and diastolic blood pressure mathematically in reference to mean arterial pressure (Shapiro et al., 1996). Measures were obtained every 2 min during each measurement period. There were a total of seven 10 min measurement periods (Figure 2): immediately after the 40-min adaptation period (Cardio 1), just after tape-stripping (Cardio 2), just after participants received instructions about the task (Cardio 3), during the task (Cardio 3), up to 5-7 min

after the task (Cardio 3), 20 min after the task (Cardio 4) and 30 min after the task (Cardio 4). Internal consistency for each cardiovascular measure within each measurement period was high (MAP α's from .85 - .94, Heart rate α's from .95 - .97, SBP α's from .90 - .95, DBP α's from .85 - .93).

Tape-stripping and wound measurement

Tape-stripping is commonly used in dermatological research to disrupt skin barrier function (Ghadially, 1998; Ghadially, Brown, Sequeira-Martin, Feingold, & Elias, 1995; Nickoloff & Naidu, 1994; Proksch, Brasch, & Sterry, 1996; Tanaka, Zhen, & Tagami, 1997). Baseline skin barrier function was measured by obtaining baseline readings of transepidermal water loss (TEWL) using an electrolytic water meter (cyberDERM, Cortex DermaLab; Media, PA) which measures the vapor pressure gradient in the air layers close to the skin surface (Grove, Grove, Zerweck, & Pierce, 1999). The probe was touched to skin on the palm side of the dominant forearm at 4 sites between 4 and 10 cm below the inside of the elbow for 1-2 min to obtain baseline measurements. Three sites were disrupted or "stripped," while one was left undisturbed, as shown in Figure 3. Measurements of the undisturbed area (the control site) indicated basal TEWL levels.

Next, cellophane tape (Tesa, Inc. 4101 Cellophane tape; Charlotte, NC) was applied repeatedly (6-51 times) to disrupted site to remove the superficial layer of cornified skin cells. Tape-stripping stopped when the TEWL level was elevated from the basal level of 5-7 g/m^2h to at least 20 g/m^2h at the disrupted site, or a maximum of 51 strips (Ghadially, Brown, Sequeira-Martin, Feingold, & Elias, 1995). For subjects 20 - 30

years of age, 31 ± 5 strippings were required to meet this criterion (Ghadially, Brown, Sequeira-Martin, Feingold, & Elias, 1995).

In this study, 43.5% of participants (N = 37) reached the maximum of 51 strips. Final TEWL measurements after tape-stripping were unavailable for 6 participants (mean number of strips = 31.5, range 24 – 51). For the remaining 79 participants, 60.8% reached the 20 g/m^{2}h criteria before 51 strips (N = 48, 56.5% of the total sample), and 39.2% did not reach the criteria before 51 strips (N = 31, 36.5% of the total sample). Among the participants who reached the 20 g/m^{2}h before the 51 strip maximum, 30.4 ± 9.26 strippings were required to reach the criterion. Mean baseline TEWL was higher for participants who did not reach the criterion (M = 7.52 g/m^{2}h, SD = 2.76) compared to participants who reached the criterion (M = 6.27 g/m^{2}h, SD = 1.96), $F(1,77) = 5.60$, p = .02. Mean TEWL after tape-stripping was 14.34 g/m^{2}h (SD = 3.57) for participants who did not reach the criterion, and 28.52 g/m^{2}h (SD = 13.62) for participants who reached the criterion. The proportion of participants who met criterion did not differ between the three experimental groups, $\chi^2(2) = 2.19$, p = .34.

Recovery of the skin barrier following tape-stripping was 50 - 60% complete 1 h later when assessed between 10:00 AM and 4:00 PM (Denda & Tsuchiya, 2000). TEWL measurements were taken at 60, 90, and 120 min after barrier disruption (Figure 2), corresponding to 35, 65, and 95 min relative to onset of the tasks. TEWL was collected from three sites on the arm (see Figure 2).

Ambient temperature and humidity influence TEWL measures, with increased temperature and humidity resulting in higher TEWL (Fullerton & Serup, 1995). During

the study, room temperature was between 20 – 27.8º C (68 – 82º F), and relative humidity was between 11 – 67%. In addition, TEWL measures were corrected as discussed below.

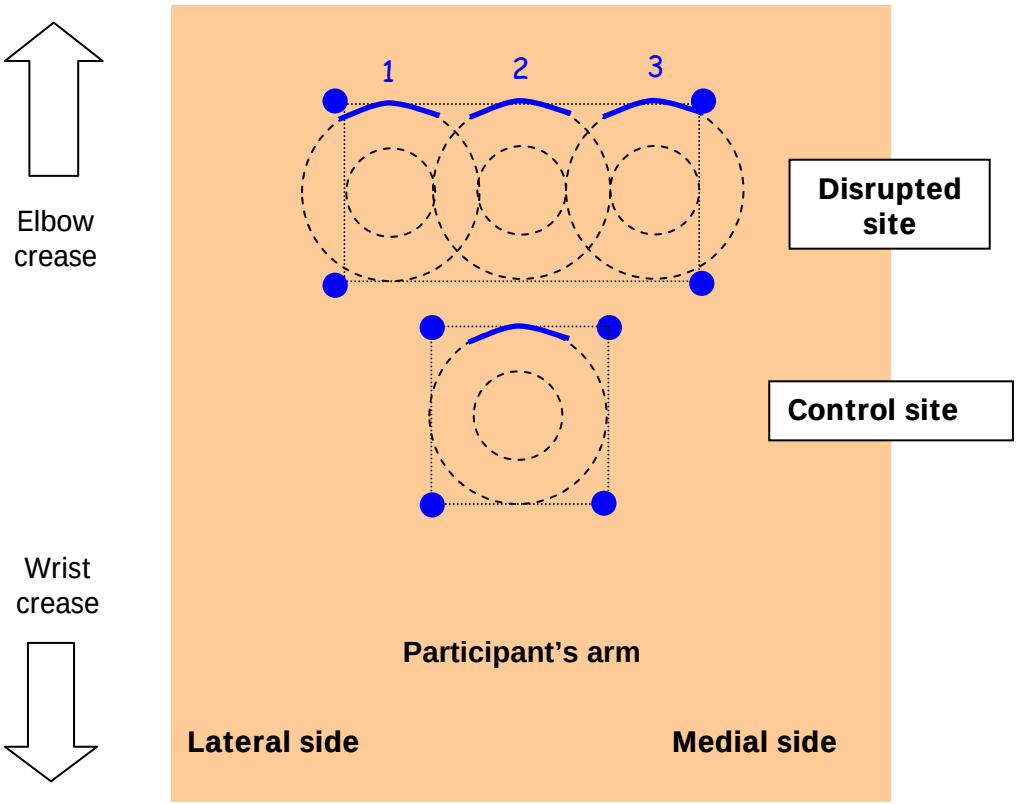

Figure 3. Diagram of tape-stripping sites. Each concentric circle is a tape-stripping site, with blue color indicating markings on the arm. The large 1 in x 2 in area was disrupted (tape-stripped), and the smaller 1 in x 1 in site was left undisturbed for control measurements. The inner portion of the concentric circle was measured using the TEWL probe.

Data analyses

Data transformations

TEWL measures

Raw TEWL values were used to compute percent recovery. Across all analyses using TEWL, skin barrier recovery was conceptualized as a percentage of baseline based on the formula used in several studies (Altemus et al., 2001; Denda & Tsuchiya, 2000):

$$\text{Skin Barrier Recovery}_{it} = \frac{\text{TEWL}_{i1} - \text{TEWL}_{it}}{\text{TEWL}_{i1} - \text{TEWL}_{i0}} \times 100\%$$

where skin barrier recovery for individual *i* at time *t* is a function of change in TEWL from the final TEWL measurement following the end of tape-stripping (time *1*) to time *t*, divided by the change from baseline unstripped TEWL (time *0*) to the end of tape-stripping at time *1*, multiplied by 100 percent.

Raw TEWL values were also corrected for local probe temperature, which reflects both room temperature and probe temperature fluctuations, with values referenced to a common reference temperature. Corrections were conducted using the following formula (Halkier-Sorensen, Thestrup-Pedersen, & Maibach, 1993):

$$\text{TEWL}_{tc} = 10[\log_{10}(\text{TEWL}_{raw}) + 0.035(30 - \text{T}^{\circ})]$$

In this formula, TEWL_{tc} indicates temperature-corrected TEWL values, TEWL_{raw} indicates uncorrected TEWL values, and T° indicates local probe temperature in °C. TEWL values from the two sites closest to one another were averaged in subsequent analyses.

In addition, each individual provided TEWL measurements from a control site, which reflects fluctuations in basal levels of TEWL at an undisturbed site. The same

temperature correction for raw values was applied to control values. $TEWL_{tc}$ was then corrected for control TEWL measures by subtracting a temperature-corrected control TEWL value ($TEWL_{control}$) from $TEWL_{tc}$. Thus, the temperature- and control-corrected TEWL value ($TEWL_{cc}$) was derived by:

$$TEWL_{cc} = TEWL_{tc} - TEWL_{control}$$

In addition to computing percent recovery and adjusting for temperature and control values, a key issue in reducing the TEWL data was determining whether to aggregate across three sites, or two sites closest in value in accordance with previous studies. To aid in this determination, I examined the reliability of using three sites vs. the two closest sites in the entire sample (99 participants). Classical test theory posits that increased items generally results in increased reliability (Kamarck, 1992), reflected in higher Cronbach's alpha statistics and higher intraclass correlations.

Table 1 shows Cronbach's α statistics computed for TEWL measurements at the 2 closest sites and all 3 sites for each time point. The table clearly shows that measurements aggregated across the two closest sites showed higher internal consistency compared to all 3 sites. Across occasions of measurement, two sites showed high test-retest reliability (Site 1 single-measurement *ICC* = .91, Site 2 *ICC* = .86) and one site showed adequate test-retest reliability (Site 3 *ICC* = .77). Based on these results, the remaining TEWL results used data from the 2 closest sites.

Cardiovascular measures

Measures of HR, SBP, and DBP were taken every 2 min during each 10 min measurement period (see *Cardiovascular measures* in the *Procedure* section and Figure 2). Measurements were aggregated within each measurement period to increase

Measurement occasion	Cronbach's α	
	2 closest sites	3 sites
Baseline	.99	.97
Post tape-stripping	.96	.88
1 h post	.93	.71
1.5 h post	.95	.77
2 h post	.96	.82

Table 1. Cronbach's α statistics for TEWL measurements, based on the 2 closest sites or all 3 sites.

reliability, in accordance with psychometric principles (Kamarck, 1992; Kamarck & Lovallo, 2003). Change scores were computed by subtracting baseline measures from post-task measures. When change scores were highly correlated with baseline measures, residualized changes scores were computed and used in subsequent analyses (Llabre, Spitzer, Saab, Ironson, & Schneiderman, 1991).

Cortisol measures

Raw cortisol values were used in subsequent analyses as they did not demonstrate significant skew. Seven of the eight salivary cortisol samples (-15, 10, 30, 45, 60, 75, and 90 min relative to the start of the tasks) were integrated into a single representation of cortisol reactivity by computing area under the curve with respect to increase (AUC_I) using the formula described by Pruessner and colleagues (Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003):

For n measurements m_i at times t_i ($i = 0,..., n$):

$$AUC_I = \left(\sum_{i=1}^{n-1} \frac{(m_{(i+1)} + m_i)t_i}{2} \right) - \left(m_1 \cdot \sum_{i=1}^{n-1} t_i \right)$$

The AUC_I reflects time-dependent change in cortisol relative to baseline, with higher numbers representing larger increases from baseline. In cases where cortisol measures were missing, AUC_I was still computed if the -15 and + 90 min samples were intact, as the formula can accommodate variations in time between measurements.

Statistical software

Throughout the data analyses, SPSS 13 (SPSS, 2004) was used for all descriptive statistics and general linear models. LISREL 8.54 (Joreskog & Sorbom, 1996) was used for multi-group multilevel models, and HLM 5.2 (Raudenbush, Bryk, Cheong, &

Congdon, 2004) was used for full-sample multilevel models. The distinction between full-sample and multi-group multilevel models is discussed below. Effect sizes are reported as *r*'s.

Multilevel modeling

Multilevel modeling provides several advantages over general linear models, including increased power by accommodating missing data. In addition, multilevel models reduce measurement error due to variable timing of measurements by treating measures as if they occurred in real time, rather than aggregating measures taken several minutes apart in different individuals and treating them like the same measurement point. In addition, multilevel models allow for specifying a model of change beyond linear change. In these models, a model of change is specified at the measurement level (level-1); for instance, cortisol was modeled at level-1 as a function of occasion of measurements nested within individuals. Across all the analyses, time was a level-1 predictor of change over occasions of measurement. In addition, multilevel models estimate intercept and slope (change over time) parameters, and if sufficient variance exists in these parameters, additional individual-level (level-2) predictors can be included to predict the variability in intercepts and slopes. For instance, Group assignment can be used to predict initial levels of cortisol and change over time.

The first step in these models was determining a model of change based on visual inspection of the data. In some cases, change was a linear and quadratic process, and in other cases, it was a linear and cubic process. The next step involved determining where to center the intercept (or initial) values. In some cases, centering the intercept at a baseline value was more appropriate; in others, centering the intercept at a "peak

response" value allowed for more relevant hypothesis testing. In addition to centering the outcome variables, I determined how to set the time scale. Using cortisol as an example, if the time scale is set in minutes, the slope parameter is interpreted as cortisol change per minute. Often it is less cumbersome computationally to change the time scale, for instance, having a time unit represent 15 min or 60 min. Across all the models, Akaike's Information Criterion (*AIC*) was used to evaluate goodness of fit to the data, with smaller values implying better fit (J. D. Singer, 1998).

Two multilevel modeling approaches were used through these analyses: a full-sample approach which included all participants in one model, using group status as a level-2 predictor intercepts and slopes, and a multi-group approach, using a multi-group multilevel model (e.g., Kiecolt-Glaser et al., 2003) with separate but simultaneous analyses for each group and no level-2 predictors. In the latter approach, group effects were examined by statistically comparing intercepts and slopes between the groups.

In the full-sample multilevel analyses, group status was represented by two dummy codes: Stress (0 = none, 1 = stress) and Support (0 = none, 1 = support). Therefore, the No Stress group was represented by Stress = 0 and Support = 0, the Stress group was represented by Stress = 1 and Support = 0, and the Stress + Support group was represented by Stress = 1 and Support = 1.

Primary hypotheses

Hypothesis 1: Stress will delay skin barrier recovery and Hypothesis 2: Support will speed skin barrier recovery

These hypotheses were tested using general linear models and multilevel models. The general linear models included two sets of analyses which differed by selection of

dependent variable. The first set of analyses used percent recovery as described in the *TEWL measures* section above as the dependent variable in a 3 x 3 mixed factor general linear model. The between-subjects factor was Group (No Stress, Stress, Stress + Support) and the repeated measures factor was Time (percent recovery at baseline, 1 h post-stripping, and 2 h post-stripping). A significant Group x Time interaction, in the direction of slower barrier recovery at 1- and 2- h post-stripping for participants in the Stress groups compared to the No Stress group would support Hypothesis 1. A significant Group x Time interaction, in the direction of faster recovery in the Stress + Support group compared to the Stress group would support Hypothesis 2. In addition to the omnibus test, Hypothesis 1 was tested with a 1 *df* planned comparison contrasting the two Stress groups with the No Stress group, and Hypothesis 2 was tested by contrasting the Stress + Support group with the Stress group (Keppel, 1991). Both contrasts used percent recovery at 2 h as the dependent variable. The second set of analyses used corrected TEWL values, described in the *TEWL measures* section as the dependent variable in a 3 x 3 mixed factor general linear model, with the same independent variables of Group and Time.

In addition to using general linear models, Hypothesis 1 and 2 were also tested using multilevel modeling. In these models, percent recovery and corrected TEWL values were the dependent variables. In this case, the level-1 model predicted each occasion of measurement as a function of baseline levels, change over time, and error. In the full-sample multilevel models the level-2 model was specified to predict initial levels and change over time as a function of Group assignment (No Stress, Stress, and Stress + Support) represented by the 2 dummy coded variables of Stress (0,1) and Support (0,1).

A significant effect of the dummy coded Stress variable, in the direction of larger increases in skin barrier recovery (increasing percent recovery, decreasing TEWL) for the No Stress group compared to the Stress and Stress + Support groups would support Hypothesis 1. A significant effect of the dummy coded Support variable, in the direction of larger increases in recovery for the Stress + Support group compared to the Stress group would support Hypothesis 2.

In the multi-group multilevel models, separate intercept and slope parameters were estimated for each group simultaneously. A statistically significant difference between parameters in the No Stress group vs. the Stress and Stress + Support groups, in the direction of greater skin barrier recovery (increasing percent recovery, decreasing TEWL) for the No Stress group vs. the Stress and Stress + Support groups would support Hypothesis 1. A statistically significant difference between parameters in the Stress vs. the Stress + Support groups, in the direction of greater skin barrier recovery for the Stress + Support vs. the Stress group would support Hypothesis 2.

Hypothesis 3: Social support will reduce physiological reactivity

This hypothesis was tested using both cardiovascular and cortisol measures. Both general linear models and multilevel models were used to test *Hypothesis 3*. Cardiovascular measures were aggregated over 7 10-min periods during the session: after the 40 min adaptation, following tape-stripping, during the 10-min preparation period before the tasks, during the tasks, after the tasks, and 20 and 30 min after the tasks. Cortisol measures were taken 8 times during the session: prior to and after tape-stripping, and 10, 30, 45, 60, 75, and 90 min after the beginning of the tasks.

Cardiovascular reactivity. There were no significant increases in cardiovascular measures from the baseline measurement (Cardio 1) to the post-stripping measurement (Cardio 2). Therefore, the change scores used in subsequent analyses were based on changes from the post-stripping measurement (Cardio 2) to the task measurement (Cardio 3, during the task). When change scores were highly correlated with baseline measures, residualized change scores were used.

Change scores served as dependent variables in a one-way general linear model with Group and Gender as between-subjects factors. A significant main effect of Group, in the direction of smaller change scores in the Stress + Support group compared to the Stress group would support Hypothesis 3. In addition to the omnibus test, this hypothesis was tested with a 1 *df* planned comparison contrasting the Stress + Support group with the Stress group.

Additional analyses of cardiovascular reactivity used multilevel models, with occasions of measurement as the level-1 unit, and individuals as the level-2 unit. In these models, cardiovascular measures aggregated across the seven 10 min measurement periods were the dependent variables. For cardiovascular reactivity, the level-1 model predicted each occasion of measurement as a function of peak reactivity during the task, change over time, and error. In the full-sample approach, the level-2 model was specified to predict peak reactivity during the task and change over time as a function of Group assignment (No Stress, Stress, and Stress + Support) represented by the 2 dummy coded variables of Stress (0,1) and Support (0,1). A significant effect of Group in the direction of lower peak reactivity and/or smaller increases in cardiovascular measures for the Stress + Support group compared to the Stress group would support the hypothesis.

In the multi-group multilevel models, separate intercept and slope parameters were estimated for each group simultaneously. A statistically significant difference between parameters in the Stress vs. the Stress + Support group, in the direction of greater cardiovascular reactivity Stress group vs. the Stress + Support group would support Hypothesis 3.

Cortisol reactivity. Cortisol AUC_I was computed as described above and was the dependent variable in a one-factor general linear model, with Group as the between-subjects factor. A significant main effect of Group, in the direction of smaller AUC_I values in the Stress + Support group compared to the Stress group would support the hypothesis. In addition to the omnibus test, this hypothesis was tested with a 1 *df* planned comparison contrasting the Stress + Support group with the Stress group.

Additional analyses of cortisol reactivity used multilevel models, with occasions of measurement as the level-1 unit, and individuals as the level-2 unit. For cortisol reactivity, the level-1 model predicted each occasion of measurement as a function of baseline cortisol (Cortisol 2), change over time, and error. In the full-sample approach, the level-2 model was specified to predict baseline cortisol and change over time as a function of Group assignment (No Stress, Stress, and Stress + Support) represented by the 2 dummy coded variables of Stress (0,1) and Support (0,1). A significant effect of Group in the direction of lower peak cortisol reactivity and/or smaller increases in cortisol over time for the Stress + Support group compared to the Stress group would support the hypothesis.

In the multi-group multilevel models, separate intercept and slope parameters were estimated for each group simultaneously. A statistically significant difference

between parameters in the Stress vs. the Stress + Support group, in the direction of greater cortisol reactivity in the Stress group vs. the Stress + Support group would support Hypothesis 3.

Hypothesis 4: Increased physiological reactivity will be related to delayed skin barrier recovery

Cardiovascular change scores and cortisol AUC_I were included as independent variables in a hierarchical linear regression predicting % skin barrier recovery at 2 h. This allowed for assessing the unique contributions of both sympathetic and glucocorticoid activity in predicting changes in skin barrier recovery. A significant relationship between physiological reactivity (cardiovascular or cortisol) and skin barrier recovery would support the hypothesis.

In addition to using regression, Hypothesis 4 was also tested using multilevel modeling. In these models, percent recovery and corrected TEWL values were the dependent variables, using full-sample approach only. The level-2 model was specified to predict initial levels and change over time as a function of cardiovascular and cortisol reactivity. Larger increases in physiological reactivity, if related to lower levels of skin barrier recovery (smaller percent recovery or higher TEWL) and decreased skin barrier recovery over time supports this hypothesis.

Hypothesis 5: Stress-induced cortisol responses will show distinct and reliable patterns of reactivity from and recovery to baseline.

Latent growth curve modeling is an efficient and informative method for studying change over time (Willett & Sayer, 1994), and is particularly useful for modeling reactivity and recovery (Llabre, Spitzer, Saab, & Schneiderman, 2001). Models were

specified with the 7 repeated measures of cortisol as observed data, and baseline levels, reactivity, and recovery as latent parameters that influenced the observed data. This flexible approach allowed for testing different models of change (e.g., linear, quadratic), and complex structures where latent parameters function as predictors, outcomes, or both. Full-information likelihood structural equation modeling was used to model latent parameters. This method takes full advantage of all available data, even if missing data (due to non-systematic influences) are present. A model of cortisol change that shows significant variance in reactivity and recovery parameters, and small relationships between reactivity and recovery patterns would support the hypothesis.

CHAPTER 3

RESULTS

Overview

I first discuss participant characteristics and group equivalence in the measures. I then discuss manipulation checks, comparing the groups on anxiety and affect, cognitive appraisals of the stressor, and ratings of the confederate. Finally, I describe the primary analyses, which are organized by hypothesis, including ancillary analyses related to skin barrier recovery.

Participant characteristics

Group comparisons

Demographic information for final study sample ($N = 85$) is shown in Table 2. The groups did not significantly differ in age or distributions of gender, education, and ethnicity. Moreover, the groups did not differ in any individual difference measures (Table 3), health behaviors (Table 4), or baseline physiological measures (Table 5), including baseline and post-tape-stripping cortisol, heart rate, systolic and diastolic blood pressure, and mean arterial pressure.

Cortisol was generally high at the beginning of the experiment and decreased over the first two samples. Baseline cortisol was not significantly related to trait positive and negative affect, depressive and anxiety symptoms over the past week, life events, perceived stress or other individual difference variables listed in Table 3 with the

Variable	No Stress ($N = 27$)	Stress ($N = 31$)	Stress + Support ($N = 27$)
Age	23.47 (5.43)	22.63 (4.14)	21.64 (3.67)
# female (% female)	14 (52%)	14 (45%)	16 (59%)
Completed education			
High school	1	2	3
Some college	17	22	14
College/University graduate	4	4	5
Grad/Professional training	4	3	5
Ethnicity			
White	16	20	20
Black	2	5	2
Asian	8	5	4
Native Hawaiian/pacific islander	0	0	1
Other	1	1	0

Note. Values are expressed as means. Values in parentheses are *SD*s.

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

Table 2. Demographic information for final study sample, by group.

Self-report measure (scale range)	No Stress ($N = 25$)	Stress ($N = 31$)	Stress + Support ($N = 27$)
Anger expression (40 – 112)	18.52 (6.37)	21.48 (9.90)	21.81 (10.96)
Fear of negative evaluation (8 – 40)	19.28 (1.58)	21.90 (1.42)	21.56 (1.52)
Hostility total scale (0 – 50)	19.12 (8.08)	20.23 (7.34)	16.96 (8.04)
ISEL: Approval (0 – 30)	25.68 (4.60)	24.58 (4.96)	23.67 (5.36)
ISEL: Belonging (0 – 30)	24.20 (4.08)	24.48 (4.09)	22.44 (5.07)
ISEL: Self-esteem (0 – 30)	22.52 (3.64)	22.61 (3.36)	20.52 (4.18)
ISEL: Tangible (0 – 30)	24.72 (4.15)	24.77 (5.53)	25.11 (4.13)
ISEL: Total (0 – 120)	97.12 (14.00)	96.45 (14.01)	92.74 (16.54)
Life events: total events	4.08 (2.10)	3.61 (2.35)	3.00 (2.17)
LOT: Optimism (0 – 12)	7.72 (2.09)	7.81 (2.57)	7.19 (2.48)
MASQ: Depressive symptoms (12 – 60)	19.32 (7.06)	22.87 (9.68)	23.11 (7.84)
MASQ: Anxious symptoms (11 – 55)	18.08 (6.32)	20.06 (7.34)	20.70 (5.59)
MASQ: Anhedonic symptoms (8 – 40)	12.80 (4.28)	15.23 (6.66)	14.89 (5.25)
MASQ: Anxious arousal (17 – 85)	19.64 (4.68)	20.26 (4.34)	20.59 (4.26)
MASQ: Positive affect (24 – 120)	44.56 (9.17)	45.81 (11.84)	42.56 (9.05)
Social status vs. US (1 – 10)	5.32 (1.25)	5.42 (1.54)	5.08 (1.38)
Social status vs. community (1 – 10)	4.72 (1.28)	5.00 (2.08)	5.41 (1.53)
Social status vs. social group (1 – 10)	3.80 (1.80)	3.90 (1.85)	4.59 (1.76)
Perceived stress (0 – 40)	12.96 (5.01)	14.29 (6.90)	17.81 (7.14)
Loneliness (20 – 80)	32.12 (7.34)	33.03 (9.66)	33.89 (9.78)

Note. Values are expressed as means, with *SD*s in parentheses.

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

Table 3. Individual difference variables in final study sample, by group.

Self-report measure	No Stress ($N = 24$)	Stress ($N = 31$)	Stress + Support ($N = 26$)
Sleep quality (PSQI, 0 - 21)	4.96 (3.03)	4.48 (1.81)	5.12 (2.30)
BMI	25.51 (5.84)	25.74 (6.92)	24.38 (5.41)
Weight change in past week	.33 (1.58)	-0.06 (1.65)	-0.35 (1.13)
Alcohol consumed in past week	2.58 (6.02)	5.19 (11.47)	2.96 (4.33)
Caffeine consumption in past 24 hr	1.38 (1.21)	1.26 (1.73)	1.00 (1.10)
Females only: estimated day of menstrual cycle	13.46 (7.83)	20.00 (17.93)	16.56 (10.84)

Note. Values are expressed as means. Values in parentheses are *SD*s.

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

Table 4. Health behavior variables in final study sample, by group.

Baseline measures	No Stress	Stress	Stress + Support
Salivary cortisol	0.17 (0.07)	0.20 (0.24)	0.25 (0.32)
(log-transformed ng/ml)	$N = 24$	$N = 27$	$N = 27$
Salivary cortisol, after tape-stripping	0.17 (0.14)	0.15 (0.14)	0.18 (0.18)
(log-transformed ng/ml)	$N = 25$	$N = 30$	$N = 27$
Cardiovascular measures	$N = 25$	$N = 27$	$N = 26$
Pulse rate (bpm)	68.52 (12.82)	72.60 (9.52)	68.65 (9.49)
Systolic blood pressure (SBP; mmHg)	107.87 (10.07)	108.25 (11.25)	106.70 (8.13)
Diastolic blood pressure (DBP; mmHg)	64.19 (6.13)	64.71 (7.75)	64.80 (5.40)
Mean arterial pressure (MAP)	80.99 (7.24)	80.76 (9.32)	81.56 (5.90)

Note. Values are expressed as means. Values in parentheses are *SD*s.

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

Table 5. Baseline physiological data for final study sample, by group.

exception of subjective social status. Participants who rated themselves as low in social status relative to other people in the United States showed elevated baseline cortisol, r = -.23, p = .05. Cortisol after tape-stripping was not significantly related to any individual difference measure, including subjective social status.

In terms of menstrual phase, 24 women were classified in the follicular phase, and 19 women were classified in the luteal phase of the menstrual cycle. The distribution of menstrual phase in each group is as follows: No Stress, 9 follicular, 4 luteal; Stress, 8 follicular, 6 luteal; Stress + Support, 7 follicular, 9 luteal. The distribution of menstrual phase did not significantly differ between groups, $\chi^2(2) = 1.90$, p = .29.

Groups differed in several tape-stripping relevant variables (Table 6). There were significant group differences in baseline TEWL across the tape-stripping sites and control site combined, $F(2,82) = 4.06$, p = .02, r = .22. The Stress + Support group showed higher baseline TEWL compared to the No Stress group (95% CI = 0.51 – 2.89), and showed marginally higher baseline TEWL than the Stress group (95% CI = -0.18 – 2.13). Baseline TEWL did not significantly differ between the Stress and No Stress groups (95% CI = -0.43 – 1.88). Although participants in the Stress group required fewer strips to reach the 20 g/m^{2}h criterion compared to the No Stress and Stress + Support group, the difference was marginal, $F(2,82) = 2.92$, p = .06, r = .19.

The groups did not differ in other arm conditions that might explain the differences in baseline TEWL and number of strips to criterion, including arm length, room temperature, room humidity, site temperature, site humidity and TEWL at the disrupted site after tape- stripping. Moreover, distribution of participants across the

Measure	No Stress ($N = 27$)	Stress ($N = 31$)	Stress + Support ($N = 27$)
Arm length (cm)	25.78 (1.78)	25.84 (2.05)	25.00 (3.61)
Number of strips to 20 g/m^{2}h criterion	42.67 (10.07)	35.32 (13.05)	40.78 (12.82)
Room temperature (°F)	74.33 (2.88)	73.87 (2.03)	74.44 (2.23)
Room humidity (%)	33.96 (12.77)	37.90 (11.51)	34.22 (13.65)
Temperature across all sites (°C)	24.75 (1.40)	25.09 (1.08)	24.93 (0.99)
Humidity across all sites	33.02 (14.06)	37.82 (12.66)	35.07 (13.60)
Baseline TEWL, disrupted site (g/m^{2}h)*	5.97 (2.16)	6.71 (2.05)	7.74 (2.64)
Baseline TEWL, control site *	5.29 (2.03)	5.98 (2.23)	6.78 (2.17)
Post tape-stripping TEWL, disrupted site	18.64 (9.71)	21.81 (14.06)	21.98 (6.83)
Month of year (median)	April	July	May

Note. Values are expressed as means. Values in parentheses are *SD*s.

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

Table 6. Tape-stripping-relevant variables for final study sample, by group.

months of the year did not differ between groups, Kruskal-Wallis test $\chi^2(2) = 3.52$, $p = .17$.

Consistent with the literature, there were seasonal differences in baseline TEWL (Rogers, Harding, Mayo, Banks, & Rawlings, 1996). Figure 4A shows baseline TEWL across all the sites was highest during Winter and Fall quarters, followed by Spring quarter, and lowest during Summer quarter, $F(3,81) = 4.13$, $p = .009$, $r = .22$. Even after accounting for quarter of the year, the group differences in baseline TEWL remained significant, $F(2,73) = 5.14$, $p = .008$, $r = .26$, and the quarterly differences in baseline TEWL were significant, $F(3,73) = 4.49$, $p = .006$, $r = .24$. There was no significant Group x quarter interaction.

The seasonal differences in TEWL coincided with seasonal changes in temperature and humidity. Figure 4B shows temperature and humidity measured by the evaporimeter probes across quarters. Measures from the probes were highly correlated with temperature and humidity readings obtained from a separate meter in the room (temperature $r = .66$, $p = .00$; humidity $r = .92$, $p = .00$). Humidity increased from 20% in winter to 50% in summer, while site temperature increased from winter levels throughout spring, summer, and fall. The changes in room temperature and humidity with seasons were due to atmospheric conditions and artificial heating and cooling of the room. Consistent with the literature, lower humidity was related to elevated baseline TEWL ($r = -.37$, $p = .001$), as drier air is related to lower epidermal lipid content, and thus greater water loss. Higher temperature was related to elevated baseline TEWL ($r = .46$, $p = .00$), as warmer conditions are related to greater water loss.

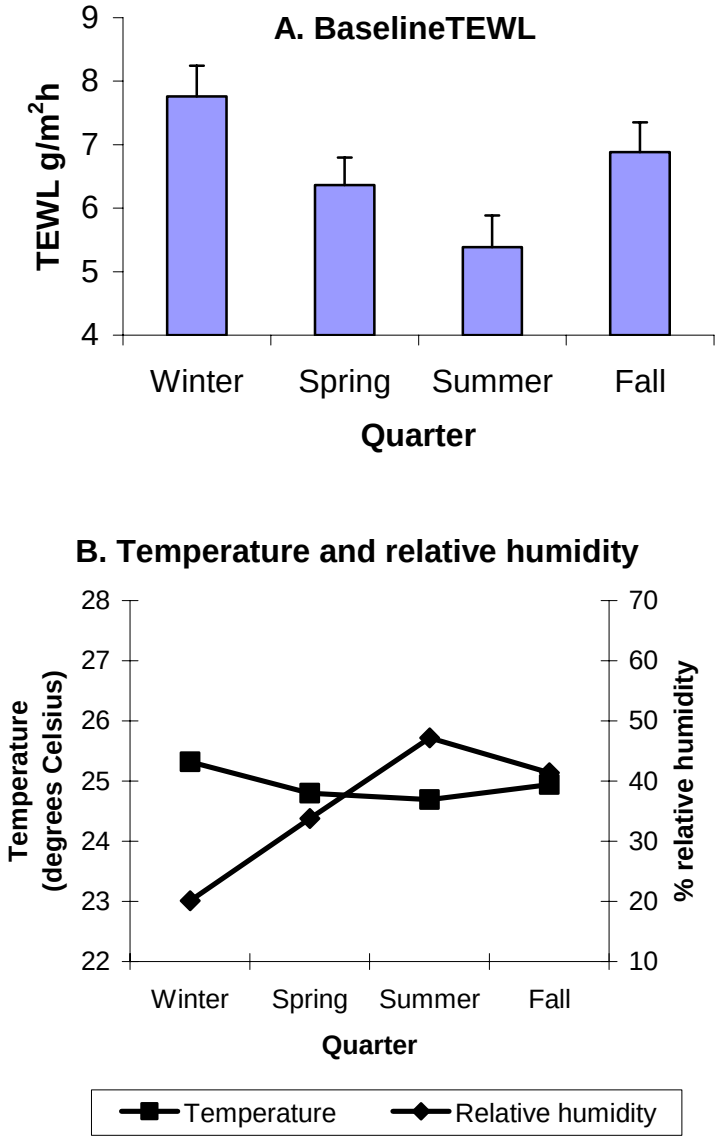

Figure 4. Quarterly variations in A) baseline TEWL, and B) probe temperature and probe relative humidity.

Room temperature and humidity changed during the course of the session by very small intervals, and there were no group differences in room conditions or change in room conditions during the session. I used multilevel modeling to examine changes in room temperature and humidity, with room conditions at 1 h post-stripping as the intercept and time scaled at 15 minute intervals. For room temperature, the intercept was 74.42º F, with a 0.03º F increase every 15 min. Group status was not significantly related to intercept room temperature and change in temperature. The intercept for relative humidity was 34.99%, with a 0.06% decrease every 15 minutes. Similar to temperature, group status was not significantly related to intercept humidity and change in humidity.

Probe temperature and humidity also changed over the course of the session, though the only significant group difference was for change in probe humidity. For temperature at the evaporimeter probes, the intercept was 25.07º C, with a -0.09º C decrease every 15 min. There was no effect of the stress and support manipulations on temperature at the sites. For humidity at the evaporimeter probes, the intercept was 39.82%, with 0.42% increase every 15 minutes. There was no significant effect of the support manipulation on humidity at the sites. However, the Stress manipulation was related to elevated intercept humidity, with exposure to the stressor related to humidity at the sites that was 6.71 percentage points higher than the No Stress group. This would be expected as the stressor probably resulted in increased perspiration.

Although probe humidity increased following the stress task in the Stress and Stress + Support groups, TEWL at the undisturbed control site was not influenced by the experimental manipulations. Multilevel analyses of control site TEWL showed an intercept of 6.80 g/m^2h (*SE* = .89) 1 h post-tape-stripping. There was a significant

decrease in control site TEWL over time, linear slope = -0.16 (*SE* = 0.04), *p* = .00, and a significant increase in control site TEWL over time^3, cubic slope = .007 (*SE* = .002), *p* = .00. There was also significant variance in intercepts, linear slopes, and cubic slopes. Group status did not predict intercepts, linear slopes, or cubic slopes for control site TEWL. Thus, while there was variability in change in control site TEWL over time, group status was unrelated to change in control site TEWL.

In summary, there were no significant differences between groups on demographic, individual difference, or health behavior variables. Groups did differ in baseline TEWL, with higher baseline TEWL in the Stress + Support group compared to the No Stress group. Variables that influence baseline TEWL such as humidity, temperature, and quarter of the year were not significantly different between groups, and accounting for quarter of the year did not change the significant group difference in baseline TEWL. While probe humidity was higher in the Stress groups, this was expected because of increased perspiration. Importantly, TEWL at the control site was unaffected by the stress manipulation despite the change in probe humidity in the Stress groups.

Gender differences

As shown in Table 7, there were no significant differences between men and women on any demographic variables. Table 8 shows that with the exception of higher approval support on the ISEL in women vs. men, there were no significant differences between men and women or gender x group interactions.

Men and women did not differ in any health behaviors (Table 9) except for change in weight and alcohol consumption. For change in weight in the past week, there was a significant interaction between group and gender. The interaction indicated

significant gender differences in change in weight in the past week in the Stress group only, with women reporting weight gain (M = +.71) and men reporting weight loss (M = -.71). Men reported more alcohol consumption in the past week compared to women. There were no other significant gender x group interactions.

Finally, there were some gender differences in baseline physiological measures (Table 10). Men showed elevated baseline SBP and MAP compared to women. Among women, the groups did not differ in baseline heart rate; among men, the Stress group showed a 9-10 beats per minute (bpm) elevation in baseline heart rate compared to the No Stress and Stress + Support groups. There were no significant gender differences in any other physiological measure. Table 11 shows no significant gender differences or gender x group interactions for tape-stripping relevant variables.

Variable	Male ($N = 41$)	Female ($N = 44$)
Age	22.81 (3.69)	22.98 (5.05)
Completed education		
High school	2	4
Some college	26	28
College/University graduate	7	6
Grad/Professional training	6	6
Ethnicity		
White	29	27
Black	1	8
Asian	9	8
Native Hawaiian/pacific islander	1	0
Other	1	1

Note. Values are expressed as means. Values in parentheses are *SD*s.

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

Table 7. Demographic information for final study sample, by gender.

Self-report measure (range)	Male ($N = 40$)	Female ($N = 43$)	Gender x Group *p*-value
Anger expression (40 – 112)	20.70 (8.14)	20.70 (9.98)	.47
Fear of negative evaluation (8 – 40)	20.74 (1.21)	21.28 (1.26)	.76
Hostility total scale (0 – 50)	20.33 (7.80)	17.44 (7.68)	.20
ISEL: Approval (0 – 30)*	23.38 (5.36)	25.77 (4.39)	.87
ISEL: Belonging (0 – 30)	23.48 (4.25)	24.60 (4.52)	.16
ISEL: Self-esteem (0 – 30)	21.14 (3.89)	21.97 (4.17)	.41
ISEL: Tangible (0 – 30)	24.28 (4.30)	25.42 (4.95)	.97
ISEL: Total (0 – 120)	93.20 (13.92)	97.53 (15.48)	.36
Life events: total events	3.38 (2.48)	3.70 (2.01)	.22
LOT: Optimism (0 – 12)	7.63 (1.94)	7.53 (2.77)	.30
MASQ: Depressive symptoms (12 – 60)	21.25 (8.59)	22.47 (8.36)	.47
MASQ: Anxious symptoms (11 – 55)	18.88 (6.34)	20.42 (6.67)	.64
MASQ: Anhedonic symptoms (8 – 40)	14.90 (5.87)	13.91 (5.38)	.33
MASQ: Anxious arousal (17 – 85)	19.75 (4.07)	20.58 (4.67)	.92
MASQ: Positive affect (24 – 120)	46.13 (9.47)	42.74 (10.66)	.29
Social status vs. US (1 – 10)	5.20 (1.40)	5.36 (1.41)	.85
Social status vs. community (1 – 10)	4.98 (1.53)	5.12 (1.85)	.83
Social status vs. social group (1 – 10)	4.10 (1.68)	4.09 (1.96)	.22
Perceived stress (0 – 40)	13.73 (5.85)	15.63 (7.15)	.13
Loneliness (20 – 80)	34.35 (9.36)	31.81 (8.55)	.24

Note. Values are expressed as means. Values in parentheses are *SD*s.

* $p < .05$

Table 8. Individual difference measures in final study sample, by gender.

Self-report measure	Male ($N = 38$)	Female ($N = 43$)	Gender x Group p-value
Sleep quality (PSQI; range 0 – 21)	4.76 (2.51)	4.88 (2.26)	.13
BMI	25.51 (4.65)	24.97 (7.31)	.46
Weight change in past week	-0.13 (1.60)	0.05 (1.40)	.01
Alcohol consumed in past week*	6.96 (10.97)	1.53 (3.38)	.32
Caffeine consumption in past 24 hr	1.32 (1.47)	1.12 (1.33)	.21

Note. Values are expressed as means. Values in parentheses are *SD*s.

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

Table 9. Health behavior variables in final study sample, by gender.

Measure	Male ($N = 33$)	Female ($N = 41$)	Gender x Group interaction
Salivary cortisol, baseline	0.25 (0.35)	0.16 (0.06)	0.44
Salivary cortisol, after tape-stripping	0.20 (0.18)	0.14 (0.13)	0.38
Heart rate	69.48 (8.48)	70.47 (12.65)	0.06
Systolic blood pressure (SBP)***	111.37 (9.20)	103.85 (9.01)	0.10
Diastolic blood pressure (DBP)	65.47 (6.16)	63.68 (6.67)	0.88
Mean arterial pressure (MAP)**	83.37 (6.78)	78.83 (7.69)	0.63

Note. Values are expressed as means. Values in parentheses are *SD*s.

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

Table 10. Baseline physiological data for final study sample, by gender.

Measure	Male ($N = 41$)	Female ($N = 44$)	Gender x Group interaction
Arm length	26.22 (3.04)	24.93 (1.90)	.24
Number of strips to criterion	38.34 (13.26)	40.36 (11.56)	.65
Room temperature	73.44 (1.95)	74.91(2.53)	.46
Room humidity	36.51 (12.07)	34.52(13.15)	.80
Baseline TEWL, disrupted site	7.11 (2.80)	6.51 (1.85)	.64
TEWL following tape-stripping, disrupted	22.59 (13.08)	19.32 (7.96)	.98

Note. Values are expressed as means. Values in parentheses are *SD*s.

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

Table 11. Tape-stripping relevant variables for final study sample, by gender.

Manipulation checks

Affect and anxiety measures throughout the session

Results for affect measured with the PANAS and anxiety symptoms measured with the STAI are shown in Figure 5. Positive affect showed a significant decrease over the session, $F(5,345) = 24.45$, $p = .00$, $r = .26$. There were no significant interactions between change in positive affect and group or gender, no significant main effects of group or gender or interactions between group and gender on positive affect averaged across the session.

Negative affect showed a significant change over the session, $F(5,345) = 9.42$, $p = .00$, $r = .16$. In addition, a significant group x time interaction, $F(10,345) = 1.92$, $p = .04$, $r = .07$, indicated that the Stress and Stress + Support groups showed a significant increase in negative affect from pre-task to post-task, followed by a decline in negative affect through the rest of the session, as shown in Figure 5. There was no significant gender x time interaction, or main effects for group and gender for negative affect averaged across the session. There was a significant group x gender interaction for negative affect across the session, $F(2,70) = 3.48$, $p = .04$, $r = .22$, indicating that male participants in the No Stress condition reported greater overall negative affect ($M = 13.22$) compared to female participants in the No Stress condition ($M = 10.61$), while there were no gender differences in overall negative affect for the Stress and Stress + Support groups.

State anxiety symptoms also showed significant change over the session, $F(5,350) = 7.12$, $p = .09$, $r = .14$. However, the group x time interaction only approached

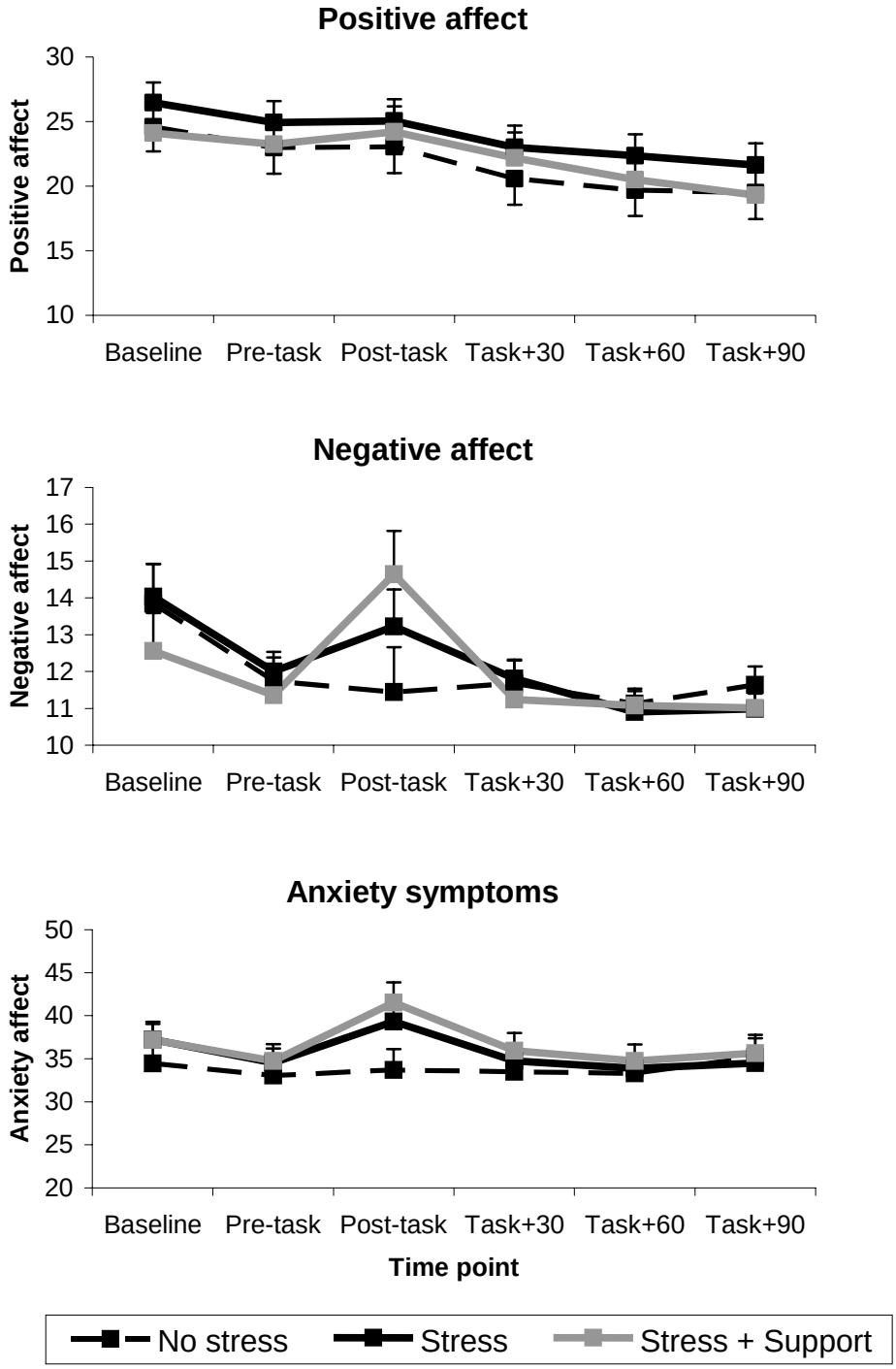

Figure 5. Positive affect, negative affect, and state anxiety symptoms across the session, by group.

significance, $F(10,350) = 1.78$, $p = .06$, $r = .07$. Figure 5 suggests that the Stress and Stress + Support groups showed an increase in anxiety from the pre-task to the post-task measurement. There were no other significant main effects or interactions.

Affect and anxiety measures before and after the task

Results for all manipulation check variables are shown in Table 12. Across all the manipulation checks, there were no significant gender differences or gender x group interactions. While positive affect change from pre- to post-task did not differ between the three groups, $F < 1$, there were group differences in negative affect change from pre-task to post-task, $F(2,76) = 3.67$, $p = .03$, $r = .22$. Specifically, the Stress + Support group showed a significant increase in negative affect pre- to post-task compared to the No Stress group (95% *CI* for difference: 0.30 – 6.56), while the Stress group did not significantly differ from the other two groups. A similar result was found for anxiety symptoms, with a significant main effect of Group, $F(2,77) = 4.74$, $p = .01$, $r = .24$ and the same significant difference between the Stress + Support group and the No Stress group (95% *CI* for difference: 1.16 – 11.39). The Stress group did show a larger increase in anxiety symptoms pre- to post-task compared to the No Stress group, but this approached significance (95% *CI* for difference: -0.45 – 9.39), and there were no differences between the Stress and Stress + Support groups.

Cognitive appraisals before and after the task

Expectations of threat and control were measured just after the task instructions, and once again prior to the task; change in these expectations across the two measurement points did not differ between the three groups. The Stress and Stress + Support groups reported more negative thoughts on the SSPS regarding the speech task

compared to the No Stress group, $F(2,80) = 6.08$, $p = .003$, $r = .27$. Follow-up comparisons indicated that the Stress and Stress + Support groups reported more negative thoughts, (Stress – No Stress 95% *CI*: 0.09 – 1.14, Stress + Support – No Stress 95% *CI*: 0.22 – 1.51). The Stress and Stress + Support groups did not differ in negative thoughts (95% *CI*: -0.76 – 0.46). The groups did not differ in number of self-reported positive thoughts on the SSPS, $F < 1$.

The Stress and Stress + Support groups also reported feeling more perceived stress during the task compared to the No Stress group, $F(2,79) = 30.62$, $p = .00$, $r = .53$ Follow-up comparisons indicated that the Stress and Stress + Support groups reported greater perceived stress compared to the No Stress group (Stress – No Stress 95% *CI*: 1.91 – 3.95, Stress + Support – No Stress 95% *CI*: 1.84 – 3.95). The Stress and Stress + Support groups did not differ in perceived stress ratings (95% *CI*: -0.95 – 1.02).

In addition to higher perceived stress, the Stress and Stress + Support groups reported lower performance satisfaction compared to the No Stress group, $F(2,79) = 10.72$, $p = .00$, $r = .35$. Follow-up comparisons confirmed that the Stress and Stress + Support groups reported feeling less satisfied with their performance compared to the No Stress group (Stress – No Stress 95% *CI*: -2.87 – 0.83, Stress + Support – No Stress 95% *CI*: -2.58 – -0.48). The Stress and Stress + Support groups did not differ in performance satisfaction ratings (95% *CI*: -0.67 – 1.31).

The Stress and Stress + Support groups also reported less perceived control, $F(2,79) = 13.12$, $p = .00$, $r = .38$, and greater perceived helplessness compared to the No Stress group, $F(2,79) = 24.30$, $p = .00$, $r = .49$. Follow-up comparisons confirmed the group effect for perceived control (Stress – No Stress 95% *CI*: -2.72 - -0.68, Stress +

Self-report measure (range)	No Stress	Stress	Stress + Support
Pre-to post-task change			
Positive affect	0.26 (4.98)	0.19 (3.65)	0.81 (5.08)
Negative affect*	-0.27 (2.12)	1.19 (3.26)	3.15 (6.53)
Anxiety symptoms	0.30 (5.49)	4.77 (6.40)	6.58 (9.41)
Threat appraisal	0.00 (0.29)	-0.42 (1.12)	-0.27 (0.72)
Coping appraisal	-0.04 (0.20)	0.19 (1.05)	0.31 (0.84)
Threat appraisal – before preparation*** (1 – 7)	1.38 (0.31)	4.00 (0.27)	4.42 (0.30)
Threat appraisal – before speech*** (1 – 7)	1.40 (0.29)	3.58 (0.26)	4.19 (0.28)
Coping appraisal – before preparation*** (1 – 7)	6.79 (0.28)	4.87 (0.25)	4.54 (0.27)
Coping appraisal – before speech*** (1 – 7)	6.72 (0.25)	5.07 (0.22)	4.89 (0.24)
SSPS: Negative thoughts** (0 – 5)	0.65 (0.74)	1.36 (1.00)	1.51 (1.06)
SSPS: Positive thoughts (0 – 5)	3.95 (0.55)	3.92 (0.68)	3.88 (0.73)
Post-task ratings (1 – 7)			
Stress rating***	1.58 (1.28)	4.52 (1.61)	4.48 (1.65)
Performance satisfaction ***	5.75 (1.15)	3.90 (1.72)	4.22 (1.60)
Perceived control ***	6.38 (1.28)	4.68 (1.78)	4.30 (1.44)
Perceived helplessness ***	1.25 (0.85)	3.94 (1.63)	3.56 (1.76)
Perceived stress during math	-	3.16 (1.75)	3.59 (1.47)
Perceived control during math*	-	5.68 (1.28)	4.89 (1.67)
Perceived helplessness during math	-	2.71 (1.77)	2.81 (1.69)

Note. Values are expressed as means. Values in parentheses are *SD*s. SSPS = Self-Statements about Public Speaking.

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

Table 12. Manipulation check variables, by group.

Support – No Stress 95% *CI*: -3.13 – -1.03) and perceived helplessness (Stress – No Stress 95% *CI*: 1.69 – 3.68, Stress + Support – No Stress 95% *CI*: 1.28 – 3.33). The Stress and Stress + Support groups did not differ in perceived control ratings (95% *CI*: -0.61 – 1.37) or perceived helplessness (95% *CI*: -0.58 – 1.34).

The final set of ratings was perceived stress, helplessness, and control during the math task, and these analyses involved comparing the Stress and Stress + Support groups only. The Stress and Stress + Support group did not differ in perceived stress, $F(1,56) = 1.01$, *ns*, or perceived helplessness during the math task, $F < 1$. However, the Stress group reported more perceived control compared to the Stress + Support group, $F(1,56) = 4.14$, $p = .05$, $r = .26$.

Confederate ratings

Figure 6 shows the average pattern of ratings of the confederate on the IAS-R for participants in the Stress + Support group. For comparison, ratings of three types of tape-recorded supportive statements (Provoking, Neutral, and Supportive) from unseen confederates in a previous study (Gallo et al., 2000) are shown in Figure 6. The IAS-R circumplex is conceptualized around two primary dimensions, love and status, corresponding to the horizontal and vertical axes respectively. Comparisons with the Gallo et al. study suggest that confederates in this study were rated as similar in status (Aloof-Introverted, Unassured-Submissive, Unassuming-Ingenuous) and lower in love (Warm-Agreeable, Gregarious-Extraverted, Assured-Dominant) as the tape-recorded support used by Gallo and colleagues. Thus, the support from confederates was more similar to the Neutral support rather than the Supportive support or Provoking support in the Gallo et al. study. There were no gender differences in ratings of the same-sex

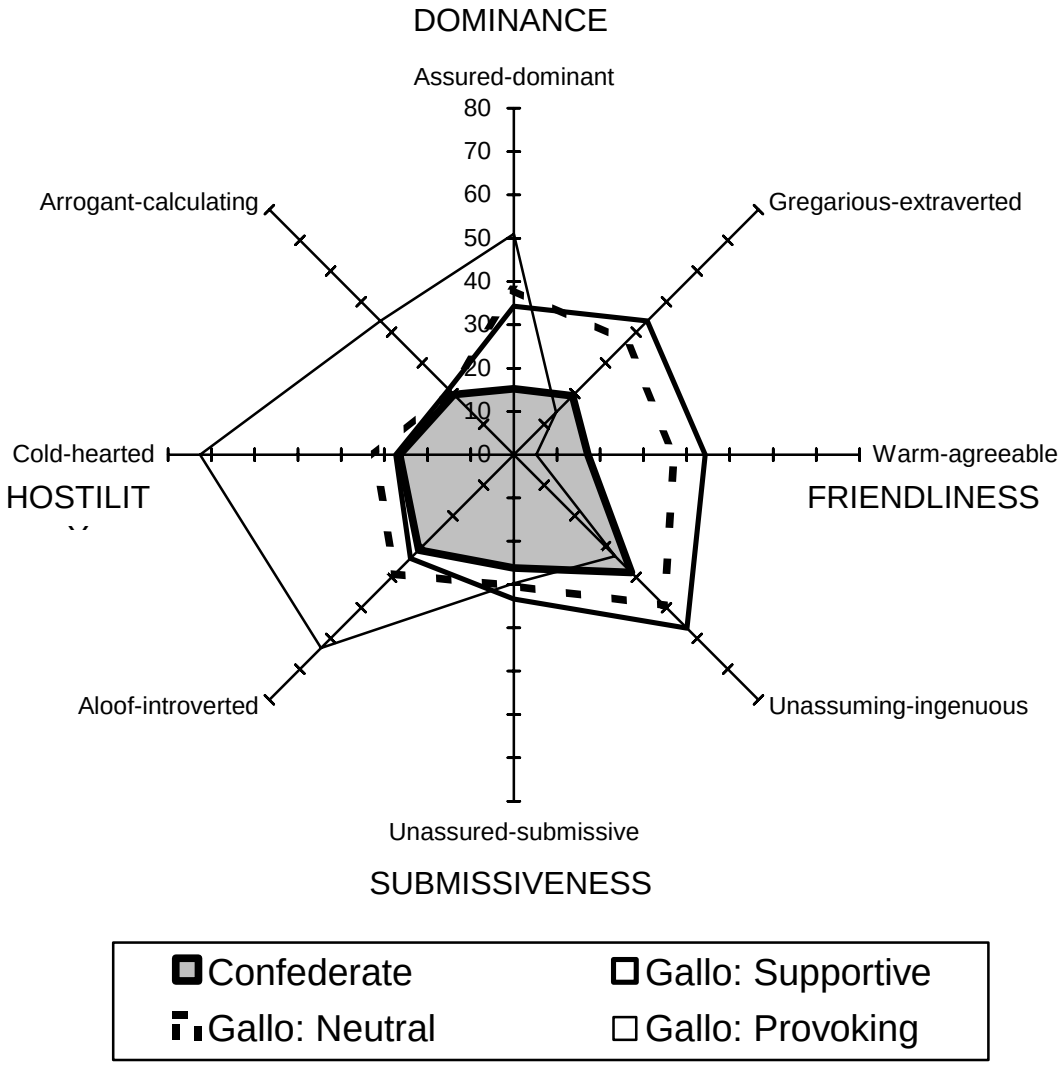

Figure 6. Ratings of the confederate on the Interpersonal Adjective Scale – Revised, in comparison to ratings of tape-recorded support by participants in the study by Gallo et al. 2000.

confederate in this study (Table 13), with the exception of higher ratings on the Arrogant-Calculating scale for the male confederate compared to the female confederates, $F(1,25) = 17.76$, $p = .00$, $r = .64$. Higher ratings were also observed on the Assured-Dominant scale for the male confederate compared to the female confederates, but this approached significance, $p = .06$.

Ratings of the confederate on the USII for participants in the Stress + Support group are also shown in Table 13. In general, participants were rated as not unsupportive (the items are on a 0 – 4 scale), and there were no gender differences in the degree of unsupportiveness of male vs. female confederates. For more meaningful comparisons, participants in the Stress + Support group rated confederates 0.5 – 1 *SD* below the average of the initial validation sample (Ingram, Betz, Mindes, Schmitt, & Smith, 2001) on all of the subscales and the total scale. In other words, interactions with the confederates were rated as less unsupportive compared to self-reports of interactions with supportive others (i.e., family and friends) in similar student samples.

Manipulation check summary

In summary, regardless of the presence or absence of support, the stress task increased negative affect, anxiety, negative thoughts about public speaking, stress ratings post-task, and helplessness ratings; and reduced satisfaction with performance and perceptions of control. There were no differences between groups for perceived threat and coping prior to the task or positive thoughts about public speaking. Thus, while the Stress manipulation was effective in increasing negative affect and stressful cognitive appraisals, the Support manipulation was not effective in altering those responses. In fact, the only significant difference between the Stress and Stress + Support groups was for

perceived control during the math task, with the Stress group reporting greater perceived control during the math task compared to the Stress + Support group.

Ratings of the confederate on the IAS-R and the USII suggest that participants perceived the confederate as "not unsupportive," but similar to the Neutral support provided in the Gallo et al. study. There were no significant gender differences on any IAS-R subscale except for higher ratings on the Arrogant-Calculating scale for the male confederate compared to the female confederates. Interactions with the confederates were rated as low in unsupportiveness. However, taken together with the manipulation check results, support from the confederate was perceived as more neutral rather than supportive, and did not effectively change affect, anxiety, or cognitive appraisals.

Scale and subscale	Male	Female
Interpersonal Adjective Scale – Revised		
Assured-dominant $p = .06$	20.76 (3.62)	11.37 (3.00)
Gregarious-extraverted	22.77 (7.31)	16.78 (6.06)
Warm-agreeable	18.99 (8.60)	15.86 (7.13)
Unassuming-ingenuous	35.28 (6.84)	40.59 (5.67)
Unassured-submissive	29.49 (3.22)	23.85 (2.67)
Aloof-introverted	35.52 (5.73)	28.18 (4.75)
Cold-hearted	33.51 (5.53)	21.61 (4.58)
Arrogant-calculating***	28.76 (2.81)	13.38 (2.33)
Unsupportive Social Interactions Inventory		
Total scale (*1.27, 1.14*)	0.46 (0.14)	0.51 (0.11)
Distancing (*0.84, 0.88*)	0.32 (0.38)	0.23 (0.34)
Bumbling (*1.33, 1.10*)	0.38 (0.48)	0.41 (0.39)
Minimizing (*1.57, 1.49*)	0.97 (0.65)	1.02 (1.16)
Blaming (*1.35, 1.10*)	0.18 (0.29)	0.33 (0.46)

Note. IAS-R ratings are standardized *t* scores ($M = 40$, $SD = 10$). Ratings in italics are from the Study 1 and Study 2 of the USII validation samples.

Table 13. Participant ratings of the confederate, by gender.

Primary analyses

Hypothesis 1: Stress exposure will result in delayed skin barrier recovery and Hypothesis 2: Social support will speed skin barrier recovery

These analyses are organized into two sections. First, I present results from general linear models including time (1, 1.5, 2 h post-stripping) and Group (No Stress, Stress, Stress + Support) as independent variables. In addition, results are presented using 1 *df* planned comparisons testing Hypothesis 1 (No Stress vs. Stress and Stress + Support) and Hypothesis 2 (Stress vs. Stress + Support). Next, I present multilevel models including time as a level-1 predictor and Group as a level-2 predictor. Prior to analyses, I screened the data for outliers that exceeded ± 3 *SD* relative to the mean, resulting in the removal of 1 participant who actually showed negative values for percent recovery. Across these analyses percent recovery, temperature-corrected, and temperature- and control-corrected TEWL were used as dependent variables in separate analyses.

General linear models testing Hypotheses 1 and 2

Table 14 shows *F*-statistics and *p*-values for each set of analyses. Results for percent recovery showed no significant effects of Group or a Group x Time interaction. Skin barrier recovery significantly increased over time, $r = .20$ - $.51$. Planned comparisons testing Hypotheses 1 and 2 were not significant.

Results for temperature corrected TEWL showed a trend for Group, $r = .18$, a significant effect of Time, $r = .23$, but no significant Group x Time interaction.

Independent variable	Percent recovery [a]			Temperature-corrected TEWL [b]			Temperature-, control-corrected TEWL[c]		
	F	*p*	*r*	*F*	*p*	*r*	*F*	*p*	*r*
Group	0.93	.40	.11	2.41	.10	.18	1.92	.15	.16
Time	75.7	.00	.51	11.2	.00	.23	8.85	.00	.20
Group x Time	0.63	.71	.05	0.11	.99	.02	0.91	.30	.07
Hypothesis 1 planned comparison	1.87	.18	.16	4.19	.04	.23	2.04	.16	.16
Hypothesis 2 planned comparison	0.001	.97	.00	3.60	.06	.21	0.15	.70	.04

Note.
[a] *df*(Group, Error) = 2,70; *df*(Time, Error) = 3,210; *df* (Group X Time, Error) = 6,210; planned comparisons *df*(1,71)
[b] *df*(Group, Error) = 2,70; *df*(Time, Error) = 3,210; *df* (Group X Time, Error) = 6,210; planned comparisons *df*(1,77)
[c] *df*(Group, Error) = 2,70; *df*(Time, Error) = 3,210; *df* (Group X Time, Error) = 6,210, planned comparisons *df*(1,77)

Table 14. *F*-table for general linear models predicting skin barrier recovery, with group and time as independent variables.

Follow-up comparisons indicated that the No Stress group showed lower temperature-corrected TEWL values compared to the Stress + Support group (95% *CI* = -0.21 – -0.01), with no significant differences between the No Stress and Stress group, or the Stress and Stress + Support group. On the other hand, the significant planned comparison testing Hypothesis 1 showed that the No Stress group had lower temperature-corrected TEWL compared to the Stress and Stress + Support groups combined (No Stress – Stress & Stress + Support 95% CI = -0.18 - -0.002). The planned comparison for Hypothesis 2 approached significance, but the direction of the effect was opposite of the predicted direction, suggesting that the Stress group had lower TEWL values compared to the Stress + Support group (95% CI = -0.18 - 0.00).

Results for temperature- and control-corrected TEWL showed no effect for Group, a significant effect of Time, $r = .20$, and no significant Group x Time interaction.

Multilevel models testing Hypotheses 1 and 2

Additional analyses of skin barrier recovery used multilevel models, which offered the advantages of accommodating missing data and variable measurement times. In this study, 2.9% of the total possible measurement points were missing. Because the post-tape-stripping measurement is necessary for computing percent recovery, having a missing post-tape-stripping measurement results in a greater proportion of missing data; as a result, 6.8% of the possible percent recovery measurement points were missing. In addition, while measurements were intended to be taken at exactly 1 h, 1.5 h, and 2 h post-stripping, there was variability in the timing of measurement, which can be accommodated by multilevel models.

Inspection of the raw data indicated that models specifying linear and quadratic (u-shaped) change were most parsimonious for percent recovery measures of wound healing, and models specifying linear and cubic (~ - shaped) change were most parsimonious for corrected TEWL measures of wound healing. The level-1 predictor in these models was the time of measurement relative to the 1 h post-tape-stripping TEWL measurement, which served as the intercept value. Centering the intercept at the 1 h post-tape-stripping TEWL measurement rather than at baseline allowed for comparing skin barrier recovery after the task between groups. In addition, centering the intercept at the true initial percent recovery value would result in an initial value of 0% across all participants, which has no variability and would not take advantage of the strengths of multilevel modeling. Time values were scaled so that linear slopes represented change per 1 h of time (i.e., -1 h, 0, .5 h, 1 h).

As discussed in the *Data analyses* section, two multilevel modeling approaches were used to evaluate wound healing: The full-sample and the multi-group approach. This section is organized by type of measure, with percent recovery measures followed by corrected TEWL measures. Within each section, results for the full-sample approach are presented first, followed by results for the multi-group approach.

Multilevel modeling percent recovery. Models with intercepts specified as random, and linear and quadratic slopes specified as random provided better fit (*AIC* = 2676) compared with models with intercepts specified as random and slopes specified as fixed (linear and quadratic fixed *AIC* = 2760; linear fixed, quadratic random *AIC* = 2716; linear random, quadratic fixed *AIC* = 2681) or models with only intercepts specified as random and no slopes specified (*AIC* = 2875).

Table 15 shows level-1 parameter estimates for percent recovery using the full-sample approach. The disrupted site was 32% healed 1 h after tape-stripping (35 min after task onset). Wound healing increased 16% per h, which was countered by an 11% decline per h^2. There was significant variance in intercepts and slopes, justifying including group status as a level-2 predictor of intercepts and slopes. Intercepts and slopes were highly correlated, such that greater healing at 1 h after tape-stripping was related to larger linear increases in healing and larger quadratic decreases in healing. Larger linear increases in healing were related to larger quadratic decreases in healing. All parameters showed low to moderate reliability.

After including group status as level-2 predictors of intercepts and slopes, the direction of results supported Hypotheses 1 and 2, with delayed healing for the Stress and Stress + Support groups vs. the No Stress group, and slightly faster healing for the Stress + Support group vs. the Stress group (Table 16). However, no group effects were significant. The only effect that approached significance was a slower linear rate of healing for the Stress and Stress + Support groups vs. the No Stress group ($p = .11$), apparent in Figure 7A. Including group status accounted for 1% of intercept variance, 7% of linear slope variance, 5% of quadratic slope variance, and 1% of within-subject variance.

The multi-group analyses supported *Hypothesis 1*. Table 17 shows separate level-1 parameter estimates for each group, indicating no significant slope variances, probably due to reduced sample size from conducting separate analyses for each group. Therefore, the parameters were modeled with intercepts random and linear and quadratic slopes fixed, shown in Table 18. In addition, a series of multivariate and univariate contrasts

Parameter	Means	Variances	Reliability
Intercept	31.80*** (2.99)	526.78*** [22.96]	.76
Linear slope	15.96*** (1.56)	99.23*** [9.96]	.42
Quadratic slope	-10.69*** (2.26)	123.61*** [11.12]	.28
Within-subjects variance		235.37*** [15.34]	

Note. Intercept values reflect TEWL (g/m^2h) 1 h after tape-stripping. Linear slopes reflect change in TEWL per 1 h of time, and quadratic slopes reflect change in TEWL per 1 h of $time^2$. Correlation between intercepts and linear slopes = .92; intercepts and quadratic slopes = -.94; linear and quadratic slopes = -.75. Values in parentheses are standard errors and values in brackets are standard deviations.

*** $p < .001$.

Table 15. Initial level-1 full-sample multilevel modeling parameter estimates for percent recovery.

Parameter	Means	Variances	Reliability
Intercept	36.48*** (3.91)	519.04*** [22.78]	.76
Stress group	-7.79 (6.89)		
Support group	3.03 (7.84)		
Linear slope	20.78*** (2.73)	91.96*** [9.59]	.41
Stress group	-5.84 (3.67) $p = .11$		
Support group	-3.05 (3.66)		
Quadratic slope	-14.17*** (2.93)	117.65*** [10.85]	.28
Stress group	4.99 (4.54)		
Support group	-1.80 (5.82)		
Within-subjects variance		231.88*** [15.23]	

Note. Intercept values reflect TEWL (g/m^2h) 1 h after tape-stripping. Linear slopes reflect change in TEWL per 1 h of time, and quadratic slopes reflect change in TEWL per 1 h of $time^2$. Stress group is coded 0 = No Stress, 1 = Stress; Support group is coded 0 = No Support, 1 = Support. Correlation between intercepts and linear slopes = .93; intercepts and quadratic slopes = -.95; linear and quadratic slopes = -.78. Values in parentheses are standard errors and values in brackets are standard deviations.

*** $p < .001$.

Table 16. Final full-sample multilevel modeling parameter estimates for % skin barrier recovery, including dummy-coded group status (stress group and support group) as level-2 predictors of intercepts and slopes.

Parameter	No Stress	Stress	Stress + Support
Means			
Intercept	38.76*** (3.54)	30.85*** (5.55)	33.53*** (5.06)
Linear slope	23.16*** (2.62)	15.94*** (3.15)	14.55*** (3.06)
Quadratic slope	-15.06*** (3.50)	-9.43** (4.09)	-11.47*** (4.42)
Variances			
Intercept	146.30 (94.52), $p = .12$	656.05** (242.38)	358.09 (187.48), $p = .06$
Linear slope	43.32 (53.11)	-5.74 (84.83)	-69.43 (77.09)
Quadratic slope	-25.07 (105.23)	16.89 (144.18)	75.75 (151.71)
Within-subject	206.92*** (55.02)	358.38*** (81.53)	386.58*** (97.40)

Note. Intercept values reflect TEWL (g/m^2h) 1 h after tape-stripping. Linear slopes reflect change in TEWL per 1 h of time, and quadratic slopes reflect change in TEWL per 1 h of $time^2$. Values in parentheses are standard errors.

*** $p < .001$.

Table 17. Initial level-1 multi-group multilevel modeling parameter estimates for % skin barrier recovery.

Parameter	No Stress	Stress	Stress + Support
Means			
Intercept	38.94*** (3.25)	31.30*** (4.52)	33.91*** (4.06)
Linear slope[a]	23.21*** (2.15)	16.10*** (2.67)	14.72*** (2.88)
Quadratic slope	-15.34*** (3.56)	-9.40* (3.89)	-12.13** (3.92)
Variances			
Intercept	99.20* (46.39)	323.62** (112.22)	160.99* (75.47)
Within-subject	227.18*** (38.52)	415.04*** (66.52)	375.61*** (63.24)

Note. Intercept values reflect TEWL (g/m^2h) 1 h after tape-stripping. Linear slopes reflect change in TEWL per 1 h of time, and quadratic slopes reflect change in TEWL per 1 h of $time^2$. Values in parentheses are standard errors. Multivariate contrasts are described below; significant univariate contrasts are indicated by subscripts. Multivariate contrasts: No Stress vs. Stress and Stress + Support: $\chi^2(3) = 12.05$, $p = .007$. No Stress vs. Stress: $\chi^2(3) = 8.77$, $p = .03$. No Stress vs. Stress + Support: $\chi^2(3) = 8.69$, $p = .03$. Stress vs. Stress + Support: $\chi^2(3) = 0.30$, $p = .96$.

[a] Univariate contrasts of linear slopes: No Stress vs. Stress and Stress + Support: $\chi^2(1) = 7.16$, $p = .007$. No Stress vs. Stress, $\chi^2(1) = 4.29$, $p = .04$. No Stress vs. Stress + Support, $\chi^2(1) = 5.57$, $p = .02$.

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

Table 18. Final level-1 multi-group multilevel modeling parameter estimates for % skin barrier recovery, including multivariate and univariate contrasts.

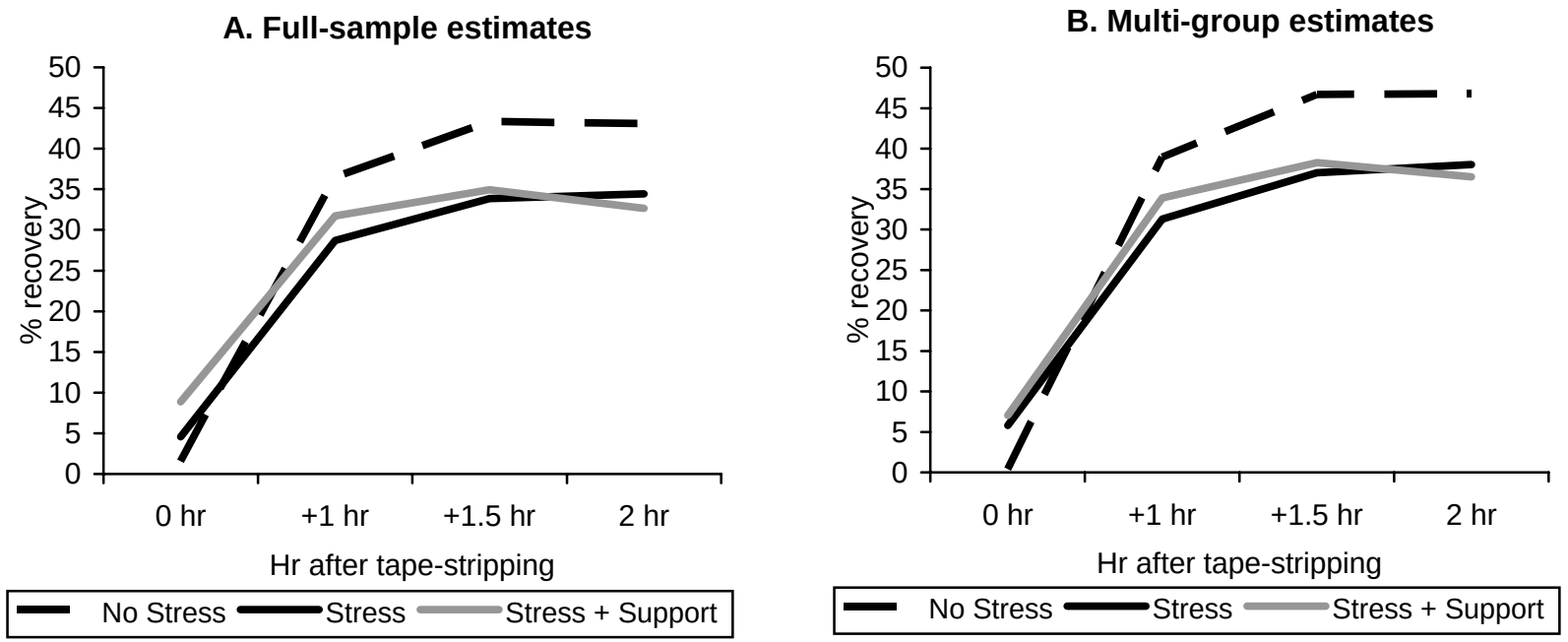

Figure 7. Predicted percent skin barrier recovery over the course of the session, by group, based on multilevel modeling estimates from A) full-sample parameter estimates from Table 16 and B) multi-group parameter estimates from Table 18.

compared the parameters between the three groups. Multivariate contrasts indicated that parameter estimates in the No Stress group significantly differed from the Stress and Stress + Support groups, both in combination (p = .007), and separately (p's = .03). The Stress and Stress + Support groups did not significantly differ on intercepts or slopes. As shown in Figure 8B, the No Stress group showed significantly steeper linear slopes (i.e., faster healing) compared to the Stress group and Stress + Support groups when combined, $\chi^2(1) = 7.16$, p = .007, and compared to the Stress and Stress + Support group separately (p's from .02 - .04).

Multilevel modeling corrected TEWL. For the corrected TEWL values, in addition to modeling intercepts and linear slopes, a cubic pattern was more suitable to the data compared to a quadratic pattern. Models with intercepts and linear and cubic slopes specified as random provided better fit (*AIC* = 2326)[4] compared with models with intercepts specified as random and slopes specified as fixed (linear and cubic fixed *AIC* = 2413; linear fixed, cubic random *AIC* = 2373; linear random, cubic fixed *AIC* = 2346) or models with intercepts specified as random and no slopes specified (*AIC* = 2531).

Table 19 shows level-1 parameter estimates for temperature-corrected TEWL and temperature- and control- corrected TEWL using the full-sample approach. Figure 8A shows the pattern of change over time. There was significant variance in intercepts and slopes, justifying including group as a level-2 predictor. The exceptions were for temperature- and control-corrected TEWL values, where linear and cubic slope variance was not significant. Intercepts and slopes were highly correlated, such that higher TEWL

[4] All *AIC* values are derived from analyses of temperature-corrected TEWL. The pattern of results is the same for and temperature- and control-corrected TEWL.

at 1 h after tape-stripping was related to larger linear decreases in TEWL and larger cubic increases in TEWL. Larger linear decreases in TEWL were related to larger cubic increases in TEWL. Intercept parameters showed high reliability, while slope parameters showed low to moderate reliability. Results for group status as level-2 predictors of intercepts and slopes are shown in Table 20. No group effects were significant.

Like the results for percent recovery, the multi-group approach also yielded a pattern suggesting some group differences (Figure 8B). Table 21 shows separate level-1 parameter estimates for temperature-corrected TEWL in each group indicating no significant variance for linear slope variances, again due to reduced sample size because of separate analyses for each group. Therefore, the parameters were modeled with intercepts random and linear slopes fixed, and intercepts and cubic slopes as random, as shown in Table 22. Multivariate contrasts indicated that parameter estimates in the No Stress group significantly differed from the Stress group and Stress + Support groups separately and in combination (*p*'s from .00 - .05). The No Stress and Stress groups and the Stress and Stress + Support groups did not significantly differ in intercepts and slopes. The No Stress group showed significantly lower intercepts compared to the Stress and Stress + Support groups, $p = .02$. However, the difference between intercepts in the No Stress and Stress groups was not significant; rather, the No Stress and Stress + Support groups were significantly different, $p = .004$. Linear and cubic slopes did not differ between the two groups.

Table 23 shows separate level-1 parameter estimates for temperature- and control-corrected TEWL in each group, indicating no significant linear or cubic slope variances for the Stress and Stress + Support groups. Therefore, the parameters were modeled with

intercepts random, linear slopes fixed, and cubic slopes random for the No Stress group only, as shown in Table 24. Multivariate contrasts showed that parameter estimates in the No Stress group significantly differed from the Stress group and Stress + Support groups in combination, and that the No Stress group significantly differed from the Stress + Support group. By contrast, the No Stress and Stress groups, and the Stress and Stress + Support groups did not significantly differ on any parameters. However, no univariate contrasts were significant, though two univariate contrasts approached significance: comparisons between the No Stress vs. the Stress and Stress + Support groups, $\chi^2(1) = 2.66$, $p = .10$, indicating that the No Stress group showed significantly lower intercepts compared to the Stress and Stress + Support groups, and between the No Stress vs. the Stress + Support group, $\chi^2(1) = 2.90$, $p = .09$, indicating lower intercepts for No Stress compared to the Stress + Support group.

Hypothesis 1 & 2 summary

Overall, there was some support for Hypothesis 1: exposure to acute stress would result in delayed wound healing. The strongest support came from the multilevel modeling analyses for percent recovery, shown in Figure 7. Analyses of temperature-corrected TEWL yielded similar conclusions, but the differences were primarily between the No Stress and the Stress + Support group. Analyses of temperature- and control-corrected TEWL also found slower healing in the Stress groups compared to the No Stress group. In contrast, there was no support for Hypothesis 2, that social support would speed skin barrier recovery.

Parameter estimate	Temperature- corrected			Temperature- and control- corrected		
	Means	Variances	Reliability	Means	Variances	Reliability
Intercept	1.34*** (0.02)	0.03*** [0.16]	.86	0.38*** (0.01)	0.02*** [0.14]	.79
Linear slope	-0.03*** (0.004)	0.0002 [0.01]	.14	-0.03*** (0.004)	0.00008 [0.009]	.05
Cubic slope	0.002*** (0.0001)	0.00*** [0.008]	.33	0.002*** (0.0001)	0.0000 [0.0007] $p = .07$	.21
Within-subject variances		0.01 [0.11]			0.03 [0.17]	

Note. Intercept values reflect TEWL 1 h after tape-stripping. Linear slopes reflect change in TEWL per 1 h of time, and cubic slopes reflect change in TEWL per 1 h of time^3. For temperature-corrected TEWL, correlation between intercepts and linear slopes = -.36; intercepts and cubic slopes = .51; linear and cubic slopes = -.97. For temperature- and control-corrected TEWL, correlation between intercepts and linear slopes = -.79; intercepts and cubic slopes = .87; linear and cubic slopes = -.97. Values in parentheses are standard errors and values in brackets are standard deviations.

*** $p < .001$.

Table 19. Initial level-1 multilevel modeling parameter estimates for temperature-corrected TEWL and temperature- and control-corrected TEWL.

Parameter	Temperature- corrected			Temperature- and control- corrected		
	Means	Variances	Reliability	Means	Variances	Reliability
Intercept	1.28*** (0.03)	0.02*** [0.15]	.86	0.33*** (0.03)	0.01*** [0.11]	.76
Stress group	0.06 (0.04)			0.05 (0.03)		
Support group	0.06 (0.05)			0.03 (0.03)		
Linear slope	-0.03*** (0.006)	0.0002** [0.01]	.14	-0.03*** (0.004)	-	
Stress group	-0.009 (0.009)			-		
Support group	0.01 (0.01)			-		
Cubic slope	0.002*** (0.0003)	0.00*** [0.0008]	.33	0.002*** (0.0002)	-	
Stress group	0.0002 (0.0004)			-		
Support group	-0.0005 (0.0003)			-		
Within-subjects variance		0.01*** [0.11]			0.02 [0.14]	

Note. Intercept values reflect TEWL 1 h after tape-stripping. Linear slopes reflect change in TEWL per 1 h of time, and cubic slopes reflect change in TEWL per 1 h of time^3. For temperature-corrected TEWL, correlation between intercepts and linear slopes = -.39; intercepts and cubic slopes = .60; linear and cubic slopes = -.96. Values in parentheses are standard errors and values in brackets are standard deviations.

** $p < .01$, *** $p < .001$.

Table 20. Full-sample multilevel modeling parameter estimates for temperature- and temperature- and control-corrected TEWL, including dummy-coded group status (stress group and support group) as level-2 predictors of intercepts and slopes.

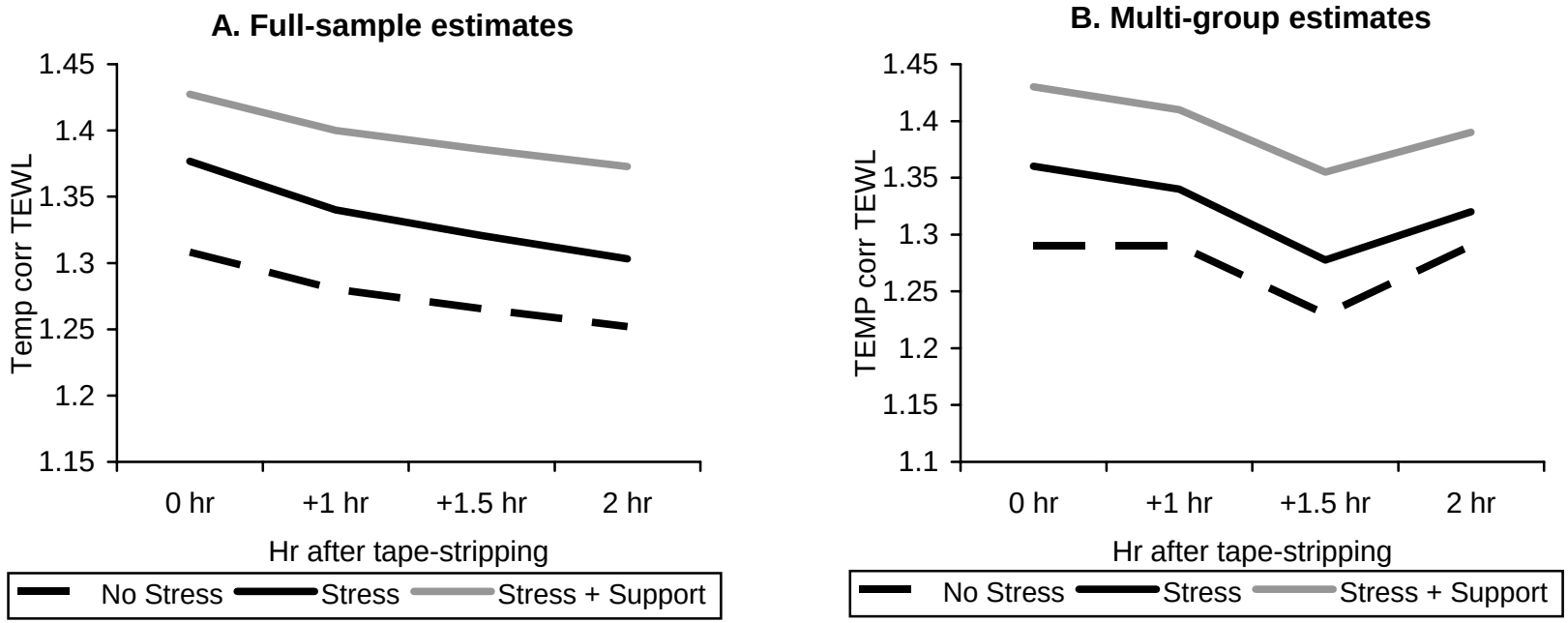

Figure 8. Predicted temperature-corrected TEWL over the course of the session, by group, based on multilevel modeling estimates from A) full-model parameter estimates from Table 20 and B) multi-group parameter estimates from Table 22. Higher TEWL values indicate poorer healing.

Parameter	No Stress	Stress	Stress + Support
Means			
Intercept	1.29*** (0.03)	1.34*** (0.04)	1.42*** (0.03)
Linear slope	-0.17*** (0.02)	-0.17*** (0.02)	-0.16*** (0.02)
Cubic slope	0.17*** (0.02)	0.15** (0.02)	0.13*** (0.02)
Variances			
Intercept	0.02***(0.006)	0.04*** (0.01)	0.02** (0.007)
Linear slope	0.001 (0.005) $p = .05$	0.004 (0.005)	0.002 (0.004)
Cubic slope	0.006 (0.003) $p = .05$	0.004 (0.002) $p = .08$	0.004* (0.002)
Within-subject	0.009*** (0.002)	0.01*** (0.002)	0.008*** (0.002)

Note. Intercept values reflect TEWL 1 h after tape-stripping. Linear slopes reflect change in TEWL per 1 h of time, and quadratic slopes reflect change in TEWL per 1 h of time^2. Values in parentheses are standard errors.

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

Table 21. Initial level-1 multi-group multilevel modeling parameter estimates for temperature-corrected TEWL.

Parameter	No Stress	Stress	Stress + Support
Means			
Intercept[a]	1.29*** (0.03)	1.34*** (0.03)	1.41*** (0.03)
Linear slope	-0.16*** (0.02)	-0.16** (0.02)	-0.14*** (0.02)
Cubic slope	0.16*** (0.02)	0.14*** (0.01)	0.12*** (0.01)
Variances			
Intercept	0.02** (0.006)	0.03*** (0.007)	0.02** (0.006)
Cubic slope	0.002** (0.008)	-	0.001*** (0.008)
Within-subject	0.009*** (0.002)	0.01*** (0.002)	0.008*** (0.001)

Note. Intercept values reflect TEWL 1 h after tape-stripping. Linear slopes reflect change in TEWL per 1 h of time, and quadratic slopes reflect change in TEWL per 1 h of time^2. Values in parentheses are standard errors. Multivariate contrasts are described below; significant univariate contrasts are indicated by subscripts. Multivariate contrasts: No Stress vs. Stress and Stress + Support: $\chi^2(3) = 14.85$, $p = .002$. No Stress vs. Stress: $\chi^2(3) = 4.51$, $p = .21$. No Stress vs. Stress + Support: $\chi^2(3) = 25.85$, $p = .00$. Stress vs. Stress + Support: $\chi^2(3) = 7.72$, $p = .05$.

[a] Univariate contrasts of intercepts: No Stress vs. Stress and Stress + Support: $\chi^2(1) = 5.28$, $p = .02$. No Stress vs. Stress + Support, $\chi^2(1) = 8.40$, $p = .004$. Stress vs. Stress + Support, $\chi^2(1) = 3.08$, $p = .08$.

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

Table 22. Final level-1 multi-group multilevel modeling parameter estimates for temperature-corrected TEWL.

Parameter	No Stress	Stress	Stress + Support
Means			
Intercept	0.34*** (0.04)	0.39*** (0.02)	0.42*** (0.03)
Linear slope	-0.16*** (0.02)	-0.14*** (0.03)	-0.15*** (0.03)
Cubic slope	0.14*** (0.02)	0.12*** (0.02)	0.12*** (0.02)
Variances			
Intercept	0.03*** (0.009)	0.01*(0.005)	0.02** (0.007)
Linear slope	0.002 (0.004)	0.0008 (0.006)	0.0008 (0.006)
Cubic slope	0.007* (0.003)	-0.002 (0.002)	0.002 (0.002)
Within-subject	0.006 *** (0.001)	0.02*** (0.004)	0.01*** (0.003)

Note. Intercept values reflect TEWL 1 h after tape-stripping. Linear slopes reflect change in TEWL per 15 min of time, and quadratic slopes reflect change in TEWL per 15 min of time^2. Values in parentheses are standard errors.

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

Table 23. Initial level-1 multi-group multilevel modeling parameter estimates for temperature- and control-corrected TEWL.

Parameter	No Stress	Stress	Stress + Support
Means			
Intercept	0.34*** (0.03)	0.39*** (0.02)	0.41*** (0.03)
Linear slope	-0.15*** (0.02)	-0.14*** (0.03)	-0.13*** (0.03)
Cubic slope	0.13*** (0.01)	0.12*** (0.02)	0.10*** (0.01)
Variances			
Intercept	0.03** (0.01)	0.01* (0.003)	0.01** (0.004)
Cubic slope	0.002** (0.0008)	-	-
Within-subject	0.006*** (0.001)	0.02*** (0.003)	0.02*** (0.003)

Note. Intercept values reflect TEWL 1 h after tape-stripping. Linear slopes reflect change in TEWL per 15 min of time, and quadratic slopes reflect change in TEWL per 15 min of time^2. Values in parentheses are standard errors. Multivariate contrasts: No Stress vs. Stress and Stress + Support: $\chi^2(3) = 12.18$, $p = .007$. No Stress vs. Stress: $\chi^2(3) = 4.52$, $p = .21$. No Stress vs. Stress + Support: $\chi^2(3) = 14.42$, $p = .002$. Stress vs. Stress + Support: $\chi^2(3) = 3.44$, $p = .33$.

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

Table 24. Final level-1 multi-group multilevel modeling parameter estimates for temperature- and control-corrected TEWL.

Ancillary analyses for skin barrier recovery

In these analyses, I examined whether additional variables were related to skin barrier recovery, particularly within the Stress and Stress + Support groups. The approach in these analyses was two-fold, and was only conducted with percent recovery measures. First, I derived the empirical Bayes estimates of intercepts and slopes from the multilevel models. I then examined relationships between the empirical Bayes estimates and specific variables of interest within each experimental group. The independent variables of interest were the individual difference variables, manipulation check variables, including cognitive appraisals and change in affect and anxiety, health behaviors, and confederate ratings for the Stress + Support group. Measures that were significantly related to any of the empirical Bayes estimates (intercepts, slopes) were then included in additional within-group multilevel models to see if they were related to intercepts and/or slopes in the context of a full multilevel model. In these analyses, I used empirical Bayes estimates from multilevel models that included percent recovery as the dependent variable.

For the Stress + Support group, two variables emerged from the analyses. Specifically, participants' ratings of the confederate on the Assured-Dominant scale of the IAS-R-C, and among female participants, self-reported day of the current menstrual cycle were significantly related to skin barrier recovery. Participants who rated confederates as more Assured-Dominant showed lower intercept estimates, $r = -.41$, $p = .04$, lower linear slope estimates, $r = -.40$, $p = .04$, and higher quadratic slope estimates, $r = .42$, $p = .03$. To further explore this relationship, I included Assured-Dominant ratings of the confederate and gender as level-2 predictors of intercepts and quadratic slopes in a multilevel model with percent recovery as the dependent variable. I did not include level-

2 predictors of linear slopes as there was no significant variance in linear slopes. This model only used data from the Stress + Support group. Higher ratings of the confederate as Assured-Dominant were related to lower skin barrier recovery 1 h after tape-stripping, unstandardized β = -0.64 (0.26), p = .02. In addition, higher Assured-Dominant ratings were related to slower healing, evidenced by greater quadratic slopes, estimate = 0.41 (0.19), p = .04. Predicted percent recovery values are shown in Figure 9. The figure clearly shows that participants in the Stress + Support group who rated the confederates as more Assured-Dominant showed slower skin barrier recovery 1 h after tape-stripping and slower recovery over the first 1.5 hours after tape-stripping. Including Assured-Dominant ratings of the confederate accounted for 23% of the variance in skin barrier recovery 1 h after tape-stripping in the Stress + Support group.

Among the female participants in the Stress + Support group, those who were closer to day 28 in the cycle showed larger linear slope estimates, r = .53, p = .04. By contrast, self-reported day of the menstrual cycle was not related to skin barrier recovery estimates for the No Stress group (r's from -.40 - .43, but p's from .17 - .22) or the Stress group (r's from -.13 - .16). Although not significant, the trend for the No Stress group was for slower healing closer to day 28 in the cycle.

To further explore the relationship between menstrual phase and skin barrier recovery in the Stress + Support group, I included self-reported day of the cycle as a level-2 predictor of intercepts in a multilevel model with percent recovery as the dependent variable. Self-reported day of the menstrual cycle was centered around the group mean of 14.53 days. This model only used data from the female participants in the Stress + Support group. I did not include level-2 predictors of linear or quadratic slopes

as there was no significant variance in either parameter. More advanced day of the menstrual cycle was marginally related to greater skin barrier recovery 1 h after tape-stripping, estimate = 0.81 (0.46), p = .11. Predicted percent recovery values are shown in Figure 10. The figure shows a trend for greater skin barrier recovery 1 hr after tape-stripping for women in the later, luteal phase of the menstrual cycle. Including day of the menstrual cycle accounted for 22% of the variance in skin barrier recovery 1 h after tape-stripping in the Stress + Support group.

In addition to effects specific to the Stress + Support group, several individual difference variables were related to healing across the groups, including anxious arousal in the past week reported on the MASQ, change in anxiety pre- to post-task on the State-Trait Anxiety Inventory, and trait positive affect reported on the PANAS. Higher anxious arousal in the past week was related to lower intercepts, estimate = -0.86 (0.30), p = .005, and smaller linear slopes, estimate = -1.03 (0.34), p = .003. In addition, there was a significant interaction between stress group status and anxious arousal in predicting linear slopes, estimate = 0.96 (0.38), p = .05. The predicted skin barrier recovery from this model is shown in Figure 11. In general, individuals reporting low anxious arousal in the past week showed faster skin barrier recovery compared to individuals reporting high anxious arousal. In addition, for individuals reporting high anxious arousal, skin barrier recovery was faster in the Stress and Stress + Support groups, whereas for individuals reporting low anxious arousal, skin barrier recovery was faster in the No Stress group.

In addition to anxious arousal in the past week, changes in state anxiety before to after the task were related to skin barrier recovery. Increased state anxiety was related to lower intercepts, estimate = -1.45 (0.43), p = .001, and lower linear slopes, estimate = -

0.83 (0.24), p = .001. In addition, there was a significant interaction between support group status and change in state anxiety in predicting linear slopes, estimate = 0.96 (0.38), p = .05. The predicted skin barrier recovery from this model is shown in Figure 12. In general, individuals reporting increasing state anxiety from pre- to post-task showed slower skin barrier recovery compared to individuals reporting no change in state anxiety. In addition, for individuals reporting increasing anxiety, skin barrier recovery was faster in the Stress + Support groups compared to the Stress and No Stress groups, whereas for individuals reporting no change in anxiety, skin barrier recovery was similar across the groups.

Finally, trait positive affect was related to skin barrier recovery. The trait positive affect measure was included later in data collection, and data from only 59 participants is available. There was a significant interaction between stress group status and trait positive affect in predicting intercepts, estimate = 2.49 (0.90), p = .008, linear slopes, estimate = 1.13 (0.49), p = 0.49, and quadratic slopes, estimate = -1.33 (0.51), p = .01. The predicted skin barrier recovery from this model is shown in Figure 13. The results indicated that levels of trait positive affect were significantly related to skin barrier recovery in the Stress and Stress + Support groups, but not the No Stress groups. Specifically, in the Stress and Stress + Support groups, individuals reporting high trait positive affect showed faster skin barrier recovery compared to individuals reporting low trait positive affect.

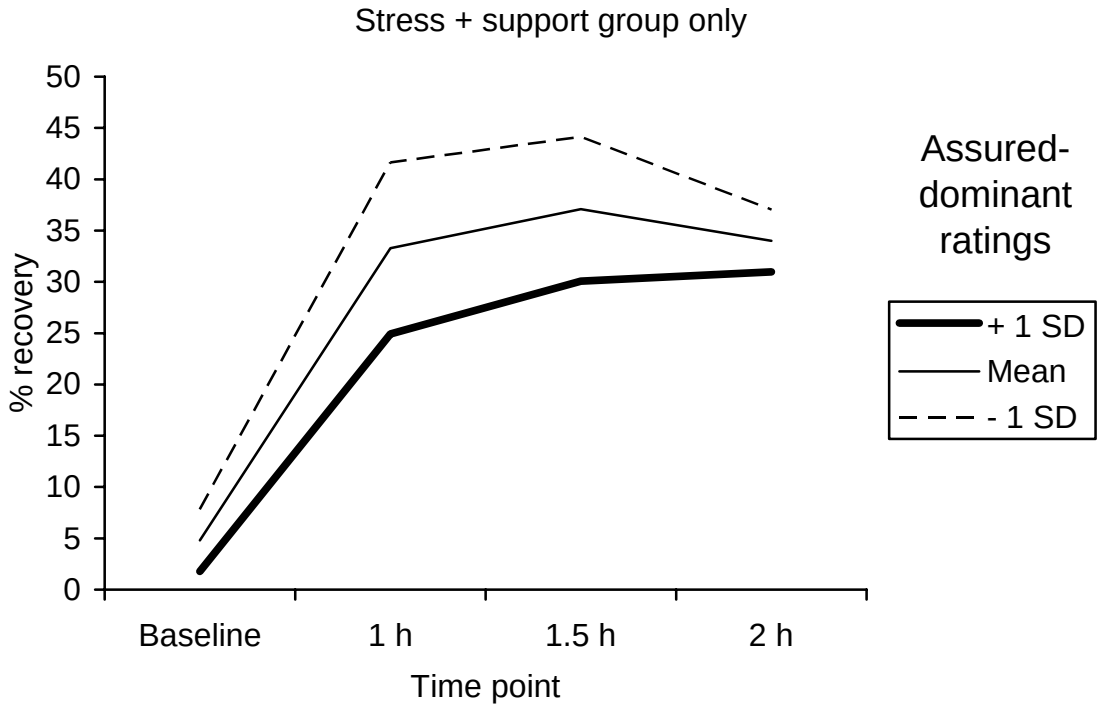

Figure 9. Predicted percent recovery over the course of the session, by ratings on the IAS-R Assured-dominant subscale, based on multilevel modeling estimates. Lines represent values at the group mean and 1 *SD* above and below the mean.

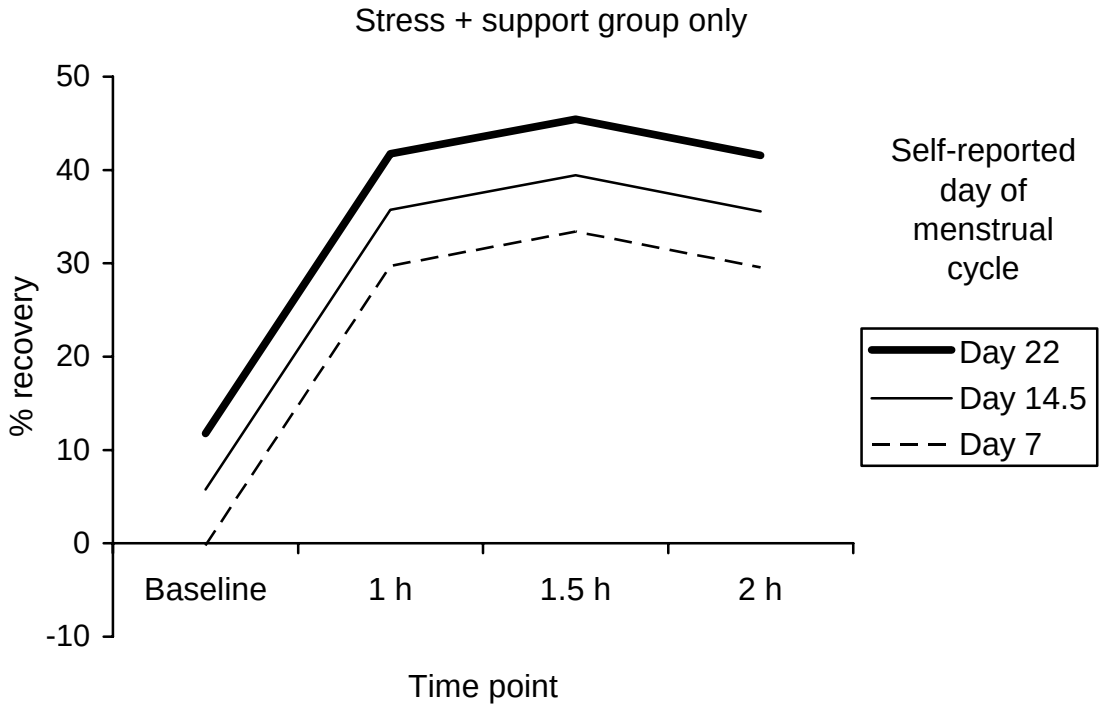

Figure 10. Predicted percent recovery over the course of the session, by self-reported day of the menstrual cycle, based on multilevel modeling estimates. Lines represent values at the group mean and 1 *SD* above and below the mean.

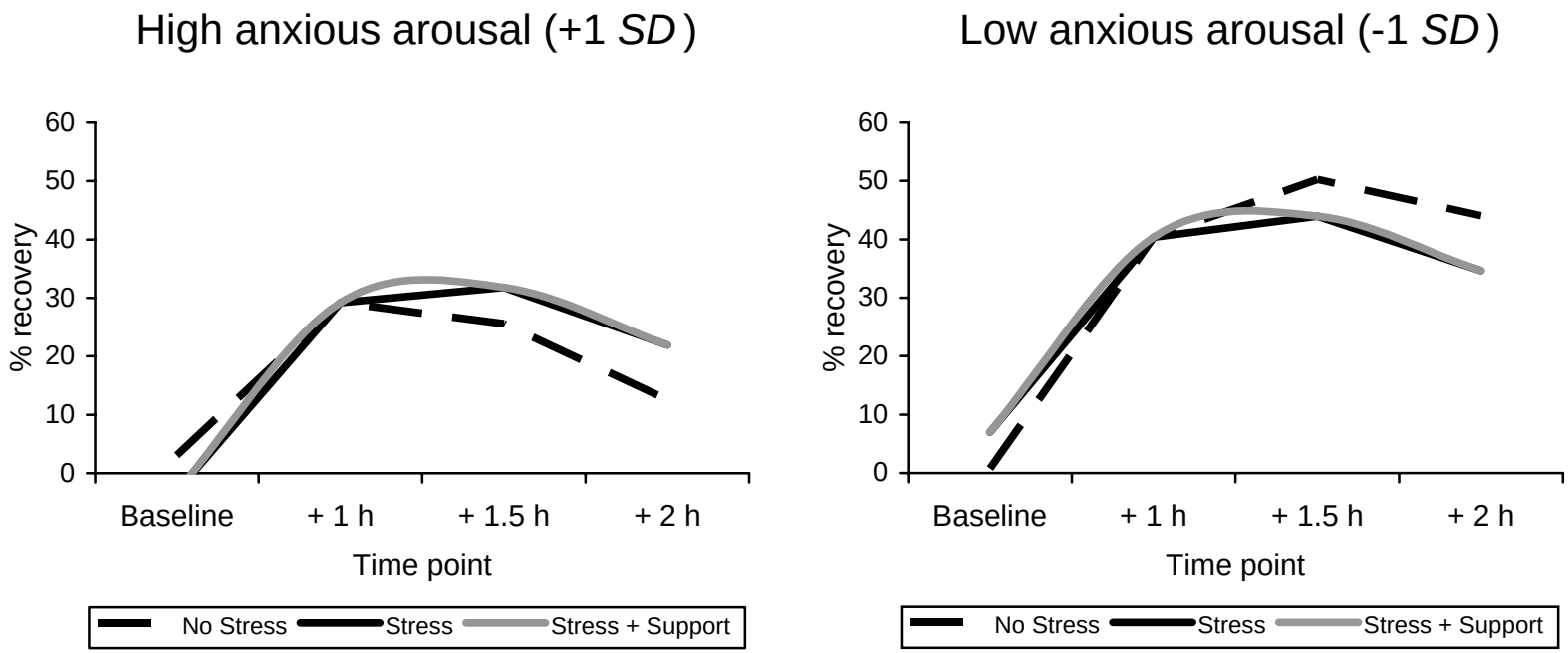

Figure 11. Predicted percent recovery over the course of the session, by group and MASQ anxious arousal scale based on multilevel modeling estimates. The plots represent predicted values for individuals ± 1 *SD* relative to the mean for anxious arousal, with separate lines for each group. Some lines may be smoothed to show overlapping predicted values.

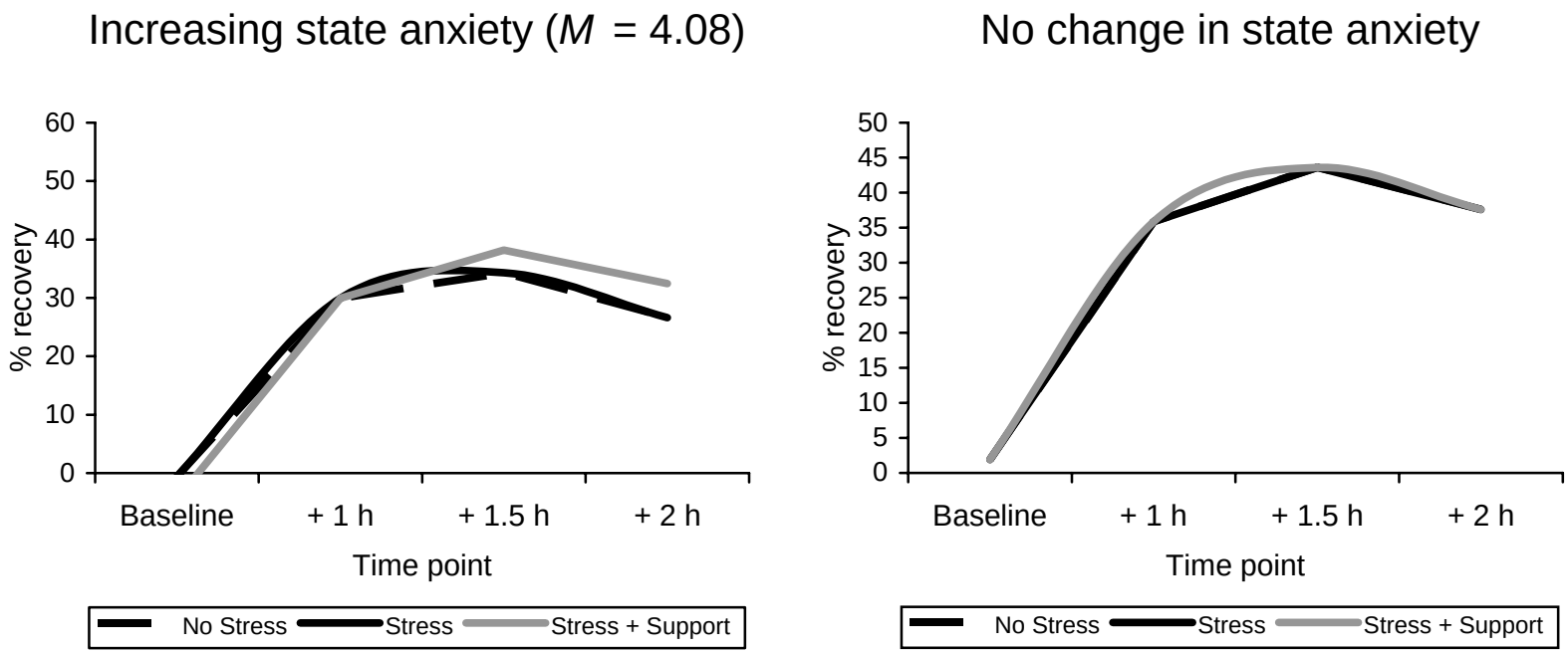

Figure 12. Predicted percent recovery over the course of the session, by group and change in state anxiety based on multilevel modeling estimates. The plots represent predicted values for individuals showing an increase in state anxiety (the mean value across the sample 4.08) and for individuals showing no change in state anxiety, with separate lines for each group. Some lines may be smoothed to show overlapping predicted values.

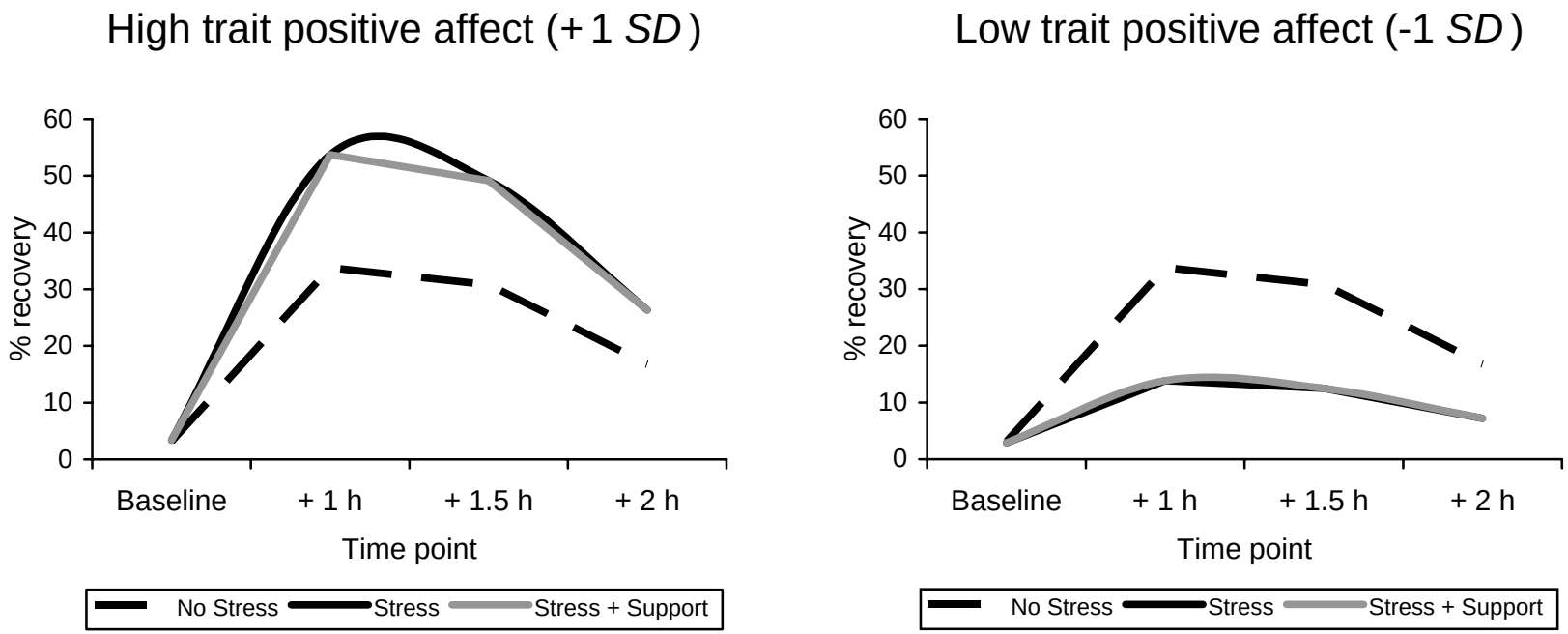

Figure 13. Predicted percent recovery over the course of the session, by group and trait positive affect based on multilevel modeling estimates. The plots represent predicted values for individuals ± 1 *SD* relative to the mean for trait positive affect, with separate lines for each group. Some lines may be smoothed to show overlapping predicted values.

Hypothesis 3: Social support will reduce physiological reactivity

These analyses tested group differences in cardiovascular and cortisol reactivity. The results are organized into general linear models followed by multilevel models, similar to the skin barrier recovery results presented above.

Cardiovascular reactivity – general linear models

Baseline cardiovascular measures taken before the stressor were not significantly related to simple change scores (*r*'s range from -.16 - .10). Therefore, simple change scores were used in these analyses. No outliers were found during data screening. All analyses are one-way general linear models with change scores as the dependent variable and Group and Gender as independent variables. Figure 14 shows the results for simple change scores, and Table 25 shows *F*-statistics for the main effects of group, in addition to 95% *CI*s for planned comparisons between each group.

Across all the cardiovascular reactivity measures using simple change, there was a significant main effect of Group (Table 25 and Figure 14). Planned comparisons indicated that the Stress and Stress + Support groups showed higher reactivity compared to the No Stress group. Cardiovascular reactivity did not significantly differ between the Stress group and the Stress + Support group. Across all the analyses, there were no significant main effects of Gender, or Gender x Group interactions.

In addition, I computed a composite cardiovascular variable with MAP and heart rate by averaging standardized MAP and heart rate values. SBP and DBP were not included because the values are derived from MAP values. Similar to the analyses above, there was a significant main effect of group, $F(2,73) = 28.80$, $p = .00$, $r = .53$, with the Stress and Stress + Support groups showing significantly larger cardiovascular reactivity

Dependent variable	Group effect		95% *CI* for group differences (Group A – Group B)		
	F	*r*	Stress – No Stress	Stress + Support – No Stress	Stress +Support – Stress
Simple change					
MAP (2,71)	32.57	.56	9.7 – 17.4	9.4 – 17.3	-4.2 – 3.8
HR (2,70)	14.06	.41	7.1 – 16.2	4.1 – 13.5	-7.6 – 1.9
SBP (2,71)	44.61	.62	17.7 – 28.3	14.7 – 25.7	-8.4 – 2.7
DBP (2,71)	18.87	.46	5.00 – 10.4	3.9 – 9.6	-3.8 – 1.9
Residualized change					
MAP (2,71)	30.15	.55	9.8 – 18.2	7.4 – 15.1	-6.1 – 1.6
HR (2,70)	13.27	.40	6.8 – 15.7	3.1 – 12.3	-8.1 – 1.1
SBP (2,71)	43.13	.61	17.9 – 28.3	12.6 – 23.4	-10.6 – 0.4
DBP (2,71)	19.64	.47	5.3 – 10.4	2.9 – 8.2	-5.0 – 0.4

Note. All *F*-statistics are significant at $p < .001$. Numbers in parentheses are *df*(Group, error). Change scores are equal to During task – Baseline. 95% *CI*s that contain 0 within the interval are not statistically significant.

Table 25. *F*-table for general linear models predicting cardiovascular reactivity by group status.

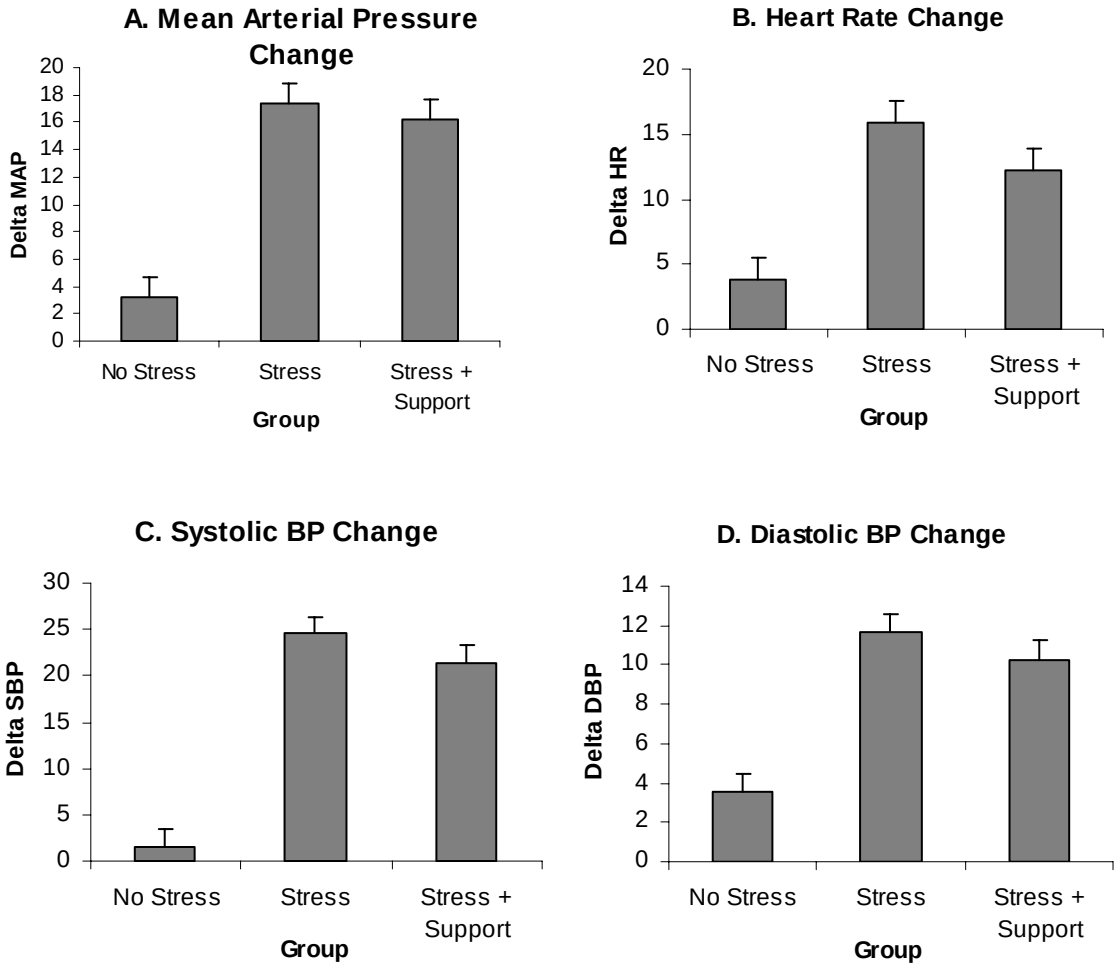

Figure 14. Simple change from baseline to task in cardiovascular measures as a function of group status. The Stress and Stress + Support groups showed significantly greater change above baseline compared to the No Stress group for all measures. There were no significant differences between the Stress and Stress + Support groups.

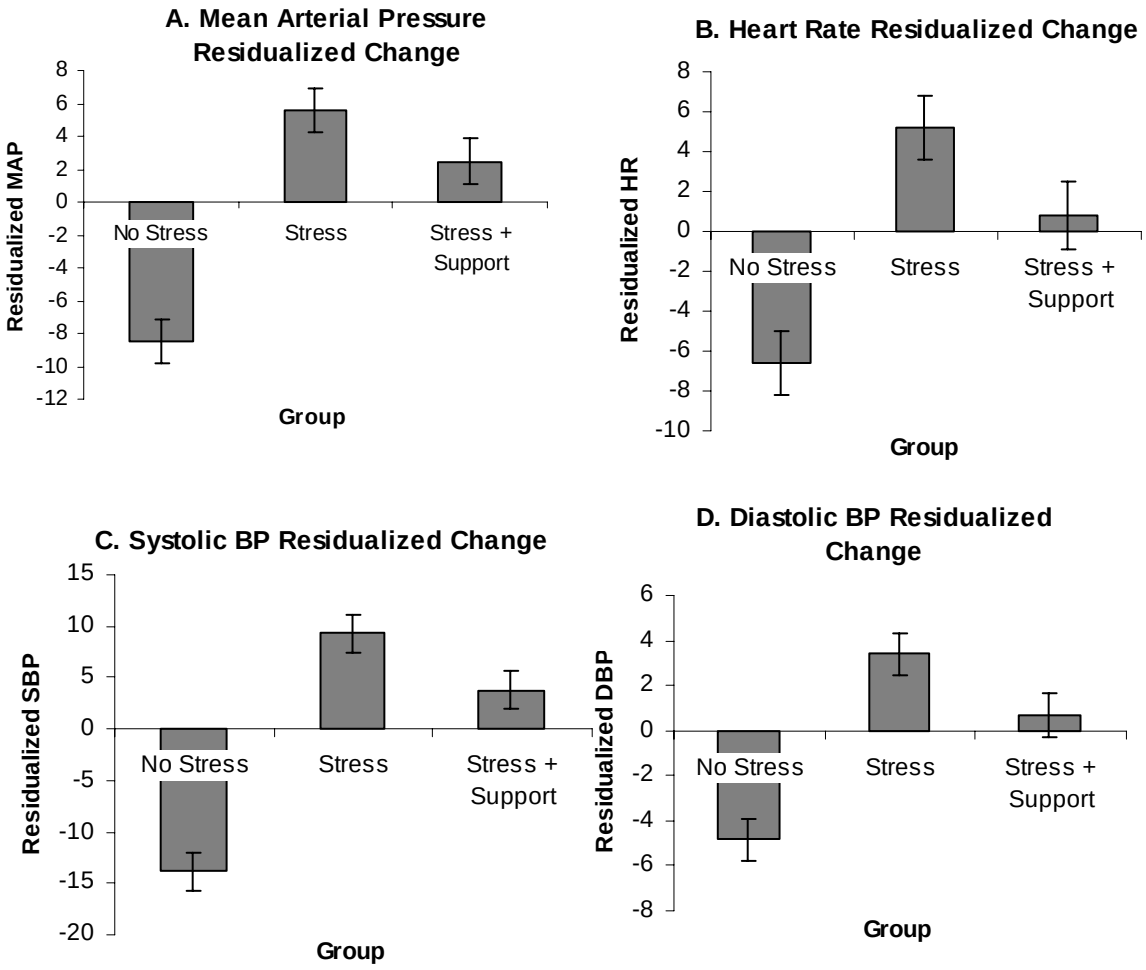

Figure 15. Residualized change from baseline to task in cardiovascular measures as a function of group status. The Stress and Stress + Support groups showed significantly greater change above baseline compared to the No Stress group for all measures. There were no significant differences between the Stress and Stress + Support groups.

compared to the No Stress group (95% *CI* for Stress – No Stress comparison, 0.99 – 1.76; Stress + Support – No Stress comparison, 0.73 – 1.51).

I conducted additional analyses using residualized change scores. Results for residualized change scores are shown in Figure 15. Across all the residualized cardiovascular reactivity measures, there was a significant main effect of Group (Table 25). Planned comparisons indicated that the Stress groups showed higher reactivity compared to the No Stress group. In addition, cardiovascular reactivity across all the measures was marginally higher for the Stress group compared to Stress + Support group. Across all the analyses, there were no significant main effects of Gender or Gender x Group interactions.

Cardiovascular reactivity – multilevel models

Additional analyses of cardiovascular reactivity used multilevel models, with occasions of measurement as the level-1 unit, and individuals as the level-2 unit. Inspection of the raw data indicated that models specifying linear and quadratic (u-shaped) change were most parsimonious. The level-1 predictor in these models was the 10-min epoch of measurement (-2, -1, 0, 1, 2, 3, 4) and the intercept was centered at the 10-min epoch during the task itself. Centering the intercept at the task measurement rather than at baseline allowed for comparing absolute levels of reactivity during the task. In these analyses, the dependent variables were cardiovascular change above baseline values.

The first set of models included all participants in one group. Models with intercepts specified as random and linear and quadratic slopes specified as fixed provided

better fit (*AIC* = 3271)[5] compared with models with intercepts and slopes specified as random (*AIC* = 3274) or models with only intercepts specified as random and no slopes (*AIC* = 3353). Because slopes were best specified as fixed, additional predictors of cardiovascular change (specifically Group assignment) could not be included as level-2 predictors of slopes. In other words, a full-sample multilevel model could not be used to examine change in cardiovascular reactivity as a function of group status. Therefore, a multi-group approach was used which allowed for statistically comparing intercepts and slopes between the groups. Results from this model are shown in Table 26 and Figure 16. In line with the analyses for simple and residualized change, the Stress and Stress + Support groups showed significantly higher cardiovascular reactivity during the tasks compared to the No Stress group, as evidenced by elevated intercept values across all the measures, and steeper linear slopes for MAP and SBP. No significant differences emerged between the Stress and Stress + Support group, with the Stress group showing higher SBP during the task compared to the Stress + Support group.

Cortisol reactivity – general linear models

Data screening identified three outliers with values for AUC_I greater than 3 *SD* above the mean, which were removed from these analyses. The final sample for these analyses was 24 in the No Stress Group, 28 in the Stress group, and 22 in the Stress + Support group.

In a one-way general linear model, with AUC_I as the dependent variable and Group and Gender as the independent variables, there were significant main effects of Group, $F(2,68) = 10.88$, $p = .00$, $r = .37$ and Gender, $F(1,68) = 10.08$, $p = .00$, $r = .36$.

[5] All *AIC* values from analyses with MAP, all other cardiovascular variables showed similar results.

Cardiovascular parameter estimate	No Stress	Stress	Stress + Support
MAP			
Intercept [a]	0.72 (0.52)	7.77*** (0.93)	6.84*** (0.76)
Linear slope [a]	0.88* (0.35)	4.91*** (0.68)	3.91** (0.77)
Quadratic slope [a]	-0.21* (0.08)	-1.34*** (0.15)	-1.17*** (0.17)
Intercept variance	3.78* (1.70)	11.89* (5.71)	-0.74 (3.24)
Within-subject variance	11.12*** (1.56)	44.64*** (6.01)	50.92*** (7.19)
Heart rate			
Intercept [a]	1.04*** (0.38)	7.47*** (1.00)	5.95*** (0.71)
Linear slope [a]	1.14*** (0.30)	3.81*** (0.72)	2.91*** (0.67)
Quadratic slope [a]	-0.28*** (0.07)	-1.04*** (0.16)	-0.83*** (0.15)
Intercept variance	1.39 (0.88)	14.41** (6.71)	1.27 (2.83)
Within-subject variance	8.16*** (1.15)	50.69*** (6.83)	39.41*** (5.56)
SBP			
Intercept [a]	0.35 (0.65)	11.23*** (1.20)	8.85*** (0.94)
Linear slope [a]	0.31 (0.48)	7.15*** (0.95)	5.70** (0.92)
Quadratic slope [a]	-0.04 (0.10)	-1.91*** (0.21)	-1.55*** (0.20)
Intercept variance	5.24 (2.68)	15.63 (10.26)	1.26 (5.00)
Within-subject variance	20.70*** (2.90)	88.26*** (11.88)	73.48*** (10.37)
DBP			
Intercept [a]	1.20* (0.55)	5.09*** (0.61)	4.42*** (0.62)
Linear slope	0.85* (0.34)	3.14*** (0.50)	1.88** (0.61)
Quadratic slope [a]	-0.20** (0.07)	-0.87*** (0.11)	-0.63*** (0.13)
Intercept variance	4.97*** (1.97)	3.83 (2.42)	0.22 (2.13)
Within-subject variance	10.31*** (1.44)	23.98*** (3.23)	32.64*** (4.61)

Note. Dependent variables are change relative to baseline measures. Intercepts reflect peak reactivity during the task, linear slopes reflect change per 10 min epoch of

Table 26 (continued).

measurement, quadratic slopes reflect change per 10 min^2 epoch of measurement. Multivariate contrasts are described below. Univariate contrasts are in subscript.

MAP multivariate contrasts: No Stress vs. Stress and Stress + Support, $\chi^2(3) = 120.58$, $p = .00$, No Stress vs. Stress, $\chi^2(3) = 83.78$, $p = .00$, No Stress vs. Stress + Support, $\chi^2(3) = 66.99$, $p = .00$, Stress vs. Stress + Support, $\chi^2(3) = 4.43$, $p = .49$.

Heart rate multivariate contrasts: No Stress vs. Stress and Stress + Support, $\chi^2(3) = 84.77$, $p = .00$, No Stress vs. Stress, $\chi^2(3) = 48.29$, $p = .00$, No Stress vs. Stress + Support, $\chi^2(3) = 52.60$, $p = .00$, Stress vs. Stress + Support, $\chi^2(3) = 2.69$, $p = .44$.

SBP multivariate contrasts: No Stress vs. Stress and Stress + Support, $\chi^2(3) = 172.04$, $p = .00$, No Stress vs. Stress, $\chi^2(3) = 94.81$, $p = .00$, No Stress vs. Stress + Support, $\chi^2(3) = 119.53$, $p = .00$, Stress vs. Stress + Support, $\chi^2(3) = 4.17$, $p = .24$.

DBP multivariate contrasts: No Stress vs. Stress and Stress + Support, $\chi^2(3) = 48.05$, $p = .00$, No Stress vs. Stress, $\chi^2(3) = 23.22$, $p = .00$, No Stress vs. Stress + Support, $\chi^2(3) = 45.43$, $p = .00$, Stress vs. Stress + Support, $\chi^2(3) = 4.15$, $p = .25$.

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

[a] Significant difference between No Stress vs. Stress and Stress + Support groups

Table 26. Multi-group multilevel modeling parameter estimates for cardiovascular reactivity.

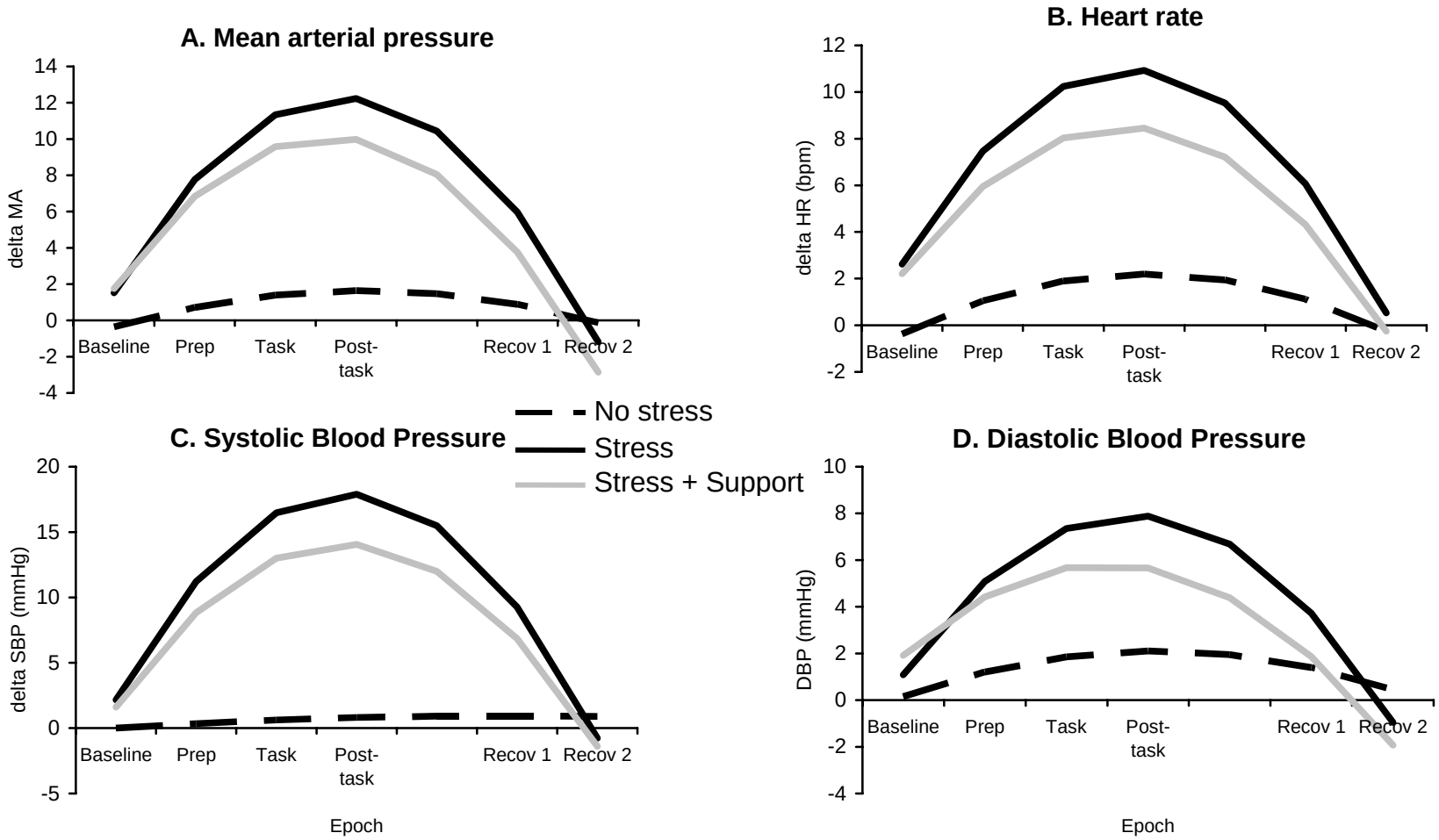

Figure 16. Predicted cardiovascular reactivity based on the multi-group multilevel modeling parameter estimates in Table 26. Recov 1 and Recov 2 correspond to 11 minute epochs within Cardio 4. Values represent change from baseline.

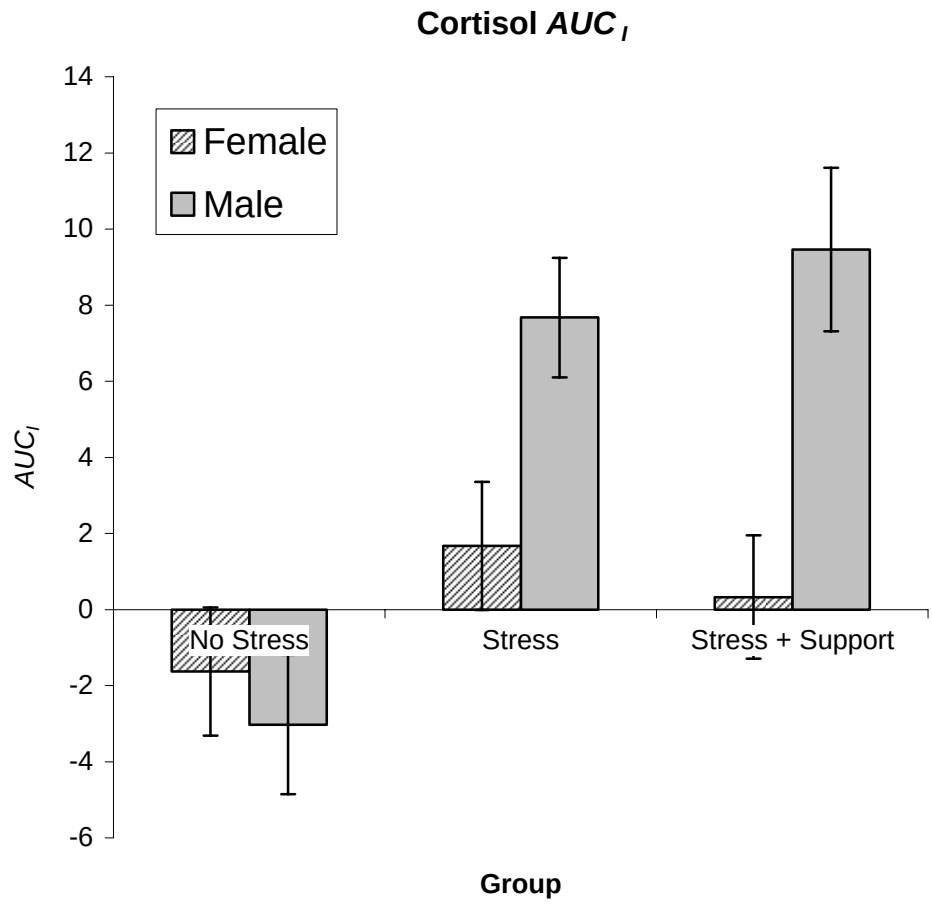

Figure 17. Cortisol reactivity assessed by AUC_I, by group and gender. Males showed in the Stress and Stress + Support groups showed larger cortisol responses compared to females. The Stress and Stress + Support groups showed greater cortisol responses compared to the No Stress group, but were not significantly different from one another.

Figure 17 summarizes the results for cortisol AUC_I. The Group x Gender interaction was significant, $F(2,68) = 4.50$, $p = .02$, $r = .25$. For the Group main effect, planned comparisons indicated that the Stress group showed higher cortisol reactivity compared to the No Stress group (Stress – No Stress difference, 95% *CI* = 3.62 – 10.38). The Stress + Support group showed higher cortisol reactivity compared to the No Stress group (Stress + Support – No Stress difference, 95% *CI* = 3.56 – 10.88). Cortisol reactivity did not significantly differ between the Stress and Stress + Support groups (Stress + Support – Stress difference, 95% *CI* = -3.31 – 3.76).

The main effect of Gender indicated that males had larger cortisol responses compared to females (male – female difference, 95% *CI* = 1.70 – 7.46). Follow up main-effects tests of the significant interaction between group and gender suggested that the effects of group were significant for male participants, $F(2,68) = 13.15$, $p = .00$, $r = .40$, but not female participants, $F(2,68) = 0.97$, $p = .39$, $r = .12$. In the male participants, the Stress and Stress + Support groups were significantly different from the No Stress group (95% *CI* for Stress – No Stress = 5.35 – 16.05; 95% *CI* for Stress + Support - No Stress: 6.22 – 18.75) but not different from each other (95% *CI* = -4.11 – 7.69).

Cortisol reactivity – multilevel models

Additional analyses of cortisol reactivity used multilevel models, with occasions of measurement as the level-1 unit, and individuals as the level-2 unit. The samples used were the last 7 of the 8 available samples, as there was significant variability in the first sample. Moreover, these analyses allowed for including 7 additional participants whose data were not sufficient for computing AUC_I, for a total of 82 participants. Models specifying linear and quadratic change were the most parsimonious. The level-1 predictor

in these models was the time of measurement relative to the start of the task (in minutes, divided by 15), and the intercept was centered at the baseline cortisol sample. Similar to the multilevel models for skin barrier recovery, I first report results from the full-sample approach, followed by results from the multi-group approach.

Models with intercepts and linear and quadratic slopes specified as random provided the best fit (*AIC* = -1602) compared with models with intercepts specified as random and slopes specified as fixed (*AIC* = -1479) and models with intercepts specified as random and either linear or quadratic slopes specified as fixed while the other was random (*AIC*'s = -1479 and -1501). Table 27 shows results from the model without any level-2 predictors. Cortisol showed a significant linear increase in cortisol of 0.01 pg/ml every 15 min, and a significant quadratic decrease in cortisol of 0.002 pg/ml every 15 min^2. There were significant individual differences in intercepts and slopes, which justified including additional level-2 predictors. Higher initial levels of cortisol were moderately related to larger linear slopes and steeper quadratic slopes. Larger linear increases in cortisol were related to larger quadratic decreases.

Table 28 shows results from the full-sample model with the inclusion of gender, group status, and their interactions as predictors of intercepts and slopes. Group and gender were not significant predictors of intercepts. The results indicated that Stress (both in the Stress and Stress + Support group) was related to larger linear increases and larger quadratic decreases in cortisol compared to No Stress. There was no significant effect of the support manipulation on change in cortisol. The significant effect of Gender indicated that males showed larger linear and quadratic changes compared to females, and the

Parameter	Mean	Variance	Reliability
Intercept	0.14*** (0.01)	0.006*** [0.08]	.81
Linear slope	0.01** (0.004)	0.001*** [0.03]	.72
Quadratic slope	-0.002** (0.0006)	0.00002*** [0.005]	.75
Within-subjects variance		0.002 [0.04]	

Note. Intercept values reflect baseline cortisol (pg/ml). Linear slopes reflect change in TEWL per 15 min of time, and quadratic slopes reflect change in TEWL per 15 min of time^2. Correlation between intercepts and linear slopes = .30; intercepts and quadratic slopes = -.36; linear and quadratic slopes = -.98. Values in parentheses are standard errors and values in brackets are standard deviations.

** $p < .01$, *** $p < .001$.

Table 27. Initial level-1 full-sample multilevel modeling parameter estimates for cortisol change.

Parameter	Mean	Variance	Reliability
Intercept	0.15*** (0.009)	0.006*** [0.08]	.81
Linear slope	-0.02*** (0.003)	0.0006*** [0.02]	.58
Stress group	0.02** (0.006)		
Support group	-0.002 (0.01)		
Gender	0.006 (0.006)		
Stress x Gender	0.04** (0.01)		
Support x Gender	-0.007 (0.02)		
Quadratic slope	0.002*** (0.0006)	0.00001*** [0.004]	.63
Stress group	-0.003** (0.001)		
Support group	0.0003 (0.001)		
Gender	-0.002 (0.001), $p = .10$		
Stress x Gender	-0.004* (0.002)		
Support x Gender	0.002 (0.003)		
Within-subject variance		0.002 [0.04]	

Note. Intercept values reflect baseline cortisol (pg/ml). Linear slopes reflect change in cortisol per 15 min of time, and quadratic slopes reflect change in cortisol per 15 min of time^2. Stress group is coded 0 = No Stress, 1 = Stress; Support group is coded 0 = No Support, 1 = Support; Gender is coded 0 = female, 1 = male. Correlation between intercepts and linear slopes = .24; intercepts and quadratic slopes = -.32; linear and quadratic slopes = -.97. Values in parentheses are standard errors and values in brackets are standard deviations.

** $p < .01$, *** $p < .001$.

Table 28. Final full-sample multilevel modeling parameter estimates, including group, gender, and group x gender interactions as predictors of cortisol change.

significant Stress x Gender interactions indicated larger effects of Stress for males compared to females, as is clearly indicated in Figure 18.

Table 29 shows results from the initial level-1 multi-group multilevel model, indicating significant variance in intercepts across all the groups, and significant variance in both linear and quadratic slopes for the Stress and Stress + Support groups. The No Stress group showed no significant variance in linear or quadratic slopes. Table 30 shows results from the multi-group multilevel model with linear and quadratic slopes fixed for the No Stress group, and random for the Stress and Stress + Support groups. Gender was included as a predictor of intercepts for all groups, and slopes for the Stress and Stress + Support groups. In addition, a series of multivariate and univariate contrasts compared mean parameters between the three groups.

Multivariate contrasts indicated that parameter estimates in the No Stress group significantly differed from the Stress group and Stress + Support groups separately (No Stress vs. Stress, $p = .04$; No Stress vs. Stress + Support, $p = .004$). Univariate contrasts indicated that the No Stress group showed higher initial cortisol levels compared to the Stress group, though this approached significance, $p = .06$. In addition, the Stress + Support group showed significantly higher initial cortisol levels compared to the Stress group, $p = .05$. Although the multivariate contrasts indicated that the No Stress group did not differ from the combined Stress and Stress + Support groups on any parameter, the univariate contrasts showed significant differences between the No Stress and the combined Stress and Stress + Support groups for linear ($p = .01$) and quadratic ($p = .03$) slopes. Linear slopes for the No Stress group were significantly different from the Stress group, $p = .02$. Figure 19 summarizes these results, showing that for male participants,

Parameter	No Stress	Stress	Stress + Support
Means			
Intercept	0.15*** (0.02)	0.14*** (0.01)	0.16*** (0.02)
Linear slope	-0.01*** (0.03)	0.04*** (0.01)	0.02* (0.01)
Quadratic slope	0.002*** (0.0005)	-0.005*** (0.001)	-0.003* (0.001)
Variances			
Intercept	0.005*** (0.001)	0.004** (0.001)	0.01** (0.004)
Linear slope	0.00003 (0.0007)	0.001** (0.0005)	0.002** (0.0007)
Quadratic slope	0.00 (0.00)	0.00004** (0.00001)	0.00003** (0.0001)
Within-subject	0.0006*** (0.0009)	0.001*** (0.0002)	0.002*** (0.0003)

Note. Intercept values reflect baseline cortisol (pg/ml). Linear slopes reflect change in cortisol per 15 min of time, and quadratic slopes reflect change in cortisol per 15 min of time^2. Values in parentheses are standard errors.

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

Table 29. Multi-group multilevel modeling level-1 parameter estimates for cortisol change.

Parameter	No Stress	Stress	Stress + Support
Means			
Intercept[a]	0.14*** (0.01)	0.10*** (0.02)	0.16*** (0.03)
Gender	0.02 (0.02)	0.07*** (0.02)	0.005 (0.04)
Linear slope[b, c]	-0.01*** (0.004)	0.008 (0.009)	0.004 (0.01)
Gender	-	0.05*** (0.01)	0.04* (0.02)
Quadratic slope[c]	0.002** (0.0006)	-0.001 (0.001)	-0.001 (0.001)
Gender	-	-0.008*** (0.002)	-0.005* (0.002)
Variances			
Intercept	0.002** (0.0006)	0.002* (0.0008)	0.01** (0.003)
Linear slope	-	0.001** (0.0003)	0.001** (0.0005)
Quadratic slope	-	0.00002** (0.00001)	0.00002** (0.00001)
Within-subject	0.001*** (0.0002)	0.001*** (0.0002)	0.002*** (0.0003)

Note. Intercept values reflect baseline cortisol (pg/ml) for female participants. Linear slopes reflect change in cortisol per 15 min, and quadratic slopes reflect change in cortisol per 15 min^2. Values in parentheses are standard errors. Multivariate contrasts: No Stress vs. Stress and Stress + Support, $\chi^2(3) = 6.26$, $p = .10$; No Stress vs. Stress, $\chi^2(3) = 8.18$, $p = .04$; No Stress vs. Stress + Support, $\chi^2(3) = 13.35$, $p = .004$; Stress vs. Stress + Support, $\chi^2(3) = 3.84$, $p = .28$.

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

[a]Significant difference between the Stress and Stress + Support group

[b]Significant difference between the No Stress and Stress group

[c]Significant difference between the No Stress vs. Stress and Stress + Support group

Table 30. Multi-group multilevel modeling parameter estimates for cortisol, including gender as a level-2 predictor.

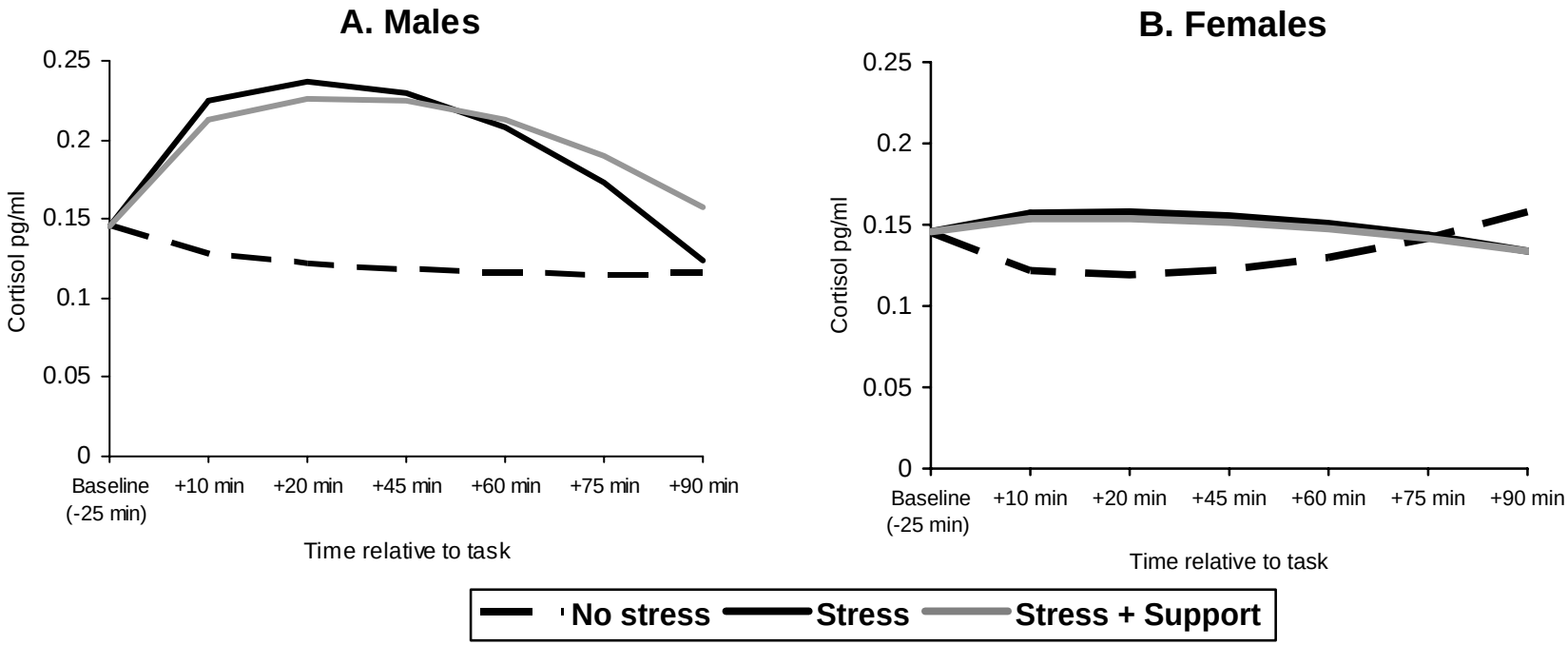

Figure 18. Predicted cortisol levels based on the full-sample multilevel model parameter estimates by group, with separate plots for A) Males and B) Females.

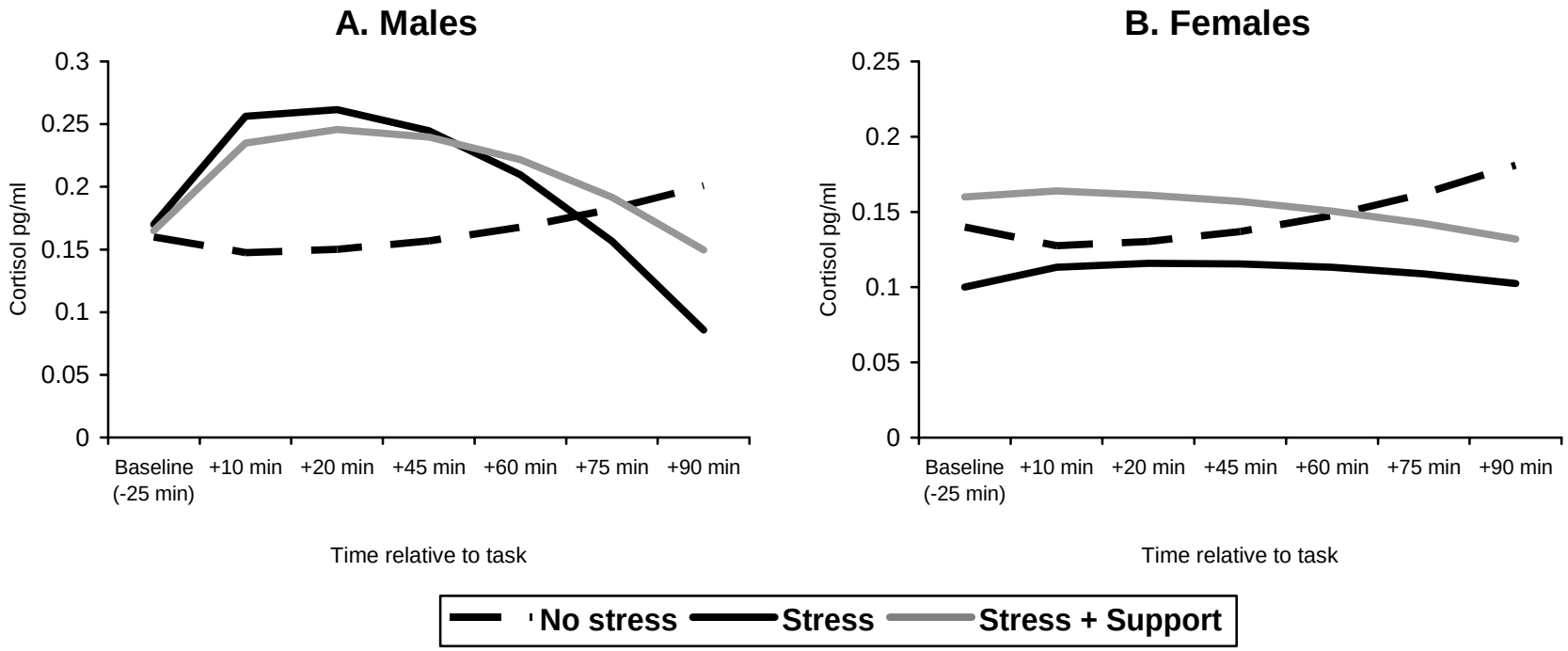

Figure 19. Predicted cortisol levels based on the multi-group multilevel model parameter estimates by group, with separate plots for A) Males and B) Females.

both the Stress and Stress + Support groups showed significant increases in cortisol compared to the No Stress group. By contrast, for female participants, the No Stress group showed higher initial levels of cortisol compared to the Stress group, and a larger increase in cortisol over time compared to the Stress and Stress + Support groups.

Gender differences in cortisol reactivity

The small effects of stress on cortisol in the female participants, though observed in other studies, were surprising. Therefore, I investigated potential explanations for this finding. The most likely variable that could explain the relationship between low cortisol reactivity during stress in the female participants was menstrual phase. Women in the follicular phase typically show attenuated cortisol reactivity in response to the TSST compared to women in the luteal phase and men (Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999). As noted earlier, the groups did not systematically differ in distribution of menstrual phase.

Including Group and Gender + menstrual phase in a general linear model with cortisol AUC_I as a dependent variable yielded significant effects for Group, $F(2,65) = 7.98$, $p = .001$, $r = .33$, Gender + menstrual phase, $F(2,65) = 5.37$, $p = .007$, $r = .28$, with no significant interaction between Group and Gender + menstrual phase, $F(4,65) = 1.00$, $p = .41$, $r = .12$. The group effect reflected the same effects described above, with the Stress and Stress + Support groups differing from the No Stress group, but not from each other. Males showed greater cortisol reactivity in general compared to women in either phase, regardless of Group assignment, as described above. However, women in the luteal phase did not significantly differ in cortisol reactivity compared to women in the

follicular phase (95% *CI* = -3.49 – 5.55). Therefore, menstrual phase could not account for the lower cortisol responses in men compared to women.

Hypothesis 3 summary

Exposure to the TSST produced the expected increases in cardiovascular and cortisol reactivity. However, contrary to Hypothesis 3, there was no significant attenuation of physiological reactivity for participants in the Stress + Support group. While the Stress group showed increases in cardiovascular reactivity that were 7 – 30% larger times the Stress + Support group, the differences were not significant. Results for residualized change approached significance, with the Stress group showing increases in cardiovascular reactivity that were 1.3 – 5.4 times the Stress + Support group. The multilevel modeling results showed no significant differences between the two groups. Similar to the cardiovascular reactivity results, the Support manipulation did not reduce cortisol reactivity. One notable finding for cortisol reactivity was the relative lack of reactivity to the TSST in women, regardless of menstrual phase, compared to men (Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999).

Hypothesis 4: Increased physiological reactivity will be related to delayed skin barrier recovery

These analyses tested whether physiological reactivity would be significantly related to skin barrier recovery. Prior testing this hypothesis, I determined the relationships among baseline cortisol, cortisol AUC_I and cardiovascular reactivity (Table 31). Elevated cardiovascular reactivity was associated with elevated cortisol reactivity, and baseline cortisol was not significantly related to cardiovascular reactivity.

Cardiovascular variable	Baseline (pre-task) cortisol	Cortisol AUC_I
Delta SBP ($N = 67$)	.02	.42***
Delta DBP ($N = 67$)	.02	.33**
Delta MAP ($N = 67$)	.01	.36**
Delta HR ($N = 66$)	.02	.41**

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

Table 31. Correlations between cortisol reactivity and cardiovascular reactivity.

Hierarchical linear regression analyses

To determine which components of physiological reactivity would be most strongly related to skin barrier recovery, I conducted a hierarchical linear regression with cardiovascular reactivity and cortisol as independent variables, and % skin barrier recovery at 2 h as the independent variable. In the first step of the model, simple change in heart rate, SBP, and DBP were included (MAP was not included because it is highly correlated with both SBP and DBP). In the second step of the model, cortisol AUC_I was included.

Percent recovery. As shown in Table 32, after step 1 (cardiovascular reactivity) R^2 = .002, and after step 2 (cortisol reactivity) R^2 = .04, and the R^2 change was not

significant (p = .17). The cardiovascular variables did not predict skin barrier recovery (β's from -.13 - .05). Increased cortisol reactivity was not significantly related to skin barrier recovery. The final model was not significant, $F(4,53) = 0.52$, p = .72.

Temperature corrected TEWL. As shown in Table 33 after step 1 (cardiovascular reactivity) R^2 = .04, and after step 2 (cortisol reactivity) R^2 = .04, and the R^2 change was not significant (p = .90). The cardiovascular variables did not predict skin barrier recovery (β's from -.17 - .29). Increased cortisol reactivity was not significantly related to skin barrier recovery. The final model was not significant, $F(4,58) = 0.58$, p = .69.

Temperature and control corrected TEWL. As shown in Table 34, after step 1 (cardiovascular reactivity) R^2 = .03, and after step 2 (cortisol reactivity) R^2 = .04, and the R^2 change was not significant (p = .53). The cardiovascular variables did not predict skin barrier recovery (β's from -.20 - .34). Increased cortisol reactivity was not significantly

Step and variable	*r*	*B*	*β*	*t*	R^2	R^2 change
1. Delta SBP	-.02	-0.16	-.08	-0.28	-	-
Delta DBP	.01	0.24	.05	0.23	-	-
Delta HR	-.002	0.10	.04	0.16	.002	.002
2. Delta SBP	-.02	-0.25	-.13	-0.43	-	-
Delta DBP	.01	0.23	.05	0.23	-	-
Delta HR	-.002	-0.05	-.02	-0.08	-	-
Cortisol AUC_I	.17	0.80	.21	1.41	.04	.036

Note. Final model: $F(4,53) = 0.52$, $p = .72$.

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

Table 32. Multiple linear regression of cortisol and cardiovascular reactivity on % skin barrier recovery at 2 h post-tape-stripping.

Step and variable	r	B	β	t	R^2	R^2 change
1. Delta SBP	.16	0.004	.29	1.07	-	-
Delta DBP	.14	0.000	.003	0.02	-	-
Delta HR	.05	-0.003	-.17	-0.84	.04	.04
2. Delta SBP	-.16	0.004	-.13	.29	-	-
Delta DBP	.14	0.000	.05	.004	-	-
Delta HR	.05	-0.003	-.02	-.16	-	-
Cortisol AUC_I	.04	0.000	.21	-.02	.04	.00

Note. Final model: $F(4,58) = 0.58$, $p = .68$.

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

Table 33. Multiple linear regression of cortisol and cardiovascular reactivity on temperature-corrected TEWL at 2 h post-tape-stripping.

Step and variable	r	B	β	t	R^2	R^2 change
1. Delta SBP	.10	0.004	.32	1.19	-	-
Delta DBP	.07	-0.002	-.06	-0.29	-	-
Delta HR	-.01	-0.004	-.22	-1.12	.03	.03
2. Delta SBP	.10	0.004	.34	1.24	-	-
Delta DBP	.07	-0.002	-.05	-0.26	-	-
Delta HR	-.01	-0.004	-.20	-0.99	-	-
Cortisol AUC_I	-.05	-0.002	-.09	-0.64	.04	.007

Final model: $F(4,58) = 0.57$, $p = .68$.

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

Table 34. Multiple linear regression of cortisol and cardiovascular reactivity on temperature- and control-corrected TEWL at 2 h post-tape-stripping.

related to skin barrier recovery. The final model was not significant, $F(4,58) = 0.57$, $p = .68$.

Multilevel modeling analyses

The models were similar to the multilevel analyses for Hypotheses 1 and 2. However, instead of including Group as a level-2 predictor of intercepts and slopes, simple change in SBP, DBP, and HR, and cortisol AUC_I were included as level-2 predictors. Between the cardiovascular and cortisol reactivity measures, there was 7.1 – 9.4% missing data. While multilevel analyses can accommodate missing data for the dependent variable, it cannot accommodate missing data for the independent variables. This resulted in only 66 participants with available data (all cardiovascular and cortisol reactivity measures intact) for the analyses.

Quantitative researchers strongly advocate that imputation or other related methods are almost always superior to listwise deletion (Babyak, 2005). Therefore, in this section I report results from 2 sets of analyses. The first set of analyses is from the 66 participants after listwise deletion of participants with incomplete cardiovascular and/or cortisol reactivity data. The second set of analyses is from the full sample of 80 participants (after removing outliers for TEWL and cortisol reactivity). In this set of analyses, missing cardiovascular and/or cortisol reactivity data was imputed using the expectation maximization (EM) approach (Little & Rubin, 1987). This approach is a two-step iterative procedure. In the expectation step, the expected value of the complete data is computed. In the maximization step, the expected values are substituted for the missing data, and a maximum likelihood function is estimated. These steps are repeated until convergence is obtained. The EM method is superior to listwise, pairwise, and mean

substitution approaches (see www.utexas.edu/its/rc/answers/general/gen25.html). However, it lacks the uncertainty component contained in raw maximum likelihood methods (used in modeling the dependent variables in multilevel models) and multiple imputation methods. The raw maximum likelihood method could not be used for missing independent variables. The most ideal method of dealing with missing data is multiple imputation, in which multiple datasets are created with imputed data and subsequently analyzed. However, this method is cumbersome, requiring analyzing data from five to ten imputed datasets, then recombining the results into one summary.

Percent recovery. Analyses including cortisol, HR, SBP, and DBP reactivity as predictors of intercepts and slopes showed a significant interaction between SBP reactivity and quadratic slopes in both the listwise deletion sample and the imputed data sample (Table 35). As shown in Figure 20, larger increases in SBP during the tasks were significantly related to faster recovery. No other physiological variables were significantly related to recovery, although there was a trend for larger increases in heart rate to be associated with slower recovery. Including physiological variables accounted for an additional 3% of the variance in intercepts, 6% of the variance in linear change, and 12% of the variance in quadratic change in the listwise deletion sample, and an additional 0.6% of the variance in intercepts, 3% of the variance in linear change, and 1% of the variance in quadratic change in the imputed data sample.

Temperature corrected TEWL. Table 36 indicates that there were no significant relationships between physiological reactivity and intercepts or cubic slopes for both the listwise deletion and imputed data samples.

Parameter	Listwise deletion sample ($N = 66$)			Imputed data sample ($N = 80$)		
	Means	Variances	Reliability	Means	Variances	Reliability
Intercept	36.09*** (6.39)	546.82*** [23.38]	.80	33.23*** (5.08)	501.56*** [22.40]	.77
Delta HR	0.54 (0.46)			0.45 (0.41)		
Delta SBP	-0.48 (0.46)			-0.43 (0.50)		
Delta DBP	-0.15 (0.84)			0.04 (0.73)		
Cortisol AUC_I	0.18 (0.54)			0.18 (0.50)		
Linear slope	19.81*** (2.91)	102.24*** [10.11]	.48	18.31*** (2.55)	86.97*** [9.33]	.42
Delta HR	-0.02 (0.21)			-0.002 (0.20)		
Delta SBP	0.16 (0.16)			0.13 (0.15)		
Delta DBP	-0.56 (0.40)			-0.44 (0.35)		
Cortisol AUC_I	0.17 (0.22)			-0.02 (0.23)		
Quadratic slope	-13.98** (4.71)	130.01*** [11.40]	.33	-12.25** (3.71)	110.96*** [10.53]	.29
Delta HR	-0.60 (0.32)			-0.53 (0.28)		
Delta SBP	0.61* (0.29)			0.58* (0.25)		
Delta DBP	-0.09 (0.60)			-0.25 (0.52)		
Cortisol AUC_I	0.14 (0.40)			0.06 (0.33)		

Table 35 (continued).

Note. Intercept values reflect TEWL 1 h after tape-stripping. Linear slopes reflect change in TEWL per 15 min of time, and quadratic slopes reflect change in TEWL per 15 min of time^2. For the listwise deletion sample, within-subjects variance = 192.84 [13.89], correlation between intercepts and linear slopes = .95; intercepts and quadratic slopes = -.95; linear and quadratic slopes = -.81. For the imputed data sample, within-subjects variance = 208.21 [14.43], correlation between intercepts and linear slopes = .95; intercepts and quadratic slopes = -.97; linear and quadratic slopes = -.86. Values in parentheses are standard errors and values in brackets are standard deviations.

** $p < .01$, *** $p < .001$.

Table 35. Multilevel modeling parameter estimates for % skin barrier recovery, including cardiovascular and cortisol reactivity as level-2 predictors of intercepts and slopes.

Parameter	Listwise deletion sample ($N = 66$)			Imputed data sample ($N = 80$)		
	Means	Variances	Reliability	Means	Variances	Reliability
Intercept	1.28*** (0.04)	0.03*** [0.16]	.89	1.30*** (0.03)	0.02*** [0.15]	.87
Delta HR	-0.002 (0.003)			-0.002 (0.003)		
Delta SBP	0.003 (0.002)			0.003 (0.002)		
Delta DBP	0.003 (0.005)			-0.0004 (0.005)		
Cortisol AUC_I	0.0006 (0.003)			0.001 (0.003)		
Linear slope	-0.13*** (0.02)			-0.12*** (0.02)		
Cubic slope	0.11*** (0.01)	0.0009*** [0.03]	.51	0.11*** (0.01)	0.0006*** [0.03]	.43
Delta HR	0.0003 (0.006)			0.0004 (0.0005)		
Delta SBP	-0.0007 (0.007)			-0.006 (0.0007)		
Delta DBP	0.002 (0.001)			0.001 (0.001)		
Cortisol AUC_I	-0.0008 (0.008)			-0.0009 (0.0006)		
Within-subjects variance		0.01 [0.11]			0.01 [0.12]	

Note. Intercept values reflect TEWL 1 h after tape-stripping. Linear slopes reflect change in TEWL per 15 min of time, cubic slopes reflect change in TEWL per 15 min of time^3. For both samples, correlation between intercepts and cubic slopes = .65. Values in parentheses are standard errors and values in brackets are standard deviations. ** $p < .01$, *** $p < .001$.

Table 36. Multilevel modeling parameter estimates for temperature-corrected TEWL, including cardiovascular and cortisol reactivity as level-2 predictors of intercepts and slopes.

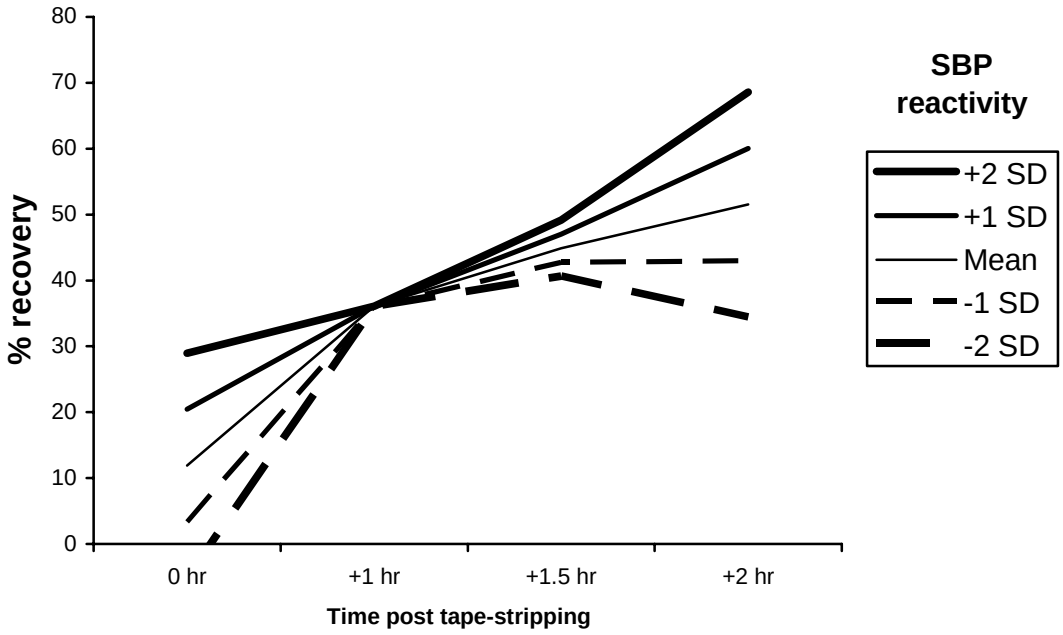

Figure 20. Predicted % skin barrier recovery during the session as a function of increasing systolic blood pressure reactivity, based on parameter estimates from Table 36.

Temperature and control corrected TEWL. Similar to the analyses for temperature-corrected TEWL, there were no significant relationships between physiological reactivity and intercepts or cubic slopes for the listwise deletion sample (Table 37). In the imputed data sample, there was one significant relationship between SBP reactivity and temperature- and control-corrected TEWL at 1 h post-tape-stripping (which approached significance in the listwise deletion sample, $p = .07$). Larger increases in SBP during the task were related to elevated temperature- and control-corrected TEWL at 1 h post-tape-stripping, suggesting that elevated SBP reactivity is related to slower healing.

Analyses with baseline cortisol

Previous studies of stress, glucocorticoids, and skin barrier recovery often disrupted the skin barrier after administration of stress or glucocorticoids. By contrast, this study disrupted the skin barrier prior to the stressor. Given that previous studies that administered glucocorticoids or stress prior to skin barrier disruption found stress- and/or glucocorticoid-related delays in skin barrier recovery, it may be possible that glucocorticoid levels prior to barrier disruption rather than reactivity to a stressor after barrier disruption are related to delays in skin barrier recovery. These analyses examined the relationship between baseline cortisol from the first cortisol sample, taken just prior to skin barrier disruption, and skin barrier recovery using multilevel modeling.

Baseline cortisol was unrelated to skin barrier recovery when assessed with percent recovery, both in the listwise deletion and EM sample. However, baseline cortisol was significantly related to cubic slopes for temperature-corrected and temperature- and control-corrected TEWL. For temperature-corrected TEWL, the relationship between

Parameter	Listwise deletion sample ($N = 66$)			Imputed data sample ($N = 80$)		
	Means	Variances	Reliability	Means	Variances	Reliability
Intercept	0.32*** (0.04)	0.02*** [0.14]	.85	0.34*** (0.04)	0.02*** [0.13]	.80
Delta HR	-0.004 (0.002)			-0.004 (0.002)		
Delta SBP	0.003 (0.002)			0.004* (0.002)		
Delta DBP	0.005 (0.004)			0.002 (0.004)		
Cortisol AUC_I	-0.003 (0.003)			-0.003 (0.002)		
Linear slope	-0.12*** (0.02)			-0.12*** (0.02)		
Cubic slope	0.09*** (0.01)	0.0008*** [0.02]	.50	0.10*** (0.01)	0.0006** [0.02]	.37
Delta HR	0.0001 (0.0006)			0.0002 (0.0005)		
Delta SBP	-0.0002 (0.0006)			-0.0003 (0.0006)		
Delta DBP	0.002 (0.001)			0.001 (0.001)		
Cortisol AUC_I	-0.0006 (0.0006)			-0.001 (0.0007)		
Within-subjects variance	0.01 [0.11]			0.02 [0.13]		

Note. Listwise deletion sample, correlation between intercepts and cubic slopes = .89; imputed data sample, correlation between intercepts and cubic slope = .92. Values in parentheses are standard errors and values in brackets are standard deviations. ** $p <$ .01, *** $p <$ .001.

Table 37. Multilevel modeling parameter estimates for temperature- and control-corrected TEWL, including cardiovascular and cortisol reactivity as level-2 predictors of intercepts and slopes.

baseline cortisol and cubic slopes approached significance in the listwise deletion sample, unstandardized $\beta = 0.17$ ($SE = 0.09$), $p = .07$, and was statistically significant in the EM sample, unstandardized $\beta = 0.2$ ($SE = 0.05$), $p = .03$. For temperature- and control-corrected TEWL, the relationship between baseline cortisol and cubic slopes was statistically significant in the listwise deletion sample, unstandardized $\beta = 0.20$ ($SE = 0.09$), $p = .03$, and the EM sample, unstandardized $\beta = 0.15$ ($SE = 0.06$), $p = .01$. The relationship between baseline cortisol and corrected TEWL was not due to a significant relationship between baseline cortisol and basal TEWL measured at the undisturbed control site, as there were no significant relationships between baseline cortisol and basal TEWL measured at the control site. Baseline cortisol accounted for between 17 – 24% of the variance in cubic slopes for corrected TEWL. As shown in Figure 21, greater baseline cortisol was related to slower healing (higher TEWL values) 2 h after tape-stripping. Additional models were run including gender and gender by baseline cortisol interactions predicting intercepts and cubic slopes. There were no significant effects of gender, or gender by baseline cortisol interactions in predicting intercepts and cubic slopes. Thus, while no results were found for percent recovery, these results suggest that baseline levels prior to stress, rather than cortisol reactivity to stress may be related to slower skin barrier recovery.

Hypothesis 4 summary

Overall, there was little support for the hypothesis that elevated physiological reactivity would predict poorer wound healing. The linear regression analyses yielded no significant findings, while the multilevel model analyses yielded two significant findings that were contrary to the hypothesis. Specifically, greater SBP responses to the task were

related to faster healing as indexed by percent recovery. Another finding for SBP reactivity suggested that greater SBP responses to the task were related to slower healing as indexed by temperature- and control-corrected TEWL. The latter finding was only observed when using imputed data, and should be interpreted with caution. No other cardiovascular reactivity measures were related to healing, and cortisol reactivity was not related to healing in any analyses. Baseline cortisol was related to slower healing as measured with corrected TEWL measures, but not percent recovery.

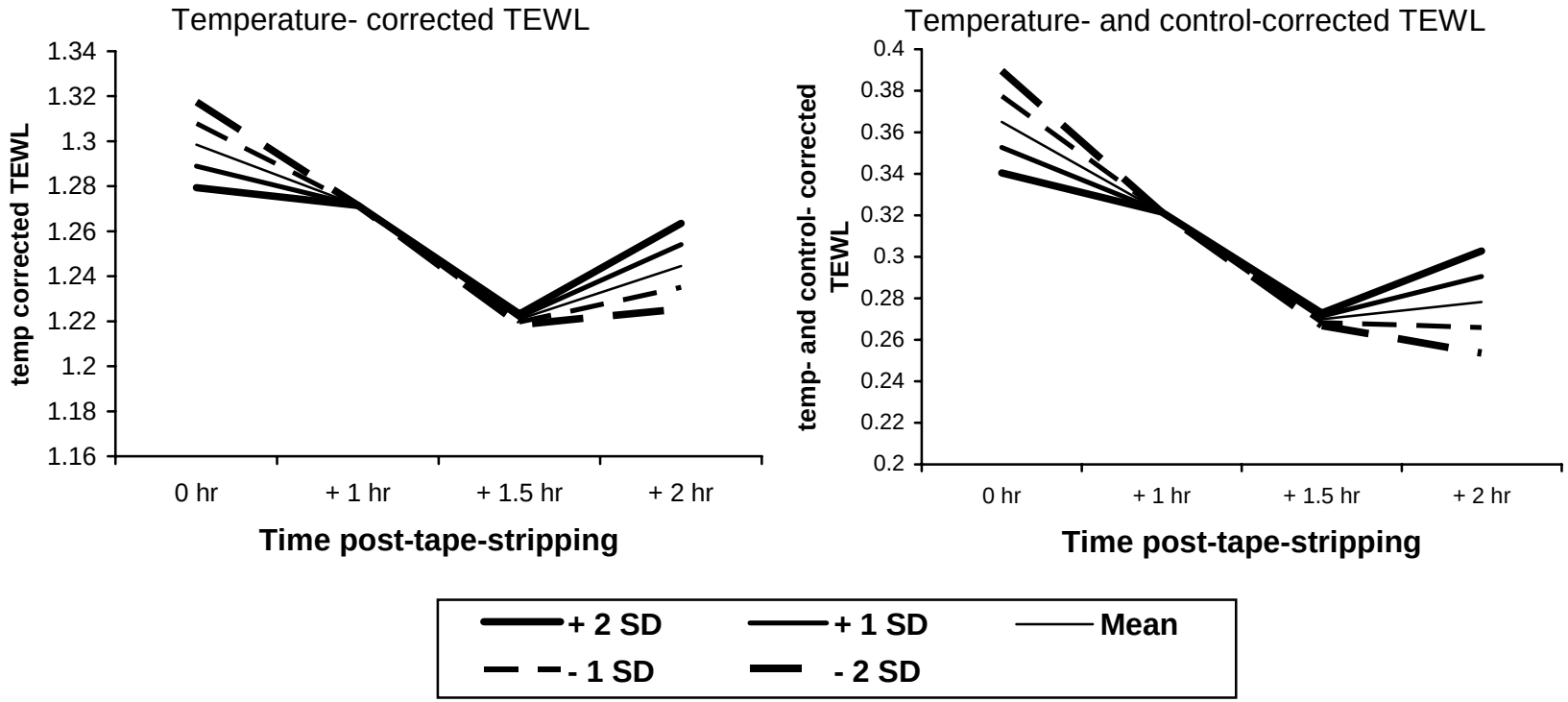

Figure 21. Predicted temperature- and temperature- and control-corrected TEWL during the session as a function of increasing baseline cortisol.

Hypothesis 5: Stress-induced cortisol responses will show distinct and reliable patterns of reactivity from and recovery to baseline

These analyses are organized according to the steps outlined by Llabre and colleagues for modeling reactivity and recovery using piecewise latent growth curve modeling (Llabre, Spitzer, Saab, & Schneiderman, 2001). Because these analyses were concerned with stress-induced cortisol responses, only data from the Stress and Stress + Support groups was used. Outliers identified in the *Hypothesis 3* section were removed, leaving a sample size of 56 participants.

The first step in these analyses was graphing the data to examine how cortisol changes over time. The raw data plots are shown in Figure 22, and are plotted separately for men and women. The general pattern of change suggested increases in cortisol from the post-stripping baseline sample (Time 1) to the sample obtained 30 min after the start of the task (Time 3). Some participants showed a purely linear pattern of change over the three time points, while others showed a quadratic pattern of change. The decrease from Time 3 to Time 7 was generally linear. As was found in the Hypothesis 3 analyses, female participants showed smaller cortisol responses compared to men.

As discussed in *Hypothesis 3* the time points were not equivalent across all participants. Latent growth curve modeling is not flexible enough to accommodate variability in measurement times. Therefore, I averaged collection time relative to the start of the task across all participants. The intended timing for the 7 samples in these analyses was -15, 10, 30, 45, 60, 75, and 90 min relative to the start of the task. The average collection times were -25, 14, 34, 49, 64, 78, and 91 min relative to the start of

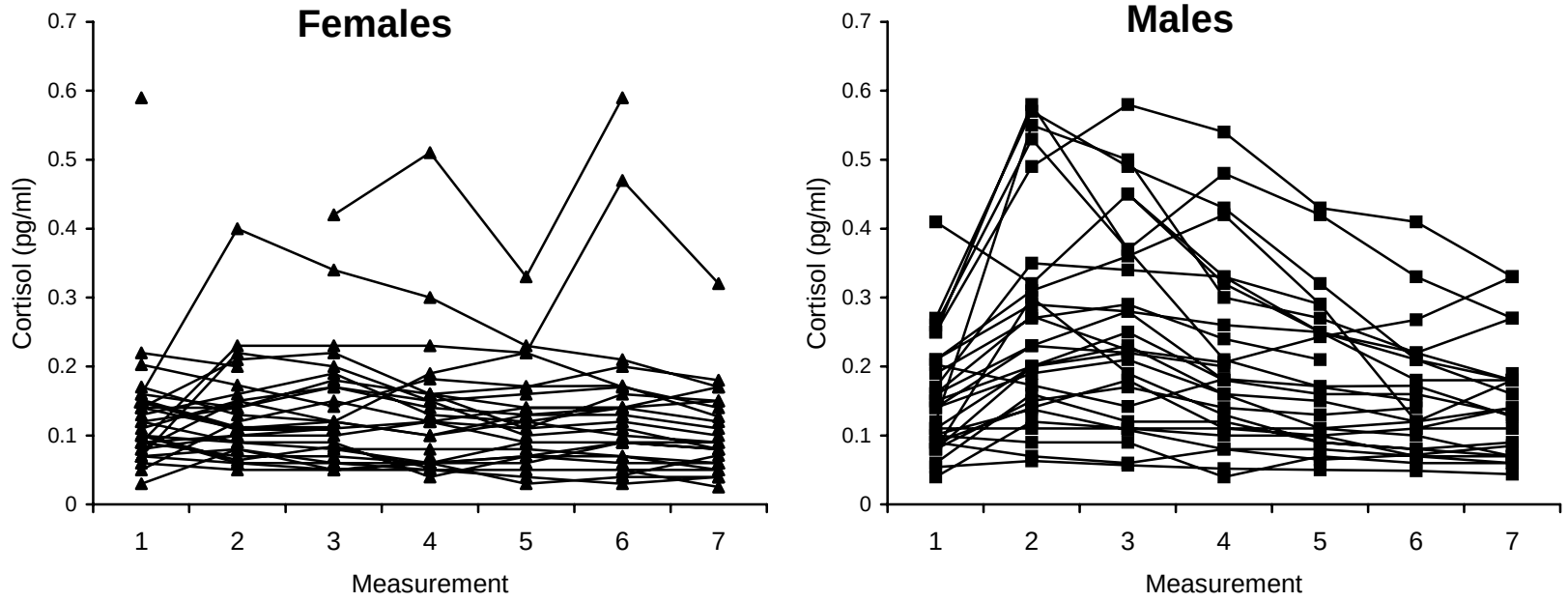

Figure 22. Individual data plots of cortisol from post-tape-stripping to 90 min after the task, by gender. Measurement 1 corresponds to Cortisol 2, the cortisol measurement after tape-stripping and just prior to receiving instructions about the task.

the task. For the remaining analyses, time was rescaled by setting Time 1 at 0 and dividing each average collection time by the minutes between Time 1 and Time 2. This was done to provide more reasonable starting values for the latent growth curve analyses, and resulted in values of 0, 1, 1.53, 1.91, 2.30, 2.66, and 3.01. In all the models, error variances for each cortisol sample were set as equal.

The second step was modeling reactivity as a function of time. Two latent variables were estimated: baseline and reactivity. The conceptual model is shown in Figure 23A, with time values (0, 1, and 1.53) included as fixed loading parameters. This model showed poor fit to the data, $\chi^2(3) = 33.40$, $p = .00$, $RMSEA = 0.43$. Thus, rather than including time values as fixed parameters, only Time 1 and Time 2 were fixed at 0 and 1 respectively. The loading for Time 3 was set as a free parameter, which was estimated by the model, rather than fixed. Thus, rather than a pure linear slope parameter, the latent reactivity variable becomes what is termed a "shape" parameter, where the loadings describe the shape of change over time, but are not necessarily reflective of change per unit of time (MacCallum, Kim, Malarkey, & Kiecolt-Glaser, 1997). This model showed good fit to the data, $\chi^2(3) = 0.53$, $p = .77$, $RMSEA = 0.00$. Parameter estimates for the model are shown in Table 38. Baseline or intercept cortisol was estimated at 0.14 pg/ml, and reactivity "shape" was estimated as 0.07, indicating an increase in cortisol over time (Figure 23B). In this model, the baseline and reactivity latent variables were not significantly correlated, $r = .12$.

The next step was modeling recovery as a function of time. Based on Figure 23 recovery was expected to be linear. Two latent variables were estimated: task and recovery. The conceptual model is shown in Figure 24, with time values included as fixed

loading parameters. The time values were scaled so that the Time 3 measurement was fixed at 0 for the recovery latent variable. This model showed poor fit to the data, $\chi^2(3) = 75.59$, $p = .00$, $RMSEA = 0.29$. All other attempts to model recovery, including freeing loading parameters and modeling change for males only did not improve fit. In addition, attempts to model reactivity and recovery simultaneously were similarly unsuccessful.

An alternative approach to addressing Hypothesis 5 is using multilevel modeling in a manner similar to the models testing *Hypothesis 3.* The primary advantage to this approach is that separate linear and quadratic slopes (Table 27) represent to some extent reactivity (the linear component, which reflects increases over time) and recovery (the quadratic component, which reflects decreases over time^2). However, this interpretation is limited by the fact that both the linear and quadratic approaches incorporate both reactivity and recovery. That is, neither linear nor quadratic slopes exclusively represent either reactivity or recovery, thus the advantage of a latent growth curve approach.

In these analyses, only data from the Stress and Stress + Support group were included. As shown in Table 39, linear and quadratic slopes were distinct from one another in that they could be modeled separately. In addition, linear and quadratic slopes showed significant variances, suggesting sufficient between-subjects variability. At the same time, linear and quadratic slopes were highly correlated ($r = -.99$), indicating that larger linear increases were always related to larger quadratic decreases. Despite this, both linear and quadratic slopes showed reasonable reliability, between .69 - .73. Thus, based on the multilevel modeling results there was mixed evidence that linear and quadratic slopes, somewhat related to reactivity and recovery, respectively, were distinct components, and there was clear evidence that linear and quadratic slopes were reliable.

Parameter	Estimate (*SE*)	*t*
Means		
Baseline	0.14*** (0.01)	11.41
Reactivity	0.07** (0.02)	4.61
Variances		
Baseline	0.007** (0.002)	4.39
Reactivity	0.01** (0.002)	4.09
Baseline/reactivity correlation	.12	0.69

Note. Baseline values reflect baseline cortisol (pg/ml). Final model showed good fit to the data, $\chi^2(3) = 0.53$, $p = .77$, $RMSEA = 0.00$.

** $p < .01$, .*** $p < .001$.

Table 38. Parameter estimates for cortisol reactivity model.

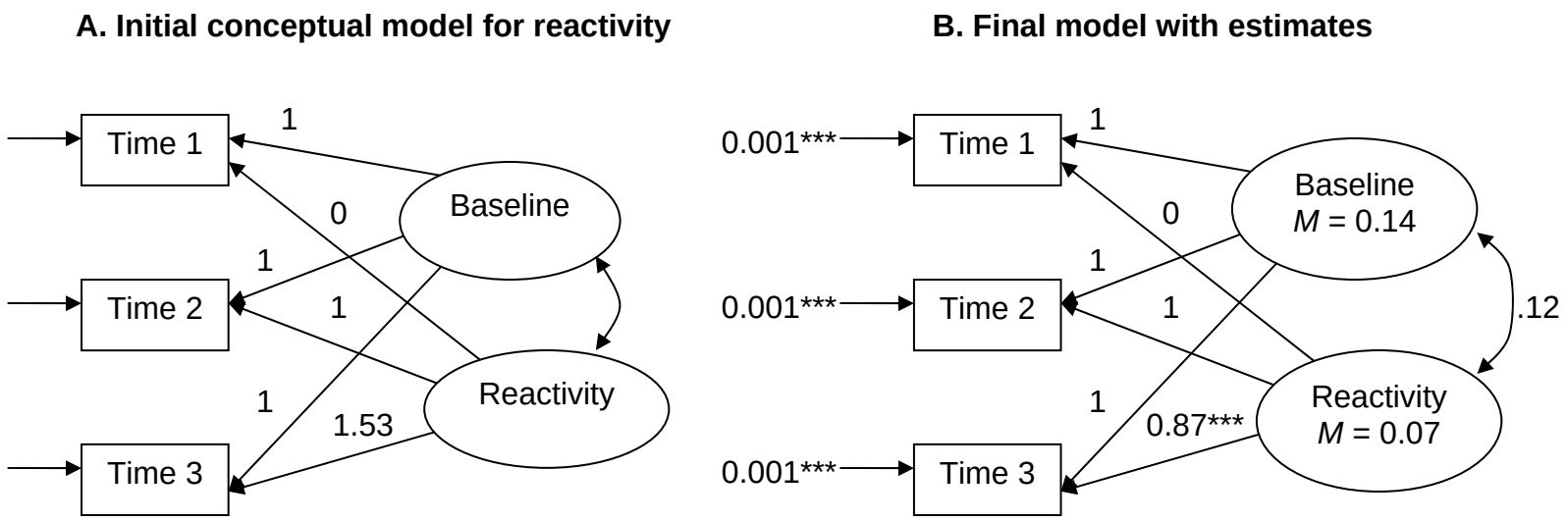

Figure 23. Latent growth curve models for cortisol reactivity. A) Initial model, B) Final model including estimates for each parameter in the model. With the exception of the correlation between the baseline and reactivity latent variables, which is a standardized estimate, all other estimates are unstandardized estimates. Final model showed good fit to the data, $\chi^2(3) = 0.53$, $p = .77$, $RMSEA = 0.00$.

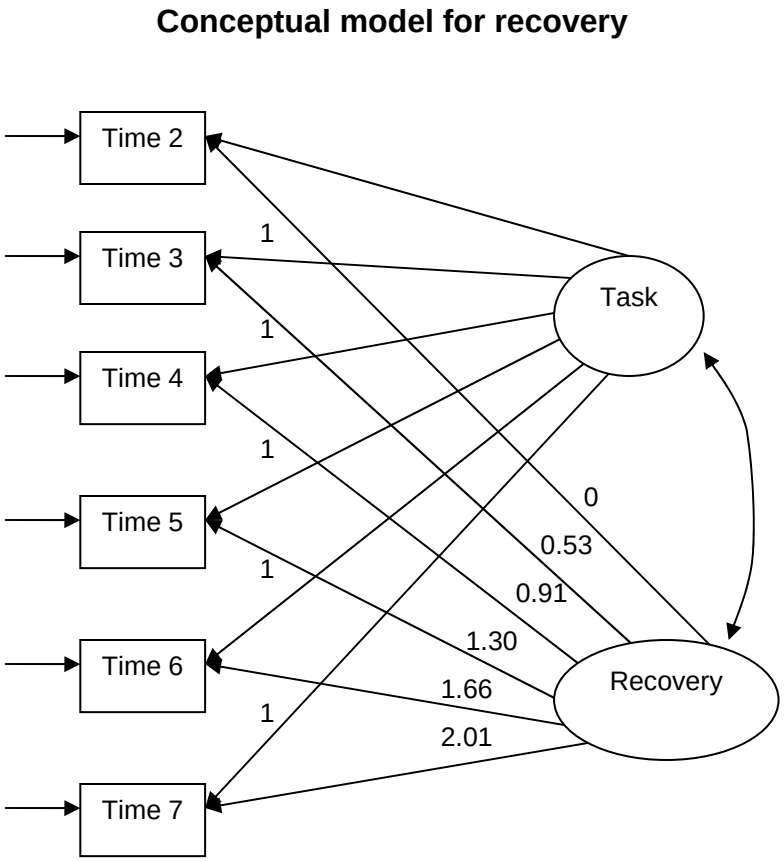

Figure 24. Conceptual latent growth curve model for cortisol recovery. No final model is displayed because no models could be reliably estimated for cortisol recovery.

Parameter	Mean	Variance	Reliability
Intercept	0.15*** (0.01)	0.007*** [0.08]	.79
Linear slope	0.03** (0.006)	0.001*** [0.03]	.69
Quadratic slope	-0.003** (0.0008)	0.00002*** [0.005]	.73
Within-subjects variance		0.002 [0.04]	

Note. Intercept values reflect baseline cortisol (pg/ml). Linear slopes reflect change in TEWL per 15 min of time, and quadratic slopes reflect change in TEWL per 15 min of $time^2$. Correlation between intercepts and linear slopes = .43; intercepts and quadratic slopes = -.41; linear and quadratic slopes = -.99. Values in parentheses are standard errors and values in brackets are standard deviations.

** $p < .01$, *** $p < .001$.

Table 39. Level-1 multilevel modeling parameter estimates for cortisol change, Stress and Stress + Support groups only.

CHAPTER 4

DISCUSSION

Overview

This study provided further evidence that acute stress in the laboratory delays skin barrier recovery after disruption. However, support provided by a confederate before an acute laboratory stressor did not reduce physiological reactivity or speed skin barrier recovery after disruption. Moreover, while acute stress delayed skin barrier recovery, increased SBP reactivity was related to faster skin barrier recovery, baseline cortisol was related to faster skin barrier recovery, and cortisol reactivity was not related to skin barrier recovery.

The remainder of this discussion is organized by Hypothesis, with detailed discussion and explanation of the results, convergence and/or divergence with previous literature, and suggestions for future research. Following the hypotheses-specific discussion, I turn to general limitations of the study, concluding with the clinical implications of studying stress, social relationships, and skin barrier recovery.

Revisiting the study hypotheses

Hypothesis 1: Stress will delay skin barrier recovery.

The results supported Hypothesis 1. Specifically, the No Stress group showed 40 - 45% recovery 2 h after tape-stripping, while the Stress and Stress + Support groups showed 30-35% recovery by the same period. Thus, this study replicates previous work

by other laboratories, which showed that public speaking or academic examination stress delayed skin barrier recovery by 10-15% after 3 h (Altemus et al., 2001; Garg et al., 2001). Similar to other work, stress-related delays in skin barrier recovery could not be accounted for by changes in basal skin barrier function, as groups did not differ in TEWL at the undisturbed control site. In addition, this study extended prior work on the relationship between stress and skin barrier recovery by demonstrating effects in a between-subjects design, rather than a within-subjects design where participants serve as their own controls.

Two types of analyses were used in examining relationships between stress and skin barrier recovery: general linear models and multilevel models. The significant effects of exposure to stress were found for the multilevel models, but not the general linear models, due to several reasons. General linear models are not capable of handling missing data, whereas the multilevel models are quite robust to missing data points. The loss of subjects likely resulted in less power in the general linear models.

In addition, the general linear models contained more within-subject variability (error variance) because TEWL measures were not taken at the exact same times across participants. For instance, the 1 h post-tape-stripping measurement was assessed on average 65 min (*SD* = 8.93 min) relative to tape-stripping. Aggregating measurements spread across a range of time points and treating them as taking place at the same time point inflates measurement error. By contrast, by treating measures across participants as occurring at a specific time rather than at the same time and accounting for increments of real time in the model of change, the multilevel models reduced measurement error and thus within-subject variability.

The significant effect of stress on skin barrier recovery was observed in the multilevel analyses that computed separate estimates for each group (multi-group analyses), rather than analyses that included participants in a single sample using group status as a level-2 predictor of skin barrier recovery (full-sample analyses). In the latter, the effect of stress approached significance. The multi-group analyses provide some insight as to why effects were not statistically significant in the full-sample analyses. In the multi-group analyses, the slope variances for the Stress and Stress + Support groups were negative. Rather than suggesting very low between-subject variability in slopes, inspection of the raw and empirical Bayes' estimates actually suggested there was greater slope variability in the Stress and Stress + Support groups. The negative variances were likely due to individuals who had slopes that were zero or negative; while they did not appear as outliers, they were sufficiently different from other individuals to result in a negative variance estimate. Ultimately, the high variability contributed to results in the full-sample analyses for group status that only approached significance.

The ancillary analyses offered interesting preliminary results regarding the role of anxiety and positive affect in stress-related changes in skin barrier recovery. Participants reporting high anxious arousal showed slower skin barrier recovery compared to participants reporting low anxious arousal. However, among individuals reporting high anxious arousal, exposure to the stressor was related to faster skin barrier recovery. In a similar vein, participants reporting increasing state anxiety over the course of the task showed slower skin barrier recovery compared to participants who reported no change or decreases in state anxiety. Moreover, among individuals reporting increased state anxiety, receiving support prior to the stressor was related to faster skin barrier recovery. Finally,

individuals exposed to the stressor who also reported high levels of trait positive affect showed faster skin barrier recovery compared to those who reported low levels of trait positive affect. Overall, these results indicate that tonic levels of anxiety and positive affect and state levels of anxiety influence stress-related changes in skin barrier recovery. These findings merit further exploration and replication in future work.

A key difference between this study and previous stress and skin barrier recovery studies is that this study measured skin barrier recovery up to 2 h after tape-stripping, rather starting measures 3 h after tape-stripping. Stress-related delays in skin barrier recovery may be larger and more clinically relevant when observed later on in the process. For instance, the delays in skin barrier recovery observed during academic examinations were observed up to 24 h after tape-stripping, but the magnitude of differences between stress and no stress periods appeared largest at 3 and 6 h (e.g., Garg et al 2001). The fact that stress-related delays in skin barrier recovery persist for up to a day, and perhaps longer, may have important clinical implications for populations with certain skin diseases like psoriasis. More generally, longer skin barrier recovery may put individuals at increased risk for exposure to foreign pathogens. Both potential consequences are discussed further in the *Clinical implications* section.

The effect of short-term stressors on skin barrier recovery has now been demonstrated in at least three independent studies in humans. Describing the mechanisms through which stress results in delayed skin barrier recovery requires separating the mechanisms into two parts: Which cells are affected and how; and how is "stress" communicated to those cells?

Eventually, the effects of stress on skin barrier recovery occur in the stratum corneum. As discussed in the *Introduction*, the stratum corneum is organized into a bricks-and-mortar, or "two-compartment" system consisting of the corneocyte "bricks" and the extracellular matrix "mortar." The extracellular mortar is rich in lipid content, and the lipids are produced by organelles called lamellar bodies, which are located in all nucleated epidermal cells, that secrete proteins and lipids into the extracellular matrix (Elias, 2005).

Skin barrier recovery involves restoring lipids to the extracellular matrix and organization of new lipids into membrane structures. Delays in skin barrier recovery likely result from a delay in lipid synthesis and processing. Indeed, recent murine studies suggest that psychological stress influences lipid synthesis and formation of lamellar bodies. Mice exposed to 42 h of continuous light and radio noise showed delayed skin barrier recovery, decreased production and secretion of lamellar bodies, and a 35-50% reduction in epidermal lipid synthesis (Choi et al., 2005). Applying topical lipids reversed these effects, which suggests that lipid synthesis and processing are a key mechanism explaining stress-related delays in skin barrier recovery. In addition to effects on lipid synthesis, stress also impaired keratinocyte proliferation and differentiation, and decreased the overall integrity of the stratum corneum.

The second key question in understanding the mechanisms of stress-related delays in skin barrier recovery is how the stress "signal" is communicated to the cells in the stratum corneum. Garg and colleagues speculated three potential mechanisms: Stress-related activation of immune and inflammatory processes in deeper skin layers, neuropeptide release from afferent nerves in the peripheral nervous system, and systemic

glucocorticoid levels (Garg et al., 2001). These mechanisms are described in further detail in the discussion of *Hypothesis 4*.

Hypothesis 2: Support will speed skin barrier recovery.

In contrast to Hypothesis 1, where there was support for stress delaying skin barrier recovery, there was no support for Hypothesis 2, that social support from a confederate would speed skin barrier recovery. The Stress and Stress + Support group showed similar rates of healing, and one planned comparison suggested that the Stress group had lower temperature-corrected TEWL values, hence faster healing, than the Stress + Support group. The best support for Hypotheses 2 came from the ancillary analyses that found that individuals in the Stress + Support group who reported increasing state anxiety during the task showed faster skin barrier recovery compared to the Stress and No Stress groups.

The absence of a social support manipulation effect on skin barrier recovery may be due to several factors, notably the efficacy of the support manipulation in reducing perceptions of threat, and the effectiveness of the confederates in providing support. Indeed, these issues are highlighted by the finding that perceptions of the confederate as assured and dominant were related to slower skin barrier recovery. The section on *Hypothesis 3* discusses these issues in more detail, including the effect of the support manipulation on physiological reactivity. Similar to skin barrier recovery, there was little evidence that the support manipulation reduced cardiovascular and cortisol reactivity.

Despite the lack of a support manipulation effect, this does not discount the role of social interaction in skin barrier recovery and wound healing more generally. Two animal studies demonstrate that social contact can influence wound healing. In an initial

study, Siberian hamsters received a punch biopsy wound, and were then exposed to 2 h of restraint stress for 14 days (Detillion, Craft, Glasper, Prendergast, & DeVries, 2004). Hamsters that were housed individually from the time of weaning (socially isolated) took longer to heal, with delays in healing up to 10 days after wounding, compared to hamsters that were housed with a female sibling from the time of weaning (pair-housed). This finding was replicated in a later study in mice species (Glasper & DeVries, 2005). The Glasper study also highlighted the effect of social disruption on wound healing observed in other studies (Sheridan, Padgett, Avitsur, & Marucha, 2004). In two monogamous mouse species, separating mice that were pair-housed for two weeks for 48 h prior to wounding resulted in delays in wound healing similar to socially isolated mice (Sheridan, Padgett, Avitsur, & Marucha, 2004).

A recent study in humans demonstrated that socially supportive interactions are related to faster wound healing relative to conflict interactions (Kiecolt-Glaser et al., 2005). In this study, couples participated in two 26 h admissions to a hospital research unit. One admission included a 20 min social support interaction task, while the other admission included a 30 min conflict interaction task. At each admission, couples received suction blister wounds, and healing was assessed up to 12 days after wounding. The rate of healing after the conflict visit was 72% of the rate of healing after the social support visit, suggesting that the conflict task resulted in stress-related delays in wound healing, or possibly that social support speeded wound healing relative to the conflict task.

The current project differs from the aforementioned studies of animal and human social interaction in one key aspect: the duration of social contact. In the animal studies,

the social contact manipulations were long-term manipulations. In the comparisons of pair-housed vs. socially isolated rodents, the two groups had literally different lifetime histories of social contact. In mice that were isolated after previous pair housing, the isolation occurred for 48 hr. In the marital study in humans, the degree of social contact was also longer (20 – 30 min) compared to the current study (10 min). Moreover, unlike the confederates and participants in this project, who had no relationship history, the participants in the marital study had a significant relationship history, having been married at least 3 years, with an average of 12.5 years of marriage in the sample. Taken together, these studies suggest that the beneficial effects of social support and the detrimental effects of social disruption on wound healing are best observed by examining social interactions over longer periods of time (hours to days), in established relationships (friends, partners), and/or prolonged social disruption (e.g., separation, divorce, loneliness). Thus, a key future direction for this work will be incorporating real-life social interactions, relationships, and stressors in understanding social facilitation or disruption of skin barrier recovery and wound healing.

Hypothesis 3: Social support will reduce physiological reactivity.

The cardiovascular results provided limited support, and the cortisol results provided no support for the hypothesis that the social support manipulation would reduce physiological reactivity. The effects of the social support intervention in this study were generally smaller compared to those observed in the literature. Effect sizes for the difference between the Stress and Stress + Support groups ranged from 0.14 - 0.39 for simple change scores and 0.41 – 0.56 for residualized change scores (Cohen's *d*). By comparison, the average effect size for social support manipulations reported by

Thorsteinsson and James (1999) ranged from 0.51 – 0.61. For comparisons between supportive confederates vs. performing alone, the effect sizes computed by Thorsteinsson and James ranged from 0.25 – 0.35 for blood pressure (vs. 0.27 - 0.29 for simple change and 0.52 - 0.56 for residualized change in this study), and 0.74 for heart rate (vs. 0.39 for simple change and 0.47 for residualized change in this study). While some effects in this study were larger than the average effect sizes reported by Thorsteinsson and James, there was likely not enough power to detect group differences given the sample size in the Stress and Stress + Support groups.

The results for residualized change, though not significant, were more in line with the study hypotheses compared to the results for simple change. Both residualized and simple change are considered reliable (Llabre, Spitzer, Saab, Ironson, & Schneiderman, 1991). However, Llabre and colleagues lean towards reporting simple change, as the values are more generalizable across tasks and studies, and are not dependent on sample characteristics. Given that simple change was not significantly related to baseline cardiovascular measures, and the recommendations of Llabre and colleagues, there is no overwhelming reason to interpret Hypothesis 3 using the residualized change data. The cardiovascular data for simple change overall did not support Hypothesis 3.

In addition to the cardiovascular results, the cortisol results did not support Hypothesis 3. While the Stress and Stress + Support groups showed significant increases in cortisol compared to the No Stress group, the Stress + Support group did not show reduced cortisol reactivity compared to the Stress group. The results from this study contrast with prior work showing decreased cortisol reactivity following support from a supportive other (Heinrichs, Baumgartner, Kirschbaum, & Ehlert, 2003; Kirschbaum,

Klauer, Filipp, & Hellhammer, 1995; Thorsteinsson, James, & Gregg, 1998). However, there are notable differences between the cited studies and the current study, and the comparisons and contrasts may explain why this study diverged from the literature.

The Thorsteinsson study was similar to this study in that a same-sex unfamiliar confederate provided support. However, the Thorsteinsson study used a computer video game as a stressor, rather than a speech task in the presence of an audience. In the Thorsteinsson study, participants in the "no support" condition played the video game with a second computer screen in their line of sight containing video of the confederate silently watching a monitor (the participant was told the confederate was watching their performance). Participants in the "support" condition played the video game, but instead of the confederate on the second screen appearing silent and neutral, the confederate provided supportive commentary throughout the stressor. This setup is akin to having a supportive audience, rather than support prior to a stressful task used in this study. Indeed, most of the studies of social support and cardiovascular reactivity that found positive results included supportive audiences, rather than support prior to the stressor.

The Heinrichs and Kirschbaum studies were similar to this study because the supportive other provided support during the 10-min preparation period prior to the Trier Social Stress Test. Unlike this study, the supportive others in the Kirschbaum study, including the unfamiliar confederate support were of the opposite-sex. In the Heinrichs study, although all participants were male and the supportive other was the participants' best friend, the best friend could be same-sex or opposite-sex. However, the authors did not report the proportion of same- vs. opposite-sex best friends in the study. Even if all the supportive others in the Heinrichs study were male, they were still quite familiar to

participants. Thus, none of the studies in the literature is directly comparable to the current study, and the effect of familiarity and gender could explain some of the positive effects of social support on cortisol reactivity observed in the literature.

One factor that may have played a role in the lack of manipulation effects is the low cortisol reactivity observed for women. Previous work has found similar results using the same stressor (Kirschbaum, Klauer, Filipp, & Hellhammer, 1995; Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999). The difference in reactivity between men and women could not be explained by menstrual phase, as there were no significant differences between women in the self-reported follicular phase vs. luteal phase. It should be noted however that self-reported menstrual phase may not be reliable or valid in all cases. The differences in reactivity could not be explained by self-reported responses to the tasks, as men and women reported similar affective responses and cognitive appraisals. One possibility is the task was perceived as more achievement-oriented with less of an element of social rejection, which may uniquely elevate cortisol responses in women (Stroud, Salovey, & Epel, 2002). However, the TSST is typically considered to elicit threat of social rejection (Dickerson & Kemeny, 2004). Another possibility is that for most of the TSSTs in this study, committees were generally composed of two women, occasionally one man and one woman, and rarely two men. The TSSTs in the Kirschbaum laboratory typically use committees with one man and one woman (Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999). The presence of all-women committees may have reduced perceived social-evaluative threat among female participants.

Thus, compared to the effect size for social support manipulations on cortisol observed in the literature (d = .83), the social support manipulation in this study had no effect in reducing cortisol reactivity (d = .16). As discussed above, the type of stressor, timing and type of support manipulation, source of support (gender and familiarity) may explain the lack of support effect in this study.

The manipulation checks generally suggest that the support manipulation had limited efficacy. While the confederates' personality characteristics were perceived as neutral, and support from the confederates was not effective in reducing self-reported stress. Figure 6 shows participants' ratings of the confederates; in general, participants rated the confederates as more cold, aloof, unassured and unassuming, rather than warm, gregarious, assured, or arrogant. Moreover, higher assured-dominant ratings were actually related to slower skin barrier recovery. Participants' positive ratings of the confederate were more similar to the Neutral tape-recorded comments used in Gallo et al. study (Gallo, Smith, & Kircher, 2000) than the Supportive or Provoking comments.

In addition to rating confederates' personality characteristics, participants also rated the degree to which confederates were perceived as unsupportive. While confederates were rated low on unsupportiveness, this does not imply that confederates were actually perceived as being "supportive."[6] Furthermore, ratings of stress and affect suggested that the support received by participants did not change participants' affect or cognitive appraisals. The Stress + Support group did not show increased positive affect or reduced negative affect and anxiety symptoms relative to the Stress group, and the two

[6] This argument is similar to the notion that low negative affect ratings do not imply high positive affect ratings, or that low positive affect ratings imply high negative affect ratings. As others have argued, interpersonal judgments and attitudes are typically bi-dimensional rather than bipolar (Cacioppo & Berntson, 1994).

groups did not differ on changes in expectations of threat or coping, positive and negative thoughts about the speech, and ratings of stress, performance satisfaction, control, or helplessness after the task.

In fact, the only difference between the Stress and Stress + Support groups was that the Stress group reported feeling more control during the math task compared to the Stress + Support group. Participants in the Stress + Support groups may have perceived interacting with the confederate as part of the stressor. Therefore, participants in the Stress + Support group perceived the math task as occurring during the last 5 min of a 20 min stressor, while participants in the Stress group perceived the math task as occurring during the last 5 min of a 10 min stressor. Perceiving the stressor as longer, combined with the uncontrollability of the math task may explain the reduced feelings of control in the Stress + Support group.

Overall, the support provided by confederates did not influence physiological reactivity or self-reported stress and affect in the hypothesized directions. While participants were "not unsupportive," they were not perceived as "supportive," that is, warm or gregarious to the point where affect and cognitive appraisals were significantly improved. Moreover, having a confederate present made the end of the stressor task seem less controllable.

There are several potential explanations for this pattern of findings. Gender differences in the response to same-sex support are not a likely explanation, as there were no significant gender x group interactions in any of the manipulation check analyses. The literature suggests that women are perceived as more supportive compared to men regardless of participant gender (Glynn, Christenfeld, & Gerin, 1999), but this study

could not test this hypothesis. In general, there were no significant differences in participants' ratings of male vs. female confederates, with the exception of higher ratings on the Arrogant-Calculating scale for the male confederate vs. the female confederates.

The perception of live confederates as less warm and gregarious compared to the tape-recorded confederates in the Gallo study may be explained by the 5 min of silent time intended to duplicate passive support during the social support manipulation. Unlike previous studies, where confederates were out of the participants' sight (Edens et al., 1992; Fontana et al., 1999; Kamarck et al., 1995; Kamarck et al., 1990; Kors et al., 1997), confederates in this study were in full sight of the participants during the silent period. Participants may have perceived the confederate's silence at the beginning of the social support manipulation as aloof and cold, accounting for the lower ratings on the gregarious and warm scales.

Another explanation for the lack of a support effect is participants' perception of the confederate as evaluative. While the participant was explicitly told by the experimenter and confederate that they were not being evaluated, confederates did complete unrelated tasks (e.g., crosswords, other reading) during the passive support, and also completed a questionnaire to track their use of supportive statements during the active support. Participants may have perceived the confederates' activities as evaluative; however, no ratings were obtained from participants about such perceptions. However, if confederates were truly perceived as evaluative, there should be larger increases in cortisol in the Stress + Support group compared to the Stress group, and this was not the case.

A final explanation for the lack of support effects is that most studies showing significant effects of social support on cardiovascular and cortisol reactivity employed support during the stressful task (Thorsteinsson & James, 1999). For instance, a number of studies included a supportive audience member during the speech, or provided supportive comments during the stressful task itself. It may be the case that support prior to a stressful task, in some situations, may actually increase reactivity. For instance, in the Kirschbaum et al. (1995) study, female participants who received support from an opposite-sex partner, and male participants who received support from an opposite-sex confederate, showed elevated cortisol reactivity. In general, the results for support provided during a stressful task are more unequivocal compared to results for support provided prior to such tasks. Thus, the timing of support is probably a key determinant of physiological responses during short-term stressors.

In this study, confederates were the source of support rather than friends or close relationships. I deemed this appropriate in this study to provide proper experimental control of relationship quality and duration, which can affect supportiveness ratings and cardiovascular reactivity (Uno, Uchino, & Smith, 2002). In addition, previous studies found that supportive confederates or audiences were rated as supportive (Anthony & O'Brien, 1999; Hilmert, Kulik, & Christenfeld, 2002; Thorsteinsson, James, & Gregg, 1998), trained confederates showed similar ratings of supportive behaviors compared to supportive friends in direct comparisons (Christenfeld et al., 1997), and support from confederates resulted in reduced physiological reactivity (Thorsteinsson & James, 1999). While support from confederates can influence physiological reactivity, there are negative findings (Anthony & O'Brien, 1999; Sheffield & Carroll, 1996).

As discussed in the *Hypothesis 2* section, the lack of a physiological effect of social support from the confederate in this study does not discount the physiological effects of social relationships in general. Confederates were included in these laboratory studies to provide experimental control over relationship variables that operate in real-world relationships. Future work should consider optimal ways to include these relationship variables in such studies, such as classifying familiar supportive others (e.g., friends) based on participants' ratings (Holt-Lunstad, Uchino, Smith, Olson-Cerny, & Nealey-Moore, 2003; Uno, Uchino, & Smith, 2002). Ultimately, what are truly needed are ecologically valid and generalizable studies of social relationships, physiology, and health as they occur in real-life settings.

Hypothesis 4: Increased cardiovascular and cortisol reactivity to acute stress will be related to delayed skin barrier recovery.

There was no empirical support for Hypothesis 4. Only SBP reactivity was significantly related to skin barrier recover; however, the direction of the relationship depended on the measure. Higher SBP reactivity was related to faster recovery using one measure of healing (percent recovery) and slower recovery based on another measure (temperature- and control-corrected TEWL). No other cardiovascular reactivity measures were related to skin barrier recovery.

The relationship between SBP and skin barrier recovery may be due to changes in skin blood flow. Elevated SBP is generally related to increased cardiac output (Shapiro et al., 1996). While it is unknown whether increased cardiac output translates into increased local skin blood flow, periods of increased skin blood flow correspond with decreased TEWL and faster skin barrier recovery after disruption. Baseline TEWL increases in the

forearm after occluding blood flow using a tourniquet, and then decreases after occlusion is terminated (Rodrigues, Pinto, Magro, Fernandes, & Alves, 2004). Skin blood flow as measured by laser Doppler imaging has a circadian and ultradian (12 h) rhythm, at its highest levels between 2 PM and 8 PM (Yosipovitch, Sackett-Lundeen, et al., 2004). Skin barrier recovery after a standard number of tape strippings was also fastest during the period between 2 PM and 8 PM.

Thus, in addition to the neuroendocrine mechanisms discussed below, change in skin blood flow is a likely mechanism involved in determining skin barrier recovery. However, the effect of increased SBP on skin barrier recovery was similar across groups, and the groups that showed an increase in SBP on average (Stress and Stress + Support groups) demonstrated delayed skin barrier recovery. Therefore, other stress-related mechanisms may have larger effects on skin barrier recovery compared to skin blood flow alone.

Moreover, cortisol reactivity assessed using AUC_I was not related to skin barrier recovery across all the analyses. Instead, elevated baseline levels of cortisol prior to skin barrier recovery were related to slower skin recovery. Glucocorticoid activity is posited as a key mechanism in the stress and wound healing relationship (Denda, Tsuchiya, Elias, & Feingold, 2000). At the same time, other research in humans found that cortisol reactivity was not significantly related to skin barrier recovery (Altemus, Rao, Dhabhar, Ding, & Granstein, 2001).

An additional finding relevant to physiological mechanisms is that women in the relatively later, luteal phase of the menstrual cycle showed faster skin barrier recovery compared to women in the earlier, follicular phase. The relationship between menstrual

phase and healing was only observed in the Stress + Support group, and was not found in the Stress or No Stress group. Few studies have examined relationships between menstrual phase and skin barrier recovery (Shah & Maibach, 2001). One study found increased skin responses as measured by TEWL during day 1 of the menstrual cycle compared to days 9 to 11 in response to skin irritation with sodium lauryl sulfate (Agner, Damm, & Skouby, 1991). Another study compared baseline TEWL on the day of lowest estrogen and progesterone secretion, during menses, with the day of highest estrogen secretion, just before ovulation (Harvell, Hussona-Saccd, & Maibach, 1992). The authors found elevated TEWL on the day of lowest estrogen and progesterone secretion compared to the day of highest estrogen secretion, suggesting poorer skin barrier function just prior to menses compared to prior to ovulation. The data from this study suggest that in the Stress + Support group, skin barrier recovery was worse during menses and the follicular phase compared to the luteal phase, which is consistent with both of the aforementioned findings. Self-reported day of the menstrual cycle was not significantly related to skin barrier recovery in the No Stress and Stress groups, and the mechanism for this is unclear.

Mechanisms of stress-related delays in skin barrier recovery

Most researchers speculate three potential mechanisms that may explain stress-related alterations in skin barrier recovery: Stress-related activation of immune and inflammatory processes in deeper skin layers, neuropeptide release from afferent nerves in the peripheral nervous system, and systemic glucocorticoid levels (Garg et al., 2001). Systemic glucocorticoid levels have received by far the most empirical attention, and in the animal literature, are strongly implicated in stress-related delays in skin barrier

recovery. While the animal literature points to stress-induced increases in glucocorticoids as a key mechanism, human studies that focus on skin barrier recovery rather than other models of wound healing (punch biopsy, blister), including this study, are at this point inconclusive.

Two studies provide evidence for systemic glucocorticoid effects on skin barrier recovery. In mice, skin barrier recovery following disruption decreases in a dose-response manner with increasing doses of intraperitoneally administered corticosterone, with significant decreases in skin barrier recovery at doses above 4.0 μg (Denda, Tsuchiya, Elias, & Feingold, 2000). A similar dose-response relationship was observed after topical administration of corticosterone. Treating skin with 10 mg/ml of topical corticosterone daily resulted in significant delays in skin barrier recovery, but only after treatment for three days. In addition to showing the effects of treatment with corticosterone, administration of the glucocorticoid receptor antagonist RU-486 blocked the effects of systemic glucocorticoid administration, social disruption stress, and immobilization-induced stress in delaying skin barrier recovery.

A later study showed that the effects of glucocorticoids on skin barrier recovery were mediated by reduced epidermal lipid synthesis and decreased integrity of the stratum corneum (Kao et al., 2003). Topical administration of glucocorticoids (0.05% clobetasol, applied daily for 3 d) in humans resulted in delays in skin barrier recovery (slightly less than 50% recovery after 24 h) compared to topical administration of vehicle (about 62% recovery after 24 h), with similar effects observed in mice. In the mouse studies, high topical doses of clobetasol delayed skin barrier recovery on the treated flank and on the contralateral (opposite) flank treated with vehicle only, suggesting that high

topical doses of glucocorticoids increased systemic levels to the point where untreated skin also showed delayed recovery.

Kao and colleagues also tested the mechanisms of glucocorticoid-related delays in skin barrier recovery. Topical glucocorticoids decreased the ability of the stratum corneum to withstand tape-stripping. In addition, topical glucocorticoid administration decreased lamellar body production and secretion in general and after barrier disruption due to decreased epidermal lipid synthesis. Systemic administration of glucocorticoids (dexamethasone, 450 μg/kg) decreased epidermal lipid synthesis in a manner comparable to topical administration. Glucocorticoid-related delays in skin barrier recovery were partially reversed by treatment with topical lipids, suggesting that inhibited lipid synthesis is a key mechanism explaining glucocorticoid effects. Beyond lipid synthesis, topical administration of glucocorticoids reduced the density of corneodesmosomes, cells derived from keratinocytes that are involved in the shedding of skin cells from the stratum corneum. Thus, the two key mechanisms underlying glucocorticoid effects on skin barrier recovery are reduced lipid synthesis and corneodesmosome density.

Two other candidate mechanisms for explaining stress and skin barrier recovery relationships are stress-related activation of immune and inflammatory processes in deeper skin layers and neuropeptide release from afferent nerves in the peripheral nervous system. Several studies have established that acute stressors, such as the task in this study, increase plasma levels of proinflammatory cytokines, including IL-6, IL-1ra (a proxy for IL-1), and TNF-α (Kunz-Ebrecht, Mohamed-Ali, Feldman, Kirschbaum, & Steptoe, 2003; Steptoe, Willemsen, Owen, Flower, & Mohamed-Ali, 2001). While proinflammatory cytokines play key roles in skin barrier recovery, including coordinating

inflammation and immune responses and possibly promoting epidermal lipid synthesis (Paquet & Pierard, 1996; Tsai et al., 1994; Ye et al., 2002), it is unclear whether elevated proinflammatory cytokines in plasma correspond to similar effects in the epidermis. Indeed, in the Altemus study, elevated proinflammatory cytokine levels in plasma, notably IL-1β and TNF- α were related to delayed skin barrier recovery, whereas elevated proinflammatory cytokines in the epidermis, at least in the initial phases of wound healing, promote skin barrier recovery (Elias, 2005; Nickoloff & Naidu, 1994). Even less is known about the role of stress in interactions between neuropeptides and epidermal cells or other hormones and epidermal cells. While numerous receptors for various neuropeptide and hormone ligands have been identified, little is known about whether stress influences specific ligand-receptor interactions in a manner that results in delayed skin barrier recovery (Slominski & Wortsman, 2000).

Future directions

Despite the elegant research in mice models, no human research on skin barrier recovery to date has established stress-induced increases in glucocorticoids as a mechanism. This study found no association between cortisol reactivity and skin barrier recovery, and the study by Altemus and colleagues found a negative, but non-significant relationship between cortisol levels during stress and subsequent skin barrier recovery (Altemus, Rao, Dhabhar, Ding, & Granstein, 2001). However, baseline levels of cortisol in this study were related to slower healing as indexed by temperature- and temperature- and control-corrected TEWL. By contrast, other models of wound healing, including punch biopsy (Ebrecht et al., 2004) and blister wounds (Glaser et al., 1999) show good evidence for glucocorticoids delaying wound healing. If the skin barrier recovery

research in mice clearly shows glucocorticoid inhibition of skin barrier recovery, and human studies using wound healing models other than skin barrier recovery provide similar evidence, why has the same effect not been observed in studies of skin barrier recovery in humans undergoing acute stress?

The discrepancy between the positive findings (animal studies, human studies involving punch biopsy and blister wound models) and the negative findings (acute stress studies of skin barrier recovery in humans) can be explained by several key differences between the studies: stressor duration and timing, glucocorticoid dosage and potency, and modes of administration. The studies of stress and skin barrier recovery in mice used stressors of prolonged duration that could qualify as chronic stressors. For instance, the stressors in the Denda studies lasted for two weeks (Denda, Tsuchiya, Elias, & Feingold, 2000; Denda, Tsuchiya, Hosoi, & Koyama, 1998). The human wound healing studies also involved stressors of longer duration, including inpatient admission at the GCRC for 24 h (Glaser et al., 1999), and more tonic conditions assessed by perceived stress measures (Ebrecht et al., 2004). The Garg et al. 2001 study involved academic examination stress, which lasts days to weeks. By contrast, the stressors in this study and the Altemus study lasted for 20-30 minutes.

The duration of the stressors is related to timing. The mice studies conducted tape-stripping after the stressors, during a period when stress-related activation of the HPA axis had already occurred or was already occurring. A similar argument could be made for the Garg et al. 2001 study of medical students. By contrast, the Altemus study and this study conducted tape-stripping before stress-related activation of the HPA axis. Thus, physiological levels of glucocorticoids prior to skin barrier disruption may have

more of an effect on skin barrier recovery than physiological levels of glucocorticoids after skin barrier disruption occurs. Indeed, in this study baseline cortisol levels prior to tape-stripping were related to slower skin barrier recovery when assessed by corrected TEWL. Therefore, the relationship between glucocortiocoid levels and skin barrier recovery may not be a matter of when glucocorticoids are measured relative to skin barrier disruption, but whether an ongoing stressor is occurring that elevates glucocorticoids prior to skin barrier disruption. Another possibility is that glucorticoid levels several hours after disruption may be related to delayed skin barrier recovery and wound healing. Using a punch biopsy model of wound healing, Ebrecht and colleagues found that larger cortisol awakening responses (measured within the first hour after waking) 1 day after receiving a punch biopsy wound were related to slower wound healing between 7 and 21 days after wounding (Ebrecht et al., 2004). By contrast, cortisol awakening responses 14 days before and after wounding were not related to wound healing.

In the skin barrier recovery studies, the dosage of glucocorticoids during stress in mice and during exogenous administrations in mice and humans was over a longer duration (e.g., daily application for 3 days in Kao et al. 2003), and in pharmacological rather than physiological doses. In general, the glucocorticoids used in these studies are also much more potent compared to endogenous glucocorticoids. A similar issue occurs with modes of administration – topical corticosteroids, by virtue of pharmacological dosage and increased proximity to target tissues, are much more potent compared to endogenous corticosteroids produced physiologically. A key question that would clarify this literature is determining the relationship between stress-induced increases in

glucocorticoids observed systemically (through saliva or plasma) and glucocorticoid levels in the epidermis. That is, if an individual has .25 pg/ml cortisol present in saliva, what is the concurrent level of cortisol present in epidermal tissues?

Similar questions could be applied to understanding the role of stress-induced increases in other hormones related to stress and reproductive hormones, for which cells in the epidermis have identified receptors. This list is numerous and covers most well-characterized neuroendocrine signals (Slominski & Wortsman, 2000). Similarly, future work could also examine the relationship between systemic increases in proinflammatory cytokines during acute or chronic stress and proinflammatory cytokine actions in the skin during skin barrier recovery.

Overall, studies showing that glucocorticoids mediate stress-related delays in skin barrier recovery used stressors of long duration that occurred before skin barrier disruption, and often high pharmacological doses of glucocorticoids. By contrast, this study and the Altemus study did not observe cortisol reactivity as a mediator of skin barrier recovery, perhaps because the stressors were of short duration, occurred after skin barrier disruption, and involved physiological doses. Instead, baseline cortisol was related to skin barrier recovery. Thus, there is clearly more work needed to identify the biological mediators of stress-related delays in skin barrier recovery in humans, their kinetics, and how these mediators operate in the epidermis.

Hypothesis 5: Stress-induced cortisol responses will show distinct and reliable patterns of reactivity from and recovery to baseline.

The planned analyses for Hypothesis 5 involving piecewise latent growth curve modeling were not successful. In particular, recovery functions could not be adequately

modeled. Multilevel modeling, while less suited for testing Hypothesis 5, but better able to handle smaller sample sizes and variable timing of samples, suggested that reactivity represented by linear slopes, and recovery represented by quadratic slopes were distinct in one sense, and indistinct in another. On one hand, linear and quadratic slopes were reliable and could be modeled separately and distinctly with sufficient variability. However, linear and quadratic slopes were almost perfectly correlated, suggesting that these components of change were not distinct.

Several factors contributed to problems with modeling recovery using latent growth curve models. The sample size was much smaller than other samples used in modeling physiological reactivity and recovery. Data from 56 participants was available in this study, compared to 99 in Llabre et al. 2001, and 167 in a later study (Llabre, Spitzer, Siegel, Saab, & Schneiderman, 2004). Moreover, the variable timing of cortisol samples in this study likely increased measurement error, while psychophysiological monitoring in the Llabre studies allowed for sampling at the exact same times across all participants. In addition, half of the sample, specifically female participants, showed lower variability in change over time. Finally, Figure 22 shows that some individuals showed cortisol changes that were not consistent with the general pattern of an increase from Time 1 to Time 3, followed by a decrease from Time 3 to Time 7. Some participants showed increases across that persisted throughout the session, while some showed even more variable cortisol responses.

A strong inverse correlation between the linear and quadratic components suggests that reactivity and recovery are not distinct. However, Llabre et al. (2001) observed similar high inverse correlations between reactivity and recovery components in

their piecewise latent growth curve models. They noted that different functions (linear vs. quadratic) were required to model reactivity and recovery similar to this study. Indeed, the raw data plots from this study clearly suggest that cortisol declines over time for many participants after the initial reactivity period, and a model with only linear slopes would not adequately model this pattern of change.

Beyond the need for different functions to model reactivity and recovery components, Llabre and colleagues note that reactivity and recovery also differed in their relationship to other variables. For instance, reactivity was predicted by appraisal of the stress task in their study, and recovery predicted systolic blood pressure in the work place. In this study, there was no evidence that group status had different influences on linear or quadratic cortisol slopes. Moreover, given that cortisol responses were not significantly related to skin barrier recovery, it is likely that the relationships between linear cortisol slopes and skin barrier recovery and quadratic cortisol slopes and skin barrier recovery would not be significant.

Thus, several limitations of this study in testing Hypothesis 5 point to directions for future research. Beyond increasing sample size, a key measurement issue for future research is more frequent sampling and ensuring low variability in sample timing across participants. While low variability in sample timing is feasible for cortisol measurements, increased sampling frequency is much more costly for biological measures like cortisol compared to the electrophysiology measures used by Llabre and colleagues. Finally, it may be the case that reactivity and recovery will always be highly correlated. Thus, the key to establishing their distinctiveness may be whether reactivity and recovery differ in their predictors and outcomes. For instance, Llabre and colleagues point out that different

types of stressors may have different effects on reactivity and recovery. Studying physiological reactivity and recovery responses to stress in larger samples and tracking subsequent health outcomes over time could allow for determining whether reactivity and recovery differ in their relationships to health outcomes.

Limitations of the current study

The limitations of this study are worth noting as they provide areas of improvement for future research in studies of acute stress, social support, and skin barrier recovery. The limitations of the social support manipulation were already discussed in *Hypothesis 4*. The remaining limitations are in the areas of design and internal validity, measurement, statistical analyses and power, and generalizability of physiological responses to stress.

One of the design strengths of the current project is the between-subjects design, which offered logistical advantages such as reducing the number of visits per participant to one, rather than two or more in previous studies. At the same time, the design made it difficult to account for sources of between-subject variability, especially for TEWL measures. For instance, there are individual differences in skin barrier function, not just in terms of baseline TEWL, but also the integrity of the stratum corneum, translating into the number of strips needed to reach a specified criterion (e.g., 20 g/m^2h). Not only does stratum corneum integrity affect the number of strips to criterion, but also the length of time it takes to tape-strip participants. Indeed, one of the reasons for variable cortisol and TEWL measurement times across participants was because in some cases it took 20 min to tape-strip a participant, and in others it took 40 min.

Furthermore, individual differences probably exist in lipid content, rates of lipid synthesis, and cytokine expression following disruption. On top of these individual differences, TEWL measures are sensitive to fluctuating temperature and humidity conditions, which varied over the course of data collection in a seasonal manner. Therefore, a within-subjects design may be more ideal in reducing sources of between-subject variability in future skin barrier recovery studies that involve comparing a stress to a no-stress condition, or a stress to a stress + support condition.

In addition to the between-subjects design, this study also differed from previous stress and skin barrier recovery studies in humans by measuring skin barrier recovery earlier than 3 h after tape-stripping. While stress-related differences in recovery were observed in this study, the effect of stress in several analyses only approached significance, partially due to the lack of slope variability within the Stress and Stress + Support groups. Measuring recovery after 3 h and perhaps up to 24 h afterwards as was done in the Garg et al. 2001 study may provide additional variability that could aid in detecting stress-related effects. Moreover, as discussed in the *Clinical implications* section, delays in skin barrier recovery over the course of 3 h or more may result in increased exposure to pathogens. Thus, it may be important to assess the duration of stress-related delays in skin barrier recovery over longer periods of time in future work.

While this study included greater numbers compared to previous stress and skin barrier recovery studies, particularly the number of participants undergoing stress, the analyses comparing the Stress and Stress + Support groups was probably underpowered. Despite this shortcoming, the use of multilevel models increased the available sample size for key analyses and accommodated the between-subject variability in sample

timing, which hampered the latent growth curve models. Thus, future work would benefit from larger sample sizes and more consistent timing of biological samples across subjects in order to maximize the possible data analytic approaches and most importantly statistical power.

Finally, a key issue for all studies using acute laboratory stressors is lab-to-life generalizability; do physiological responses in the laboratory mirror responses in real-life situations outside the laboratory? For cardiovascular reactivity, lab-to-life generalizability studies are "characterized by inconsistent findings" (Kamarck and Lovallo, 2003). However, the authors noted that a number of methodological problems were not addressed in the prevailing literature. For instance, Kamarck and Lovallo noted that most studies assessed relationships between laboratory reactivity (e.g., change during the task) and average ambulatory heart rate and blood pressure over the course of the day. The limitation to this approach is that averaging cardiovascular responses over the entire day masks potential relationships between laboratory reactivity and specific types of stressors during the day. The authors suggested using an approach taken by Kamarck and colleagues, in which the authors examined the relationship between laboratory reactivity and ambulatory cardiovascular measures during specific types of activities, notably activities rated along dimensions of demand and controllability (Kamarck, Schwartz, Janicki, Shiffman, & Raynor, 2003). The authors found that greater blood pressure reactivity in lab was related to higher ambulatory blood pressure during specific daily activities rated as highly demanding or uncontrollable. Thus, there is some evidence suggesting that laboratory cardiovascular reactivity generalizes to specific types of stressors.

While some evidence exists regarding the generalizability of cardiovascular reactivity, the same cannot be said of cortisol reactivity (Dickerson & Kemeny, 2004). To date, no published studies have examined whether cortisol responses to laboratory stressors are related to cortisol responses in daily life experience. While a number of studies have examined either, none has examined both in relation to each other. Thus, the generalizability of cortisol responses in the laboratory remains an open question for future work.

Clinical implications

While delays in skin barrier recovery have the potential to be clinically important for most persons, the clinical implications of stress-related delays in skin barrier recovery are particularly important for "vulnerable" populations. Vulnerable primarily refers to populations for whom delayed skin barrier recovery may exacerbate existing disease or increase risk for additional health problems, including individuals with specific types of skin diseases and older adults.

Vulnerable populations primarily include individuals with diseases of the skin barrier, such as psoriasis, contact dermatitis, and atopic dermatitis; notably, abnormal T cell responses are implicated in these diseases. Psoriasis is a chronic inflammatory skin disorder characterized by exaggerated proliferation of keratinocytes, resulting in itchy, scaly, and inflamed skin, with a prevalence of about 2% in most Western countries (Gelfand et al., 2005). The various dermatitis conditions involve rash and itching in response to stimuli like chemical exposure or allergens.

For example, stress-related delays in skin barrier recovery may play a role in the "Koebner phenomenon," in which psoriatic patients show psoriatic lesions in uninvolved

skin following skin trauma (Weiss, Shemer, & Trau, 2002). Such skin trauma includes lacerations, pressure, shaving, and tape-stripping. Delayed skin barrier recovery may increase the likelihood that a psoriatic lesion forms at the site of skin trauma by prolonging inflammatory processes that result in abnormal T-cell responses and exaggerated proliferation of keratinocytes. Exposure to acute or chronic stress may further exacerbate delays in skin barrier recovery following skin trauma, further prolonging the inflammatory processes that result in exaggerated keratinocyte proliferation and psoriasis symptoms.

Another example of the clinical implications of delayed skin barrier recovery comes from recent developments in understanding allergic contact dermatitis, notably allergies to peanuts and other food proteins in mice models (Strid, Hourihane, Kimber, Callard, & Strobel, 2004). Researchers in this area make a distinction between epicutaneous exposure to antigen, which involves exposing the skin to antigen following removal of the stratum corneum through tape-stripping, versus subcutaneous exposure to antigen, which involves injection of antigen into the dermal layer. In these studies epicutaneous exposure occurred 24 h after skin barrier disruption, which is notable because Garg and colleagues found that examination stress delayed skin barrier recovery up to 24 h after disruption (Garg et al., 2001).

Epicutaneous exposure to ovalbumin or peanut protein resulted in markedly different immune responses compared to exposure through intact skin (Strid, Hourihane, Kimber, Callard, & Strobel, 2004). Specifically, epicutaenous exposure resulted in increased levels of immunoglobulin (Ig) G and IgE antibodies and elevated delayed-type hypersensitivity (DTH) responses up to 20 days after exposure compared to exposure

through intact skin. Moreover, IgG, IgE, and DTH responses were lower following subcutaneous injection compared to epicutaneous exposure to these purified proteins. In general, epicutaneous exposure resulted in a Th2-type immune response to protein antigens, while subcutaneous exposure resulted in a Th1-type response. Th2-type responses are the key immune responses associated with developing allergic reactions (Ngoc, Gold, Tzianabos, Weiss, & Celedon, 2005).

Thus, delays in skin barrier recovery may increase the likelihood of epicutaneous exposure to protein antigens. Increased likelihood of epicutaneous exposure may further increase the likelihood of developing Th2-type responses and the development or maintenance of allergic contact dermatitis. Moreover, in individuals with existing allergic contact dermatitis, disruption of the skin barrier may be associated with enhanced responses to allergens after exposure.

Beyond the clinical consequences for individuals with skin diseases, delayed skin barrier recovery more generally disrupts the protective function of the skin. Several lipids in the stratum corneum serve antimicrobial functions (Elias, 2005). Thus, delays in skin barrier recovery increase the likelihood of exposure to infectious pathogens in two ways: allowing in more pathogens by weakening the physical barrier, and decreasing antimicrobial killing through reduced lipid synthesis. Moreover, recent animal work suggests that social disruption and restraint stress induces translocation of bacteria present on the skin surface to lymph nodes in mice (Bailey, Engler, & Sheridan, 2006). Thus, a combination of prolonged stress such as social disruption results in both decreased skin barrier function, delays in skin barrier recovery following damage, and increased invasion of bacteria present on the skin into the body. Taken together, the

combination of stress and skin barrier damage may result in increased likelihood of infection.

The consequences of stress-related delays in skin barrier recovery are also important for older adults. The skin barrier is more susceptible to disruption through tape-stripping in older adults, with only 18 ± 2 tape-strippings required to reach 20 g/m^2h TEWL in adults older than 80 yr, vs. 31 ± 5 in adults younger than 30 (Ghadially, Brown, Sequeira-Martin, Feingold, & Elias, 1995). Skin barrier recovery is also delayed in older adults, with 15% recovery by 24 h after acetone disruption compared to 50% recovery in younger adults (Ghadially, Brown, Sequeira-Martin, Feingold, & Elias, 1995). Coupled with an aging and less effective immune system, age-related increases in inflammation, and increased likelihood of injury and subsequent exposure to infection, further stress-related impairment of skin barrier recovery presents another challenge to older adults, particularly chronically stressed older adults.

Acute laboratory and academic exam stress delay skin barrier recovery, a clinically relevant health outcome. While the human work suggests that these short-term stressors are sufficient to delay skin barrier recovery, the animal research indicates that long-term, chronic stressors also delay skin barrier recovery. Thus, a key future direction for this work is examining skin barrier recovery in chronically stressed populations. Chronic stress from caregiving is related to slower wound healing (Kiecolt-Glaser, Marucha, Malarkey, Mercado, & Glaser, 1995), and enduring negative aspects of marriage are also related to slower wound healing (Kiecolt-Glaser et al., 2005).

The health consequences of stress-related delays in skin barrier recovery are of particular concern for vulnerable populations, including individuals with skin diseases

and older adults. For example, retrospective and prospective studies of stress and psoriatic flares suggest that stressful life events are related to increased severity of psoriatic symptoms (Al'Abadie, Kent, & Gawkrodger, 1994; Gaston, Lassonde, Bernier-Buzzanga, Hodgins, & Crombez, 1987). Psychosocial factors, including stress, may also disrupt dermatological treatments. In a sample of 122 psoriasis patients receiving photochemotherapy treatment, individuals reporting high levels of chronic worry took 1.8 times longer to clear psoriatic lesions compared to individuals reporting low levels of chronic worry (Fortune et al., 2003). Thus, future work should explore delayed skin barrier recovery as a potential mechanism linking psychosocial factors and/or stressful events to symptom exacerbation.

Moreover, skin barrier recovery could be easily included as an outcome variable in treatment-outcome studies of stress management interventions, providing key experimental data on the role of stress and skin barrier recovery. For example, a mindfulness meditation-based stress reduction intervention increased rates of skin clearing in patients with psoriasis who were undergoing phototherapy and photochemotherapy treatments (Kabat-Zinn et al., 1998). Moreover, one study of patients at a dermatology clinic found that 40% could be classified with a psychiatric disorder (Wessely & Lewis, 1989). Thus, a further direction for this work is whether treatment for psychiatric disorders in dermatological patients reduces skin disease symptoms in affected patients, and whether skin barrier recovery is influenced by effective psychosocial or pharmacological treatment.

Conclusions

The over-arching goal of this project was to address the question of how social relationships get "under our skin" and affect health. My study addressed this question by uniting two lines of research that are typically conducted separately: observational studies evaluating the effects of social support on clinically relevant health outcomes, such as coronary artery calcification; and laboratory experiments examining the physiological effects of social support. I combined these two lines of research by measuring both physiological responses to stress and social support, and more importantly, the health outcome of skin barrier recovery.

While there was no effect of social support on skin barrier recovery or physiological reactivity, this study provides important insights into future work on stress, social support, and skin barrier recovery. This study replicated the effects of short-term laboratory stressors on skin barrier recovery, further establishing the relevance of skin barrier recovery for future research on stress and restorative processes like wound healing. In addition, this study showed that individual differences in anxious arousal and positive affect, and state anxiety symptoms may have important influences on stress-related changes in skin barrier recovery. The limited effectiveness of the social support manipulations in influencing wound healing relative to real-world and longer-term supports (stable social conditions in rodents, marital support in humans) and stressors (social disruption in rodents, academic exams and marital discord in humans) suggests that future work would benefit from a focus on real-world conditions. Real-world sources of support could include actual friends and/or close relationships, and real-world stressors could include enduring life stressors and potentially psychopathology. Finally, the lack of

relationship between cortisol reactivity and skin barrier recovery, now observed in two independent studies, suggests that future systematic work is needed to firmly establish relationships between physiological levels of glucocorticoids and skin barrier recovery in humans. Thus, there is fertile ground in studying psychosocial influences on skin barrier recovery and wound healing, and focusing on social relationships and physiology will likely become a worthwhile endeavor.

BIBLIOGRAPHY

Adler, N. E., Epel, E. S., Castellazzo, G., & Ickovics, J. R. (2000). Relationship of subjective and objective social status with psychological and physiological functioning: Preliminary data in healthy white women. *Health Psychology, 19*, 586-592.

Agner, T., Damm, P., & Skouby, S. O. (1991). Menstrual cycle and skin reactivity. *Journal of the American Academy of Dermatology, 24*, 566-570.

Al'Abadie, M. S., Kent, G. G., & Gawkrodger, D. J. (1994). The relationship between stress and the onset and exacerbation of psoriasis and other skin conditions. *British Journal of Dermatology, 130*, 199-203.

Allen, K., Blascovich, J., & Mendes, W. B. (2002). Cardiovascular reactivity and the presence of pets, friends, and spouses: The truth about cats and dogs. *Psychosomatic Medicine, 64*, 727-739.

Allen, K. M., Blascovich, J., Tomaka, J., & Kelsey, R. M. (1991). Presence of human friends and pet dogs as moderators of autonomic responses to stress in women. *Journal of Personality and Social Psychology, 61*, 582-589.

Altemus, M., Rao, B., Dhabhar, F. S., Ding, W., & Granstein, R. D. (2001). Stress-induced changes in skin barrier function in healthy women. *Journal of Investigative Dermatology, 117*, 309-317.

Anthony, J. L., & O'Brien, W. H. (1999). An evaluation of the impact of social support manipulations on cardiovascular reactivity to laboratory stressors. *Behavioral Medicine, 25*, 78-88.

Babyak, M. A. (2005). *Some key developments in data analysis.* Paper presented at the annual meeting of the American Psychosomatic Society, Vancouver, BC, Canada.

Baecke, J. A. H., Burema, J., & Frijters, J. E. R. (1982). A short questionnaire for the measurement of habitual physical activity in epidemiological studies. *American Journal of Clinical Nutrition, 36*, 936-942.

Bailey, M. T., Engler, H., & Sheridan, J. F. (2006). Stress induces the translocation of cutaneous and gastrointestinal microflora to secondary lymphoid organs of C57BL/6 mice. *Journal of Neuroimmunology, 171*, 29-37.

Bashir, S. J., Chew, A., Anigbogu, A., Dreher, F., & Maibach, H. I. (2001). Physical and physiological effects of stratum corneum tape stripping. *Skin Research and Technology, 7*, 40-48.

Bauer, F. W., Boezeman, J. B., Rijzewijk, J. J., & de Grood, R. M. (1989). Topical corticosteroids delay the proliferative response to sellotape stripping. *Skin Pharmacology and Applied Skin Physiology, 2*, 204-209.

Benschop, R. J., Geenen, R., Mills, P. J., Naliboff, B. D., Kiecolt-Glaser, J. K., Herbert, T. B., et al. (1998). Cardiovascular and immune responses to acute psychological stress in young and older women: A meta-analysis. *Psychosomatic Medicine, 60*, 290-296.

Berkman, L. F., & Breslow, L. (1983). *Health and ways of living: The Alameda county study*. New York: Oxford University Press.

Breier, A., Albus, M., Pickar, D., Zahn, T., Owen, M., & Paul, S. (1987). Controllable and uncontrollable stress in humans: Alterations in mood and neuroendocrine and psychophysiological function. *American Journal of Psychiatry, 144*, 1419-1425.

Brookings, J. B., & Bolton, B. (1988). Confirmatory factor analysis of the Interpersonal Support Evaluation List. *American Journal of Community Psychology, 16*, 137-147.

Buysse, D. J., Reynolds III, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiatry Research, 28*, 193-213.

Cacioppo, J. T., & Berntson, G. G. (1994). Relationship between attitudes and evaluative space: A critical review, with emphasis on the separability of positive and negative substrates. *Psychological Bulletin, 115*(3), 401-423.

Cacioppo, J. T., Berntson, G. G., Malarkey, W. B., Kiecolt-Glaser, J. K., Sheridan, J. F., Poehlmann, K. M., et al. (1998). Autonomic, neuroendocrine, and immune responses to psychological stress: The reactivity hypothesis. *Annals of the New York Academy of Sciences, 840*, 664-673.

Cacioppo, J. T., Berntson, G. G., Sheridan, J. F., & McClintock, M. K. (2000). Multilevel integrative analyses of human behavior: Social neuroscience and the complementing nature of social and biological approaches. *Psychological Bulletin, 126*, 829-843.

Cacioppo, J. T., & Petty, R. E. (1981). Social psychological procedures for cognitive response assessment: The thought-listing technique. In T. V. Merluzzi, C. R.

Glass & M. Genest (Eds.), *Cognitive Assessment* (pp. 309-342). New York: Guilford Press.

Cacioppo, J. T., von Hippel, W., & Ernst, J. M. (1997). Mapping cognitive structures and processes through verbal content: The thought-listing technique. *Journal of Consulting and Clinical Psychology, 65*(6), 928-940.

Champion, R. H., Rook, A., Wilkinson, D. S., & Ebling, F. J. G. (Eds.). (1998). *Rook/Wilkinson/Ebling textbook of dermatology* (6th ed. Vol. 1). Oxford, England: Blackwell Science.

Choi, E. H., Brown, B. E., Crumrine, D., Chang, S., Man, M. Q., Elias, P. M., et al. (2005). Mechanisms by which psychologic stress alters cutaneous permeability barrier homeostasis and stratum corneum integrity. *Journal of Investigative Dermatology, 124*, 587-595.

Christenfeld, N., Gerin, W., Linden, W., Sanders, M., Mathur, J., Deich, J. D., et al. (1997). Social support effects on cardiovascular reactivity: Is a stranger as effective as a friend? *Psychosomatic Medicine, 59*, 388-398.

Christenfeld, N., Glynn, L. M., & Gerin, W. (2000). On the reliable assessment of cardiovascular recovery: An application of curve-fitting techniques. *Psychophysiology, 37*, 543-550.

Cohen, S., Kamarck, T., & Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior, 24*, 385-396.

Cohen, S., Mermelstein, R., Kamarck, T., & Hoberman, H. M. (1985). Measuring the functional components of social support. In I. G. Sarason & B. R. Sarason (Eds.), *Social support: Theory, research and applications* (pp. 73-94). Dordrecht, Netherlands: Martinus Nijhoff.

Cohen, S., Underwood, L., & Gottlieb, B. H. (2000). Social relationships and health. In S. Cohen, L. Underwood & B. H. Gottlieb (Eds.), *Social support measurement and intervention* (pp. 3-25). New York: Oxford University Press.

Cook, W. W., & Medley, D. M. (1954). Proposed hostility and pharisaic-virtue scales for the MMPI. *Journal of Applied Psychology, 38*, 414-418.

Crawford, J. R., & Henry, J. D. (2004). The Positive and Negative Affect Schedule (PANAS): Construct validity, measurement properties and normative data in a large non-clinical sample. *British Journal of Clinical Psychology, 43*, 245-265.

Deffenbacher, J. L., Oetting, E. R., Lynch, R. S., & Morris, C. D. (1996). The expression of anger and its consequences. *Behaviour Research and Therapy, 34*, 575-590.

Denda, M., & Tsuchiya, T. (2000). Barrier recovery rate varies time-dependently in human skin. *British Journal of Dermatology, 142*, 881-884.

Denda, M., Tsuchiya, T., Elias, P. M., & Feingold, K. (2000). Stress alters cutaneous permeability barrier homeostasis. *American Journal of Physiology: Regulatory, Integrative, and Comparative Physiology, 278*, R367-R372.

Denda, M., Tsuchiya, T., Hosoi, J., & Koyama, J. (1998). Immobilization-induced and crowded environment-induced stress delay barrier recovery in murine skin. *British Journal of Dermatology, 138*, 780-785.

Detillion, C. E., Craft, T. K., Glasper, E. R., Prendergast, B. J., & DeVries, A. C. (2004). Social facilitation of wound healing. *Psychoneuroendocrinology, 29*, 1004-1011.

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and meta-analytic review. *Psychological Bulletin, 130*, 355-391.

Dohrenwend, B. S., Krasnofff, L., Askenasy, A. R., & Dohrenwend, B. P. (1978). Exemplification of a method for scaling life events: The PERI Life Events Scale. *Journal of health and Social Behavior, 19*, 205-229.

Earle, T. L., Linden, W., & Weinberg, J. (1999). Differential effects of harassment on cardiovascular and salivary cortisol stress reactivity and recovery in women and men. *Journal of Psychosomatic Research, 46*, 125-141.

Ebrecht, M., Hextall, J., Kirtley, L., Taylor, A., Dyson, M., & Weinman, J. (2004). Perceived stress and cortisol levels predict speed of wound healing in healthy male adults. *Psychoneuroendocrinology, 29*, 798-809.

Edens, J., Larkin, K., & Abel, J. (1992). The effect of social support and physical touch on cardiovascular reactions to mental stress. *Journal of Psychosomatic Research, 36*, 371-382.

Elias, P. M. (2005). Stratum corneum defensive functions: An integrated view. *Journal of Investigative Dermatology, 125*, 183-200.

Elias, P. M., Wood, L. C., & Feingold, K. R. (1997). Relationship of the epidermal permeability barrier to irritant contact dermatitis. *Contact Dermatitis, 17*, 417-430.

Erdfelder, E., Faul, F., & Buchner, A. (1996). GPOWER: A general power analysis program. *Behavior Research Methods, Instruments, and Computers, 28*, 1-11.

Feigelson, M. E., & Dwight, S. A. (2000). Can asking questions by computer improve the candidness of responding? A meta-analytic perspective. *Consulting Psychology Journal: Practice and Research, 52*, 248-255.

Fenske, N. A., & Lober, C. W. (1986). Structural and functional changes of normal aging skin. *Journal of the American Academy of Dermatology, 15*, 571-585.

Fillenbaum, G. G., & Smyer, M. A. (1981). The development, validity, and reliability of the OARS Multidimensional Functional Assessment Questionnaire. *Journal of Gerontology, 36*(4), 428-434.

Fluhr, J. W., Dickel, H., Kuss, O., Weyher, I., Diepgen, T. L., & Berardesca, E. (2002). Impact of anatomical location on barrier recovery, surface pH and stratum corneum hydration after acute barrier disruption. *British Journal of Dermatology, 146*, 770-776.

Fluhr, J. W., Gloor, M., Lehmann, L., Lazzerini, S., Distante, F., & Berardesca, E. (1999). Glycerol accelerates recovery of barrier function in vivo. *Acta Dermato-Venereologica, 79*, 418-421.

Fontana, A. M., Diegnan, T., Villeneuve, A., & Lepore, S. J. (1999). Nonevaluative social support reduces cardiovascular reactivity in young women during acutely stressful performance situations. *Journal of Behavioral Medicine, 22*, 75-91.

Fortune, D. G., Richards, H. L., Kirby, B., McElhone, K., Markham, T., Rogers, S., et al. (2003). Psychological distress impairs clearance of psoriasis in patients treated with photochemotherapy. *Archives of Dermatology, 139*, 752-756.

Frankenhaeuser, M. (1986). A psychobiological framework for research on human stress and coping. In M. H. Appley & R. Trumbull (Eds.), *Dynamics of stress: Physiological, psychological, and social perspectives* (pp. 101-116). New York: Plenum.

Fullerton, A., & Serup, J. (1995). Transepidermal water loss measurements in patch test assessment: The need for standardisation. *Current Problems in Dermatology, 23*, 152-158.

Gallo, L. C., Smith, T. W., & Kircher, J. C. (2000). Cardiovascular and electrodermal responses to support and provocation: Interpersonal methods in the study of psychophysiological reactivity. *Psychophysiology, 37*, 289-301.

Garg, A., Chren, M., Sands, L. P., Matsui, M. S., Marenus, K. D., Feingold, K. R., et al. (2001). Psychological stress perturbs epidermal permeability barrier homeostasis. *Archives of Dermatology, 137*, 53-59.

Gaston, L., Lassonde, M., Bernier-Buzzanga, J., Hodgins, S., & Crombez, J. C. (1987). Psoriasis and stress: A prospective study. *Journal of the American Academy of Dermatology, 17*, 82-86.

Gelfand, J. M., Weinstein, R., Porter, S. B., Neimann, A. L., Berlin, J. A., & Margolis, D. J. (2005). Prevalence and treatment of psoriasis in the United Kingdom: A population-based study. *Archives of dermatology, 141*, 1537-1541.

Gerin, W., Milner, D., Chawla, S., & Pickering, T. G. (1995). Social support as a moderator of cardiovascular reactivity in women: A test of the direct effects and buffering hypotheses. *Psychosomatic Medicine, 57*, 16-22.

Gerin, W., Pieper, C., Levy, R., & Pickering, T. G. (1992). Social support in social interaction: A moderator of cardiovascular reactivity. *Psychosomatic Medicine, 54*, 324-336.

Ghadially, R. (1998). Aging and the epidermal permeability barrier: implications for contact dermatitis. *American Journal of Contact Dermatitis, 9*, 162-169.

Ghadially, R., Brown, B. E., Sequeira-Martin, S. M., Feingold, K. R., & Elias, P. M. (1995). The aged epidermal permeability barrier: Structural, functional, and lipid biochemical abnormalities in humans and a senescent murine model. *Journal of Clinical Investigation, 95*, 2281-2290.

Girdler, S. S., Pedersen, C. A., Stern, R. A., & Light, K. C. (1993). Menstrual cycle and premenstrual syndrome: Modifiers of cardiovascular reactivity in women. *Health Psychology, 12*, 180-192.

Glaser, R., Kiecolt-Glaser, J. K., Marucha, P. T., MacCallum, R. C., Laskowski, B. F., & Malarkey, W. B. (1999). Stress-related changes in proinflammatory cytokine production in wounds. *Archives of General Psychiatry, 56*, 450-456.

Glasper, E. R., & DeVries, A. C. (2005). Social structure influences effects of pair-housing on wound healing. *Brain, Behavior, and Immunity, 19*, 61-68.

Glynn, L. M., Christenfeld, N., & Gerin, W. (1999). Gender, social support, and cardiovascular responses to stress. *Psychosomatic Medicine, 61*, 234-242.

Grove, G. L., Grove, M. J., Zerweck, C., & Pierce, E. (1999). Computerized evaporimetry using the DermaLab TEWL probe. *Skin Research and Technology, 5*, 9-13.

Gruenewald, T. L., Kemeny, M. E., & Aziz, N. (in press). Subjective social status moderates cortisol responses to social threat. *Brain, Behavior, and Immunity*.

Halkier-Sorensen, L., Thestrup-Pedersen, K., & Maibach, H. I. (1993). Equation for conversion of transepidermal water loss (TEWL) to a common reference temperature: what is the slope? *Contact Dermatitis, 29*, 280-281.

Harvell, J. D., Hussona-Saccd, I., & Maibach, H. I. (1992). Changes in transepidermal water loss and cutaneous blood flow during the menstrual cycle. *Contact Dermatitis, 27*, 294-301.

Harvell, J. D., & Maibach, H. I. (1994). Percutaneous absorption and inflammation in aged skin: A review. *Journal of the American Academy of Dermatology, 31*, 1015-1021.

Heinrichs, M., Baumgartner, T., Kirschbaum, C., & Ehlert, U. (2003). Social support and oxytocin interact to suppress cortisol and subjective responses to psychosocial stress. *Biological Psychiatry, 54*, 1025-1034.

Hilmert, C. J., Kulik, J. A., & Christenfeld, N. (2002). The varied impact of social support on cardiovascular reactivity. *Basic and Applied Social Psychology, 24*, 229-240.

Hintze, J. (2001). NCSS and PASS. Kaysville, Utah: Number Cruncher Statistical Systems.

Hocking Schuler, J. L., & O'Brien, W. H. (1997). Cardiovascular recovery from stress and hypertension risk factors: A meta-analytic review. *Psychophysiology, 34*, 649-659.

Hofmann, S. G., & DiBartolo, P. M. (2000). An instrument to assess self-statements during public speaking: Scale development and preliminary psychometric properties. *Behavior Therapy, 31*, 499-515.

Holt-Lunstad, J., Uchino, B. N., Smith, T. W., Olson-Cerny, C., & Nealey-Moore, J. B. (2003). Social relationships and ambulatory blood pressure: Structural and qualitative predictors of cardiovascular function during everyday social interactions. *Health Psychology, 22*, 388-397.

House, J. S., Landis, K. R., & Umberson, D. (1988). Social relationships and health. *Science, 241*, 540-545.

Huzaira, M., Ruis, F., Rajadhyaksha, M., Anderson, R. R., & Gonzalez, S. (2001). Topographic variations in normal skin, as viewed by in vivo reflectance confocal microscopy. *Journal of Investigative Dermatology, 116*, 846-852.

Ingram, K. M., Betz, N. E., Mindes, E. J., Schmitt, M. M., & Smith, N. G. (2001). Unsupportive responses from others concerning a stressful life event:

Development of the Unsupportive Social Interactions Inventory. *Journal of Social and Clinical Psychology, 20*, 173-207.

Joreskog, K., & Sorbom, D. (1996). LISREL 8: User's reference guide. Chicago: Scientific Software International.

Kabat-Zinn, J., Wheeler, E., Light, T., Skillings, A., Scharf, M. J., Cropley, T. G., et al. (1998). Influence of a mindfulness meditation-based stress reduction intervention on rates of skin clearing in patients with moderate to severe psoriasis undergoing phototherapy (UVB) and photochemotherapy (PUVA). *Psychosomatic Medicine, 60*, 625-632.

Kamarck, T. W. (1992). Recent developments in the study of cardiovascular reactivity: Contributions from psychometric theory and social psychology. *Psychophysiology, 29*, 491-503.

Kamarck, T. W., Annunziato, B., & Amateau, L. M. (1995). Affiliation moderates the effects of social threat on stress-related cardiovascular responses: Boundary conditions for a laboratory model of social support. *Psychosomatic Medicine, 57*, 183-194.

Kamarck, T. W., & Lovallo, W. R. (2003). Cardiovascular reactivity to psychological challenge: Conceptual and measurement considerations. *Psychosomatic Medicine, 65*, 9-21.

Kamarck, T. W., Manuck, S. B., & Jennings, J. R. (1990). Social support reduces cardiovascular reactivity to psychological challenge: A laboratory model. *Psychosomatic Medicine, 52*, 42-58.

Kamarck, T. W., Peterman, A. H., & Raynor, D. A. (1998). The effects of the social environment on stress-related cardiovascular activation: Current findings, prospects, and implications. *Annals of Behavioral Medicine, 20*, 247-256.

Kamarck, T. W., Schwartz, J. E., Janicki, D. L., Shiffman, S., & Raynor, D. A. (2003). Correspondence between laboratory and ambulatory measures of cardiovascular reactivity: A multilevel modeling approach. *Psychophysiology, 40*, 675-683.

Kao, J. S., Fluhr, J. W., Man, M. Q., Fowler, A. J., Hachem, J. P., Crumrine, D., et al. (2003). Short-term glucocorticoid treatment compromises both permeability barrier homeostasis and stratum corneum integrity: Inhibition of epidermal lipid synthesis accounts for functional abnormalities. *Journal of Investigative Dermatology, 120*, 456-464.

Kenouch, S., Lombes, M., Delahaye, F., Eugene, E., Bonvalet, J., & Farman, N. (1994). Human skin as target for aldosterone: Coexpression of mineralocorticoid

receptors and 11β-hydroxysteroid dehydrogenase. *Journal of Clinical Endocrinology and Metabolism, 79*, 1334-1341.

Keppel, G. (1991). *Design and analysis: A researcher's handbook*. Upper Saddle River, NJ: Prentice-Hall, Inc.

Kiecolt-Glaser, J. K., Cacioppo, J. T., Malarkey, W. B., & Glaser, R. (1992). Acute psychological stressors and short-term immune changes: What, why, for whom, and to what extent? *Psychosomatic Medicine, 54*, 680-685.

Kiecolt-Glaser, J. K., & Glaser, R. (1988). Methodological issues in behavioral immunology research with humans. *Brain, Behavior, and Immunity, 2*, 67-78.

Kiecolt-Glaser, J. K., & Greenberg, B. (1984). Social support as a moderator of the aftereffects of stress in female psychiatric inpatients. *Journal of Abnormal Psychology, 93*(2), 192-199.

Kiecolt-Glaser, J. K., Loving, T. J., Stowell, J. R., Malarkey, W. B., Lemeshow, S., Dickinson, S. L., et al. (2005). Hostile marital interactions, proinflammatory cytokine production, and wound healing. *Archives of General Psychiatry, 62*, 1377-1384.

Kiecolt-Glaser, J. K., Marucha, P. T., Malarkey, W. B., Mercado, A. M., & Glaser, R. (1995). Slowing of wound healing by psychological stress. *Lancet, 346*, 1194-1196.

Kiecolt-Glaser, J. K., Preacher, K. J., MacCallum, R. C., Atkinson, C., Malarkey, W. B., & Glaser, R. (2003). Chronic stress and age-related increases in the proinflammatory cytokine interleukin-6. *Proceedings of the National Academy of Sciences of the United States of America, 100*, 9090-9095.

Kirschbaum, C., & Hellhammer, D. H. (1994). Salivary cortisol in psychoneuroendocrine research: Recent developments and applications. *Psychoneuroendocrinology, 19*, 313-333.

Kirschbaum, C., Klauer, T., Filipp, S. H., & Hellhammer, D. H. (1995). Sex-specific effects of social support on cortisol and subjective responses to acute psychological stress. *Psychosomatic Medicine, 21*, 525-531.

Kirschbaum, C., Kudielka, B. M., Gaab, J., Schommer, N. C., & Hellhammer, D. H. (1999). Impact of gender, menstrual cycle phase, and oral contraceptives on the activity of the hypothalamus-pituitary-adrenal axis. *Psychosomatic Medicine, 61*, 154-162.

Kirschbaum, C., Pirke, K., & Hellhammer, D. H. (1993). The 'Trier Social Stress Test' - A tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology, 28*, 76-81.

Kors, D. J., Linden, W., & Gerin, W. (1997). Evaluation interferes with social support: Effects on cardiovascular stress reactivity in women. *Journal of Social and Clinical Psychology, 16*, 1-23.

Krantz, D. S., & McCeney, M. K. (2002). Effects of psychological and social factors on organic disease: A critical assessment of research on coronary heart disease. *Annual Review of Psychology, 53*, 541-569.

Kunz-Ebrecht, S. R., Mohamed-Ali, V., Feldman, P. J., Kirschbaum, C., & Steptoe, A. (2003). Cortisol responses to mild psychological stress are inversely with proinflammatory cytokines. *Brain, Behavior, and Immunity, 17*, 373-383.

Leary, M. R. (1983). A brief version of the Fear of Negative Evaluation Scale. *Personality and Social Psychology Bulletin, 9*, 371-375.

Lepore, S. J. (1995). Cynicism, social support, and cardiovascular reactivity. *Health Psychology, 14*, 210-216.

Lepore, S. J. (1998). Problems and prospects for the social support-reactivity hypothesis. *Annals of Behavioral Medicine, 20*, 257-269.

Lepore, S. J., Mata Allen, K. A., & Evans, G. W. (1993). Social support lowers cardiovascular reactivity to an acute stressor. *Psychosomatic Medicine, 55*, 518-524.

Linden, W., Earle, T. L., Gerin, W., & Christenfeld, N. (1997). Physiological stress reactivity and recovery: Conceptual siblings separated at birth? *Journal of Psychosomatic Research, 42*, 117-135.

Linden, W., Gerin, W., & Davidson, K. (2003). Cardiovascular reactivity: Status quo and a research agenda for the new millennium. *Psychosomatic Medicine, 65*, 5-8.

Little, R. J. A., & Rubin, D. A. (1987). *Statistical analysis with missing data*. New York: John Wiley and Sons.

Llabre, M. M., Spitzer, S., Siegel, S., Saab, P. G., & Schneiderman, N. (2004). Applying latent growth curve modeling to the investigation of individual differences in cardiovascular recovery from stress. *Psychosomatic Medicine, 66*, 29-41.

Llabre, M. M., Spitzer, S. B., Saab, P. G., Ironson, G. H., & Schneiderman, N. (1991). The reliability and specificity of delta versus residualized change as measures of

cardiovascular reactivity to behavioral challenges. *Psychophysiology, 28*, 701-711.

Llabre, M. M., Spitzer, S. B., Saab, P. G., & Schneiderman, N. (2001). Piecewise latent growth curve modeling of systolic blood pressure reactivity and recovery from the cold pressor test. *Psychophysiology, 38*, 951-960.

Lowry, S. F. (1993). Cytokine mediators of immunity and inflammation. *Archives of Surgery, 128*, 1235-1241.

MacCallum, R. C., Kim, C., Malarkey, W. B., & Kiecolt-Glaser, J. K. (1997). Studying multivariate change using multilevel models and latent curve models. *Multivariate Behavioral Research, 32*, 215-253.

Marucha, P. T., Kiecolt-Glaser, J. K., & Favagehi, M. (1998). Mucosal wound healing is impaired by examination stress. *Psychosomatic Medicine, 60*, 362-365.

Matthews, K. A., Gump, B. B., & Owens, J. F. (2001). Chronic stress influences cardiovascular and neuroendocrine responses during acute stress and recovery, especially in men. *Health Psychology, 20*, 403-410.

McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *New England Journal of Medicine, 388*, 171-179.

McNair, D. M., Lorr, M., & Dropelman, L. F. (1981). *Profile of Mood States*. San Diego: Educational and Testing Service.

Nagurney, A., III. (2001). *The effects of social support and social control on cardiovascular reactivity.* Unpublished master's thesis, Arizona State University, Tempe, Arizona.

Ngoc, P. L., Gold, D. R., Tzianabos, A. O., Weiss, S. T., & Celedon, J. C. (2005). Cytokines, allergy, and asthma. *Current Opinion in Allergy and Clinical Immunology, 5*, 161-166.

Nickoloff, B. J., & Naidu, Y. (1994). Perturbation of epidermal barrier function correlates with initiation of cytokine cascade in human skin. *Journal of the American Academy of Dermatology, 30*, 535-546.

Norlen, L., Nicander, I., Rozell, B. L., Ollmar, S., & Forslind, B. (1999). Inter- and Intra-individual differences in human stratum corneum lipid content related to physical parameters of skin barrier function *in vivo*. *Journal of Investigative Dermatology, 112*, 72-77.

O'Sullivan, R. L., Lipper, G., & Lerner, E. A. (1998). The neuro-immuno-cutaneous-endocrine network: relationship of mind and skin. *Archives of Dermatology, 134*, 1431-1435.

Paquet, P., & Pierard, G. E. (1996). Interleukin-6 and the skin. *International Archives of Allergy and Applied Immunology, 109*, 308-317.

Polefrone, J. M., & Manuck, S. B. (1988). Effects of menstrual phase and parental history of hypertension on cardiovascular response to cognitive challenge. *Psychosomatic Medicine, 50*, 23-36.

Pope, M. K., Smith, T. W., & Rhodewalt, F. (1990). Cognitive, behavioral, and affective correlates of the Cook and Medley Hostility Scale. *Journal of Personality Assessment, 54*, 501-514.

Pressman, S., Cohen, S., Miller, G. E., Barkin, A., Rabin, B. S., & Treanor, J. J. (2005). Loneliness, social network size, and immune response to influenza vaccination in college freshmen. *Health Psychology, 24*, 297-306.

Proksch, E., Brasch, J., & Sterry, W. (1996). Integrity of the permeability barrier regulates epidermal Langerhans cell density. *British Journal of Dermatology, 134*, 630-638.

Proksch, E., Feingold, K. R., Mao-Qiang, M., & Elias, P. M. (1991). Barrier function regulates epidermal DNA synthesis. *Journal of Clinical Investigation, 87*, 1668-1673.

Pruessner, J. C., Kirschbaum, C., Meinlschmid, G., & Hellhammer, D. H. (2003). Two formulas for computation of the area under the curve represent measures of total hormone concentration versus time-dependent change. *Psychoneuroendocrinology, 28*, 916-931.

Raikkonen, K., Matthews, K. A., Flory, J. D., Owens, J. F., & Gump, B. B. (1999). Effects of optimism, pessimism, and trait anxiety on ambulatory blood pressure and mood during everyday life. *Journal of Personality and Social Psychology, 76*, 104-113.

Raudenbush, S. W., Bryk, A. S., Cheong, Y. F., & Congdon, R. (2004). *HLM 6: Hierarchical linear and nonlinear modeling*. Lincolnwood, IL: Scientific Software International.

Reed, J. T., Ghadially, R., & Elias, P. M. (1995). Skin type, but neither race nor gender, influence epidermal permeability barrier function. *Archives of Dermatology, 131*, 1134-1138.

Reilly, D. M., & Green, M. R. (1999). Eicosanoid and cytokine levels in acute skin irritation in response to tape stripping and capsaicin. *Acta Dermato-Venereologica, 79*, 187-190.

Rodebaugh, T. L., Woods, C. M., Thissen, D. M., Heimberg, R. G., Chambless, D. L., & Rapee, R. M. (2004). More information from fewer questions: The factor structure and item properties of the original and Brief Fear of Negative Evaluation Scale. *Psychological Assessment, 16*, 169-181.

Rodrigues, L. M., Pinto, P. C., Magro, J. M., Fernandes, M., & Alves, J. (2004). Exploring the influence of skin perfusion on transepidermal water loss. *Skin Research and Technology, 10,* 257-262.

Rogers, J., Harding, C., Mayo, A., Banks, J., & Rawlings, A. (1996). Stratum corneum lipids: The effect of ageing and the seasons. *Archives of Dermatological Research, 288*, 765-770.

Roy, M. P., Kirschbaum, C., & Steptoe, A. (2001). Psychological, cardiovascular, and metabolic correlates of individual differences in cortisol stress recovery in young men. *Psychoneuroendocrinology, 26*, 375-391.

Russell, D., Peplau, L. A., & Cutrona, C. E. (1980). The revised UCLA loneliness Scale: Concurrent and discriminant validity evidence. *Journal of Personality and Social Psychology, 39*, 472-480.

Rutledge, T., Linden, W., & Paul, D. (2000). Cardiovascular recovery from acute laboratory stress: Reliability and concurrent validity. *Psychosomatic Medicine, 62*, 648-654.

Sarason, I. G., & Sarason, B. R. (1986). Experimentally provided social support. *Journal of Personality and Social Psychology, 50*, 1222-1225.

Schallreuter, K. U., Lemke, K. R., Pittelkow, M. R., Wood, J. M., Korner, C., & Malik, R. (1995). Catecholamines in human keratinocyte differentiation. *Journal of Investigative Dermatology, 104*, 953-957.

Scheier, M. F., Carver, C. S., & Bridges, M. W. (1994). Distinguishing optimism from neuroticism (and trait anxiety, self-mastery, and self-esteem): A reevaluation of the Life Orientation Test. *Journal of Personality and Social Psychology, 67*, 1063-1078.

Schwartz, A. R., Gerin, W., Davidson, K. W., Pickering, T. G., Brosschot, J. F., Thayer, J. F., et al. (2003). Toward a causal model of cardiovascular responses to stress and the development of cardiovascular disease. *Psychosomatic Medicine, 65*, 22-35.

Serres, M., Viac, J., & Schmitt, D. (1996). Glucocorticoid receptor localization in human epidermal cells. *Archives of Dermatological Research, 288*, 140-146.

Shah, M. G., & Maibach, H. I. (2001). Estrogen and skin: An overview. *American Journal of Clinical Dermatology, 2*, 143-150.

Shapiro, D., Jamner, L. D., Lane, J. D., Light, K. C., Myrtek, M. Sawada, Y., & Steptoe, A. (1996). Blood pressure publication guidelines. *Psychophysiology, 33,* 1-12.

Sheffield, D., & Carroll, D. (1994). Social support and cardiovascular reactions to active laboratory stressors. *Psychology and Health, 9*, 305-316.

Sheffield, D., & Carroll, D. (1996). Task-induced cardiovascular activity and the presence of a supportive or undermining other. *Psychology and Health, 11*, 583-591.

Sheridan, J. F., Padgett, D. A., Avitsur, R., & Marucha, P. T. (2004). Experimental models of stress and wound healing. *World Journal of Surgery, 28*, 327-330.

Singer, A. J., & Clark, R. A. F. (1999). Cutaneous wound healing. *New England Journal of Medicine, 341*(10), 738-746.

Singer, J. D. (1998). Using SAS PROC MIXED to fit multilevel models, hierarchical models, and individual growth curve models. *Journal of Educational & Behavioral Statistics, 24*, 323-355.

Slominski, A., & Wortsman, J. (2000). Neuroendocrinology of the skin. *Endocrine Reviews, 21*, 457-487.

Smith, J. C., Bradley, M. M., & Lang, P. J. (2005). State anxiety and affective physiology: Effects of sustained exposure to affective pictures. *Biological Psychology, 69*, 247-260.

Smith, T. W., Gallo, L. C., Goble, L., Ngu, L. Q., & Stark, K. A. (1998). Agency, communion, and cardiovascular reactivity during marital interaction. *Health Psychology, 17*, 537-545.

Smith, T. W., Limon, J. P., Gallo, L. C., & Ngu, L. Q. (1996). Interpersonal control and cardiovascular reactivity: Goals, behavioral expression, and the moderating effects of sex. *Journal of Personality and Social Psychology, 70*, 1012-1024.

Smith, T. W., & Ruiz, J. M. (2002). Psychosocial influences on the development and course of coronary heart disease: Current status and implications for research and practice. *Journal of Consulting and Clinical Psychology, 70*, 548-568.

Snydersmith, M. A., & Cacioppo, J. T. (1992). Parsing complex social factors to determine component effects: I. Autonomic activity and reactivity as a function of human association. *Journal of Social and Clinical Psychology, 11*, 263-278.

Spielberger, C. D., Gorsuch, R. L., Lushene, R. E., Vagg, P. R., & Jacobs, G. A. (1983). *Manual for the State-Trait Anxiety Inventory(Form Y)*. Palo Alto, CA: Consulting Psychologists Press.

Spielberger, C. D., Krasner, S. S., & Solomon, E. P. (1988). The experience and expression of anger: Construction and validation of an anger expression scale. In M. A. Chesney & R. H. Rosenman (Eds.), *Anger and hostility in cardiovascular and behavioral disorders* (pp. 5 - 30). Washington, DC: Hemisphere.

SPSS. (2004). SPSS 13.0 for Windows. Chicago, IL: SPSS.

Steinkraus, V., Steinfath, M., Korner, C., & Mensing, H. (1992). Binding of beta-adrenergic receptors in human skin. *Journal of Investigative Dermatology, 98*, 475-780.

Steptoe, A., Willemsen, G., Owen, N., Flower, L., & Mohamed-Ali, V. (2001). Acute mental stress elicits delayed increases in circulating inflammatory cytokine levels. *Clinical Science, 101*, 185-192.

Strid, J., Hourihane, J., Kimber, I., Callard, R., & Strobel, S. (2004). Disruption of the stratum corneum allows potent epicutaneous immunization with protein antigens resulting in a dominant systemic Th2 response. *European Journal of Immunology, 34*, 2100-2109.

Stroud, L. R., Salovey, P., & Epel, E. S. (2002). Sex differences in stress responses: Social rejection versus achievement stress. *Biological Psychiatry, 52*, 318-327.

Tagami, H., & Yoshikuni, K. (1985). Interrelationship between water-barrier and reservoir functions of pathologic stratum corneum. *Archives of Dermatological Research, 121*, 642-645.

Tanaka, M., Zhen, Y. X., & Tagami, H. (1997). Normal recovery of the stratum corneum barrier function following damage induced by tape stripping in patients with atopic dermatitis. *British Journal of Dermatology, 136*, 966-967.

Thorsteinsson, E. B., & James, J. E. (1999). A meta-analysis of the effects of experimental manipulations of social support during laboratory stress. *Psychology and Health, 14*, 869-886.

Thorsteinsson, E. B., James, J. E., & Gregg, M. E. (1998). Effects of video-relayed social support on hemodynamic reactivity and salivary cortisol during laboratory-based behavioral challenge. *Health Psychology, 17*, 436-444.

Tomaka, J., Blascovich, J., Kelsey, R. M., & Leitten, C. L. (1993). Subjective, physiological, and behavioral effects of threat and challenge appraisal. *Journal of Personality and Social Psychology, 65*, 248-260.

Trieber, F. A., Kamarck, T., Schneiderman, N., Sheffield, D., Kapuku, G., & Taylor, T. (2003). Cardiovascular reactivity and development of preclinical and clinical disease states. *Psychosomatic Medicine, 64*, 46-62.

Tsai, J. C., Feingold, K. R., Crumrine, D., Wood, L. C., Grunfeld, C., & Elias, P. M. (1994). Permeability barrier disruption alters the localization and expression of TNF alpha protein in the epidermis. *Archives of Dermatological Research, 286*, 242-248.

Uchino, B. N., Cacioppo, J. T., & Kiecolt-Glaser, J. K. (1996). The relationship between social support and physiological processes: A review with emphasis on underlying mechanisms and implications for health. *Psychological Bulletin, 119*, 488-531.

Uchino, B. N., & Garvey, T. S. (1997). The availability of social support reduces cardiovascular reactivity to acute psychological stress. *Journal of Behavioral Medicine, 20*, 15-27.

Uno, D., Uchino, B. N., & Smith, T. W. (2002). Relationship quality moderates the effect of social support given by close friends on cardiovascular reactivity in women. *International Journal of Behavioral Medicine, 9*, 243-262.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54*(6), 1063-1070.

Watson, D., Clark, L. A., Weber, K., Assenheimer, J. S., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model: II. Exploring the symptom structure of anxiety and depression in student, adult, and patient samples. *Journal of Abnormal Psychology, 104*, 15-25.

Watson, D., Weber, K., Assenheimer, J. S., Clark, L. A., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model: I. Evaluating the convergent and discriminant validity of anxiety and depression symptom scales. *Journal of Abnormal Psychology, 104*(1), 3-14.

Weeks, J. W., Heimberg, R. G., Fresco, D. M., Hart, T. A., Turk, C. L., Schneier, F. R., et al. (2005). Empirical validation and psychometric evaluation of the brief fear of negative evaluation scale in patients with social anxiety disorder. *Psychological Assessment, 17*, 179-190.

Weiss, G., Shemer, A., & Trau, H. (2002). The Koebner phenomenon: Review of the literature. *Journal of the European Academy of Dermatology and Venereology, 16*, 241-248.

Wessely, S. C., & Lewis, G. H. (1989). The classification of psychiatric morbidity in attenders at a dermatology clinic. *British Journal of Psychiatry, 155*, 686-691.

Wiggins, J. S., Trapnell, P., & Phillips, N. (1988). Psychometric and geometric characteristics of the revised interpersonal adjective scales (IAS-R). *Multivariate Behavioral Research, 23*, 517-530.

Willett, J. B., & Sayer, A. G. (1994). Using covariance structure analysis to detect correlates and predictors of individual change over time. *Psychological Bulletin, 116*, 363-381.

Wills, T. A., & Shinar, O. (2000). Measuring perceived and received social support. In S. Cohen, L. Underwood & B. H. Gottlieb (Eds.), *Social support measurement and intervention* (pp. 86-135). New York: Oxford University Press.

Witte, M. B., & Barbul, A. (1997). General principles of wound healing. *Surgical Clinics of North America, 77*, 509-528.

Ye, J., Garg, A., Calhoun, C., Feingold, K. R., Elias, P. M., & Ghadially, R. (2002). Alterations in cytokine regulation in aged epidermis: Implications for permeability barrier homeostasis and inflammation. I. IL-1 gene family. *Experimental Dermatology, 11*, 209-216.

Yoshikawa, N., Imokawa, G., Akimoto, K., Jin, K., Higaki, Y., & Kawashima, M. (1994). Regional analysis of ceramides within the stratum corneum in relation to seasonal changes. *Dermatology, 188*, 207-214.

Yosipovitch, G., Sackett-Lundeen, L., Goon, A., Huak, C. Y., Goh, C. L., & Haus, E. (2004). Circadian and ultradian (12 h) variations of skin blood flow and barrier function in non-irritated and irritated skin – effect of topical corticosteroids. *Journal of Investigative Dermatology, 122,* 824-829.

Yosipovitch, G., Xiong, G. L., Haus, E., Sackett-Lundeen, L., Ashkenazi, I., & Maibach, H. (1998). Time-dependent variations of the skin barrier function in humans: Transepidermal water loss, stratum corneum hydration, skin surface pH, and skin temperature. *Journal of the American Academy of Dermatology, 110*, 20-23.

APPENDIX A

CONFEDERATE SOCIAL SUPPORT BEHAVIORS

Supportive behaviors

Functions	Definitions	Benefits	Specific Examples
Emotional support	Allowing discussion of feelings, expression of concerns or worries	Alters threat appraisals, enhances self-esteem, reduces anxiety/depression, motivates coping	"Remember, it will all be over in a few minutes" "It's okay to feel anxious about this" "I definitely understand what you're going through" "You'll do fine." "I know you'll be able to get through this"
Instrumental support	Providing tangible assistance	Solves practical problems, allows increased time for coping efforts	"You should structure your speech into 3 parts: Your background, what you bring to this position, and what you like about the position."

Supportive behaviors (continued).

Informational support	Providing information about resources, advice about effective actions	Increases amount of useable information available to individual, leads to more effective coping	"I've found that writing a brief outline of your main points is helpful" "One thing you can do is come up with 3 items for each main idea of your speech." "It helps to speak at a slightly slower pace, because that makes you look comfortable"
Validation	Providing information on normativeness of individual's behavior and/or feelings, relative status in population	Decreases perceived deviancy, allows acceptance of feelings, provides favorable comparisons	Other participants who have gone through this also feel pretty nervous, so what you're feeling is quite normal" "It's okay if you stumble a little bit in there; everybody gets nervous when giving a speech in front of people they don't know."

Note. Functions, definitions, and benefits taken from Table 4.1, (Wills & Shinar, 2000). Some of the specific examples are from previous work (Nagurney, 2001).

Supportive questions and responses

Functions	Questions	Responses
Emotional support	"How are you feeling?"	"You sound worried"
	"Do you feel nervous?"	"I understand"
Instrumental support	"How are you organizing your speech?"	"That sounds like a good way to do it"
	"What did you write down in your notes?"	"Good idea!"

APPENDIX B

SELF-REPORT QUESTIONNAIRES

Packet 1, administered during 40 min habituation period. Contents include:

- Recent Medication use, menstrual phase questions
- Positive and Negative Affect Schedule (PANAS), Trait version (Watson, Clark, & Tellegen, 1988)
- Mood and Anxiety Symptoms Questionnaire (Watson, Clark et al., 1995; Watson, Weber et al., 1995)
- Brief Fear of Negative Evaluation scale (Leary, 1983)
- Revised UCLA Loneliness Scale (Russell, Peplau, & Cutrona, 1980)
- Cook-Medley Hostility Scale (Cook & Medley, 1954)
- Spielberger Anger Expression Scale (Spielberger, Krasner, & Solomon, 1988)
- MacArthur scale of subjective social status (Adler, Epel, Castellazzo, & Ickovics, 2000)
- Interpersonal Support Evaluation List (Cohen, Mermelstein, Kamarck, & Hoberman, 1985)
- Life Orientation Test – Revised (Scheier, Carver, & Bridges, 1994)
- Perceived Stress Scale (Cohen, Kamarck, & Mermelstein, 1983)
- PANAS-1 (Also administered in Packets 1,2,5,6,7,8).
- Spielberger State-Trait Anxiety Inventory, State version (STANX-1, also administered in Packets 1,2,5,6,7,8).

Recent Medication use

1. How many standard aspirin tablets (325 mg/pill) have you taken over the last 72 hours? ___ over the last 48 hours? ___ over the last 24 hours? ___

2. How many extra strength aspirin tablets (500 mg/pill) have you taken over the last 72 hours? ___ over the last 48 hours? ___ over the last 24 hours? ___

3. How many standard ibuprofen tablets (200 mg/pill) have you taken over the last 72 hours? ___ over the last 48 hours? ___ over the last 24 hours? ___

4. For women: When was the first day of your most recent menstrual cycle?

   Month: ___ Day: ____ Year: ____

   OR

   ___ postmenopausal, or no longer menstruating.

5. Over the past 3 months, what has been the typical length of your entire menstrual cycle (how many days are there typically between the start of two consecutive menstrual periods)? ____ days

6. Over the past three months, has your cycle changed from month to month?

   No/Yes. If YES, please explain:

7. In a year, a woman will typically experience 12 menstrual periods (1 a month for 12 months). According to this standard, how many menstrual periods (if any) have you missed in the past year? _____

8. Do you have any menstrual or gynecological problems for which you have seen a doctor? No/Yes. If YES, please explain.

9. A woman's menstrual period (number of days a woman actually bleeds) tends to last typically anywhere from 4 to 8 days. Over the past three months, what has been the typical length of your menstrual period (number of days you actually bleed)?

___ days

10. Please list below any irregularities or problems with your menstrual cycle that have not been covered in previous questions on this questionnaire:

Positive and Negative Affect Schedule, Trait version (PANAS-T)

This scale consists of a number of words that describe different feelings and emotions. Read each item and then fill in the circle that indicates how you feel IN GENERAL, THAT IS, ON THE AVERAGE.

	Very slightly or not at all	A little	Moderately	Quite a bit	Extremely
Interested	☐	☐	☐	☐	☐
Distressed	☐	☐	☐	☐	☐
Excited	☐	☐	☐	☐	☐
Upset	☐	☐	☐	☐	☐
Strong	☐	☐	☐	☐	☐
Guilty	☐	☐	☐	☐	☐
Scared	☐	☐	☐	☐	☐
Hostile	☐	☐	☐	☐	☐
Enthusiastic	☐	☐	☐	☐	☐
Proud	☐	☐	☐	☐	☐
Irritable	☐	☐	☐	☐	☐
Alert	☐	☐	☐	☐	☐
Ashamed	☐	☐	☐	☐	☐
Inspired	☐	☐	☐	☐	☐
Nervous	☐	☐	☐	☐	☐
Determined	☐	☐	☐	☐	☐
Attentive	☐	☐	☐	☐	☐
Jittery	☐	☐	☐	☐	☐
Active	☐	☐	☐	☐	☐
Afraid	☐	☐	☐	☐	☐

Mood and Anxiety Symptoms Questionnaire

Below is a list of feelings, sensations, problems, and experiences that people have. Read each item and then mark the appropriate choice. Use the choice that best describes how much you have felt or experienced things this way this past week, including today. Use this scale when answering:

1. not at all 2. a little bit 3. moderately 4. quite a bit 5. extremely

1. Felt sad	24. Hands were cold and sweaty	46. Had a very dry mouth
2. Startled easily	25. Felt withdrawn from other people	47. Felt like I had a lot of interesting things to do
3. Felt cheerful	26. Felt keyed up, "on edge"	48. Was afraid I was going to die
4. Felt afraid	27. Felt like I had a lot of energy	49. Felt like I had accomplished a lot
5. Felt discouraged	28. Was trembling or shaking	50. Felt like it took extra effort to get started
6. Hands were shaky	29. Felt inferior to others	51. Felt like nothing was very enjoyable
7. Felt optimistic	30. Had trouble swallowing	52. Heart was racing or pounding
8. Had diarrhea	31. Felt like crying	53. Felt like I had a lot to look forward to
9. Felt worthless	32. Was unable to relax	54. Felt numbness or tingling in my body
10. Felt really happy	33. Felt really slowed down	55. Felt tense or "high-strung"
11. Felt nervous	34. Was disappointed in myself	56. Felt hopeful about the future
12. Felt depressed	35. Felt nauseous	57. Felt like there wasn't anything interesting or fun to do
13. Was short of breath	36. Felt hopeless	58. Seemed to move quickly and easily
14. Felt uneasy	37. Felt dizzy or lightheaded	59. Muscles were tense or sore
15. Was proud of myself	38. Felt sluggish or tired	60. Felt really good about myself
16. Had a lump in my throat	39. Felt really "up" or lively	61. Thought about death or suicide
17. Felt faint	40. Had pain in my chest	62. Had to urinate frequently
18. Felt unattractive	41. Felt really bored	
19. Had hot or cold spells	42. Felt like I was choking	
20. Had an upset stomach	43. Looked forward to things with enjoyment	
21. Felt like a failure	44. Muscles twitched or trembled	
22. Felt like I was having a lot of fun	45. Felt pessimistic about the future	
23. Blamed myself for a lot of things		

Brief Fear of Negative Evaluation scale

Using the following rating scale, please indicate in the blank next to each statement the degree to which the following items are characteristic of you:

1----------2----------3----------4----------5

Not at all characteristic of me — Extremely characteristic of me

1. I worry about what people will think of me even when I know it doesn't make any difference.
2. I am unconcerned even if I know people are forming an unfavorable impression of me.
3. I am frequently afraid of other people noting my shortcomings.
4. I rarely worry about what kind of impression I am making on someone.
5. I am afraid that others will not approve of me.
6. I am afraid that others will find fault with me.
7. Other people's opinions of me do not bother me.
8. When I am talking to someone, I worry about what they may be thinking about me.
9. I am usually worried about what kind of impression I make.
10. If I know someone is judging me, it has little effect on me.
11. Sometimes I think I am too concerned with what other people think of me.
12. I often worry that I will say or do the wrong thing.

Revised UCLA Loneliness Scale

Directions: Indicate how often you feel the way described in each of the following statements. Mark one answer for each statement.

	Never	Rarely	Sometimes	Often
1. I feel in tune with the people around me.	☐	☐	☐	☐
2. I lack companionship.	☐	☐	☐	☐
3. There is no one I can turn to.	☐	☐	☐	☐
4. I do not feel alone.	☐	☐	☐	☐
5. I feel part of a group of friends.	☐	☐	☐	☐
6. I have a lot in common with the people around me.	☐	☐	☐	☐
7. I am no longer close to anyone.	☐	☐	☐	☐
8. My interests and ideas are not shared by those around me.	☐	☐	☐	☐
9. I am an outgoing person.	☐	☐	☐	☐
10. There are people I feel close to.	☐	☐	☐	☐
11. I feel left out.	☐	☐	☐	☐
12. My social relationships are superficial.	☐	☐	☐	☐
13. No one really knows me well.	☐	☐	☐	☐
14. I feel isolated from others.	☐	☐	☐	☐
15. I can find companionship when I want it.	☐	☐	☐	☐
16. There are people who really understand me.	☐	☐	☐	☐
17. I am unhappy being so withdrawn.	☐	☐	☐	☐
18. People are around me but not with me.	☐	☐	☐	☐
19. There are people I can talk to.	☐	☐	☐	☐
20. There are people I can turn to.	☐	☐	☐	☐

Cook-Medley Hostility Scale

Indicate if each statement is usually true or usually false for you by filling in the circle under the column labeled true or the column labeled false.

1. When someone does me wrong, I feel I should pay him/her back if I can, just for the principle of the thing.
2. I prefer to pass by school friends, or people I know but have not seen for a long time, unless they speak to me first.
3. I have often had to take orders from someone who did not know as much as I did.
4. I think a great many people exaggerate their misfortunes in order to gain the sympathy and help of others.
5. It takes a lot of argument to convince most people of the truth.
6. I think most people would lie to get ahead.
7. Someone has it in for me.
8. Most people are honest chiefly through fear of being caught.
9. Most people will use somewhat unfair means to gain profit or an advantage rather than to lose it.
10. I commonly wonder what hidden reason another person may have for doing something nice for me.
11. It makes me impatient to have people ask my advice or otherwise interrupt me when I am working on something important.
12. I feel that I have often been punished without cause.
13. I am against giving money to beggars.
14. Some of my family have habits that bother and annoy me very much.
15. No one cares much about what happens to you.
16. My relatives are nearly all in sympathy with me.
17. My way of doing things is apt to be misunderstood by others.
18. I do not blame anyone for trying to grab everything he/she can get in this world.
19. Most people make friends because friends are likely to be useful to them.
20. I am sure I am being talked about.
21. I am likely not to speak to people until they speak to me.
22. Most people inwardly dislike putting themselves out to help other people.
23. I tend to be on my guard with people who are somewhat more friendly than I had expected.
24. I have sometimes stayed away from another person because I feared doing or saying something that I might regret afterwards.
25. People often disappoint me.
26. I like to keep people guessing what I'm going to do next.

Cook Medley Hostility Scale (continued).

27. I frequently ask people for advice.
28. I am not easily angered.
29. I have often met people who were supposed to be experts who were no better than I.
30. I would certainly enjoy beating a crook at his/her own game.
31. It makes me feel like a failure when I hear of the success of someone I know well.
32. I have at times had to be rough with people who were rude or annoying.
33. People generally demand more respect for their own rights than they are willing to allow for others.
34. There are certain people whom I dislike so much that I am inwardly pleased when they are catching it for something they have done.
35. I am often inclined to go out of my way to win a point with someone who has opposed me.
36. I am quite often not in on the gossip and talk of the group I belong to.
37. The man who had most to do with me when I was a child (such as my father, stepfather, etc.) was very strict with me.
38. I have often found people jealous of my good ideas, just because they had not thought of them first.
39. When a man is with a woman, he is usually thinking about things related to her sex.
40. I do not try to cover up my poor opinion or pity of a person so that he won't know how I feel.
41. I have frequently worked under people who seem to have things arranged so that they get credit for good work but are able to pass off their mistakes on to those under them.
42. I strongly defend my own opinions as a rule.
43. People can pretty easily change me even though I thought that my mind was already made up on a subject.
44. Sometimes I am sure that other people can tell what I am thinking.
45. A large number of people are guilty of bad sexual conduct.
46. When I take a new job, I like to be tipped off on who should be gotten next to.
47. Strangers look at me critically.
48. I can be friendly with people who do things which I consider wrong.
49. It is safer to trust nobody.
50. I do not blame a person for taking advantage of someone who lays himself open to it.

Spielberger Anger Expression Scale

Everyone feels angry or furious from time to time, but people differ in the ways that they react when they are angry. A number of statements are listed below which people have used to describe their reactions when they feel angry or furious. Read each statement and then fill in the circle to the right of the statement that indicates how often you generally react or behave in the manner described.

	Almost never	Sometimes	Often	Almost always
1. I control my temper	☐	☐	☐	☐
2. I express my anger	☐	☐	☐	☐
3. I keep things in	☐	☐	☐	☐
4. I am patient with others	☐	☐	☐	☐
5. I pout or sulk	☐	☐	☐	☐
6. I withdraw from people	☐	☐	☐	☐
7. I make sarcastic remarks to others	☐	☐	☐	☐
8. I keep my cool	☐	☐	☐	☐
9. I do things like slam doors	☐	☐	☐	☐
10. I boil inside, but I don't show it	☐	☐	☐	☐
11. I control my behavior	☐	☐	☐	☐
12. I argue with others	☐	☐	☐	☐
13. I tend to harbor grudges that I don't tell anyone about	☐	☐	☐	☐
14. I strike out at whatever infuriates me	☐	☐	☐	☐
15. I can stop myself from losing my temper	☐	☐	☐	☐
16. I am secretly quite critical of others	☐	☐	☐	☐
17. I am angrier than I am willing to admit	☐	☐	☐	☐
18. I calm down faster than most other people	☐	☐	☐	☐
19. I say nasty things	☐	☐	☐	☐
20. I try to be tolerant and understanding	☐	☐	☐	☐
21. I'm irritated a great deal more than people are aware of	☐	☐	☐	☐
22. I lose my temper	☐	☐	☐	☐
23. If someone annoys me, I'm apt to tell him or her how I feel	☐	☐	☐	☐
24. I control my angry feelings	☐	☐	☐	☐

MacArthur scale of subjective social status.

Each type of social status question includes a picture of a ladder with 10 possible rungs.

Social status vs. the United States

Think of this ladder as representing where people stand in the United States. At the top of the ladder are the people who are the best off--those who have the most money, the most education and the most respected jobs. At the bottom are the people who are the worst off

--who have the least money, least education, and the least respected jobs or no job. The higher up you are on this ladder, the closer you are to the people at the very top; the lower you are, the closer you are to the people at the bottom. Where would you place yourself on this ladder? Please fill in the circle on the rung where you think you stand at this time in your life, relative to other people in the United States.

Social status vs. your community

Think of this ladder as representing where people stand in their communities. People define community in different ways: Please define it in whatever way is most meaningful to you. At the top of the ladder are the people who have the highest standing in their community. At the bottom are the people who have the lowest standing in their community. Where would you place yourself on this ladder? Please fill in the circle on the rung where you think you stand at this time in your life, relative to other people in your community.

Social status vs. your social group

Think of this ladder as representing where people stand in their social group. People define social groups in different ways: Please define it in whatever way is most meaningful to you. At the top of the ladder are the people who have the highest standing in their social group. At the bottom are the people who have the lowest standing in their social group. In the box below, briefly define the social group you used when filling out this form: _____

Where would you place yourself on this ladder? Please fill in the circle on the rung where you think you stand at this time in your life, relative to other people in your social group.

Interpersonal Support Evaluation List

Each of these statements may or may not be true about you and your feelings about people around you. For each statement, please place an "X" in the circle under the response that best reflects your feelings.

	Definitely True	Probably True	Probably False	Definitely False
1. There are several people that I trust to help solve my problems.	☐	☐	☐	☐
2. If I needed help fixing an appliance or repairing my car, there is someone who would help me.	☐	☐	☐	☐
3. Most of my friends are more interesting than I am.	☐	☐	☐	☐
4. There is someone who takes pride in my accomplishments.	☐	☐	☐	☐
5. When I feel lonely, there are several people I could call and talk to.	☐	☐	☐	☐
6. There is no one that I feel comfortable going to for advice about very intimate problems.	☐	☐	☐	☐
7. I often talk with family or friends.	☐	☐	☐	☐
8. Most people I know think highly of me.	☐	☐	☐	☐
9. If I needed a ride to the airport very early in the morning, I would have a hard time finding anyone to take me.	☐	☐	☐	☐
10. I feel like I'm not always included by my circle of friends.	☐	☐	☐	☐
11. There is really no one who can give me an objective view of how I'm handling my problems.	☐	☐	☐	☐
12. There are several different people with whom I enjoy spending time with.	☐	☐	☐	☐

Interpersonal Support Evaluation List (continued).

	Definitely True	Probably True	Probably False	Definitely False
13. I think that my friends feel that I'm not very good at helping them solve problems.	☐	☐	☐	☐
14. If I were sick and needed someone (friend, family member, or acquaintance) to drive me to the doctor, I would have trouble finding someone.	☐	☐	☐	☐
15. If I wanted to go out of town (e.g., to the mountains, beach, or country) for the day I would have a hard time finding someone to go with me.	☐	☐	☐	☐
16. If I needed a place to stay for a week because of an emergency (for example, water or electricity out in my apartment or house), I could easily find someone who would put me up.	☐	☐	☐	☐
17. I feel that there is no one with whom I can share my most private worries and fears with.	☐	☐	☐	☐
18. If I were sick, I could easily find someone to help me with my daily chores.	☐	☐	☐	☐
19. There is someone I can turn to for advice about handling problems with my family.	☐	☐	☐	☐
20. I am as good at doing things as most other people are.	☐	☐	☐	☐
21. If I decide one afternoon that I would like to go to a movie that evening, I could find someone to go with me.	☐	☐	☐	☐
22. When I need suggestions on how to deal with a personal problem I know someone I can turn to.	☐	☐	☐	☐

Interpersonal Support Evaluation List (continued).

	Definitely True	Probably True	Probably False	Definitely False
23. If I needed a quick emergency loan of $100, there is someone (friend, relative, or acquaintance) I could get it from.	☐	☐	☐	☐
24. In general, people don't have much confidence in me.	☐	☐	☐	☐
25. Most people I know don't enjoy the same things that I do.	☐	☐	☐	☐
26. There is someone I could turn to for advice about making career plans or changing my job.	☐	☐	☐	☐
27. I don't often get invited to do things with others.	☐	☐	☐	☐
28. Most of my friends are more successful at making changes in their lives than I am.	☐	☐	☐	☐
29. If I had to go out of town for a few weeks, it would be difficult to find someone who would look after my house or apartment (the plants, pets, garden, etc.).	☐	☐	☐	☐
30. There is really no one I can trust to give me good financial advice.	☐	☐	☐	☐
31. If I wanted to have lunch with someone, I could easily find someone to join me.	☐	☐	☐	☐
32. I am more satisfied with my life than most people are with theirs.	☐	☐	☐	☐
33. If I got stranded 10 miles out of town, there is someone I could call to come and get me.	☐	☐	☐	☐
34. No one I know would throw a birthday party for me.	☐	☐	☐	☐
35. It would be difficult to find someone who would lend me their car for a few hours.	☐	☐	☐	☐

Interpersonal Support Evaluation List (continued).

36. If a family crisis arose, it would be difficult to find someone who could give me good advice about how to handle it.	☐	☐	☐	☐
37. I am closer to my friends than most other people are to theirs.	☐	☐	☐	☐
38. There is at least one person I know whose advice I really trust.	☐	☐	☐	☐
39. If I needed some help in moving to a new home, I would have a hard time finding someone to help me.	☐	☐	☐	☐
40. I have a hard time keeping pace with my friends.	☐	☐	☐	☐

Life Orientation Test – Revised

Please indicate the extent to which you agree or disagree with each of the statements below. Please be as honest and accurate as you can; do not let your responses to one question influence your responses to other questions. There are no right or wrong answers.

	Strongly disagree	Disagree	Neutral	Agree	Strongly Agree
1. In uncertain times, I usually expect the best.	☐	☐	☐	☐	☐
2. It's easy for me to relax.	☐	☐	☐	☐	☐
3. If something can go wrong for me, it will.	☐	☐	☐	☐	☐
4. I'm always optimistic about my future.	☐	☐	☐	☐	☐
5. I enjoy my friends a lot.	☐	☐	☐	☐	☐
6. It's important for me to keep busy.	☐	☐	☐	☐	☐
7. I hardly ever expect things to go my way.	☐	☐	☐	☐	☐
8. I don't get upset too easily.	☐	☐	☐	☐	☐
9. I rarely count on good things happening to me.	☐	☐	☐	☐	☐
10 Overall, I expect more good things to happen to me than bad.	☐	☐	☐	☐	☐

Perceived Stress Scale

The questions in this scale ask you about your feelings and thoughts DURING THE LAST WEEK. In each case, please indicate by filling in the circle beside the statement that best represents how often you felt or thought a certain way.

	Never	Almost never	Sometimes	Fairly often	Very often
1. In the last week, how often have you been upset because of something that happened unexpectedly?	☐	☐	☐	☐	☐
2. In the last week, how often have you felt you were unable to control the important things in your life?	☐	☐	☐	☐	☐
3. In the last week, how often have you felt nervous and "stressed?"	☐	☐	☐	☐	☐
4. In the last week, how often have you felt confident about your ability to handle your personal problems?	☐	☐	☐	☐	☐
5. In the last week, how often have you felt that things were going your way?	☐	☐	☐	☐	☐
6. In the last week, how often have you felt that you could not cope with all the things that you had to do?	☐	☐	☐	☐	☐

Perceived Stress Scale (continued).

	Never	Almost never	Sometimes	Fairly often	Very often
7. In the last week, how often have you been able to control irritations in your life?	☐	☐	☐	☐	☐
8. In the last week, how often have you felt that you were on top of things?	☐	☐	☐	☐	☐
9. In the last week, how often have you been angered because of things that were outside of your control?	☐	☐	☐	☐	☐
10. In the last week, how often have you felt difficulties piling up so high that you could not overcome them?	☐	☐	☐	☐	☐

Positive and Negative Affect Schedule, state version

Administered in Packets 1,2,5,6,7,8

This scale consists of a number of words that describe different feelings and emotions. Read each item and then fill in the circle that indicates how you feel RIGHT NOW, THAT IS, AT THE PRESENT MOMENT.

	Very slightly or not at all	A little	Moderately	Quite a bit
Interested	☐	☐	☐	☐
Distressed	☐	☐	☐	☐
Excited	☐	☐	☐	☐
Upset	☐	☐	☐	☐
Strong	☐	☐	☐	☐
Guilty	☐	☐	☐	☐
Scared	☐	☐	☐	☐
Hostile	☐	☐	☐	☐
Enthusiastic	☐	☐	☐	☐
Proud	☐	☐	☐	☐
Irritable	☐	☐	☐	☐
Alert	☐	☐	☐	☐
Ashamed	☐	☐	☐	☐
Inspired	☐	☐	☐	☐
Nervous	☐	☐	☐	☐
Determined	☐	☐	☐	☐
Attentive	☐	☐	☐	☐
Jittery	☐	☐	☐	☐
Active	☐	☐	☐	☐
Afraid	☐	☐	☐	☐
Extremely	☐	☐	☐	☐

Spielberger State-Trait Anxiety Inventory, state version

Administered in Packets 1,2,5,6,7,8

A number of statements which people have used to describe themselves are given below. Read each statement and then fill in the circle to the right of the statement that most appropriately indicates how you feel right now, at this moment. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best.

	Not at all	Somewhat	Moderately	Very much
1. I feel calm.	☐	☐	☐	☐
2. I feel secure.	☐	☐	☐	☐
3. I am tense.	☐	☐	☐	☐
4. I am regretful.	☐	☐	☐	☐
5. I feel at ease.	☐	☐	☐	☐
6. I feel upset.	☐	☐	☐	☐
7. I am presently worrying over possible misfortunes.	☐	☐	☐	☐
8. I feel rested.	☐	☐	☐	☐
9. I feel anxious.	☐	☐	☐	☐
10. I feel comfortable.	☐	☐	☐	☐
11. I feel self-confident.	☐	☐	☐	☐
12. I feel nervous.	☐	☐	☐	☐
13. I am jittery.	☐	☐	☐	☐
14. I feel “high strung”	☐	☐	☐	☐
15. I am relaxed.	☐	☐	☐	☐
16. I feel content.	☐	☐	☐	☐
17. I am worried.	☐	☐	☐	☐
18. I feel over-excited and “rattled.”	☐	☐	☐	☐
19. I feel joyful.	☐	☐	☐	☐
20. I feel pleasant.	☐	☐	☐	☐

Packet 2, administered after tape-stripping. Contents include:

- Tape stripping comfort questions:

  Please indicate on the scale below how discomforting/painful you found the tape stripping:

  No Pain Mild Discomforting Distressing Horrible Excruciating

- PANAS-2
- STANX-2

Packet 3, administered after instructions for the task. Contents include:

- Perceived threat and control appraisals (Tomaka, Blascovich, Kelsey, & Leitten, 1993) :

  1. On a scale of 1-7, with 1 being the least threatening and 7 being the most threatening, how threatening do you expect the upcoming task to be?

  2. On a scale of 1-7, with 1 being the least able and 7 being the most able, how able are you to cope with this task?

Packet 4, administered after preparation period. Contents include:

- Perceived threat and control appraisals:

  1. On a scale of 1-7, with 1 being the least threatening and 7 being the most threatening, how threatening do you expect the upcoming task to be?

  2. On a scale of 1-7, with 1 being the least able and 7 being the most able, how able are you to cope with this task?

- Self-statements during Public Speaking (Hofmann & DiBartolo, 2000)

Self-statements during Public Speaking

Instructions: How much do you agree with the statements given below?

Please rate the degree of your agreement on a scale between 0 (if you do not agree at all) to 5 (if you agree extremely with the statement).

1. What do I have to lose, it's worth a try.
2. I'm a loser.
3. This is an awkward situation but I can handle it.
4. A failure in this situation would be more proof of my incapacity.
5. Even if things don't go well, it's no catastrophe.
6. I can handle everything.
7. What I say will probably sound stupid.
8. I'll probably "bomb out" anyway.
9. Instead of worrying I could concentrate on what I want to say.
10. I feel awkward and dumb; they're bound to notice.

Packet 5, administered after the task is complete. Contents include:

- Post-task appraisals (Breier et al., 1987)
- PANAS-3
- STANX-3
- Interpersonal Adjective Checklist – Revised, (Wiggins, Trapnell, & Phillips, 1988)

Post-task appraisals

The following items deal with your experience with the task you just completed. The first items inquire about feelings you have about your performance of the task.

1. How stressful did you find performing the previous task?

(1 = Not at all stressful, 7 = Extremely stressful)

2. How satisfied are you with your performance on the task?

(1 = Not at all, 7 = Extremely satisfied)

3. How much control did you feel you had during the performance of the task?

(1 = No control, 7 = Full control)

4. To what degree did you experience feelings of helplessness during your performance of the task?

(1 = No feelings of helplessness, 7 = Extreme feelings of helplessness)

This section should be completed for those doing the TSST session only:

5. How stressful did you find the math task?

(1 = Not at all stressful, 7 = Extremely stressful)

6. How much control did you feel you had during the math task?

(1 = No control, 7 = Full control)

7. To what degree did you experience feelings of helplessness during the math task?

(1 = No feelings of helplessness, 7 = Extreme feelings of helplessness)

Interpersonal Adjective Checklist – Revised

Directions: The following is a list of adjectives. Please rate the accuracy of each adjective in describing the person who was with you before your speech.

(0 = Extremely inaccurate, 8 = Extremely accurate)

1. Accommodating	17. Dominant	33. Outgoing	49. Unauthoritative
2. Antisocial	18. Domineering	34. Perky	50. Unbold
3. Assertive	19. Enthusiastic	35. Persistent	51. Uncalculating
4. Bashful	20. Extraverted	36. Ruthless	52. Uncharitable
5. Boastful	21. Firm	37. Self-assured	53. Uncheery
6. Boastless	22.Forceful	38. Self-confident	54. Uncrafty
7. Calculating	23. Forceless	39. Shy	55. Uncunning
8. Charitable	24. Friendly	40. Sly	56. Undemanding
9. Cheerful	25. Gentlehearted	41. Softhearted	57. Unneighborly
10. Cocky	26. Hardhearted	42. Sympathetic	58. Unsly
11. Coldhearted	27. Introverted	43. Tender	59. Unsociable
12. Crafty	28. Ironhearted	44. Tenderhearted	60. Unsparkling
13. Cruel	29. Jovial	45. Timid	61. Unsympathetic
14. Cunning	30. Kind	46. Tricky	62. Unwily
15. Dissocial	31. Meek	47. Unaggressive	63. Warmthless
16. Distant	32. Neighborly	48. Unargumentative	64. Wily

Packet 6, administered 1 h after tape-stripping. Contents include:

- PANAS-4
- STANX-4
- Unsupportiveness Social Interactions Inventory (Ingram, Betz, Mindes, Schmitt, & Smith, 2001)

Unsupportive Social Interactions Inventory

Instructions: The following is a list of possible responses the person with you before your speech may have used when talking with you. Please rate each item by indicating how much of the response you received, on a scale from 0 (none) to 4 (a lot).

1. "I told you so" or similar comment.
2. "Should or shouldn't have" comments about my role in the speech.
3. Asked "why" questions about my role in the speech.
4. Blaming me, trying to make me feel responsible for the speech.
5. Changed the subject before I wanted to.
6. Did not seem to know what to say, or seemed afraid of saying or doing the "wrong" thing.
7. Did not seem to want to hear about it.
8. Did things for me that I wanted to do and could have done myself.
9. Discouraged me from expressing feelings such as anger, hurt, or sadness.
10. Felt that I should focus on the present or the future and that I should forget about what has happened and get on with my life.
11. Felt that I should stop worrying about the event and just forget about it.
12. Felt that I was overreacting.
13. Felt that it could have been worse or was not as bad as I thought.
14. From voice tone, expression, or body language, I got the feeling he or she was uncomfortable talking about it.
15. Refused to provide the type of support I was asking for.
16. Refused to take me seriously.
17. Responded with uninvited physical touching (e.g., hugging).
18. Said I should look on the bright side.
19. Seemed disappointed in me
20. Seemed to be telling me what he or she thought I wanted to hear.
21. Told me that I had gotten myself in the situation in the first place, and now must deal with the consequences.
22. Told me to be strong, to keep my chin up, or that it should not let it bother me.
23. Tried to cheer me up when I was not ready to.
24. When I was talking about it, person didn't give me enough time, or made me feel like I should hurry.

Packet 7, administered 1.5 h after tape-stripping. Contents include:

- PANAS-5
- STANX-5
- Psychiatric Epidemiological Research Inventory Life Events Scale (Dohrenwend, Krasnofff, Askenasy, & Dohrenwend, 1978)

Psychiatric Epidemiological Research Inventory Life Events Scale

Below are questions about a number of events that commonly happen in people's lives. Each question is concerned with whether an event has happened to you and, in some cases, your spouse during the last 12 months. For several of the events, the questions we ask below may remind you of rather painful feelings. They are, however, extremely important to people when they do happen, so please try to answer.

1. Have you moved during the last 12 months? Yes No

    (IF YES) Overall, would you say that your moving was a good or bad experience?

    Very good/moderately good/slightly good/slightly bad/moderately bad/very bad

2. Did someone you were close to die within the last 12 months? Yes No

    (IF YES) Who? (more than one response is possible)

    Spouse or intimate friend/brother or sister/parent/child/spouse's parent/friend /other relative

3. Did you break up with a close friend during the last 12 months? Yes No

    (IF YES) Overall, would you say your break up was a good or bad experience?

    very good/moderately good/slightly good/slightly bad/moderately bad/very bad

4. Have you had any important relationship, for example, with your spouse or a close friend, get significantly worse during the last 12 months? (This should not include the relationship referred to in item 3 above.) Yes/No

(IF YES) With whom? (more than one response is possible)

Boss/parent/friend/child/spouse/other relative

5. Have you, a very close friend, or close family member had an accident that required emergency medical treatment during the last 12 months? Yes No

(IF YES) Who? (more than one response is possible.)

You/spouse or intimate friend/brother or sister/parent/child/spouse's parent/ other relative/ friend

6. Have you, a very close friend, or close family member been hospitalized for a serious (life threatening) illness during the last 12 months? Yes No

(IF YES) Who? (more than one is possible.)

You/spouse or intimate friend/brother or sister/parent/child/spouse's parent/ other relative/ friend

7. Have you or your spouse retired, lost or changed jobs, or been involuntarily unemployed during the last 12 months? Yes No

b. (IF YES) Who?

Your spouse/both of you

c. How would you rate your feelings about leaving your job (or your spouse leaving his/her job)? If both of you lost or changed jobs, answer only for you.

Very good/moderately good/slightly good/slightly bad/moderately bad/very bad

8. During the last 12 months, have you or your spouse suffered a significant business or investment loss, or has a business you owned failed?

(IF YES) Who?

You/spouse/both of you

9. During the last 12 months, have you or your spouse had any serious problems or disappointments at work or in an educational course (university, training program, etc.)? (IF YES) Who?

You/spouse/both of you

10. Has there been a significant change in your personal finances during the last 12 months? Yes/No

(IF YES) Has the change been for the better or worse?

Better/Worse

11. Has your house been broken into and/or burglarized during the last 12 months?

Yes/No

12. Have you or your spouse or other member of your immediate family been assaulted or mugged during the last 12 months? Yes/No

(IF YES) Who? (more than one answer is possible.)

You/brother or sister/spouse/child/parent/other

13. Has the behavior of any member of your family been a significant problem for you during the last 12 months? Yes/No

(IF YES) Who? (more than one answer is possible.)

Spouse/brother or sister/child/parent

14. Have you or your spouse had to appear in court during the last 12 months as either a defendant, a witness in a criminal case, or as a party to a suit? Yes/No

b. (IF YES) Who?

You/spouse/both of you

c. How would you rate the court experience?

Very good/moderately good/slightly good/slightly bad/moderately bad/very bad

15. Have you had a pet (animal) to whom you were attached, die, get lost, or had to give it away during the last 12 months? Yes/No

16. Other than the events we have already asked about, have any other important things happened to you during the last 12 months that made that period significantly different from a typical year? Yes/No

(IF YES) You can list up to three events. Please do not feel obliged to include an additional event or events unless they were significant.

Event 1: To whom?

Spouse/brother or sister/parent/child/friend/other relative/you

What happened?

How would you rate your feelings about this event?

Very good/moderately good/slightly good/slightly bad/moderately bad/very bad

17. Event 2: To whom? What happened?

How would you rate your feelings about this event?

18. Event 3: To whom? What happened?

How would you rate your feelings about this event?

Packet 8, administered at the end of the session. Contents include:

- PANAS-6
- STANX-6
- Recent health behaviors questionnaire
- Pittsburgh Sleep Quality Index (Buysse, Reynolds III, Monk, Berman, & Kupfer, 1989)

Recent health behaviors questionnaire

1. If there has been any change in your weight in the last week, please indicate.

   ____ pounds lost ____ pounds gained

   Are you trying to lose or gain weight? No/Yes

2. How many alcoholic drinks have you had in the last seven days? ___ alcoholic drinks

3. How many alcoholic drinks have you had in the last 48 hours? ___ alcoholic drinks in the last 24 hours? ___ alcoholic drinks

4. How many caffeinated beverages (coffee, tea, soda pop) have you had in the last 24 hours? ___ total caffeinated beverages

5. Do you currently take birth control pills or do you currently receive birth control skin patches? No/Yes. If YES, please list medication and usage:

6. What time did you last eat? : _____ a.m. p.m.

   If less than two hours ago, what did you eat?:

   OR ____ I ate more than two hours ago.

Pittsburgh Sleep Quality Index

1. During the past month, when have you usually gone to bed at night? _____

2. During the past month, how long (in minutes) has it usually taken you to fall asleep each night? ____

3. During the past month, when have you usually gotten up in the morning? _____

4. During the past month, how many hours of *actual sleep* did you get at night? (This may be different from the amount of hours you spend in bed.) _____

5. During the past month, how often have you had trouble sleeping because you...

	Not during the past month	Less than once a week	Once or twice a week	Three or more times a week
(a) Cannot get to sleep within 30 minutes	☐	☐	☐	☐
(b) Wake up in the middle of the night or early morning	☐	☐	☐	☐
(c) Have to get up to use the bathroom	☐	☐	☐	☐
(d) Cannot breathe comfortably	☐	☐	☐	☐
(e) Cough or snore loudly	☐	☐	☐	☐
(f) Feel too cold	☐	☐	☐	☐
(g) Feel too hot	☐	☐	☐	☐
(h) Had bad dreams	☐	☐	☐	☐
(i) Have pain	☐	☐	☐	☐
(j) Other reason(s), please describe:				
How often have you had trouble sleeping because of the problem(s) you listed in (j)?	☐	☐	☐	☐

6. During the past month, how would you rate your sleep quality overall?

Very good/Fairly good/Fairly bad/Very bad

Pittsburgh Sleep Quality Index (continued).

	Not during the past month	Less than once a week	Once or twice a week	Three or more times a week
7. During the past month, how often have you taken medicine (prescribed or "over the counter") to help you sleep?	☐	☐	☐	☐
8. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activities?	☐	☐	☐	☐

9. During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?

No problem at all/Only a very slight problem/Somewhat of a problem/A very big problem