INTEGRATING SOCIAL RELATIONSHIP DYNAMICS INTO THE ASSOCIATION
BETWEEN EMOTIONAL DISTURBANCES AND PHYSICAL HEALTH: A STRESS
PSYCHOPHYSIOLOGICAL APPROACH

A Dissertation in

Biobehavioral Health

by

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Abstract

Interpersonal stress from conflictive social interactions and emotional disturbances are robust psychosocial predictors of physical health. However, there has been little research on the joint effects of social relationships and emotion on health. The current dissertation provided two theoretical models to explain biopsychosocial mechanisms by which emotional factors and social dynamics interact and relate to affect physical health outcomes, including general health status, inflammation, and sleep.

The first study of this dissertation involved an examination of the effects of having multiple negative social relationships on inflammatory cytokine responses to stress and whether trait hostility aggravates the effects of negative relationships over and above effects of depressed mood. Participants were healthy young adults who were challenged with a laboratory stress protocol to measure physiological responses after stress; they were also asked about negativity in their relationships with a romantic partner, roommates, friends, and family as well as levels of trait hostility and depressed mood. Results showed that those who reported negativity in multiple social relationships showed greater IL-6 and TNF-α responses to stress; they also had higher mean IL-6 across time, and less reactivity in systolic and diastolic blood pressure. Differences in mean IL-6 and diastolic blood pressure (but not changes in IL-6 or TNF-α to stress) were accounted for by either depressed mood or hostility. Overall, this research indicates that having multiple negative relationships may exacerbate acute inflammatory responses to a laboratory stressor.

The second study focused on the relationship with a romantic partner and how negative and positive emotional states may interact with couple interactions to affect sleep of chronic pain patients. Knee osteoarthritis patients and their partners were assessed for their daily positive and negative emotional states, three different partner responses to pain behaviors (empathic, punishing or solicitous), and subjective sleep quality across 14 consecutive days. Results
demonstrated that higher daily negative emotional states and lower positive emotional states were associated with poorer sleep of patients. Interactions between emotional states and partner solicitous responses were observed, such that higher solicitousness strengthened the association between both negative and positive emotional states and sleep. Thus, the second study demonstrated that day to day fluctuations in negative and positive emotional states can interact with partner behaviors to affect nightly sleep quality among knee osteoarthritis patients.

The third study examined whether different aspects of negativity in marital relationships mediate the association between depressive symptoms and physical health. Adults with a married spouse or partner were asked about their depressive symptoms at baseline, three indicators of negativity in their relationship (marital risk, spousal emotional strain, and marital disagreement) at 9 year follow-up, and physical health outcomes at 10 year follow-up, including three markers of inflammation (CRP, IL-6, and sIL-6r), sleep quality, and numbers of physical symptoms and chronic conditions. The results showed that depressive symptoms at Time 1 were associated with greater marital risk at Time 2 and lower sleep quality at Time 3, and that marital risk and sleep quality in turn were associated with greater number of physical symptoms and chronic conditions at Time 3. Age and gender moderations for these associations were observed. These results demonstrate that impaired marital relationships and sleep can explain the processes by which depressive symptoms aggravate physical health over time.

Findings connect the emotion and social relationship literatures and explain how those processes are closely connected to affect physical health. Applying the stress amplifying model of social relationship and stress generation model of depression, this dissertation found specific pathways for how social interactions can aggravate the impact of emotional disturbances on health and how emotional disorders may have long lasting effects on health by impairing the marital relationship.
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Chapter 1

Introduction and Background: Interpersonal relationship processes, emotion, and health

Interpersonal stress and trait/state emotion are the most robust psychosocial predictors of physical health status (Kiecolt-Glaser, Gouin, & Hantsoo, 2010; Pressman & Cohen, 2005; Raison & Miller, 2003; Uchino, 2006). Social integration, levels of social support, and social conflict are known to affect cardiovascular, endocrine, and immune functioning as well as morbidity and mortality (for a review see, Cohen & Janicki Deverts, 2009). A separate body of literature shows that mood disorders and negative mood states are much more prevalent among individuals with physical illness – including cardiovascular, gastrointestinal, autoimmune diseases, and chronic pain conditions (for a review, see Balon, 2006; Härter, Conway, & Merikangas, 2003) and mood disorders and negative mood predict pain, other symptoms and adverse outcomes associated with those types of diseases (Balon, 2006; Robinson & Riley, 1999).

Despite the seeming influence of both social relationship processes and affective states on physical health, there has been little research on the joint effects of social relationships and emotion on health. Such research is important because social relationships and mood are intimately intertwined and likely interact to predict health. For instance, social relationships are critically affected by an individual’s emotional status and by symptoms of mood disorders (Joiner, 2000). Also, social interactions often engender specific emotions, and the expression and regulation of those emotions is shaped by social contexts (Clore & Robinson, 2012). The current dissertation provides two theoretical models (Figure 1.1 & 1.2) to explain biopsychosocial mechanisms by which emotional factors and social dynamics relate to affect physical health outcomes and health behaviors.
This dissertation is expected to fill in gaps in the literature on social relationships, emotion, and health in several significant ways. Previous literature has demonstrated that higher perceived social support (from social relationships) and lower levels of social stress are associated generally with better health status (Uchino, Cacioppo, & Kiecolt-Glaser, 1996). This dissertation aims to advance this literature by examining social relationship processes in a more detailed fashion than has typically been done. For example, I will examine social interactions in multiple relationship areas (e.g., relationships with a romantic partner, family, and friends) and how negative social interactions across relationship types can have aggregated effects on health. Further, few studies have examined how physiological factors can act as mechanisms of an interaction between social relationships and emotional tendencies on physical health outcomes (Slavich, O’Donovan, Epel, & Kemeny, 2010). Thus, another way in which this dissertation will bridge gaps in the existing social relationship and emotion literature is by examining how the stress response system connects emotional distress and social relationship processes with health outcomes. Lastly, much of the current literature takes the view that relationships between social relationships, emotion, and health are bi-directional and are generally interconnected with each other; although this may be true, it hinders testing specific pathways in the relationships and their directionality. The two theoretical models in this dissertation provide a set of testable hypotheses that specify directionality and specific pathways linking emotional factors, social relationship processes, and health outcomes.

**Health outcomes of this dissertation**

Among various indicators of health, this dissertation will focus on inflammation, sleep, and general physical health status. Inflammation has received considerable empirical attention due to its critical role in many chronic diseases, such as cardiovascular diseases, cancer, type II diabetes, arthritis, Alzheimer’s disease, and osteoporosis (Aggarwal, Shishodia, Sandur, Pandey, & Sethi, 2006; Ershler & Keller, 2000; Kiecolt-Glaser et al., 2010). Growing evidence suggests that not just immunological stimuli
but also psychosocial and behavioral factors can trigger processes that lead to chronic inflammation. Depression and social stress have been shown to be the most robust psychosocial predictors for chronic inflammatory status (Kiecolt-Glaser et al., 2010). The detrimental effects of depression and social stress on health status are apparent among those with chronic illness. For example, disability due to chronic pain condition is directly influenced by negative affect of those with chronic pain conditions (Robinson & Riley, 1999).

Sleep is closely linked with inflammation as sleep is one the most critical behavioral factors for chronic inflammation. Previous studies have shown that sleep deprivation is associated with changes in inflammatory processes, indicated by increased numbers of monocytes and elevated levels of circulating interleukin-6 (IL-6) and tumor necrosis factor’s soluble receptor p55 immediately after experimentally induced sleep deprivation of healthy adults (for a review, see Mullington, Hinze-Selch, & Pollmächer, 2001). Both short and longer durations of sleep are associated with a vast range of health problems, including increased pain sensitivity, and fatigue, and prospectively predicted all-cause mortality according to a meta-analysis involving 1,382,999 participants from 8 different countries (Cappuccio, D'Elia, Strazzullo, & Miller, 2010). Thus, this dissertation examines inflammatory markers and sleep quality as important health indicators, especially in the context of adults living with and without chronic disease conditions, in addition to perceived health.

**Stress response system: Potential physiological mechanisms**

Psychological stress can activate inflammatory response system even in the absence of infection or injury (for reviews, see Maes, 2001; Steptoe, Hamer, & Chida, 2007). For example, Steptoe and colleagues showed that circulating serum IL-6 was increased after 2 hours from a laboratory induced psychological stress among healthy adults (Steptoe, Willemsen, Owen, Flower, & Mohamed-Ali, 2001); in contrast, individuals in a control (non-stress) condition of this study who watched nature video clips
instead of stress tasks did not show any increase in circulating inflammatory markers over time. The stress response system is considered to be a key physiological mechanism that can explain how social relationships and emotional experience “get under the skin” to affect inflammation and health. The two major stress response systems – the Sympatho-Adrenal-Medullary (SAM) axis and the Hypothalamic-Pituitary-Adrenal (HPA) axis – are involved in inflammatory responses to a psychological stress.

Relevant to the SAM axis, norepinephrine is produced in the medulla of the adrenal gland in response to psychological stress and can activate production of nuclear factor kappa-B (NF-kB), which is a gene transcription factor for a range of pro-inflammatory cytokines (for review see, Kiecolt-Glaser et al., 2010). Glucocorticoids, such as CRH and cortisol, are produced in HPA axis and act to suppress the immune activation of leukocytes and to inhibit the production of cytokines and other inflammatory mediators (Chrousos, 2000). Although there are opposite actions of SAM and HPA axis, inflammation is typically inhibited after acute stress by the anti-inflammatory actions of glucocorticoids in healthy men and women.

Under conditions of chronic stress, the way in which the stress response system and inflammatory systems are connected can become dysregulated. One way that this can happen is that immune cells of individuals who experience chronic stress can develop resistance to the effects of glucocorticoids (Cohen et al., 2012). Inflammatory responses to stress of such individuals can be excessive and be prolonged compared to individuals without chronic stress (Kiecolt-Glaser et al., 2005; Kudielka, Buske-Kirschbaum, Hellhammer, & Kirschbaum, 2004; Miller, Chen, & Zhou, 2007; Miller, Cohen, & Ritchey, 2002). Social stressors are strongly linked with excessive inflammatory responses to acute laboratory stressors (for review, see Miller, Rohleder, & Cole, 2009; Steptoe et al., 2007). Together, previous studies suggest that emotional distress and social interactions contribute to health problems via dysregulated actions of the stress response system. Next, I will more thoroughly review the existing literature on the effects of social relationships and emotional factors on health.
Effects of social relationships on health

Substantial research has examined the role of social relationships in health in multiple ways. Reviewing the entire literature on social relationships and health is beyond the scope of this dissertation. Thus, this dissertation focuses on reviewing the literature relevant to the impact of social integration, loneliness, social support, and social conflicts on general health status and also on inflammation and sleep quality.

Social integration, loneliness, and health

One important indicator of social relationships that has been linked with health is social integration, which refers to the extent to which an individual is connected within a social network, including friends, family, and clubs or organizations in the community (Barrera, 1986). A wide range of epidemiological studies have reported that social integration is strongly associated with better physical health outcomes, including longevity, lower morbidity and mortality as well as better mental health status (for review see, Graham, Christian, & Kiecolt-Glaser, 2007; Seeman, 2001; Seeman, Lusignolo, Albert, & Berkman, 2001). The Alameda county study measured levels of social connectedness by how many relationships participants reported (e.g., a spouse, close friends, and relatives) and how many social activities they were engaged in, such as regular participation in church or other groups. At their 8 year follow-up, both men and women across all age groups showed an inverse association between social integration and mortality independent of baseline health status, functioning, and sociodemographic characteristics (Berkman & Breslow, 1983). Subsequent studies confirmed this finding among middle aged and older adults across the U.S. as well as with samples from Sweden and Finland (House, Robbins, & Metzner, 1982; Kaplan et al., 1988; Orth-Gomer & Johnson, 1987). Social integration is also associated with better prognosis after myocardial infarction (Williams et al., 1992).
In terms of inflammation, lower levels of IL-6 and C-reactive protein (both inflammatory markers) have been observed among older men with high levels of social integration (Ford, Loucks, & Berkman, 2006; Loucks, Berkman, Gruenewald, & Seeman, 2006; Sbarra, 2009). Genome wide association studies revealed that socially isolated individuals show underexpression of genes involved in anti-inflammatory responses and overexpression of genes involved in pro-inflammatory responses (Cole et al., 2007). Social isolated individuals appear to have less sensitive immunosuppressive reaction to glucocorticoids compared to less socially isolated individuals (Cole, 2008), indicating one possible physiological mechanism for their sustained inflammatory state.

**Loneliness and health**

“Feeling lonely” is different from an objective state of social isolation. Loneliness can be defined as feeling that one’s social needs are not satisfied by one’s current social relationships (Hawkley & Cacioppo, 2003). Recent studies have shown that loneliness predicts poor health outcomes, including poor immune functioning (Hawkley & Cacioppo, 2003). Loneliness measured prior to a mammogram was higher among women who were later diagnosed as having breast cancer, suggesting a potential link between loneliness and immune functioning (Fox, Harper, Hyner, & Lyle, 1994). Loneliness has also been associated recently with fibrinogen, which is a known blood coagulant that is involved in inflammatory processes related to vascular wall impairment and thus linked with cardiovascular disease (Ernst, 1994). Another study examined the independent effects of loneliness from social isolation using data from the English Longitudinal Study of Ageing found that lonely older adults were more likely to engage in multiple health risk behaviors including being inactive and smoking (Shankar, McMunn, Banks, & Steptoe, 2011). The study also found that social isolation was associated with greater levels of CRP and fibrinogen, which was mainly driven by the marital status of participants (Shankar et al., 2011). Greater increases in fibrinogen in response to a laboratory stressor were observed among middle aged adults.
(Steptoe, Owen, Kunz-Ebrecht, & Brydon, 2004). Thus, overall, a few studies suggest that there may be a higher inflammatory reactivity to psychological stress among lonely individuals.

Cacciopo and Hawkley examined underlying mechanisms that account for the association between perceived loneliness and health (2003). They found that stress and repair/maintenance systems explain the association between loneliness and health outcomes. Stress can explain the association between social isolation and health in two different ways. First, loneliness could be a long term consequence of insecure attachment in childhood or in adulthood. Insecure attachment would predispose individuals to be insecure, anxious, or suspicious about their social relationships and in turn produce more frequent activation of the SAM and HPA systems to a subtle cue of social strains. Although there are few studies that examine this mechanism, Caccioppo and colleagues examined the association between attachment style and physiological reactivity to stress. They reported that young adults who felt socially isolated were more likely to have insecure attachment styles; however, attachment styles were not significantly associated with autonomic and neuroendocrine measures of stress reactivity in this study (Cacioppo et al., 2000). Another way in which stress may explain the association between social isolation and health is that social isolation is itself is a stressor that causes negative affect and lessens feelings of self-worth (Cacioppo et al., 2002), which is in turn associated with SAM and HPA activation. A relationship between negative/positive affect and SAM/HPA axis has been reported (Pressman & Cohen, 2005; Solomon, 1969), but previous studies have not examined whether social isolation explains the activation of the stress response system via influencing levels of negative/positive affect.

**Social support and health**

Having reliable support from others is another aspect of social relationships that is typically thought to benefit health. Social support can be defined by “support provided by other people and [which is] accessible within interpersonal context” (Cooke, Rossmann, Hamilton, & Patterson, 1988). Social
support is associated with improved health from diverse health issues, including lower risk for cardiovascular diseases, cancer, and infectious illness (Uchino et al., 1996). Social support may improve health by mitigating the effects of adverse stress on immune function. Circulating levels of IL-6 were negatively associated with overall social support among elderly women (Friedman, 2010). People with a low level of social support have low levels of autonomic activity (e.g., low resting blood pressure), poor immunosurveillance (e.g., weak natural killer cell lysis), and higher basal level of stress hormones, compared to those with a high level of support (Uchino et al., 1996). Social support is especially critical for health when it provides comfort and resources in time of need. Social support also appears to be associated with better sleep quality among caregivers of a family member with dementia (Brummett et al., 2006). Pregnant women during their third trimester showed evidence of higher inflammation (via CRP) if they had low levels of social support compared to counterparts with high levels of support (Coussons-Read, Okun, & Nettles, 2007).

There are four broad forms of social support, reflecting different aspects of support provided: emotional support, instrumental support, informational support, and appraisal support (Cooke et al., 1988). A high level of intimacy combined with the frequent use of instrumental support—which refers to providing aid in kind, money, labor, or any direct help—in social relationships were associated with lower plasma IL-6, and lower expression of NF-κB among ovarian cancer patients (Lutgendorf et al., 2009). However, a substantial amount of research in individuals with chronic pain reports that even well intended support may lead to negative outcomes when it is more than what is needed or if it is not the needed kind of support (for review see, Leonard, Cano, & Johansen, 2006). Different types of social support have also been investigated for their impact on sleep quality. For example, instrumental support was associated with more sleep disturbances among HIV positive adults, whereas empathizing—a kind of emotional support of providing empathy, caring, love, trust, esteem, concern, and listening—was
associated with less sleep disturbance, suggesting that only certain kinds of support is beneficial for sleep of HIV positive adults. Overall, studies suggest that social support is associated with lower levels of inflammatory markers and better sleep quality among both healthy individuals and individuals with diseases.

**Social conflict and health**

Social relationships can also become a source of stress when they generate conflict and carry with them too many demands or criticism. Having conflicts within the family, such as constantly quarrelling parents, can lead offspring to develop vulnerability for mental and physical health problems (for review see, Repetti, Taylor, & Seeman, 2002). In marital relationships, low level of marital satisfaction and aversive marital interactions are associated with poor perceived health of couples (Kiecolt-Glaser & Newton, 2001). Social conflict is also linked with suppressed immune functioning as well as elevated inflammation. Cohen and colleagues showed that people with severe and enduring social conflicts are twice as likely to develop a cold after being exposed to a common cold virus compared to others without conflicts (Cohen et al., 1998a). Also, high levels of daily or general interpersonal stress were linked with elevated level of pro-inflammatory markers among both healthy adults and those with rheumatoid arthritis (Davis et al., 2008; G. E. Miller et al., 2009). The effects of social conflict appear to exist over and above effects of loneliness and lack of social network support, factors which have been linked with both of social conflict and immune function (for a review, see Hawkley, Bosch, Engeland, Marucha, & Cacioppo, 2007; Pressman et al., 2005).

There is also an acute effect of social conflict on inflammation. Experimental studies have induced a marital conflict by asking couples to discuss a provocative topic (as previously determined for them as couple) in a laboratory session to examine the acute effect of social conflicts. In a study by Kiecolt-Glaser and colleagues, puncture wounds were induced on the skin of participants, the production
of proinflammatory cytokines was measured at the wound site, and the speed of wound healing after the marital conflict session was assessed; Couples who showed hostile interactions during the marital conflict session had greater production of IL-6 in response to the skin wound as well as slower wound healing than those with less hostile interactions (Kiecolt-Glaser et al., 2005).

There is particularly strong evidence that chronic stress from relationships, such as social conflict that goes unresolved for an extended period of time, is associated with impaired immune function. Unresolved chronic social conflict has been associated with an over-exaggerated inflammatory cytokine profile (Friedman, 2010; Fuligni et al., 2009). In a study of female adolescents, although chronic social strain did not predict changes in circulating levels of IL-6 and CRP, it was associated with heightened reactivity in inflammatory signaling pathways (Miller, Maletic, & Raison, 2009); Specifically, chronic social stress in that study predicted greater increases in the gene products involved in inflammatory signals (i.e., messenger RNA of NF-κB and glucocorticoid receptor-α) and in an in-vitro test for IL-6 production of leukocytes to bacterial endotoxins [Lipopolysaccharide (LPS) stimulated production of IL-6] over a 6 month follow-up (A. H. Miller et al., 2009). Other studies of chronic stress have examined caregivers of chronic disease patients, such as those caring for individuals with cancer and Alzheimer’s’ disease; such caregivers show a faster rate of increase in inflammatory markers over years of their lifespan, compared to individuals in the same age group without caregiving burden (Kiecolt-Glaser et al., 2003). Caregivers of children with pediatric cancer showed reduced ability to inhibit LPS stimulated IL-6 production via the actions of glucocorticoids, suggesting that reduced sensitivity of neuroendocrine system to inflammatory signals is a possible physiological mechanism underlying the chronic inflammation among caregivers (Miller et al., 2002). Overall, these studies suggest that there may be a dynamic interaction between social and immunological challenges that influences inflammation-related pathogenesis.
Social conflict may also affect health via affecting sleep. Interestingly, there are few studies that have examined how social conflict with a romantic partner affects sleep quality, even though majority of adults in the U.S. sleep with a significant other (Troxel, Robles, Hall, & Buysse, 2007). However, experiencing daily or overall higher levels of interpersonal conflict in general is associated with greater sleep disturbances among healthy adults (Brissette & Cohen, 2002; Fortunato & Harsh, 2006). Thus, it seems likely that social conflicts either directly aggravate physical health by provoking acute or chronic dysregulations in the immune system or indirectly exacerbate physical health by inducing sleep disturbances among adults.

**Emotion and health**

**Definitions of emotion related constructs**

There is lack of consistency in defining emotion related constructs in the existing literature. Prior to my review of the literature linking emotion and health, I provide here distinctions in key constructs, such as the distinction between state and trait affect as well as basic affect and cognitive-affective orientation.

Basic affect refers to a set of universal emotions, such as anger, fear, and happiness. Each kind of basic affect is known to exist across cultures and is recognizable by its distinctive characteristics such as arousal, valence, facial expression, and motivational set (Ekman, 2005). State affect refers to relatively short-term bouts of emotional experience whereas trait affect can be defined as a stable individual characteristic for one’s typical level of a certain emotion (Pressman & Cohen, 2005). Affect can be characterized by its valence (i.e. positive or negative affect), and it is known that positive affect is not a mere absence of negative affect but should be considered as an independent category of affect (Diener & Emmons, 1984). Although some distinguish between the terms “emotion”, “affect”, and “mood” in terms
of duration and whether it was generated by a specific cause, the three terms will be used interchangeably in this dissertation due to lack of consistent use of the three terminologies in the literature.

Importantly, basic affect can be expanded to include cognitive-affective orientation to describe more complicated emotional tendencies of individuals (Izard, 2007; Mayne, 1999). Cognitive-affective orientation is an elaborated set of cognitions and behaviors that are associated with basic affect like fear, anger, and sadness. Leventhal and Scherer (1987) proposed that basic affect becomes associated with memories and behavioral repertoires over a lifetime and thus be consolidated into cognitive-affective orientation involving distinctive way of emotional and cognitive processes. For example, hostility can be defined as a cognitive-affective orientation originated from anger and involve cognitive framework of “enduring attitude of ill will and negative view of others”. Also, depression can be viewed as “internal, global, and stable negative attributions and cognitive framework that abets faulty information processing [that results in a sense of self worthlessness]” (Beck & Alford, 2009). Thus, although depression and hostility cannot be considered emotions, they are strongly linked with consistent emotional tendencies.

**The impact of emotion on physical health outcomes**

Both negative and positive affect have been reliably associated with health outcomes. Negative emotion is associated with greater risk of major chronic diseases, such as coronary heart disease and cancer, and increased mortality among middle aged and older adults (Gallo & Matthews, 2003; Wilson, Bienias, Mendes de Leon, Evans, & Bennett, 2003). Specifically, anger is one of the strongest psychosocial risk factors for coronary heart disease and premature death even after controlling for baseline health conditions and demographic risk factors such as age and SES (Smith, Glazer, Ruiz, & Gallo, 2004). Levels of depression and anxiety predict future coronary events and all-cause mortality and appear to have a dose response effect on mortality risk (Gallo & Matthews, 2003).
Benefits of expressing negative emotion

Experiencing negative affect does not necessarily result in adverse health outcomes. Indeed, Mayne and other colleagues (1999) suggest that negative affect can exert health benefits when it is properly expressed. For example, the benefit of expressing negative affect has been extensively examined among cancer patients and those with HIV sero-positive status. Anger expression appeared to mediate the relationship between optimism and better immune function of prostate cancer patients, as measured by natural killer cell cytotoxicity (Penedo et al., 2006). Among HIV sero-positive individuals, those who showed greater emotional and physiological arousal during an experimentally induced emotional recall session had better NK cell function and survived longer than non-reactive individuals (O’ Leary, Temoshok, Sweet, & Jenkins, 1989). In a similar vein, the health benefits of talking or writing about the most upsetting and traumtic experience of one’s life have been examined in a series of studies stemming from research by Pennebaker and colleagues (1990). A meta-analysis for the effects of expressive writing confirmed that healthy adults who expressed negative affect associated with the traumatic experiences showed better mental and physical health status than those who were in the control condition (Smyth, 1998). A randomized clinical trial on the effects of expressive writing among patients with rheumatoid arthritis and asthma showed that the benefits of expressive writing on reductions in disease severity from baseline to 4 months follow-up (Smyth, Stone, Hurewitz, & Kaell, 1999). Despite the known benefits of emotional expression on overall health, previous studies showed that cancer patients often report lower levels of anger than their healthy counterparts of comparable age and gender but tend to suppress their expression of anger when they feel it (Thomas et al., 2000). All in all, previous research suggests that rather than mere experience of negative emotion, individuals’ inability to properly express and regulate the negative emotion may be linked to adverse physical health outcome. Thus, it would be meaningful to
examine the social contexts when individuals express and regulate their emotion to further specifying the process by which negative emotional experiences affect physical health.

**Positive emotion and health**

The impact of emotion on health is not limited to negative emotion. Recent studies have demonstrated the benefit of positive affect on health. A meta-analysis suggests that positive affect is associated with decreased mortality among community residing older adults after controlling for age, socioeconomic status, and baseline health status (Pressman & Cohen, 2005). Also, high levels of positive affect predict a lower risk of developing coronary artery diseases, infectious illness, and injuries among adults, as well as a lower likelihood of rehospitalization after heart attack (Pressman & Cohen, 2005).

Interestingly, lab induced positive affect with acute arousal (e.g., excitement) appears to be result in worse pulmonary functioning among asthma patients and more bowel mobility among patients with irritable bowel syndrome than neutral mood, suggesting that short-term inducement of positive affect may be related to short-term negative consequences in certain circumstances (Ritz, George, & Dahme, 2000; Whorwell, Houghton, Taylor, & Maxton, 1992).

**Emotion and immune function**

Do positive and negative affect have a direct effect on immune functioning? Both positive and negative affect can provoke immune activation indicated by production of secretory immunoglobulin and increases in peripheral white blood cells (Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002). Previous studies have shown that negative affect is often linked with increased levels of inflammatory markers, such as CRP and pro-inflammatory cytokines. A meta-analysis of 180 studies suggests that depression is also associated with increased circulating pro-inflammatory cytokines (Zorrilla et al., 2001). Similarly, individuals with anxiety disorders also appear to have elevated basal levels of inflammatory markers such as CRP and IL-6 (Howren, Lamkin, & Suls, 2009; O’Donovan et al., 2010). A recent study showed that
changes in the level of negative affect (e.g., anxiety and anger) in response to a laboratory stressor were associated with increases in IL-6 (Carroll et al., 2011). Also, depressed men show excessive activation in their inflammatory response to an acute psychosocial stress, with greater increases in IL-6 and NF-κB compared to healthy participants (Pace, Mletzko, Oyetunde, Musselman, & et al., 2006). Thus, overall, studies suggest that both circulating levels of inflammatory markers and inflammatory stress responses can be influenced by negative affect and mood disorders.

In contrast, inducing positive affect could reduce the levels of pro-inflammatory cytokines. One study with healthy young adults suggests that levels of Tumor Necrosis Factor-α (TNF-α) – another inflammatory cytokine – was decreased after positive mood induction (Mittwoch-Jaffe, Shalit, Srendi, & Yehuda, 1995). Another study showed that patients with rheumatoid arthritis showed decreases in IL-6 and interferon-γ after watching comedic performance (Yoshino, 1996).

**Emotion and health behaviors**

Negative and positive affect can influence health via health behaviors. Negative affect such as anger, anxiety, and depression is associated with various health compromising behaviors, including overeating or binge eating, alcohol use, and poor sleep quality of healthy adults (Cooper, Frone, Russell, & Mudar, 1995; Edman, Yates, Aruguete, & DeBord, 2005; Thomsen, Yung Mehlsen, Christensen, & Zachariae, 2003). Among various health behaviors, sleep has been received growing attention due to its substantial role to regulate the body’s diurnal rhythm and overall restoration processes. A recent review suggested a link between insomnia and mood disorders, including anxiety and depression and proposed that emotional reactivity to stressors in life can cause neurobiological arousal that contribute to insomnia (Baglioni, Spiegelhalder, Lombardo, & Riemann, 2010). Interestingly, people with insomnia showed increases in the levels of negative mood in the evening whereas those without insomnia showed decreases (Buysse et al., 2007). Positive affect assessed by ecological momentary assessment every 20 minute
across a day was inversely associated with sleep problems among middle aged adults in White Hall II study (Steptoe, Wardle, & Marmot, 2005). Thus, previous studies suggest that emotional states, traits, and disorders are associated with physical health outcomes or indirectly influence physical health via changing health behaviors and sleep quality.

**Integrating emotion into the relationship between social relationships and health and examining detailed processes of social relationships**

The research reviewed in the previous sections provides grounds to expect a direct pathway from negative and positive affect to health and from social relationship characteristics to health. The present dissertation will go further than this by examining how state emotion or cognitive-affective orientation interacts with social relationships to affect health and also assessing social relationship characteristics in a more detailed fashion. Figures 1.1 and 1.2 (below) suggest two theoretical models by which state emotion and mood disorder can be integrated into the relationship between social relationship processes and health. This section provides an overview of the theories that support the pathways in these models.

**Moderation by social interaction characteristics**

The model presented in Figure 1.1 predicts that the association between state emotion and health outcomes will be moderated by whether ongoing social interactions are supportive or stressful. This model was developed on the basis of two theoretical perspectives relevant to how stress affects health: the stress buffering theory and the amplifying theory of social relationships. Based on stress and coping theory (Lazarus, 1999), the stress buffering theory proposes that social relationships reduce stress via providing additional resources to cope with stress. Stress buffering theory further suggests that supportive social interactions may reduce or eliminate the effect of negative affect on health by changing appraisals and promoting effective coping behaviors. This perspective appears to be particularly relevant to how
social support improves health. Individuals who experience stress but who also perceive availability of social support from their social network show less morbidity and mortality than those who do not perceive adequate support (Cohen, 2004; Uchino et al., 1996). At the level of stress appraisals, perceived social support may reduce the threatening aspect of a stressful situation and increase one’s perceived capacity to deal with the situation (Cohen & Wills, 1985; Thoits, 1995; Wethington & Kessler, 1986). The changed interpretation of the stressful situation can alter affective states, which can lead to more adaptive behavioral responses to the stressor and better health outcomes (e.g., Wills & Cleary, 1996).

In contrast to the stress buffering model, social relationships can amplify the effects of negative affect when they become a source of stress (Cohen, 2004). A recent study by Slatcher and colleagues examined this among married couples. Wives showed the strong association between their daily level of work-related worries and daily cortisol awakening responses if they reported low levels of marital satisfaction and lack of disclosure to a spouse (Slatcher, Robles, Repetti, & Fellows, 2010). Results of this study thus suggest that a wives’ interaction with her husband can change the degree to which worry of a wife influences on cortisol secretion in the morning.

In keeping with both the amplifying theory of social relationships that is illustrated by this study as well as with stress buffering theory, Figure 1.1 thus presents a model in which social interactions, broadly speaking, are proposed to moderate the association between emotion and health. Specifically, individual’s levels of emotional states can directly affect physical health outcomes and sleep quality. Social interactions are expected to affect the emotional state and also modulate the degree to which emotional states affect health outcomes by generating social stress or support.

**Mediation by social relationship characteristics**

While Figure 1.1 illustrated how social relationships may moderate pathways between emotion and health, Figure 1.2 illustrates how social relationship processes may mediate the pathway between
mood disorders, such as depression, and health outcomes. There are two major theories to explain the well-known association between depression and impaired social environment (i.e. lack of social support and poor relationship quality). First, the erosive perspective assumes that depressive individuals experience a loss of social resources during any given period of depression due to their inability to engage in social relationships (Joiner, 2000). On the other hand, the self-propagatory perspective proposes that depressive individuals not only passively lose their social resources but also actively generate social stressors via maladaptive patterns of interpersonal behaviors (Joiner, 2000). Previous studies have shown an association between lack of social support and depression and have discussed the results based on the erosive perspective. Although a few studies have examined how certain social behaviors of individuals degrade their social relationships and affect onset and recurrent symptoms of depression, they have not examined how the social relationship processes of depressed individuals are linked with deleterious patterns of sleep and markers of inflammation.

Figure 1.2 describes the pathways by which long-term mood disorders affect physical health outcomes by changing patterns of social interactions. If the social interactions involving those with depression generate social stress and impair the social support quality, the social stress and impaired support are expected to result in adverse health outcomes, such as systemic inflammation and sleep disturbances.

The Role of Personality

In addition to mood disorders, personality factors can play a role in the relationship between state emotion, social relationships, and health. Both Figures 1.1 and 1.2 thus illustrate how personality that is related to emotional states, traits, and disorders (e.g., particularly, hostility) may relate to emotional state, occurrence of mood disorder, social interactions as well as health behaviors.
Certain personality traits such as hostility and extraversion can set up the tendencies that may either provoke or hinder stressful social interactions. The transactional model and stress reactivity models provide two explanations for how hostility is linked with social relationship processes and health outcomes. The transactional model stipulates that hostile people generate interpersonal stress and undermine their existing social support through cynical thought processes and aggressive actions toward others. Thus, hostility may amplify the effect of social conflict on physiological stress responses by creating and maintaining a more taxing and less supportive social environment (Smith, 1992). In addition, the stress reactivity model stipulates that hostile individuals may be emotionally and physiologically reactive to social stressors because they are likely to attribute the stressors to intentional mistreatment and provocation by others (Smith, 1992). There is considerable support for this model in the realm of cardiovascular reactivity. For example, trait hostility was associated with increased systolic blood pressure during marital conflict discussion in a laboratory setting (Kiecolt-Glaser et al., 2005). Trait hostility also interacted with the frequency of negative social interactions for diastolic blood pressure (DBP): Only people with high hostility showed an association between negative social interaction and DBP levels (Brondolo et al., 2003). However, few studies have examined how inflammatory responses to stress are influenced by both negative social relationships and hostility.

**Purpose of the dissertation**

In sum, negative affect and mood disorders are linked with dysregulated inflammation, poorer perceived health, and poor sleep quality. The primary goal of this dissertation is to examine how the strength of the association between mood and health is affected by the characteristics of social relationship processes. The three studies that are included in the dissertation will test the relationships between emotional factors and health as well as the moderating and mediating role of social relationship
processes. Personality factors that are known to be related to emotional and social tendencies (e.g., trait hostility) will also be tested for the degree to which those personality factors aggravate the quality of close social relationships and functioning of the stress response system, which in turn may lead to systemic inflammation and poor sleep quality.

This dissertation is designed to bridge gaps in the current emotion and social relationship literatures by examining a series of pathways by which emotional factors and social relationships dynamically affect health outcomes. In order to examine the different sets of pathways, three separate datasets were utilized in the next three chapters. The first study of this dissertation examined the negative social interactions within multiple areas of close relationships by utilizing the dataset from the Cycling Study (PI: Dr. Laura Klein). The cycling study involved a laboratory stress protocol to measure physiological responses after stress among healthy young adults. These data were used to examine the effects of having multiple negative social relationships on inflammatory cytokine responses to stress and whether trait hostility aggravates the effects of negative social relationships over and above the levels of depression. The second and third studies focus on the relationship with a romantic partner and how emotional states and disorders may interact with relationship characteristics to affect physical health. The second study involves analyses with a dataset from the Daily Diary Study (PI Dr. Lynn Martire), which measured daily positive and negative affect, partner’s responses to pain behaviors, and subjective sleep quality among older adults with knee osteoarthritis and their partner across 14 consecutive days. As described in Chapter 3, this dataset provided a unique opportunity to track daily fluctuations of affective states, couple interactions, and sleep quality and the moderating effect of daily couple interactions on the association between daily affective states and daily sleep quality. Finally, the third study involves analyses with a dataset derived from the Midlife in the United States (MIDUS) study, a nationally representative sample of adults in the U.S.; use of these data enabled investigation of whether the
different aspects of negativity in marital relationship mediate the association between depressive symptoms and markers of inflammation, as well as the number of physical symptoms and chronic conditions.

Following chapters two through four, which describe in detail the rationale, methods, and results of all of these three studies and which include discussion of their findings, I provide an overall discussion of how findings from all three studies relate to each other. In this general discussion I further discuss how these three studies support the pathways in the two theoretical models presented in this dissertation (Figures 1.1 & 1.2) and to what extent they fulfill the main goals of this dissertation including examining a) social relationships in a detailed fashion, b) the role of physiological and psychological stress mechanisms underlying the interaction between emotional factors and social relationship on physical health, and c) the mediating and moderating roles of social relationships that explain how and under what circumstances emotional factors affect physical health.
Figure 1.1 Theoretical model of how social interactions may moderate associations between emotional state and health

Figure 1.2 Theoretical model of how social interactions may partially mediate the degree to which mood disorders affect on health
Chapter 2 The role of multiple negative social relationships and hostility on cytokine responses to stress

Abstract

Study 1 of this dissertation examined the unique impact of perceived negativity in multiple social relationships on cardiovascular, endocrine, and inflammatory responses to a laboratory stressor. Fifty-six healthy young men ($n=20$) and women ($n=36$) were challenged with the Trier Social Stress Task. Two inflammatory cytokines (IL-6 and TNF-$\alpha$), cortisol, blood pressure, and heart rate were obtained at baseline, 15min post-stress, and 75min post-stress. Participants also completed questionnaires to determine perceived negativity among four types of relationships (romantic partner, family, close friend, and roommates), trait hostility, and depressed mood. Via hierarchical cluster analysis, those who reported negativity across relationships with roommates, family, and friends showed greater IL-6 responses to stress. Those who reported negativity across relationships with a romantic partner, family, and friends had higher mean IL-6 across time, a greater increase in TNF-$\alpha$ from 15min to 75min post stress, and less reactivity in systolic and diastolic blood pressure. Differences in mean IL-6 and diastolic blood pressure were accounted for by either depressed mood or hostility, whereas differences in the cytokine stress responses remained significant after controlling for those factors. Overall, this study indicates that having multiple negative relationships may exacerbate acute inflammatory responses to a laboratory stressor independent of hostility and depressed mood.
Introduction

In this chapter, the impact of negative social relationships on physiological stress response to a psychosocial stressor was examined, as well as whether trait hostility interacts with social relationships to predict physiological responses to stress. This study achieved two major goals of this dissertation by assessing negativity in multiple social relationship areas and also by utilizing measures for the physiological reactivity to a laboratory stressor as physical health outcomes. Several key pathways in Figure 1.2 were tested in this study. First, this study examined whether the different patterns of negative social relationship patterns generate social stress that can lead to dysregulated physiological stress responses. In addition, this study tested whether trait hostility may further amplify the degree to which negative social relationship patterns affect physiological stress response.

Inflammation and health

Among the multiple physiological systems involved in stress responses, inflammatory cytokine responses to stress, which are part of the immune system, are the outcomes on which this chapter is focused. As briefly reviewed in the previous chapter, inflammation is a natural component of immune responses that are activated by internal and external immunological stimuli, such as acute infection or injury (Ershler & Keller, 2000). In the short term, activation of inflammation is a part of an adaptive immune response that helps fight off pathogens and expedites the healing process by initiating a cascade of lymphocyte activation and acute-phase protein synthesis. However, chronic activation of inflammatory status is related to some serious types of pathogenesis, such as chronic pain conditions and the development of tumor and atherosclerosis (Kiecolt-Glaser et al., 2010). Inflammation has also been linked with exacerbated pain responses and chronic pain conditions, as well as development of chronic diseases such as cardiovascular disease, type II diabetes, arthritis, Alzheimer’s disease, osteoporosis, and cancer (Aggarwal et al., 2006; Ershler & Keller, 2000; Marchand, Perretti, & McMahon, 2005). There is growing
evidence to show that not just immunological stimuli but also psychosocial factors can trigger processes that lead to chronic inflammation.

**Social relationships and inflammation**

Stress from interpersonal relationships has a powerful impact on immune function and chronic inflammatory status (Graham et al., 2007; Kiecolt-Glaser et al., 2010; Slavich, O'Donovan, Epel, & Kemeny, 2010). Negative social exchanges have been tied to immune-related responses that are linked with poorer overall health (Chiang, Eisenberger, Seeman, & Taylor, 2012; Edwards, Hershberger, Russell, & Markert, 2001; Kiecolt-Glaser & Newton, 2001), and those who suffer from chronic social conflict often have dysregulated immune function (Cohen et al., 1998b; Davis et al., 2008; Friedman, Karlamangla, Almeida, & Seeman, 2010). Interpersonal stress appears to have a long lasting impact on health in part by contributing to chronically elevated inflammation, which can confer risk of diverse age-related diseases (Ershler & Keller, 2000; Ridker, 2000); for example, chronic interpersonal strain has predicted higher levels of pro-inflammatory cytokines, neurochemical messengers that could trigger maladaptive systemic inflammatory status, 6 months later (G. E. Miller et al., 2009). However, the majority of studies on immune responses to social conflict have focused on a particular type of relationship, such that the effect of having conflict within multiple relationships is largely unknown. Further, the degree to which multiple social conflicts affect inflammatory responses to stress and whether those responses are amplified by trait hostility are important issues that are not well understood.

Experimental studies have demonstrated that acute social conflict can influence immune responses. For example, engaging in a hostile discussion with one’s spouse is associated with elevated levels of circulating pro-inflammatory cytokines and slowed wound healing (Kiecolt-Glaser & Newton, 2001). Given such findings and the documented association between chronic interpersonal stress and poor health, it is reasonable to speculate that the frequent occurrence of multiple acute conflicts could therefore
contribute to poor health. Consistent with this, the frequency of negative social exchanges with close others has been associated negatively with physical and mental health among college students and is predictive of depressed mood in a sample of married adults (Edwards et al., 2001; Joiner & Timmons, 2009). However, individuals are often involved in multiple social relationships, each with a different degree of negative social exchange. For example, people with intense marital conflict do not necessarily have many negative social exchanges with their family and friends. To appreciate the combined impact of multiple social relationships with different degrees of negativity on health, it is important to examine multiple social dyads for their degree of negative social exchange and their effects on health-related outcomes.

**Negative social relationships and inflammatory responses to stress**

One physiological mechanism that may explain the negative effects of social conflict on health is repeated physiological activation of inflammatory stress responses and delayed recovery to stress (Seeman & McEwen, 1996). A recent study demonstrated that daily negative social interactions predicted elevated levels of two markers of inflammation – interleukin-6 (IL-6) and soluble tumor necrosis factor-α receptor II (sTNF-αRII) derived from oral mucosal transudate – in response to an experimental stressor (Chiang et al., 2012). Under social conflict, inflammatory responses to stress may be also maintained by actions of the sympathetic-adrenal-medullary (SAM) system and the hypothalamic-pituitary-adrenal (HPA) axis (e.g., via cortisol) (Lovallo, 2005; Miller et al., 2007).

**Potential role of hostility and depression**

In addition to a relatively direct effect of social conflict via stress activation, it is important to consider individual characteristics that tend to go along with negative social relationships such as hostility. In particular, hostility, which is a trait that involves cynicism, hostile attributions, aggressive responding, and hostile emotions (Boyle, Jackson, & Suarez, 2007; Smith, 1992), appears to interact with social
conflicts to affect cardiovascular stress responses; hostile individuals tend to show greater blood pressure responses to a laboratory induced conflict (Kiecolt-Glaser et al., 2005) or frequent negative social interactions (Brondolo et al., 2003).

Yet another way in which social conflict may influence immune function is by aggravating existing biological and psychological vulnerability to depression and thereby inducing dysregulation of the inflammatory response system (Kiecolt-Glaser et al., 2010; Slavich, O’Donovan, et al., 2010). Experimental studies have shown that depressive symptoms are associated with an excessive inflammatory state via amplified reactivity of the inflammatory system to psychosocial stressors. Brummett and colleagues (2010) examined the effect of depressive symptoms on stress reactivity of IL-6 and C-reactive protein (CRP) among healthy adults and found that women with depressive symptoms had higher reactivity of both of those inflammatory biomarkers after an anger and sadness emotional recall task than those with low depressive symptoms. In addition, the HPA axis response to stress, which is intimately involved with regulating the inflammatory response, is dysregulated among depressed individuals (for reviews see, Burke, Davis, Otte, & Mohr, 2005; Raison, Capuron, & Miller, 2006). However, despite the possibility for hostility and depressed mood to be related to the connection between relationship stress and health, few studies have examined whether negative social relationships influence inflammatory responses to an acute stressor over and above levels of hostility and depressed mood.

Purpose of Study 1

One goal of this first study is to examine effects of negative social exchanges in multiple relationships – specifically relationships with a romantic partner, a close friend, close family, and roommates – on responses to experimental stressors, with an emphasis on inflammatory responses. For example, people with intense marital conflict may or may not have negative social exchanges with other members of their family or with friends. To appreciate the combined impact of multiple social
relationships with different degrees of perceived negativity on health, it is important to examine multiple social dyads for their degree of negative social exchange and their effects on health-related outcomes. This approach will thus fulfill the goal of this dissertation to examine social relationships in a detailed fashion.

In this study, negative social relationship patterns in relationships with a romantic partner, a close friend, family, and roommates were examined to determine if they significantly predict physiological responses above and beyond effects of age, body mass index (BMI), gender, and menstrual cycle status of female participants. Individuals with more negative relationships relative to others are expected to show a different pattern of cardiovascular, endocrine, inflammatory response to stress that has been observed in previous studies of relationship conflict (Brondolo et al., 2003; Friedman et al., 2010; Kiecolt-Glaser et al., 2005; Seeman & McEwen, 1996). The pathways in the second theoretical model that are related to each hypothesis of this study are presented in Figure 2.1. Specifically, I predict that:

H₁: Individuals with conflict in more relationships than others will have exaggerated or prolonged inflammatory response to stress (i.e., poorer recovery) in terms of two cytokines with inflammatory properties, IL-6 and TNF-α (pathway ‘H₁’ in Figure 2.1).

H₂: Individuals with more negative relationships will show greater increases in blood pressure during the stressor, with sustained elevation during the recovery (pathway ‘H₂’ in Figure 2.1).

H₃: Individuals with more negative relationships will show greater increases in cortisol after being exposed to the laboratory stressor (pathway ‘H₃’ in Figure 2.1).

Another goal of this first study is to examine whether trait hostility modulates the effects of negative relationships on stress response. I hypothesize that:
H₄: Hostility will amplify the effects of conflictive relationships on cardiovascular, hormonal, and inflammatory responses: Individuals with a combination of multiple negative relationships and high hostility are expected to show the greatest stress reactivity in blood pressure and more prolonged elevation in cortisol, and the greatest increases in pro-inflammatory cytokines in response to the stressor (pathway ‘H₄’ in Figure 2.1).

Depressed mood will also be taken into account in order to verify that the effects of hostility are not driven by higher levels of depressed mood among hostile individuals, which have been reported in the previous studies (Biaggio & Godwin, 1987); this is represented by the pathway ‘H₅’ in Figure 2.1. Finally, interactions based on both gender will be explored because gender are known to modulate cardiovascular, endocrine, and inflammatory responses (Brummett et al., 2010; Darnall & Suarez, 2009).

Method

Participants

As a part of a larger study on hormonal status and acute physiological responses to a laboratory stressor (PI: Laura C. Klein funded by the NSF SBR9905157 and Penn State Internal funding 223-15-3605), fifty six healthy participants (36 women, 20 men), aged 18-30 years (mean = 21.05 ± 0.37) were recruited to participate in a study examining hormonal responses to an experimental stressor. Participants were recruited via advertisements in the local newspaper and flyers posted in the local community and on the campus of The Pennsylvania State University. An initial telephone interview was conducted by a trained research assistant to determine the eligibility of participants. Exclusion criteria included tobacco use, BMI ≥ 30, psychiatric hospitalization within the past year, psychotropic medication use within the previous eight weeks, the use of medications such as corticosteroids within the prior three months,
hormonal contraceptives, beta-blocker or other medications for controlling blood pressure, and inhaled beta agonists. People who scored higher than 16 on the Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977) or with history of depression were not eligible for the study. In addition, people with a history of heart disease, diabetes, and neurological disorder were excluded along with women who reported any possibility for pregnancy and issues of hormonal dysregulation. These inclusion and exclusion criteria were chosen in order to minimize the effects of variables known to affect cardiovascular, immune, or endocrine function. Women came to the laboratory during either the late luteal (n = 19) or follicular (n = 17) phase of their menstrual cycle. Following telephone screening, participant eligibility was confirmed and the next onset of the luteal and follicular phases was determined by self-reported date of the last menstrual period and menstrual cycle length (Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999; Whetzel, Corwin, & Klein, 2007).

**Procedures**

All participants went through a modified Trier Social Stress Task protocol, which included a 15-min baseline rest period, a 30-min stress period (13 and ½-minute speech task and 15-minute mental arithmetic task), and a recovery period (Kirschbaum, Pirke, & Hellhammer, 1993). Blood samples were collected at three time points during the study: 1) at the end of the baseline period, immediately prior to the stress protocol, 2) 15 minutes, and 3) 75 minutes following completion of the stress protocol. From the end of the stress period, while waiting for their final two blood draws, participants filled out questionnaires and then sat quietly and listened to music. Blood pressure and heart rate were collected at least every 5 minutes throughout the protocol, along with repeated mood assessments and a series of psychosocial measures that included information about interpersonal relationships.

**Laboratory Protocol andStressor.** Eligible participants arrived at a General Clinical Research Center (GCRC) at 1300 hrs and were met by a trained research assistant who first obtained informed consent.
Next, participants were interviewed by a certified nurse practitioner to confirm health status and study eligibility. Body weight and height also were measured. Next, participants were asked to complete questionnaires, after which a standard blood pressure cuff (Dinamap Compact Blood Pressure Monitor, Critikon, Tampa, FL) was placed on the participant’s dominant arm, and a trained nurse inserted an indwelling catheter in the non-dominant arm using standard techniques. After a 10 minute acclimation period, participants were asked to sit quietly for 15 minutes while baseline blood pressure readings were taken automatically. Next, a baseline blood sample (20cc) was drawn.

In the modified Trier Social Stress Task, which followed the baseline blood draw, participants were given 10 minutes to prepare a 3½-minute speech about a personal failure that had had a negative consequence on their life. They delivered the speech in front of a video camera and were told that a recording would be later observed by a panel of psychologists. Participants were prompted by the experimenter to continue talking if they finished their speech in less than the allotted time. Immediately after the speech, the experimenter asked participants to complete a serial subtraction task as fast and as accurately as possible (4 min), followed by several math word questions that increased in difficulty (3.5 mins), and then a different serial subtraction task (4 min). The experimenter delivered timed prompts to urge participants to work more quickly and to tell them to start over if they delivered the wrong response. This stress session took 30 minutes total. No video recording actually was made of the speech and participants were debriefed about this deception at the end of the laboratory session.

Blood pressure and heart rate were recorded every two minutes throughout the stress and recovery periods. The baseline blood sample was used to determine basal levels of cortisol, IL-6, and TNF-α; blood samples at 15 and 75 minutes after the stress period were used to determine cortisol, IL-6, and TNF-α. Participants completed several post-stress measures of mood at the end of the study, after which the catheter was removed. Participants received monetary compensation. All study procedures
were reviewed and approved by The Pennsylvania State University Institutional Review Board.

Measures

Negative Social Relationships. A 25-item measure was used to assess negative social relationships, which was based on an existing questionnaire that included five items about negative social interactions with a spouse or significant other (Schuster, Kessler, & Aseltine, 1990). The present study used those same five items to ask about negative social interactions among a) roommates, b) a spouse/significant other, c) close family members, d) their closest friend, and e) their children. The items ask about the frequency of social exchanges involving disagreements and tension, with responses ranging from 0 (never) to 5 (very often). The alpha reliability of the original scale was 0.76 (Schuster et al., 1990), and the present sample showed Chronbach–α of 0.84, 0.89, 0.84, 0.81 for the spouse, family, roommate, and friends subscales, respectively. The subscale about relationship with children was not retained from analyses because no participant reported having children in this sample.

Hostility. The well-validated Cook-Medley hostility questionnaire (CMHQ; Cook & Medley, 1954) was used to assess the tendency to react and think in a hostile manner. The scale has 50 true-false items, which are aggregated into a total score ranging from 0 to 50. The internal consistency was .83 for this sample.

Negative mood. A six item negative mood scale was administered four times (baseline, immediately after the stressor, 15 min, and 75 min after the stress) to check the effect of the experimental stressor on mood. The scale was part of a larger adjective checklist scale and consisted of words describing negative and positive mood (e.g., nervous, happy, irritated, and stressed) with 7 point Likert scale ranging from 1 (Not at all) to 7 (Very much). The scale showed a good internal consistency across the four measurements of the present study (Chronbach–α = 0.74, 0.88, 0.84, and 0.86, respectively).

The Center for Epidemiologic Studies Depression Scale (CES-D). The Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977) was used to measure depressed mood. The CES-D has
been used to measure clinical and sub-clinical levels of depression and effectively identifies depression among healthy individuals (Radloff, 1977). Item responses are from 0 to 3, with 3 representing the greatest frequency of depressed symptoms over the past week. On a scale of 0 to 60, a higher score indicates more depressed mood.

**Biochemical Analysis.**

**Blood handling.** For preparation of serum, blood was drawn into separate collection tubes that contained no additive. Serum tubes were allowed to sit at room temperature for 15 minutes before centrifugation ($1500 \times g$ at $4^\circ C$ for 15 minutes). Following centrifugation, serum was aliquoted into separate 100 µL microtubes and frozen at $-80^\circ C$ for later assay.

**Serum Cortisol, IL-6, and TNF-α.** Serum cortisol, IL-6 and TNF-α were assayed at The Pennsylvania State University GCRC Core Laboratory. Serum cortisol levels were determined using commercially available enzyme immunoassay kits (EIA; Diagnostic Systems Laboratories, Inc., Webster, TX); serum IL-6 and TNF-α levels were determined by EIA using published procedures by the GCRC (Corwin & Cannon, 1999). All samples were tested in duplicate in a single assay batch; all assay plates were balanced by cycle phase (men, women: luteal, follicular). Duplicate tests that varied by more than 5% error were subject to repeat testing. The average of duplicate tests is reported for each biomarker assay.

**Blood Pressure, Heart Rate measurements.** Aggregation across two measures of basal resting SBP and DBP in a laboratory setting has been shown to provide within-subject reliability of $+0.90$ or better (Llabre et al., 1988). Therefore, SBP and DBP readings, along with HR, were averaged across each experimental time period to derive mean baseline (6 readings), stress period (15 readings), and recovery (12 readings) measures.

**Data Analysis**

SPSS 20.0 was used for all analyses. Study variables were screened for outliers and non-
normality, and cortisol, IL-6, and TNF-α were natural log transformed to correct for skewness. Preliminary descriptive analyses were then run on key study variables.

I considered using two different ways to examine the patterns of negative relationships in multiple relationship areas. One of this ways is to use median scores of negative relationship variables to divide people with highly negative relationships from those without; the second is hierarchical cluster analysis. Hierarchical cluster analysis provides a particularly robust way to recognize group of individuals who share similar patterns across multiple reference variables and can be used even when the cluster groups do not have equal sample sizes; unlike median split, this technique does not rely on one numeric value but uses the distance between all pairs of the observed values (Clatworthy, Buick, Hankins, Weinman, & Horne, 2005; Klecka, 1980). Thus, hierarchical cluster analysis was applied to the analyses of this study. Individuals were identified into different clusters with different degree of perceived negativity across the relationships with roommates, a romantic partner, close family, and the closest friend. Out of 56 participants, one woman did not provide sufficient data to compute the cluster by negative relationship analyses and was therefore excluded from analyses. While 25 participants reported having all of the four relationships, 13 participants did not have a roommate, and 13 others did not have a romantic partner. Thus, two separate hierarchical cluster analyses were run, the first including individuals who reported having roommates, a close friend, and family (n = 38), and the second cluster analysis including those who reported having a romantic partner, a close friend, and family (n = 38). Four participants who did not report a relationship with either a romantic partner or roommates had to be excluded from these cluster analyses. There was no significant difference between the four participants and the rest of the samples in any of the psychological characteristics and physiological outcome variables we examined in the study.

For both cluster analyses, the same three steps were used to identify and validate the number of
groups in the cluster models. In the first step, the variables for the perceived negativity in the three core relationships were entered into the hierarchical cluster analysis model. Ward’s method was selected for clustering for the model with the similarity measure of squared Euclidean distance used to decide the number of groups in the cluster model (Clatworthy, Buick, Hankins, Weinman, & Horne, 2005; Romesburg, 1984). In the second step, we conducted discriminant function analysis to confirm that to what extent the membership of each group was replicated, the resulting groups in cluster model were different from each other in terms of perceived negativity in the relationships, and which of the negative relationship areas contributed most to differentiating the groups in the cluster model (Klecka, 1980). In the third step, we further confirmed the validity of the cluster classification via $F$ tests to determine whether the groups in the cluster model differed by age, gender, the four negative relationship variables, or depressed mood or hostility.

Repeated-measures analysis of variance (RM ANOVA) was then used to examine the effect of the different negative social relationship clusters on SBP/DBP/HR, cortisol, and cytokine responses to stress. Greenhouse-Geisser corrected $p$ values were reported if criteria for sphericity were not met. The partial eta-squared ($\eta_p^2$) were given as measures of effect size for the significant ANOVA results (Cohen, 1988). When there was a significant effect of two cluster models on any outcome variable, post hoc tests with Bonferroni correction were used to examine which groups in the cluster models were significantly different from each other. As post hoc tests for the time effects, difference scores were calculated for the stress response measures between each pair of the three time points (e.g., from baseline to 15min after stress) in order to examine change during each time interval. Due to their known impact on inflammation, age, BMI, gender, and menstrual cycle of women were controlled in all analyses. Three dummy coded variables representing a) men, b) women in the luteal period, and c) women in the follicular period were generated and entered in analyses to control for gender and women’s menstrual cycle status. Finally,
depressed mood and hostility were additionally controlled for significant findings to examine whether the effects were independent of those characteristics.

Results

Preliminary analyses

The means and standard deviations (SDs) of demographics and study variables are presented in Table 2.1. Seventy one percent were Caucasian \( (n = 40) \), 7% were African American \( (n = 4) \), 7% were Asian \( (n = 4) \), 3.5% were Hispanic \( (n = 2) \), and 10.7% were self-described as “other” \( (n = 6) \). All but one participant had graduated from high school; 64% had some college education, and 22% of the participants had more than a college education.

Cluster analysis for negative relationship profiles

Individuals were classified as having different degrees of perceived negativity across multiple interpersonal relationship areas. First a cluster analysis was run on the 38 participants who reported relationships with roommates, family, and a close friend. This analysis identified three clusters (Table 2.2 (a)): “a low conflict group” \( (n = 30) \) characterized by low perceived negativity across all the relationship areas, “a multiple conflict group” \( (n = 3) \) characterized by high perceived negativity across all the relationship areas, and “a family conflict group” \( (n = 5) \) characterized primarily by high perceived negativity in family but low perceived negativity among roommates and friends. The \( F \) tests confirmed that the three groups in this cluster analysis were significantly different in levels of perceived negativity across the three relationship areas \( (ps < .001) \). A discriminant function analysis verified the cluster structure \( (\chi^2(6, n = 38) = 70.69, p < .001) \) and that 94.7% \( (36 \text{ cases out of } 38) \) of the original grouped cases were correctly identified; further, the relationships with family and roommates were the strongest predictors for the first and second functions of this analysis, respectively. The first and second functions
accounted for 73.4% and 26.6% of the variance explained by the model. The gender distribution was significantly different across the 3 groups ($p = .05$), which was largely driven by the multiple conflict group having only three men and no women. Age was not different across the 3 groups. The multiple conflict group demonstrated significantly higher levels of depressed mood and hostility than the other groups, with descending levels in the family conflict group and low conflict group ($ps < .01$).

Another cluster analysis was run on the 38 participants who had a romantic partner, family, and a close friend. It yielded two groups (Table 2.2 (b)): “a low conflict group” ($n = 29$) characterized by consistently low perceived negativity across all relationship areas (romantic partner, family, and friends), whereas “a multiple conflict group” ($n = 9$) had high perceived negativity across all relationship areas. The multiple conflict group in this two cluster model included only one of the three individuals who comprised the multiple conflict group in the other cluster model. $F$ tests confirmed that the two groups in this cluster model were significantly different in levels of perceived negativity in these relationships ($ps < .05$). A discriminant function analysis verified the cluster structure ($\chi^2(3, n = 38) = 53.56, p < .001$), and 97.4% of the original grouped cases (37 cases out of 38) were correctly identified. The variable for the relationship with family was the strongest predictor for the first function. The distribution of gender and age was not significantly different across the 2 groups. As with the three cluster model that included those with roommates, the multiple conflict group in this two cluster model (which instead included those with romantic partners) demonstrated higher levels of depressed mood and hostility than the low conflict group ($ps < .01$).

**Manipulation checks for the stress protocols**

Participants’ negative mood increased in response to the stressor ($F(3, 153) = 34.71, p < .001$, $\eta^2 = .28$). We also observed significant increases in SBP, DBP, and HR in response to the stressor ($F(2, 110) = 197.48, p < .001, \eta^2 = .78$; $F(2, 110) = 206.88, p < .001, \eta^2 = .79$; $F(2, 110) = 136.92, p < .001$,
$\eta^2_p = .71$, respectively). The levels of serum cortisol did not significantly increase in response to the experimental stressor but showed a significant effect of time, likely driven primarily by the diurnal rhythm of cortisol ($F(2, 106) = 9.88, p < .001, \eta^2_p = .16$). There was no significant time effect on either IL-6 or TNF-$\alpha$ levels.

**Stress responses by negative social relationships**

**IL-6.** There was a significant time by perceived negativity interaction using the three cluster model (which included those with relationships with roommates, friends, and family) on IL-6 ($F(4, 58) = 8.53, p < .01, \eta^2_p = .37$). Post hoc tests confirmed that only individuals in the multiple conflict group of this cluster model showed significantly greater increases in IL-6 from baseline to 15 min after stress ($p < .01$) and from baseline to 75 min after stress ($p < .01$) compared to those in the family conflict or low conflict groups (Figure 2.2). Results remained significant after controlling for depression or hostility ($p < .01$).

In the two cluster model (which included those with relationships with a romantic partner, friends, and family) mean IL-6 across the three time-points was significantly different between the two groups ($F(1, 31) = 5.11, p < .05, \eta^2_p = .14$); individuals in the multiple conflict group showed a higher mean level of IL-6 than those in the low conflict group ($p < .05$). The mean IL-6 result was reduced to non-significance after controlling for depression or hostility ($F(1, 26) = 2.85, p = .10$; $F(1, 30) = 2.01, p = .16$ respectively). There was no significant hostility by cluster model interaction on IL-6 responses to stress.

**TNF-$\alpha$.** There was a marginally significant time by perceived negativity interaction using the two cluster model on TNF-$\alpha$ responses to the stressor ($F(2, 56) = 2.80, p = .07; \eta^2_p = .09$). Upon examination, individuals in the multiple conflict group (negativity among romantic partner, friends, and family) showed significantly greater increases in TNF-$\alpha$ from 15 min to 75 min after stress after controlling for baseline TNF-$\alpha$ and other covariates ($F(1, 27) = 6.81, p < .05; \eta^2_p = .20$); this result remained significant after controlling for depression or hostility ($p < .05$). This difference remained significant after removing
the one individual in the multiple conflict group in the two cluster model who was also included in the multiple conflict group in the three cluster model (where greater IL-6 responses to stress were observed). There was no main effect in the three cluster model groups on TNF-α stress response. Hostility also did not interact with any of the cluster models to affect TNF-α stress responses.

**BP/HR and Cortisol.** There were significant time by perceived negativity interactions on SBP and DBP responses to stress using the two cluster model in the unexpected directions \((F(2, 64) = 6.69, p < .01, \eta^2_p = .17; F(2, 64) = 6.18, p < .01, \eta^2_p = .16)\). *Post hoc* tests for the time by perceived negativity interaction showed that those in the multiple conflict group showed less increase in SBP and DBP from baseline to 15 min after stress \((ps < .01-.05)\), and less decrease in them from 15 min to 75 min after stress \((ps < .01)\), compared to those in the low conflict group (Figure 2.3, (a) & (b)). Controlling for hostility (but not depressed mood) reduced this time by perceived negativity interaction on DBP to non-significance. The SBP and DBP responses were not predicted by the three cluster model of negative relationships. Neither of the negative relationship cluster models significantly predicted HR and cortisol responses to the stressor, and there was no significant interaction between hostility and the cluster models on HR and cortisol responses to stress.

**Moderation by gender**

There was a significant time by gender by two cluster model interaction on IL-6 response to stress \((F(2, 62) = 6.14, p = .02, \eta^2_p = .16)\). Only men \((n = 3)\) in the cluster with negativity in all of the relationships with a romantic partner, family, and friends evidenced increases in IL-6 at baseline and 15 min post stress, whereas women \((n = 6)\) with multiple conflicts did not \((F(1, 31) = 3.46, p < .05, \eta^2_p = .17)\).

There was also significant gender moderation for the time by the two cluster model on TNF-α response to stress \((F(2, 56) = 4.40, p = .02, \eta^2_p = .14)\). The levels of TNF-α decreased from baseline to 15
min and increased from 15 min to 75 min after stress among men in the multiple conflict group (n = 2) whereas women in the group (n = 6) did not show changes in the level of TNF-α from baseline to 15 min but showed increases from 15 min to 75 min post stress. The post hoc test suggests that the gender difference was driven by the different pattern of change in TNF-α from baseline to 15 min post stress ($F(1, 29) = 11.70, p < .01, \eta^2 = .29$). Gender did not moderate either IL-6 or cardiovascular findings.

**Discussion**

Stress is a routine part of daily life and relationship stress is often the most common and arguably the strongest type of stressor most people experience (For a review see, Kiecolt-Glaser et al., 2010). Social conflict has been associated consistently with poorer health and various stress-related biomarkers (Graham et al., 2007; Kiecolt-Glaser & Newton, 2001). The majority of past studies showing connections between relationship stress and biomarkers have focused on a particular type of relationship or broad characterizations of relationship quality or network size. The present research advances such past studies by examining the effect of perceived negativity across multiple interpersonal relationships and whether the effects of multiple negative relationships are independent of depressed mood and trait hostility. Another important aspect of the present research is that I examined the effect of relationships on inflammatory responses to stress (in addition to cardiovascular and endocrine responses), as opposed to collecting only basal levels of inflammation and other cardiovascular and endocrine biomarkers. A better appreciation for how social conflict may alter stress responsiveness of the body is critical to understanding how and why it is associated with poorer physical health (Seeman & McEwen, 1996).

As expected based on hypothesis 1 ($H_1$), the results of the present research suggest that there are differences in acute inflammatory cytokine responses to stress depending on the pattern of multiple negative social relationships individuals reported within the four relationship areas examined, which were
romantic partner, close family, a close friend, and roommates. Those who reported perceived negativity in their relationships with roommates, family, and friends ($n = 3$) showed increases in IL-6 to stress. Similarly, people who reported negative relationships with a romantic partner, family, and friends ($n = 9$) showed increases in TNF-α from 15 min to 75 min post stress after controlling for baseline TNF-α. Both the IL-6 and the TNF-α results remained significant after controlling for depressed mood or hostility. Age, BMI, gender, and menstrual cycle status among female participants were also controlled in all analyses. As discussed further below, the multiple conflict groups in these analyses were based on small numbers of participants and results should thus be interpreted with caution. However, these multiple conflict groups were largely distinct from each other. The greater increase in TNF-α between 15 min to 75 min post-stress remained the significant after removing the one overlapping individual. Thus, the TNF-α results show what could be considered some replication of the effects of perceived negativity in multiple social relationship areas on IL-6, via a different and slightly larger subset of individuals.

The findings of the present research complement the results of a recent study showing that daily levels of negative and competitive social interactions were associated with inflammatory responses to stress as measured by IL-6 and sTNF-αRII (Chiang et al., 2012). In terms of direction, the inflammatory stress reactivity of individuals with multiple negative relationships is also consistent with previous studies of the association between acute social conflict and inflammation (For review see, Graham et al., 2007; Kiecolt-Glaser et al., 2005). Psychological stress effects on inflammatory cytokines responses are likely explained by multiple aspects of complex, interrelated physiological systems. For example, chronic stress is related to dysregulation of the inflammatory stress response due to decreased glucocorticoid receptor sensitivity (Corwin, 2013; Miller et al., 2008; Pace, 2012) or perhaps to down-regulation of cholinergic anti-inflammatory pathways that are presumed to suppress proinflammatory cytokine production after acute stress (Tracey, 2002).
Participants who reported negative relationships with a romantic partner, family, and friends (but not those who reported negativity with roommates, family, and friends) showed higher mean IL-6 levels, but this finding was reduced to non-significance when controlling for either depressed mood or hostility. While depressed mood and hostility did not amplify the effects of relationship perceived negativity on inflammatory cytokine responses to stress as proposed in $H_4$, depressed mood and hostility did account for the higher mean IL-6 levels observed in individuals with high perceived negativity across those relationship types (despite the fact that the current sample did not include clinically depressed individuals). Unsurprisingly, individuals with perceived negativity across all relationships examined also reported higher depressed mood and hostility compared to others. Thus, another way in which perceived negativity across relationships might contribute to inflammation is by elevating inflammatory levels over time via depressed mood and hostile personality. This is consistent with previous research, which suggests that depression and hostility are associated with increased circulating markers of inflammation among adults (Graham et al., 2006; Suarez, Lewis, & Kuhn, 2002; Zorrilla et al., 2001).

Of note, the association between the number of negative relationships and cytokine stress responses may vary by gender. The stress reactivity of IL-6 was largely driven by men with multiple negative relationships in the present research. It is possible that men are more stress responsive when they have certain patterns of naturally existing negative relationships. Men with multiple negative relationships also showed unexpected decreases from baseline to 15 min post stress, compared to women with multiple negative relationships who did not show changes during that time interval. However, these results must be interpreted with caution because they were based on a very small sample.

Blood pressure reactivity to stress was also associated with multiple negative relationships in the present research but in unexpected directions from $H_2$: Having negative relationships with a romantic partner, family, and friends was expected to be associated with greater reactivity in SBP and DBP to a
laboratory stressor. However, the results showed that negative relationships were associated with less reactivity in SBP and DBP. The association between DBP reactivity and negative relationships appears to be partially explained by hostility. A meta-analysis of 729 studies showed that anxiety, neuroticism, or negative affect has been associated with decreased cardiovascular reactivity to a laboratory stressor whereas hostility, aggression, or type-A personality has been associated with increased cardiovascular reactivity (Chida & Hamer, 2008). People with multiple social conflicts appear to have both tendencies toward negative affect and hostility.

I also did not find the support for H3 that predicted a significant effect of negative relationship endorsement on cortisol responses. This null effect might be related to the particular experimental stress paradigm used in which the public speech part of the stress task was conducted in front of a video camera instead of a panel of judges, a protocol which can reduce the intensity of stress response from the Trier (For a review see, Dickerson & Kemeny, 2004; Kirschbaum et al., 1993). Also, the timing of the catheter insertion and blood draws might explain the null finding. Future research is needed to confirm whether and under what conditions and on what timeline individuals with multiple negative relationships show different patterns of cortisol responses to an experimental stressor and to what extent those cortisol responses are intertwined with inflammatory cytokine responses.

Limitations

The clinical implications of the study are limited in several ways. The present research was conducted with a small sample of healthy young adults. Most strikingly, there were only three individuals who reported multiple negative relationships in all relationship categories in the three cluster model (roommates, family, and friends), who drove effects on IL-6 responses to stress, and nine individuals who reported multiple negative relationships in the two cluster model (a romantic partner, family, and friends), who drove effects on TNF-α. Importantly, issues with unequal sample sizes across groups were
minimized in the present research by our usage of cluster analyses and by our application of the most appropriate and conservative statistical adjustments when using ANOVA (Clatworthy, 2005; Girden, 1992; Huynh, 1978). Moreover, the individuals with multiple conflicts do not appear to be consistent outliers in terms of their levels of IL-6 and TNF-α. For instance, in the three cluster model, the natural log transformed IL-6 levels of the multiple conflict group at baseline ranged from 2.70-4.80 with mean 3.95 ($SD = 1.10$), which was slightly lower than those of the family conflict group (Range 3.35-5.48; Mean = 4.25, $SD = 0.78$) and the low conflict group (Range 2.28-6.95; Mean = 3.41, $SD = 1.40$). Thus, participants in the multiple conflict group came to the lab without elevated IL-6 compared to the others, but were the ones who showed increases in IL-6 after being exposed to the laboratory stressor.

It will be important to confirm the results of the present study using larger and more diverse samples, particularly a greater number of individuals reporting negative relationships across multiple social relationship areas than was available in the present study. It would also be of value to utilize clinically diverse samples, such as those with existing inflammatory conditions, and to include assessments of clinical health outcomes.

**Conclusion**

Although having some degree of social conflict is an unavoidable part of everyday life, the present study provides preliminary evidence that having conflict across multiple social relationships might be harmful, as it is associated with greater inflammatory reactivity to a psychosocial stressor. The effect of having multiple social relationships on inflammatory responses to stress appears to be independent of any effect of hostility or depressed mood (both of which were higher among those with multiple social conflicts). However, depressed mood and hostility likely contribute to health risk that is associated with having multiple social conflict; the elevated depressed mood and hostility observed among those with multiple social conflicts accounted for their having higher mean levels of inflammation,
which might confer risk that is associated with systemic inflammation including illness symptomatology, frailty, and disease (Chrousos, 2000). Therefore, the findings of this study provide evidence to support pathway H₁ in Figure 2.1, which proposes a link between social stress (from multiple negative relationships in this case) and physiological changes that can negatively affect physical health; findings also lend support for pathway H₄ and H₅ in the same figure, which proposes that hostility and depressed mood, can contribute to outcomes of relevance to physical health (inflammatory responses to stress in this case) directly. Taken as a whole, the present study emphasizes the importance of examining the role of negative close relationships in inflammatory stress response in a detailed fashion. Specifically, it suggests that having multiple negative relationships may put individuals at particular risk of developing disease due to exacerbated acute inflammatory reactivity to psychosocial stress.

This study enabled me to examine the effects of negative social dynamics in not just in one relationship but in multiple relationships, in that I compared individuals with multiple negative social relationships to those without negative social relationships. However, individuals may have social conflicts that create negativity in their interpersonal relationships in a certain point of their lives but then resolve the conflicts over time. This kind of change (e.g., in levels of social conflict within the same individual over time) was not been able to be examined in the present study. In the next study, I will zoom in to daily lives of chronic pain patients and their partners and observe each couple’s day to day interpersonal and emotional experiences over 22 days. Specifically, the next study will examine whether the fluctuations in positive and negative emotional states of chronic pain patients and different couple interactions affect the sleep quality of the patients. Thus, the next study will provide the chance to compare different daily social and emotional experiences within the same individual and how daily experience affects one of the critical health behaviors, sleep, in different ways.
### Table 2.1 Sample characteristics and mean levels or percentage of key study variables

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Total (N = 56)</th>
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<tr>
<td></td>
<td>M or %</td>
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<tr>
<td><strong>Characteristics</strong></td>
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<tr>
<td>Age (yrs)</td>
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<td>Women (%)</td>
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<td>Cycling status among women</td>
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<td>Luteal (%)</td>
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<td>Follicular (%)</td>
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<td>TNF-α (pg/mL)</td>
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</table>

*Note.* SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate; mmHg = millimeters of mercury; bpm = beats per minute; µg/dL = micrograms per deciliter; pg/mL = picograms per milliliter.
Table 2.2 The level of perceived negativity in each relationship area for the cluster groups, generated (a) by relationships with roommates, family, and friends and (b) by relationships with a romantic partner, family, and friends

(a)

<table>
<thead>
<tr>
<th>Perceived negativity in the relationships with</th>
<th>The Three Cluster Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Low conflict (n = 30)</td>
</tr>
<tr>
<td></td>
<td>M</td>
</tr>
<tr>
<td>Roommates</td>
<td>8.60</td>
</tr>
<tr>
<td>Family</td>
<td>8.60</td>
</tr>
<tr>
<td>Friends</td>
<td>6.53</td>
</tr>
</tbody>
</table>

(b)

<table>
<thead>
<tr>
<th>Perceived negativity in the relationships with</th>
<th>The Two Cluster Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Low conflict (n = 29)</td>
</tr>
<tr>
<td></td>
<td>M</td>
</tr>
<tr>
<td>Romantic partner</td>
<td>7.86</td>
</tr>
<tr>
<td>Family</td>
<td>8.07</td>
</tr>
<tr>
<td>Friends</td>
<td>6.72</td>
</tr>
</tbody>
</table>
Figure 2.1 The three pathways being tested in the first study

Note. $H_{1,2,3}$: Individuals with conflict in more relationships than others will have exaggerated inflammatory response to stress (i.e., poorer recovery) in terms of two cytokines with inflammatory properties, IL-6 and TNF-α.

$H_4$: Individuals with more negative relationships will show greater increases in blood pressure during the stressor, with sustained elevation during the recovery.

$H_3$: Individuals with more negative relationships will show greater increases in cortisol after being exposed to the laboratory stressor.

$H_4$: Hostility will amplify the effects of conflictive relationships on cardiovascular, hormonal, and inflammatory responses.

$H_5$: Depressed mood will also be taken into account in order to verify that the effects of hostility are not driven by higher levels of depressed mood among hostile individuals.
Figure 2.2 Changes in serum IL-6 levels by the negative relationship groups in the three cluster model

Note. LN = Natural Log transformation; the natural log transformed levels of serum IL-6 were presented.
Figure 2.3 (a) systolic and (b) diastolic blood pressure response to stress by the negative relationship groups in the two cluster model

(a)

(b)
Chapter 3

The effects of daily mood and couple interactions on sleep quality and perceived health of individuals with chronic pain

Abstract

Poor sleep quality is associated with symptom severity and pain-related disability among chronic pain patients. Although negative mood and social interactions with an intimate partner appear to influence sleep quality of healthy adults, few studies have examined these associations among arthritis patients or across days. The present study aims to examine the unique effect of daily negative and positive mood on subjective sleep quality of knee arthritis patients (N=152) and whether daily spousal responses moderate this association. Patients (mean age 65.78 ± 9.99, and 58.6% female) and their spouses responded to questionnaires 3 times per day for 22 consecutive days. In addition to patients’ self-reported mood and sleep quality, degree to which spouses reported being empathic, punishing or solicitous toward their spouse was assessed. Controlling for patients’ age, gender, BMI, education, daily pain, comorbidities, and use of sleep medication, multilevel modeling analyses showed main effects of daily negative mood on the same night’s sleep quality and of positive mood on the degree to which sleep was perceived to be refreshing. Interactions between mood and solicitous responses were observed, such that higher solicitousness was associated with poorer sleep quality on the days patients had higher negative mood. In addition, on days when patients’ positive mood was low and partners provided solicitous responses more than usual, patients reported less refreshing sleep. These results demonstrate that negative and positive mood fluctuations can interact with spousal behaviors to affect nightly subjective sleep quality among older adults with knee osteoarthritis. Findings advance our understanding of how emotional and social factors can dynamically affect health.
Introduction

The second study in my dissertation focused on older adults with knee osteoarthritis who are receiving support from their partner. Osteoarthritis (OA) is the most common joint disorder in the world (Arden & Nevitt, 2006). In the United States, 27 million adults have clinical OA, and nearly half of adults are likely to develop OA in their lifetime (Lawrence et al., 2008; Murphy et al., 2008). OA often causes loss of functioning, disability among older adults and is the second leading cause of work disability among adults over 50 years old in the US (Arden & Nevitt, 2006). Because OA is a significant source of pain and interference with daily living, individuals with OA often experience emotional distress and disturbances in sleep and depend on support of a partner to cope with them. The living contexts of individuals with OA and partners thus provide ample opportunity to examine the interaction between mood and couple relationships on sleep of those with chronic pain. I expect that mood and social relationships have a particularly powerful influence on health outcomes among people with chronic pain because their existing pain conditions would make them vulnerable and responsive to certain patterns of emotional distress and social interaction.

This research focuses on the degree to which daily couple interactions moderate the association between daily affective states and daily sleep quality. Thus, it tested several key pathways in Figure 1.1. Specifically, the pathway between mood and sleep quality was examined, as well as the degree to which social relationship dynamics moderate that association.

Sleep, emotion, and pain connections in chronic pain

Poor sleep quality is associated with pain severity and physical disability among people with chronic pain, which is typically defined as pain lasting longer than 3 months (Naughton, Ashworth, & Skevington, 2007). About 50-70% of individuals with chronic pain suffer from substantial sleep disturbances and there is a vicious cycle between poor sleep and heightened pain sensitivity among those
with chronic pain (Smith & Haythornthwaite, 2004). Previous studies have primarily focused on identifying factors that affect sleep quality at the individual level, such as emotional states and various health behaviors. Negative mood, particularly depressive mood, is consistently associated with poor subjective sleep quality, severe pain, and disability among individuals with chronic pain (Naughton, Ashworth, & Skevington, 2007). In contrast, how sleep can be influenced by social interactions with others has not received enough empirical attention (Troxel et al., 2007). According to the 2005 National Sleep Foundation poll, 61% of adults sleep with a significant other, and at least one quarter of them report that their intimate relationships are adversely affected by their own or their partner’s sleep problems (Troxel et al., 2007). Social interactions between individuals with chronic pain and their spouses can influence each other’s sleep quality and other health behaviors (Leonard, Cano, & Johansen, 2006). Despite the known influence of social interactions and mood on the overall health of individuals with chronic pain, few studies have examined how daily stressful and/or supportive couple interactions influence the association between daily mood and sleep quality of those with chronic pain.

**The link between mood and chronic pain**

Previous research suggests that mood disorders, such as depression, are much more prevalent in individuals with chronic pain than in healthy individuals (Robinson & Riley, 1999). In a study with older adults with and without various chronic disease conditions including rheumatoid arthritis (RA) and diabetes mellitus, older adults with a chronic disease condition reported more depressed symptoms than their aged matched counter parts without disease ( Penninx et al, 1998). Adult RA patients with Major Depressive Disorder (MDD) appear to have more severe pain symptoms, disturbed sleep, and increased levels of indicators for cell mediated immunity (e.g., more HLADR T cells in the blood) than RA patients without MDD (Çakibay, 2004).
Not only mood disorders but day to day experiences of negative and positive mood are also closed linked with changes in pain severity and overall health among individuals with chronic pain. Previous studies have also shown that anxiety and fear about pain conditions of those with chronic or acute pain conditions are associated with more attention to pain sensations, worse scores on physical functioning tests, and higher risk of developing of pain related disabilities later (For a review, see Keefe et al., 2004). Although a few studies examined how both positive and negative emotion affects health of those with chronic pain, Zautra and colleagues conducted a series of studies to investigate the role of negative and positive emotion in pain perception among individuals with chronic pain conditions. For example, individuals with rheumatoid arthritis or fibromyalgia who reported negative mood also had elevated levels of pain (Zautra, Smith, Affleck, and Tennen, 2001). In a later study that included weekly measurements of positive and negative mood along with levels of perceived stress and pain, individuals with chronic pain conditions (e.g., rheumatoid arthritis, fibromyalgia, and osteoarthritis) with high average levels of positive mood across the weeks showed lower levels of pain (Zautra, Johnson, and Davis, 2005). Individuals with high average positive affect exhibited a weakened correlation between negative mood and pain and between stressful events and pain compared to those with lower average levels of positive mood. Thus, prior research suggests that positive mood can help people to be more resilient in the face of emotional distress in terms of its effect on pain.

**Social Support Dynamics in Chronic Pain**

As reviewed in the previous chapter, perceived social support in general seems to buffer the deleterious effects of stress from chronic pain conditions on health outcomes such as depression and disabilities. Chronic pain conditions can generate serious psychological distress when people with chronic pain perceive their pain symptoms as severe, incontrollable, unpredictable, and as substantially interfering with life (Banks & Kerns, 1996). By helping change the perception of pain and by increasing resources to
control over pain, social support is likely to reduce stress from the pain condition and in turn promote health of people with chronic pain. Among various kinds of supportive response of family members of those with chronic pain, empathic responses of a partner (e.g., listening to talk about pain; trying to put her/himself in the situation) seem to have beneficial effects on the health of those with chronic pain, although there is a lack of research in effects of empathic responses to pain on health outcomes of those with chronic pain. Specially, individuals with chronic pain appear to greatly benefit from support provision of a partner for their pain behaviors only when the partner has an accurate perception of the pain they experience (Martire et al., 2006). On the other hand, the inaccurate perception of how much pain the patient is experiencing can lead the partner to provide support that is not matched with what they need, which is shown to aggravate the health of pain patients (Turk, Kerns, and Rosenberg, 1992).

In addition to the support provider’s inaccurate evaluation of how much and what kind of support is needed to the patients, there are two other mechanisms that explain how even well intended social support can aggravate the health of chronic pain patients. First, there is also a large literature on how receiving attention from family about pain behaviors may actually reinforce chronic pain patients to engage in more pain behaviors, on the basis of operant conditioning theory and family system theory. Receiving help and attention for pain conditions from close others such as a spouse or parents may provide rewards to the patients that may lead to increased pain behaviors to gain further care and attention from them. This vicious cycle has been applied to explain the adverse impact of solicitous responses to pain behaviors (e.g., taking over her/his duties; getting her/him to take rest) on health of chronic pain patients. Previous studies show that spouses’ solicitous responses to pain behaviors are associated with more observed pain behaviors as well as higher levels of pain intensity (Turk, Kerns, and Rosenberg, 1992). Also, people with chronic pain reported to experience mixed feeling about receiving solicitous responses—feeling cared but also feeling guilty and becoming a burden for the support provider (Newton-
John & Williams, 2006). Such research indicates that even well intended supportive behaviors may not benefit the individuals with chronic pain if the support reinforces their pain behaviors or discourage them from pursuing the day to day activities despite the pain.

Negative social interactions and unsatisfying social relationships are persistently associated with negative health outcomes among those with chronic pain. Negative social interactions are associated with more psychological distress, pain severity, disability, and later onset of depressive disorders among those with chronic pain (Leonhard, Cano, & Johanson, 2006 for a review). Sometimes when an individual expresses that they are in pain (with words or behaviors) they receive what are called “punishing responses” from someone to whom they are close, such as a spouse. Punishing responses to pain expression/behaviors involve ignoring and angry or irritated reactions. Punishing responses of close others are likely to trigger psychological distress in individuals with chronic pain. Further, disclosure and validation of the pain experience can be suppressed by receiving punishing responses from close others. The lack of disclosure and validation in on-going pain experiences induced by punishing responses of close others can generate yet more stress from pain symptoms and prevent the person with pain from utilizing collective coping strategies for managing chronic pain conditions. This pattern of behaviors may then lead to aggravated pain, sleep quality, and overall health status (Leonhard, Cano, & Johanson, 2006; Lazarus & Folkman, 1984).

**Potential interaction between mood and couple relationships in affecting health**

It appears likely that mood and social interactions dynamically interact and jointly affect physical health outcomes of individuals with chronic pain by affecting their psychological distress. As illustrated in Figure 1.1, supportive and negative social interactions can become a source of positive and negative mood of those with chronic pain and thus help determine the relative amount of psychological distress that an individual perceive. Previous studies of healthy adults showed that negative social interactions
within intimate relationships are associated with depressed mood and later onset of a depressed disorder (Shuster, Kessler, & Aseltine, 1996; Joiner & Timmons, 2009). In addition, individuals’ level of positive and negative mood can be either amplified or reduced by subsequent social interactions (Cohen, 1996), thus also manipulating social stress and available social support as suggested in Figure 1.1. For example, having supportive and empathic responses from a spouse may reduce the level of negative mood generated by stressful situations, whereas having negative or overly protective solicitous responses from a spouse is likely to amplify the level of negative mood and stress.

**Purpose of the study**

For this study, I examined how daily negative and positive mood affect daily sleep quality and perceived health status of individuals with knee osteoarthritis and how the association is further influenced by the three different types of daily spousal responses to pain behaviors. The pathway in the model that is related to each hypothesis is labeled in Figure 3.1. Previous literature suggests that negative emotional states reflect the perceived psychological distress of those with chronic pain, which may penetrate into night time and disturb their sleep. On the other hands, positive emotional states are shown to buffer the effects of stressful events on sleep quality and are expected to enhance sleep quality of those with chronic pain. Specifically, I predicted that:

$H_1$: Daily negative mood will predict worse sleep quality the next day of individuals with knee osteoarthritis (path ‘$H_1$’ in Figure 3.1)

$H_2$: Daily positive mood will predict better daily sleep quality of individuals with knee osteoarthritis (path ‘$H_2$’ in Figure 3.1)

Next, I also examined whether spousal responses to pain behaviors (i.e. solicitous, empathic, and punishing response) account for the association between mood and sleep. Due to the lack of previous
research on positive emotional states, the specific patterns of interactions with spousal responses to pain were only established for negative emotional states. Previous literature indicated the adverse impact of spousal solicitous and punishing responses to pain on health of chronic pain patients. Thus, solicitous and punishing responses are expected to amplify the adverse effects of negative emotional states on sleep of those with chronic pain. However, spousal empathic responses to pain behaviors are expected to benefit the patients and mitigate the impact of negative emotional states on sleep of chronic pain patients. The following three hypotheses present specific interactions that were examined.

H₃: Daily spousal solicitous response will amplify the effect of daily negative mood on daily sleep quality of individuals with knee osteoarthritis (path ‘H₃’ in Figure 3.1)

H₄: Daily spousal punishing response will amplify the effect of daily negative mood on daily sleep quality of individuals with knee osteoarthritis (path ‘H₄’ in Figure 3.1)

H₅: Daily spousal empathic responses will reduce the effect of daily negative mood on daily sleep quality of individuals with knee osteoarthritis (path ‘H₅’ in Figure 3.1)

Method

Study Design

Data used in the present study are from a larger study on the effects of couple interaction on physical activities of older adults with knee osteoarthritis (OA) (L. M. Martire et al., 2013). The larger study involved three surveys conducted during in person interviews and 22 days of daily diary measurements immediately after the first survey. The current research utilized the data from the first survey and the diary measurements.
Participants

One hundred and fifty two patients with knee osteoarthritis and their partners (i.e., 304 individuals) participated in the main study. The age range of the patients was 50-95 with mean age 65.78 ± 9.99 years, and 58.6% of the patients were women. In order to be eligible for the study, the patients had to have been diagnosed with knee OA by a physician, to have experienced moderate to severe intensity of arthritis pain, and to be married or in a long-term relationship in which they share the same residence with their partner. The exclusion criteria for patients include comorbidity with fibromyalgia or rheumatoid arthritis, use of wheelchair to get around, and a plan for hip or knee surgery within the next 6 months. Couples were also excluded from participation if their partner had arthritis with moderate to severe intensity of pain, used a wheelchair, or required assistance for personal care. Both members of the couples were cognitively functional and were free of any major hearing, speech, or language problems. Participants were recruited via research registries for rheumatology clinic patients and older adults interested in research, flyers distributed to University of Pittsburgh staff and faculty, and word of mouth. Of six hundred and six couples who were contacted, 221 couples declined to participate and 233 couples were not eligible primarily due to their lack of OA on knee and less than moderate levels of pain intensity. Among 152 couples, 143 couples completed the diary assessment of the study, mostly heterosexual couples but including three same sex couples. The demographic characteristics of the patients and the partners are presented in Table 3.1.

Procedures

A trained research assistant visited the participating couples in their home for the first survey and conducted separate interviews for the patient and the partner. During the interview, couples were asked to respond to a set of questionnaires to measure, among other things, health status, physical comorbidity, and arthritis symptoms. Couples were also trained to use a hand-held computer (the Palm TX) for daily
diary measurements, which was clearly labeled with each participant’s name. Participants were asked to respond to the dairy questionnaires three times per day; 1) within 60 minutes of rising in the morning (i.e., beginning-of-day), 2) between 2:00 and 4:00 p.m. (i.e., afternoon), and 3) upon retiring at night (i.e., end-of-day). The current mood and the level of pain were assessed three times per day; social interactions with a partner were measured only at the end of day survey. The items on sleep were measured only at the morning survey. Couples were instructed to follow the timeline of the surveys each day and provide independent responses from their partner.

**Measures**

**Positive and negative mood.** Positive and negative mood was measured three times per day with 9 item-adjective mood ratings (Thomas and Diener, 1990). Patients were asked to what extent they had each of nine positive or negative moods (e.g., happy, angry, and frustrated) over the past 30 minutes on a 6 point scale from not at all to extremely. The sum of four items assessing positive mood and five items assessing negative mood was calculated, and mood from the end of the day measure was used for the analyses.

**Partner’s responses to pain behaviors.** The three different types of responses of the partner to the patients’ pain behaviors (e.g., solicitous, punishing, and empathic responses) were assessed at the end of the day. The items for solicitous and punishing responses were modified from the subscales of The West Haven-Yale Multidimensional Pain Inventory (WHYMPI; Kerns & Turk, 1985) and those for empathic responses from the questionnaire developed by Stephens and colleagues (Stephens, Martire, Cremeans-Smith, Druley, & Wojno, 2006). Partners indicate how often they responded to the patient in certain ways today when they sensed that their partner was in pain, using a three point scale from 1 = not at all to 3 = very much. The items assessing solicitous responses include taking over his/her duties and getting her/him to rest, whereas the items for punishing responses involve ignoring and getting angry. Empathic responses of the partner were measured by items like “how often did you listen to what the patient was
feeling?” The solicitous responses that were reported by partners were used in this study in order to obtain the objective measurements for how frequently solicitous responses were provided to pain patients, without bias induced by patients’ own mood or levels of stress.

**Sleep quality.** Daily sleep quality was measured by two items. The first item was adapted from the subjective sleep quality item in the Pittsburgh Sleep Quality Index (Buysse et al., 1989), which asks ‘how would you rate the quality of your sleep last night’ on a scale from 0 = Very good to 3 = very bad). The second item asked for ratings of how refreshing sleep was the night before on a scale from 0 = not at all to 6 = extremely. The second item used the subjective sleep item of PSQI, which provides a measure global sleep quality that has been validated for assessing global appreciation of sleep (Akerstedt, Hume, Minors, & Waterhouse, 1994).

**Daily pain.** Patients’ reports of pain or tenderness in 10 different joints a scale from 0 = no pain/tenderness to 3 = severe pain/tenderness were collected at the end of day using the Rapid Assessment of Disease Activity in Rheumatology (RADAR; Mason et al., 1992). The RADAR has a good convergent validity with clinician’s assessment of pain symptoms and is sensitive to changes in the levels of pain over time (Mason et al., 1992).

**Covariates.** Age, gender, Body Mass Index (BMI), years of education, cormorbid conditions, and daily pain were included in the present analysis as covariates. Years of education were calculated by the number of years in schools based on the highest degree of participants. Cormorbid conditions were measured by asking participants if they had ever been told that they have chronic conditions included high blood pressure, coronary artery disease, ulcer, diabetes, cancer, etc. The daily levels of pain at the end of each day were assessed by asking participants how would they describe the pain/tenderness in their 10 different joints in the past 30 min on a 4 point Likert scale from 0 = none to 3 = severe.
Data Analysis

All the analyses were conducted using SAS 9.3. Preliminary descriptive analyses for means, standard deviations, and bivariate correlations were conducted on key study variables. Next, multilevel modeling analysis (MLM) were applied to examine daily fluctuations in mood, spousal responses to pain behaviors, and sleep quality over time and how they are related to perceived health status using SAS PROC MIXED.

There are a number of reasons why MLM analysis is appropriate to use with daily diary data. First, it is designed to analyze data that violate the assumption of independent observations (Smith, 1992). This is important for the present study because it included repeated measurement of study variables three times a day for 22 days, thus observations within the same individual are related to each other. Also, the observations that were measured at closer time points (e.g., pain perception at day 1 and day 2) may be more strongly associated to each other than observations at more distant time points (i.e. pain perception at day 1 and day 16). MLM can deal with this kind of interdependence (or nested structure) by defining the error structure of the estimated model to reflect the repeated measurement design of data and by adjusting for the possible temporal association between closer time points, thereby providing unbiased estimations (Bolger, Davis, & Rafaeri, 2003; Singer, 1998). In order to do that, this study will set up a comparison for chi-square likelihood test scores between a model with unstructured covariance structure, in which the random intercepts are allowed to be correlated, and with an autocorrelation structure that assumes the correlation between proximal time points. If the model with the autocorrelation (AR) structure fits better with data than the model with unstructured covariance, the temporal association between nearby time points would be assumed and the AR structure would be selected for the final model. In addition, the diary design using MLM analysis is superior to traditional designs in analyzing within-person processes over time. Using MLM analysis with diary data “helps determine the antecedents, correlates, and consequences of daily experiences” and allows us to use participants as their own control
(Smith, 1992). For instance, it is possible to examine the within subject association between negative mood at time 1 and sleep at time 2 (or lagged time point), controlling for each participant’s level of pain or negative mood at time 2.

The multilevel model of this study will be defined by a level 1 equation with within- subject variables and level 2 equations with between subject variables. First, the level 1 equation will examine whether daily negative or positive mood predicts sleep quality and refreshing sleep and whether one of the three spousal responses moderates the association between mood and sleep measures. Regarding the timing of the measures, the evening measures of mood and spousal responses and the next day morning report of the sleep measures will be utilized. To examine the main effect of negative or positive mood on sleep quality, daily negative and positive mood will be entered at level 1 to predict sleep quality and refreshing sleep. The interaction between mood and spousal responses will be examined by adding the interaction term between negative or positive mood and one of the three spousal responses to the level 1 of the MLM model. Then, if there is a significant interaction effect, the pattern of the interaction will be further examined by comparing the slope estimate and the standard error of negative or positive mood when fixing the spousal response at +1 standard deviation (SD) above and -1 SD below the average. The pattern of interactions will be plotted for the significant moderating effects. In all MLM analyses of this study, the level 2 equation will include controlling variables such as gender, age, education, physical comorbidity, and use of sleep medication. The daily level of pain will also be controlled for in the analyses and will be entered at level 1. Regarding to centering of the variables, daily measures of this study will be centered with within-person centering and level 2 covariates will be grand mean centered. Because I utilized reports by patients and their spouse, each dyad of a couple was used as the unit for all of the within person analyses, including within person correlations and multilevel analyses. The
exemplary SAS codes that were used in the analyses were provided in the Appendix N. The exemplary equations for the MLM analyses and SAS code are listed below.

**Equation for the main effect of negative mood and solicitous response on sleep quality**

Level 1: \( \text{Sleep}_i = \beta_0 + \beta_1 \text{Negative}_i + \beta_2 \text{Positive}_i + \beta_3 \text{age}_j + \beta_4 \text{gender}_j + \beta_5 \text{education}_j + \beta_6 \text{BMI}_j + \beta_7 \text{Comorbidity}_j + \beta_8 \text{pain}_i + r_{ij} \)

Level 2: \( \beta_0 = \gamma_{00} + u_{0j} \)

\( \beta_1 = \gamma_{10} \)

\( \beta_2 = \gamma_{20} \)

\( \beta_3 = \gamma_{30} \)

\( \beta_4 = \gamma_{40} \)

\( \beta_5 = \gamma_{50} \)

\( \beta_6 = \gamma_{60} \)

\( \beta_7 = \gamma_{70} \)

In this equation, sleep_quality for person \( j \) at time \( i \) is a function of a person’s intercept, their daily deviation in negative mood at time \( i-1 \), and their daily deviation in positive affect at time \( i-1 \) after controlling for age, gender, years of education, BMI, comorbid conditions, daily level of pain, use of sleep medicine and the levels of positive and negative mood when participants report sleep quality. The fixed effects for both of negative mood and positive mood are going to be estimated in addition to the estimated fixed effects of covariates. The random intercept \( (u_{0j}) \) for sleep quality means that the model is going to estimate how much individuals differ with each other for their baseline levels of sleep quality; in other words, the baseline sleep quality is allowed to be random across individuals.
Equation for the interaction effect between negative mood and solicitous response

Level 1: \( \text{Sleep}_i = \beta_0i + \beta_1i \text{Negative}_i + \beta_2i \text{Solicitous}_i + \beta_3i \text{Age}_i + \beta_4i \text{Gender}_i + \beta_5i \text{Education}_i + \beta_6i \text{BMI}_i + \beta_7i \text{Comorbidity}_i + \beta_8i \text{Pain}_i + \beta_9i \text{Sleep_medicine}_i + e_i \)

\( \text{Negative}_i * \text{Solicitous}_i + \beta_1i \text{Age}_i + \beta_2i \text{Gender}_i + \beta_3i \text{Education}_i + \beta_4i \text{BMI}_i + \beta_5i \text{Comorbidity}_i + \beta_6i \text{Pain}_i + \beta_7i \text{Sleep_medicine}_i + e_i \)

Level 2: \( \beta_{0i} = \gamma_{00} + \upsilon_{0i} \)

\( \beta_{1i} = \gamma_{10} \)

\( \beta_{2i} = \gamma_{20} \)

\( \beta_{3i} = \gamma_{30} \)

\( \beta_{4i} = \gamma_{40} \)

\( \beta_{5i} = \gamma_{50} \)

\( \beta_{6i} = \gamma_{60} \)

\( \beta_{7i} = \gamma_{70} \)

\( \beta_{8i} = \gamma_{80} \)

The equation for examining the interaction between negative mood and solicitous spousal responses is similar to that for the main effect model except that the interaction term \( \text{Negative}_i * \text{Solicitous}_i \) was included to the equation in addition to the main effect terms of negative mood and solicitous response.

Results
**Preliminary analysis**

The descriptive statistics results using person mean variables showed that knee OA patients reported fairly good quality of sleep on the average across days ($M = 0.97$, $SD = 0.43$, range = 0-2.50) and feeling moderately refreshed after sleep ($M = 3.57$, $SD = 1.01$, range = 0.72-6.00). Levels of average daily positive emotional state were higher than that of their average negative emotional state ($M = 10.81$, $SD = 5.69$ for positive emotional state; $M = 2.50$, $SD = 3.62$ for negative emotional state). Partners of knee OA patients reported using daily empathic response the most frequently ($M = 5.24$, $SD = 3.62$), followed by solicitous and punishing response ($M = 4.41$, $SD = 1.15$; $M = 3.26$, $SD = 0.62$, respectively). Patients, on average, reported daily experiences of substantial pain and tenderness on at least two loci of the 11 parts of the body asked about on the scale, with mean levels of 5.88 and $SD = 4.73$.

The within person correlation was estimated among the study variables to show how much each pair of two daily variables covary over time. As shown in Table 3.2, all correlations were significant except the correlation between feeling refreshed after sleep and spouse empathic response. However, significant but weak correlations were not interpreted as meaningful as they were calculated by using a large number of the observations (no. of observations used = 3821). Particularly strong within person correlations were observed between daily negative and positive emotional state ($r = -.42$, $p < .01$), between solicitous and empathic response ($r = .40$, $p < .01$), and between sleep quality and feeling refreshed after sleep ($r = -.65$, $p < .01$).

**Main effect of daily positive and negative emotional states**

Table 3.3 presents the coefficients for the fixed effects for positive and negative emotional states and covariates in the multilevel model. Daily negative emotional state was associated with lower daily sleep quality, such that on days when those with knee OA experienced one SD higher level of negative emotional state than usual, they reported 0.016 times worse sleep quality. Daily positive emotional state
was associated with more refreshing feeling after sleep, such that on days when those with knee OA experienced one SD higher levels of positive emotional state than usual, they reported 0.025 times more refreshing sleep. Among the covariates, only use of sleep medication was significantly associated with lower sleep quality.

**Interaction between daily affect and three spouse responses to pain**

There was significant interaction between daily positive emotional state and daily spouse solicitous response to predict feeling refreshed after sleep (Estimate = 0.019, \( p < .05 \)). The estimates of predictors including the interaction term and covariates for the model are presented in Table 3.4. As shown in Figure 3.2, daily spousal solicitous responses appear to amplify the positive association between daily positive emotional state and feeling refreshed after sleep. Participants reported more refreshed after sleep when they felt higher levels of positive emotional state than average or lower levels of positive emotional state. In addition, receiving more than usual solicitous responses from a spouse further strengthened the association between the levels of daily positive emotional state and feeling refreshed after sleep; when patients experienced more positive emotional states and also receiving more solicitous responses from a spouse than on usual days, patients reported feeling more refreshing after sleep than when they experienced only positive emotional states without receiving solicitous responses.

There was a marginally significant interaction between daily negative affect and spouse solicitous response to predict sleep quality (Estimate = 0.013, \( p = .06 \)). Table 3.5 presents the estimates of predictors including the interaction term and covariates for the model. As shown in Figure 3.3, having more than usual daily spouse solicitous response enlarged the adverse effect of daily negative emotional state on sleep quality. Patients reported poorer sleep quality on days when they experienced higher negative emotional state than usual. Receiving more solicitous response from spouse further amplified the differences in sleep quality between days with high and low negative emotional state.
Discussion

Utilizing a daily diary survey of chronic pain patients and their partners across a 14 day period, this chapter examined how daily negative and positive emotional states of chronic pain patients and three different partner responses to pain (solicitous, empathic, and punishing response) predict two indicators of sleep (self-reported sleep quality and feeling refreshed after sleep). As expected, negative emotional states were associated with adverse effects on sleep, whereas positive emotional states were associated with the benefiting effects on sleep among older adults with chronic pain. This study advances the existing literature on emotion, social relationships, and health of those with chronic pain by showing that the way in which a romantic partner responds to pain behaviors of a chronic patient can interact with that person’s daily emotional states to predict his or her sleep quality. By applying the daily diary design and multilevel modeling analysis, this study revealed how day to day fluctuations of emotional states within each chronic pain patient and their partner’s responses to their pain behaviors affected the same night’s sleep quality for the patients. This approach has particular strength in two different ways. First, the results are robust from many potential confounding factors that differently affect each individual because different days of the same individual were compared to each other to find the resulting patterns of associations, as opposed to comparing patterns between different individuals. Second, the emotional states of each day were coupled with the same day’s partner responses to predict the sleep quality of that night, such that it was possible to examine close temporal associations between emotional states, spousal response, and sleep.

The main effects of daily positive and negative emotional state on sleep of those with chronic pain

As predicted in hypothesis 1 (H1), negative emotional state of chronic pain patients on a certain day significantly influenced their subjective ratings of sleep quality of the night of the same day: On the days when chronic pain patients reported experiencing more negative mood than usual for them, they had
poorer sleep quality that night. These results are consistent with previous literature showing adverse effects of negative emotion on quality and duration of sleep among people with and without chronic pain (Harris & Dawson-Hughes, 1993; Naughton et al., 2007; Waters, Adams, Binks, & Varnado, 1993b). However, this study replicated the finding in a more subtle way by showing the daily association between emotional states and sleep within the context of older adults. The results indicate that experiences of negative emotion during daytime can carry over to the night and affect sleep quality of those with chronic pain, which may in turn exacerbate the pain symptoms the next day.

Daily positive emotional state was associated with the daily experiences of refreshing feeling after sleep, which supports what was hypothesized in H2. People with chronic pain reported more refreshed after sleep on the days when they felt more positive emotion than usual for them. This is in concordance with previous studies on the association between daily positive emotional states and chronic pain showing that daily positive emotional state predicts less pain symptoms among women with rheumatoid arthritis and fibromyalgia (Strand et al., 2006; Zautra, Johnson, & Davis, 2005). Positive emotional state may promote more refreshing sleep by reducing pain perception of pain symptoms among those with chronic pain. Also, previous research suggests that the subjective feeling of refreshment after sleep may tap into different patterns of sleep than evaluating the overall quality of sleep. Further feeling refreshed was predicted only by daily positive emotional state after controlling for negative mood and other covariates. Previous studies on the correspondence between subjective measures of sleep quality and objective measures of sleep suggest that the rating of sleep quality is correlated with the total hours of sleep, whereas feeling refreshed after sleep was linked with how fragmented the sleep was (Åkerstedt, Hume, Minors, & Waterhouse, 1997; Martire, Keefe, Schulz, Parris Stephens, & Mogle, 2013). Thus, it may be that daily experiences of positive mood, independently from negative mood, are associated with better and less fragmented sleep among chronic pain patients.
Moderation by spouse’s solicitous response to pain behaviors

The association between daily negative emotional state and daily sleep quality was further amplified when partners reported that they gave solicitous responses to the patients’ pain behaviors, which is in concordance with H₃. There was a stronger association between higher levels of daily negative emotional state and poorer sleep quality on the days the partner reported providing more solicitous responses than usual for them. Similarly, higher than usual solicitous responses of partners strengthened the association between patients’ daily positive emotional state and feeling more refreshed after sleep: The degree to which high levels of daily positive emotional state predicted daily experience of more refreshing sleep was stronger on the days when patients’ high positive emotional state was combined with partners’ report on providing more solicitous responses than usual.

The finding that partners’ solicitous responses can aggravate the impact of negative mood on disturbed sleep is consistent with previous research on solicitousness. For example, solicitous responses—characterized by helping behaviors such as getting patients medication and taking over the tasks of patients—are associated with pain severity/physical disability of chronic pain patients (Flor, Turk, & Rudy, 1989; Kerns, Haythornthwaite, Southwick, & Giller Jr, 1990; Lousberg, Schmidt, & Groenman, 1992; Romano et al., 1995). The present study advances the literature by showing a complex interaction between mood, solicitousness, and sleep at a daily process level among older adults with chronic pain and their partners. There are two possible explanations for the additive effects of having both poor mood (either negative mood or lack of positive mood) and receiving more solicitous responses from one’s spouse. First, previous literature based on the operant conditioning theory suggests that receiving solicitous responses may aggravate pain by providing positive reinforcement (by providing more attention and helping behaviors in response to pain behaviors) for pain behaviors (for a review, see Leonard et al., 2006). Those with chronic pain may continue and even increase the pain behaviors in response to this
kind of reinforcement from their partner, which can increase pain sensitivity and thus generate more negative mood of those with chronic pain. Second, solicitous responses seem to be negatively perceived by those with chronic pain. A qualitative study in which chronic pain patients and their partners were interviewed demonstrated that people with chronic pain consistently favored partners’ responses that helped or encouraged them to persist with tasks despite pain, compared to solicitous responses such as simply offering help or taking over the tasks (Newton-John & Williams, 2006). While pain patients felt cared for when they received solicitous responses from their partner, also often they felt guilty and like they were a burden.

It is also possible that the spouse who delivers solicitous responses may do that with negative attitude that generates distress for those with chronic pain. Newton-John and colleagues study (2006) found that solicitous responses were sometimes linked with aggression or frustration of a partner and have classified that into ‘hostile solicitous response’ separately from other solicitous responses. Interestingly, in their study patients reported higher frequencies of receiving hostile solicitous responses than the partner recognized that they did, which suggests that patients may perceive substantial negativity around solicitousness. Therefore, previous literature provides two explanations why partner’s solicitous response amplified the effects of daily mood on sleep in this study; solicitousness may be accompanied with a negative attitude of a partner or solicitousness may lead pain patients to increase pain behaviors.

The specific interaction between daily positive emotional state and solicitous response, where solicitous responses in combination with positive states appeared to strengthen the association between positive mood and feeling refreshed after sleep was not expected. Although solicitous spousal response to chronic pain patients appears to have adverse effects on pain symptoms (Leonard, Cano, & Johansen, 2006), the results suggests that solicitous response can strengthen the beneficial effects of patients’ positive emotional states on feeling refreshed after sleep. It is possible that patients’ own positive mood
may lead them to perceive the solicitous response in a pleasant way. Also, patients’ positive emotion may well be transmitted to their spouse and generate a greater positive emotional state for the spouse (Larsen & Almeida, 1999), which may have influenced how positive the spouse was when delivering the solicitous response to patients.

Contrary to expectations that were specified in $H_4$ and $H_5$, neither daily punishing nor empathic responses modulated the effects of daily mood on either indicator of daily sleep. It should be noted that the reports by partners (as opposed to reports from chronic pain patients) about how much they provided punishing and empathic responses to pain behaviors were utilized in this study. Several studies reported that there are discrepancies between those with chronic pain and the partner in reporting different responses to pain behaviors (Lousberg et al., 1992; Pence, Cano, Thorn, & Ward, 2006). It appears that chronic pain patients’ own perception of punishing response from the spouse is associated with pain severity rather than partners’ report on how frequently they responded to pain behaviors in punishing ways (Pence et al., 2006). Therefore, studies that compare reports of patients and their spouses imply that future studies are needed to examine whether patients’ perception of spousal punishing response would amplify the adverse effects of daily negative emotional states on the same night’s sleep of chronic pain patients.

The associations between mood and sleep were not influenced by empathic responses because the partner consistently provided empathy toward pain, regardless of the mood status of those with chronic pain. Regarding the empathic response, the present results showed that empathic responses of the partner seem to be the most frequently used way to respond to the pain behaviors of the spouse with chronic pain among the three responses examined.

Although it was not tested in this study, there may be an alternative direction of association between sleep, emotional state, and partner’s response. For example, some recent studies have examined
the bi-directional association between daily mood and sleep and suggested the effects of sleep deprivation on increased negative emotional states the next day (O'Brien et al., 2011). Also, sleep deprivation was associated with impaired ability to recognize and respond to other’s emotion. It can be inferred that poor sleep quality of chronic pain patients may lead to increased negative emotional states and more negative social interactions with a partner the next day. However, the direction of associations that were tested in this study was set up by the social stress/buffering hypothesis that focuses on the role of social interactions in either exacerbating or mitigating the impact of psychological distress on health. Future studies are expected to reveal how poor sleep of chronic pain patients may affect their subsequent emotional states and social interactions by testing the alternative direction.

**Limitations**

This study has several limitations. The present study focuses on examining subjective measure of sleep quality, which may be influenced by reporting bias, and did not include the assessment of objective sleep measures. However, the items that were used in this study are proven to be effective at identifying individuals with clinical insomnia (Alsaadi et al., 2013) and are moderately correlated with physiological measures of sleep, including EEG measures of different stages of sleep and changes in the body temperature during sleep (Akerstedt et al., 1994). Also, the self-reported measure for sleep allowed the repeated measure for 22 days in the natural environment of participants, which enhances the generalizability of the results, compared to the laboratory studies that involve objective measure of sleep quality such as sleep polysomnography. Moreover, self-reported sleep is predictive of major chronic disease conditions (Chien et al., 2010; Troxel et al., 2010) and subjective feelings after sleep (such as feeling refreshed) may be particularly important for psychological well-being (Kawada, 2012). Future studies would benefit from utilizing both subjective and objective sleep quality, if it is feasible to measure objective sleep quality non-invasively and over time in naturalistic settings.
In addition, spousal report of their own behaviors in response to patients’ pain were used in this study, as opposed to patients’ own report of their perceptions of their spouses’ responses. There were advantages to this approach, which minimizes reporting bias. However, as discussed above, the impact of negative spousal response to pain may depend on how much patients perceive negativity in their spouse’s behavior or attitudes. Thus, adding patients’ own perception for the solicitous, punishing, and empathic response of a spouse in future research may further enrich understanding of how mood and spousal behaviors interact to affect sleep of those with chronic pain.

**Conclusion**

This research examined the dynamic interaction of daily mood and partner responses to pain behaviors on health status and daily sleep quality of older adults with knee osteoarthritis. These types of interactions have not been studied with couples’ daily experiences over time. Day to day fluctuations of sleep quality are associated with aggravated pain symptoms of chronic pain patients and thus are regarded as an important target for biobehavioral interventions for enhancing health and well-being. The present results support pathway ‘a’ shown in Figure 3.1 by indicating that daily fluctuations in negative and positive mood of chronic pain patients can be carried over to that night’s sleep and perceptions of that sleep. The present results also support pathway ‘b’ in Figure 3.1 by illustrating how partners of chronic pain patients can influence mood and sleep connections via their responses to pain behaviors. Among the three kinds of partner’s responses that were examined (solicitous, empathic, and punishing), daily solicitous response to pain behaviors appeared to amplify the effect of daily negative emotional states on poorer sleep quality and the effect of low daily positive emotional states on less refreshing sleep. Thus, this study indicates that the daily patterns of sleep of chronic pain patients may be improved by helping them regulate their mood and by providing partners with advice about how to use solicitous response to pain behaviors in more discrete and effective ways.
This study provided an in-depth analysis of how relationship dynamics and emotional states both individually and in tandem influence an important health behavior (sleep) that is strongly linked with overall physical health. However, this study was unable to identify the physiological mechanisms underlying how mood and couple dynamics get under the skin and influence physical health outcomes across different age groups. In the next chapter, I examine an important potential physiological mechanism underlying the impact of psychosocial processes on physical health outcomes, including markers of inflammation and numbers of physical symptoms/chronic conditions in addition to sleep. Specifically, the next chapter reports on how negative marital relationships affect physical health outcomes and whether the marital relationships account for the association between depressive symptoms and later physical health outcomes using longitudinal data of married adults.
Table 3.1 Demographic characteristics of participants and their partners (N = 143)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Individuals with knee OA†</th>
<th>Partners</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M (SD) or %</td>
<td>M (SD) or %</td>
</tr>
<tr>
<td>Age</td>
<td>65.5 (9.6)</td>
<td>65.4 (11.4)</td>
</tr>
<tr>
<td>Gender = Men</td>
<td>42%</td>
<td>58%</td>
</tr>
<tr>
<td>Years of education</td>
<td>16.0 (2.0)</td>
<td>15.8 (2.1)</td>
</tr>
<tr>
<td>Race = Caucasian</td>
<td>87.4%</td>
<td>86%</td>
</tr>
<tr>
<td>Years married/in relationship</td>
<td>34.6 (16.6)</td>
<td></td>
</tr>
<tr>
<td>Mean range for household income</td>
<td>$40,000-59,999</td>
<td></td>
</tr>
<tr>
<td>Employed</td>
<td>42%</td>
<td>45.5%</td>
</tr>
<tr>
<td>Body mass index</td>
<td>31.3 (5.9)</td>
<td>28.7 (4.7)</td>
</tr>
<tr>
<td>Duration of knee OA (years)</td>
<td>12.8 (11.4)</td>
<td></td>
</tr>
</tbody>
</table>

Note. OA = osteoarthritis. The employment status was asked by whether a participant is currently working for pay.
Table 3.2 Within dyad correlations among daily measures of positive and negative emotional states, sleep quality, feeling refreshed after sleep, spouse punishing, solicitous, and empathic responses

<table>
<thead>
<tr>
<th></th>
<th>Daily PE</th>
<th>Daily NE</th>
<th>Sleep quality</th>
<th>Refreshed after sleep</th>
<th>Punishing response</th>
<th>Solicitous response</th>
<th>Empathic response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daily PE</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Daily NE</td>
<td>-.42**</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleep quality</td>
<td>-.03**</td>
<td>.03**</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Refreshed after sleep</td>
<td>.08**</td>
<td>-.07**</td>
<td>-.65**</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Punishing response</td>
<td>-.03**</td>
<td>-.04**</td>
<td>.18**</td>
<td>.06**</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Solicitous response</td>
<td>.003**</td>
<td>.05**</td>
<td>.05**</td>
<td>-.02**</td>
<td>-.01**</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Empathic response</td>
<td>.053**</td>
<td>.01**</td>
<td>.04**</td>
<td>.02</td>
<td>-.02**</td>
<td>.40**</td>
<td>1</td>
</tr>
</tbody>
</table>

*Note. PE = Positive emotional states, NE = Negative emotional states

*p < .05; **p < .01; ***p < .001
Table 3.3 The main effect model for the effects of daily positive and negative affect on sleep quality and feeling refreshed after sleep

<table>
<thead>
<tr>
<th>Fixed effects</th>
<th>Sleep quality</th>
<th>Feeling refreshed after sleep</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Estimate</td>
<td>SE</td>
</tr>
<tr>
<td>Intercept</td>
<td>.81***</td>
<td>.12</td>
</tr>
<tr>
<td>Age</td>
<td>-.001</td>
<td>.004</td>
</tr>
<tr>
<td>Gender</td>
<td>.07</td>
<td>.08</td>
</tr>
<tr>
<td>Comorbidities</td>
<td>-.03</td>
<td>.02</td>
</tr>
<tr>
<td>Daily pain</td>
<td>-.003</td>
<td>.01</td>
</tr>
<tr>
<td>Years of education</td>
<td>-.01</td>
<td>.01</td>
</tr>
<tr>
<td>Body Mass Index</td>
<td>.002</td>
<td>.01</td>
</tr>
<tr>
<td>Use of sleep medicine</td>
<td>.15*</td>
<td>.08</td>
</tr>
<tr>
<td>Daily PE</td>
<td>-.001</td>
<td>.01</td>
</tr>
<tr>
<td>Daily NE</td>
<td>.02*</td>
<td>.01</td>
</tr>
</tbody>
</table>

Note. $N_{\text{patients}} = 134, N_{\text{observations}} = 1607$. Gender: 1 = men, 2 = women, PE = Positive emotional states, NE = Negative emotional states

*p < 0.05; **p < 0.01; ***p < .001
Table 3.4 The interaction effect model for the interaction between daily positive affect and daily spouse solicitous response on feeling refreshed after sleep

<table>
<thead>
<tr>
<th>Fixed effects</th>
<th>Feeling refreshed after sleep</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Estimate</td>
</tr>
<tr>
<td>Intercept</td>
<td>3.61</td>
</tr>
<tr>
<td>Covariates</td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>.03</td>
</tr>
<tr>
<td>Gender</td>
<td>.06</td>
</tr>
<tr>
<td>Cormorbid health conditions</td>
<td>-.03</td>
</tr>
<tr>
<td>Daily pain</td>
<td>-.01</td>
</tr>
<tr>
<td>Years of education</td>
<td>.05</td>
</tr>
<tr>
<td>Body Mass Index</td>
<td>-.02</td>
</tr>
<tr>
<td>Use of sleep medication</td>
<td>-.25</td>
</tr>
<tr>
<td>Daily PE</td>
<td>.03**</td>
</tr>
<tr>
<td>Daily solicitous response</td>
<td>-.05</td>
</tr>
<tr>
<td>Daily PE × solicitous response</td>
<td>.02*</td>
</tr>
</tbody>
</table>

*N* _patients_ = 134, *N* _observations_ = 1548. Gender: 1 = men, 2 = women, PE = Positive emotional states

*p < 0.05; **p < 0.01; ***p < .001
Table 3.5 The interaction effect model of daily negative emotional states and spouse solicitous response to pain on sleep quality

<table>
<thead>
<tr>
<th>Fixed effects</th>
<th>Sleep quality</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Estimate</td>
</tr>
<tr>
<td>Intercept</td>
<td>.83***</td>
</tr>
<tr>
<td>Age</td>
<td>-.003</td>
</tr>
<tr>
<td>Gender</td>
<td>.06</td>
</tr>
<tr>
<td>Cormorbid health conditions</td>
<td>-.03</td>
</tr>
<tr>
<td>Daily pain</td>
<td>-.004</td>
</tr>
<tr>
<td>Years of education</td>
<td>-.02</td>
</tr>
<tr>
<td>Body Mass Index</td>
<td>.002</td>
</tr>
<tr>
<td>Use of sleep medication</td>
<td>.16*</td>
</tr>
<tr>
<td>Daily NE</td>
<td>.01*</td>
</tr>
<tr>
<td>Daily solicitous response</td>
<td>.03</td>
</tr>
<tr>
<td>Daily NE × solicitous response</td>
<td>.01</td>
</tr>
</tbody>
</table>

*N_{patients} = 134, N_{observations} = 1607. Gender: 1 = men, 2 = women, NE = Negative emotional states

*p < 0.05; **p < 0.01; ***p < .001
Note. $H_1$: Daily negative mood will predict worse sleep quality the next day of individuals with knee osteoarthritis

$H_2$: Daily positive mood will predict better daily sleep quality of individuals with knee osteoarthritis

$H_3$: Daily spousal solicitous response will amplify the effect of daily negative mood on daily sleep quality of individuals with knee osteoarthritis

$H_4$: Daily spousal punishing response will amplify the effect of daily negative mood on daily sleep quality of individuals with knee osteoarthritis

$H_5$: Daily spousal empathic responses will reduce the effect of daily negative mood on daily sleep quality of individuals with knee osteoarthritis
Figure 3.2 The moderating effect of daily spouse solicitous response for feeling refreshed after sleep

![Graph showing the moderating effect of daily spouse solicitous response on feeling refreshed after sleep.](image)

*Note.* Estimated values are presented for the within-individual association between daily positive emotional states and feeling refreshed after sleep with high or low daily solicitous response (one SD above/below the average daily solicitous response). Error bars present the standard error for the estimated values.
Figure 3.3 The moderating effect of spouse solicitous response for subjective sleep quality

Note. Estimated values were presented for the within-individual association between daily negative emotional states and subjective sleep quality of those with chronic pain with high or low daily solicitous response (one SD above and below the average solicitous response). Error bars present the standard error for the estimated values.
Chapter 4

The role of negative marital relationships and depression in self-reported health and markers of inflammation

Abstract

Although depression and marital relationship conflict are known to have a robust impact on the physical health of adults, few studies have examined how depressive symptoms of individuals and negative marital relationship dynamics may be interconnected to affect physical health outcomes. Using nationally representative data from the Midlife in the United States study, this study examined the effects of depressive symptoms at baseline on physical health outcomes at 10 years follow-up and whether negativity in marital relationships at 9 years follow-up accounts for the associations. Adults with a married spouse or partner (mean age 56.75 ± 11.94, and 49.3% female) responded to questionnaires at baseline and that assessed depressive symptoms at baseline, and questionnaires 9 years later that assessed three indicators of marital relationship (marital risk, spousal emotional strain, and marital disagreement). Participants also came in for an overnight hospital visit to assess physical health outcomes at a 10 year follow-up point, including three markers of inflammation (CRP, IL-6, and sIL-6r), sleep quality, and self-reported number of physical symptoms and chronic conditions. Controlling for age, gender, BMI, medication use, and baseline chronic conditions, structural equation modeling analyses showed that depressive symptoms were associated with greater marital risk and lower sleep quality; marital risk and sleep quality in turn were associated with greater number of physical symptoms and chronic conditions. The effects of marital risk and sleep on the association between depressive symptoms and health problems differed by gender and age group: the depressive symptoms of older adults and men were particularly strongly related to poor sleep quality and increased marital risk. Overall, these results
demonstrate that impaired marital relationships and sleep can help explain the processes by which depressive symptoms of individuals aggravate physical health over time. Findings connect the emotion and social relationship literature and explain how those processes are closely connected to affect physical health.
Introduction

As previously reviewed, chronic inflammation has been associated with all-cause mortality in adults and development of major chronic diseases such as cardiovascular disease, type II diabetes, arthritis, and cancer (Aggarwal, Shishodia, Sandur, Pandey, & Sethi, 2006; Ershler & Keller, 2000; Kiecolt-Glaser, Gouin, & Hantsoo, 2010). Depression and interpersonal stressors are known to be associated with systemic inflammation by causing dysregulation in the stress response system (For reviews, see Irwin & Miller, 2007; Raison & Miller, 2003). However, previous research often disregards the fact that individuals with depression tend to report impairments in the quality of their close relationships, and they are particularly vulnerable to developing recurrent depression and systemic inflammation if they experience conflict in their close relationships. There is no prospective study, to my knowledge, that examines whether changes in quality of close relationships account for the association between depression and inflammatory status years later.

The effects of depression on systemic inflammation and sleep

Stress is associated with not only with inflammation but with depression. In fact, stress, depression, and inflammation seem to be intertwined in complex ways (Hammen, 2005). Recent studies have shown that depressive individuals often have cytokine abnormalities that may affect both the immune and central nervous system (Irwin & Miller, 2007). A meta-analysis of 180 studies suggests that depression is associated with increased circulating proinflammatory cytokines (Zorrilla, et al., 2001). Also, adult depressed individuals show excessive activation in their inflammatory response to an acute psychosocial stressor, with greater increases in IL-6 and NF-κB compared to healthy participants (Pace, et al., 2006). Thus, depression is linked with both chronic inflammatory state as well as excessive inflammatory responses to a psychosocial stressor.
The connection between depression and sleep problems is also well established in previous literature. A recent review by Palagni and colleagues suggests that depression is not only associated with sleep disturbances but also associated with altered sleep architecture, which can be characterized by decreases in slow wave sleep and dysregulations in rapid eye movement (REM) sleep including shortened REM sleep latency and increased REM sleep duration and density (Palagini, Baglioni, Ciapparelli, Gemignani, & Riemann, 2013). Not just clinical depression, but negative emotion (such as worry and anxiety) and daily stressful events also predict disturbed sleep on the same night, which suggest that less intense mood disturbances and stress may also have some acute impact on sleep (Waters, Adams, Binks, & Varnado, 1993a).

**Spousal relationship, depression, and systemic inflammation**

Depression has been linked with individuals’ impaired ability to engage in social relationships and dysregulated immune responses to stress (Miller, et al., 2005). There are two major theories to explain the well-known association between depression and impaired social environment (i.e., lack of social support and poor relationship quality). First, the erosive perspective assumes that depressive individuals experience a loss of social resources during any given period of depression due to their inability to engage in social relationships (Joiner, 2000). On the other hand, the self-propagatory perspective proposes that depressive individuals not only passively lose their social resources but also actively generate social stressors via maladaptive patterns of interpersonal behaviors (Joiner, 2000). For example, depressed individuals are known to have two specific types of interpersonal feedback seeking that are perceived as aversive by others: excessive reassurance seeking and negative feedback seeking. Excessive reassurance seeking is a tendency of depressed individuals to repeatedly validate their self-worth by others’ feedback. Previous studies have found a high level of reassurance seeking behaviors among depressed individuals (Davila, 2001; Joiner & Metalsky, 2001). Initially, others may respond
positively to reassurance seeking by providing support and making positive comments; however, depressed individuals often attribute such reassurance to others’ feelings of obligation and thus seek additional reassurance (Coyne, 1976). As unsatisfiable reassurance seeking continues, partners of depressed individuals may become frustrated and become more and more likely to reject the relationship with depressed ones. Reassurance seeking is likely to induce development of maladaptive social dependency and causes partner dissatisfaction in the social relationships of depressed individuals (Blatt, Quinlan, Chevron, McDonald, & Zuroff, 1982).

If such negative interactions and insatiable feedback seeking continue across the long-term, not only are existing depressive symptoms often exacerbated, but also a dysregulation in the inflammatory response system may result, leading to sustained elevation in pro-inflammatory cytokines (Kiecolt-Glaser et al., 2010; Robles & Kiecolt-Glaser, 2003a). Among different kinds of close relationships, marital relationships have received the most empirical attention in their impact on mental and physical health because marriage plays a central role in the lives of most adults in the U.S. (Bouhuys, Flentge, Oldehinkel, & van den Berg, 2004; Miller, Rohleder, Stetler, & Kirschbaum, 2005). It would be valuable to better understand how depressed symptoms of individuals may lead to changes in inflammation via changes in marital relationship functioning.

Among many different aspects of marital relationships, this 3rd study will focus on three indicators of negativity: partner/spouse strain, partner/spouse disagreement, and marital instability. Partner/spouse strain can be defined as an individuals’ perception of the degree to which their relationship with their spouse could be characterized as criticizing, irritating, and unreliable in nature (Walen & Lachman, 2000). Partner/spouse disagreement refers to differences in values and opinions about financial, recreational, household matters between spouses (Grzywacz & Marks, 2000). Finally, marital instability
refers to cognitive and affective states along the related actions that precede the decision to terminate a marriage (Booth, Johnson, & Edwards, 1983).

**Gender differences in impact of depression and marital relationships on health**

Previous studies have shown that women are more likely to be depressed and may be more vulnerable to social stressors than men (Kiecolt-Glaser & Newton, 2001; Nolen-Hoeksema, 2001; Robles & Kiecolt-Glaser, 2003b). Women across early adolescence and adulthood are twice more likely to develop depression, with lifetime prevalence of 21.3% compared to 12.7% in men (Nolen-Hoeksema, 2001). Also, women’s health status appears to be more adversely affected by having marital conflict (for review see, Kiecolt-Glaser & Newton, 2001). However, previous research has not examined whether women with depression are more likely to have impairment of their marital relationships and inflammatory status than are men with depression.

**Purpose of the study**

Using data from a large, nationally representative dataset that included relevant data on heterosexual married couples, the primary goal of the present research was to examine the degree to which depression at a baseline time point (Time 1) predicted markers of inflammation and physical symptoms and chronic conditions ten years later (Time 3), and whether the degree to which indicators of negativity in a marital relationship measured about one year before the inflammatory markers (i.e., 9 years post-baseline; Time 2) explain this association. The pathway in the model that is related to each hypothesis is labeled in Figure 4.1. Individuals’ depressed symptoms at Time 1 were expected to be associated with elevated levels of inflammatory markers, poor sleep quality, and worse physical health status at the 10 year follow-up time point, based on previous research on the effects of depressive symptoms on dysregulation of the stress response system and health compromising behaviors. Also,
depressive symptoms of individuals were expected to aggravate the three indicators of marital relationships (marital disagreement, spousal emotional strain, and marital risk) that are examined in this study because of typical stress generating behaviors and social dependency that depressed individuals tend to exhibit. The impaired marital relationship may not serve the typical protective role of a close relationship and may become a source of stress that leads to elevated levels of inflammation and poor physical health status. Therefore, I examined the unique and prospective impact of negativity in marital relationship (via three different indicators) on levels of inflammatory markers and whether the negativity mediated the association between depression and later elevations in inflammatory markers. Specifically, I predicted:

H1: Depression at Time 1 will be associated with levels of inflammatory markers, sleep quality, and number of physical symptoms and chronic conditions at Time 3 (pathway ‘h’ in Figure 4.1).

H2: Depression at Time 1 will be associated with negativity in marital/partner relationships at Time 2 (pathway ‘i’ in Figure 4.1).

H3: The negativity in marital relationships at Time 2 will predict inflammatory markers, sleep quality, and number of physical symptoms and chronic conditions at Time 3 (pathway ‘j’ in Figure 4.1).

H4: The negativity in marital relationships at Time 2 will mediate the association between depression at Time 1 and inflammatory markers, sleep quality, and number of physical symptoms and chronic conditions at Time 3.

Another goal of this study was to examine whether age and gender moderate associations between depression, marital relationship negativity, and the physical health outcomes. Women are expected to experience more depressed symptoms and to have greater impact of negative marital relationships on physical health outcomes. Age moderation will be explored due to a lack of previous research, and I examined the following hypothesis related to gender moderation.
H₅: The depression of women and negativity in the marital relationship will have a greater impact on inflammatory markers and physical symptoms/chronic conditions than men.

Methods

Participants and Procedure

Two waves of data were utilized from the National Study of Midlife in the United States (MIDUS). The study has a national sample of households in the 48 contiguous states, which was obtained through a stratified (by age and gender), multistage probability sampling methods (Brim, Ryff, & Kessler, 2004). Over 7,000 Americans aged 25-74 participated in Wave 1 of MIDUS in 1995-1996. Telephone interviews and mailed self-administered questionnaires (SAQs) were used in Wave 1 to obtain behavioral, psychological, social, and health information. In 2004-2006, about 75% of the original sample participated in a second wave of data collection. In addition to telephone interviews and SAQs at Wave 2, a subsample of 1,255 participants provided biological data, which was collected at an overnight stay at one of three General Clinical Research Centers (GCRC; University of California Los Angeles, University of Wisconsin, and Georgetown University) about one month after completing the main survey. The fasting blood samples were collected from participants at 7am in the morning on the second day at the GCRC.

Marital status information was utilized from two waves of the MIDUS data to identify individuals who were married or were living with someone in a steady marriage-like relationship and who also provided biological data at wave 2. After excluding twin and sibling samples, both women and men who reported that they lived with a partner across all ages were included in analyses. This provided a sample of 456 (71.3% of the total sample of the biological data) with mean age of 56.7 (SD = 11.94), 49.3% women. Other demographic characteristics of participants are listed in Table 4.
Measures

**Depressive symptoms.** Depressive symptoms were measured with the World Health Organization’s Composite International Diagnostic Interview Short Form (CIDI-SF, Kessler, Andrews, Mroczek, Ustun, & Wittchen, 1998) at Wave 1 and Wave 2. The CIDI-SF has a section on depressive episode in the past 12 months, which involves questions on whether the respondent has experienced feeling sad, blue, or depressed for 2 weeks or longer, how long the depressed mood lasted during the usual day of the 2 weeks period, and experiences of depressive symptoms and anhedonia, such as losing interests in most things. The CIDI-SF is a brief screening survey designed for assessing mental disorders according to the definition of the Diagnostic Criteria for Research of ICD-10 and the third version of the American Psychological Association (APA)’s Diagnostic and Statistical Manual of Major Depressive Disorder (DSM-III-R; APA, 1987). Psychometric studies showed the good agreement between CIDI-SF and the full CIDI, and the full CIDI has good test-retest reliability and concordance with clinical diagnosis (Kessler, Zhao, Blazer, & Swartz, 1997; Wittchen, 1994). Items on experiences of anhedonia in the depressive episode section were not used in the analysis of this study because lack of positive responses to those items in the sample.

**Negativity in marital relationship.** Negativity in the marital relationship was assessed at the main survey of wave 2 via three related but distinct scales: the marital risk scale, the spouse emotional strain scale, and the marital disagreement scale. The marital risk scale is consisted of two items that tap into global perception of risk in maintaining the marital status (Booth, Johnson, & Edwards, 1983); items include ‘During the past year, how often have you thought your relationship might be in trouble?’ on a 5 point Likert scale (1 = Never to 5 = Always) and ‘(Realistically) what do you think the chances are that you and your partner will eventually separate?’ on a 4 point Likert scale (1 = Very likely to 4 = Not likely at all). Partner/spouse emotional strain was administered to assess the frequency of experiencing conflicts,
tension, and irritation while interacting with a partner by using the scale developed by Schuster, Kessler, and Aseltine (1990). The partner emotional strain scale includes 6 items such as ‘How often does he or she criticize you?’ and ‘How often does he or she make you feel tense?'; all questions on this scale are on a 4 point Likert scale (1 = often to 4 = never for strain items; 1 = A lot to 4 = Not at all for support items).

Finally, how much cognitive disagreement individuals experience with the partner was measured by Spouse/Partner disagreement scale (Grzywacz & Marks, 2000). This scale asks about the degree of disagreement that people have with partner on financial matters, household tasks, and leisure time activities on a 4 point Likert scale (1 = A lot; 2 = Some; 3 = A little; 4 = not at all).

**Self-reported physical symptoms and chronic conditions.** Self-reported physical health was measured by the number of physical symptoms and chronic conditions reported by participants during the hospital visit of wave 2. The scale assessed the experiences of various health conditions, including high blood pressure, circulation problems, anemia, tuberculosis, cancer, diabetes mellitus, arthritis, and stroke using Yes/No/Unsure/Inapplicable response options. All the items asked about physical health conditions. One item on depression was excluded from the analysis to avoid overlap with depression assessments. The number of physical symptoms and chronic conditions was calculated by counting the number of reported symptoms and chronic conditions.

**Sleep quality.** Sleep quality was assessed by Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). The PSQI consists of 19 items and measures seven components of sleep during the past month, including subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping meds, and daytime dysfunction. A global sleep score is calculated by adding the scores of the seven component scores. The scale has good internal consistency, test-retest reliability, and good agreement with clinical diagnosis for sleep disorders (Buysse et al., 1989). The global sleep score was used in the preliminary analyses such as correlations, and the seven
components scores were used to form a measurement model of sleep quality in analyses using structural equation modeling.

**Covariates.** The covariates that were added in the present analyses includes age, gender, Body Mass Index, depressive symptoms at Time 2, chronic conditions at Time 1, and medication use. The depressive symptoms at Time 2 were assessed by the same measure that was used at Time 1. Chronic conditions at Time 1 were assessed by asking participants whether ever had chronic conditions including asthma, ulcer, high blood pressure, migraine, stroke etc. Medication use was coded as 0 (no) and 1 (yes) in each of three categories of drugs: anti-inflammatory medicine (e.g. Non-Steroidal Anti-inflammatory Drugs like ibuprofen and interferon gamma suppressants), anti-lipid medicine (e.g., statins), and estrogen.

**Biochemical analyses**

Inflammatory markers were obtained in the MIDUS study via fasting blood samples. Those used as outcome variables in the present analyses were C-reactive protein (CRP), Interleukin-6 (IL-6), and soluble Interleukin-6 receptor (sIL-6r). CRP is an inflammatory marker that plays a key role in the innate immune response and is known to be a predictor for cardiovascular disease risk that is associated with systemic inflammation (Ridker, 2003). As noted earlier in this dissertation, IL-6 is a multifunctional cytokine that regulates the acute phase response of the immune system and inflammation, and sIL-6r is known to coordinate the actions of IL-6 by forming a ligand-receptor complex with IL-6. High levels of IL-6 and sIL-6r were observed in inflammatory disease conditions such as rheumatoid arthritis (Jones, Horiuchi, Topley, Yamamoto, & Fuller, 2001; Mihara, Hashizume, Yoshida, Suzuki, & Shiina, 2012). Samples were processed at the three GCRCs using standardized procedures and then were prepared as frozen samples for bioassays (see additional details about the methods for obtaining each biomarker below).
**Serum IL-6.** Serum IL-6 was assayed at the MIDUS Biocore Lab using commercially available Enzyme-Linked Immunosolvent Assay (ELISA) (Quantikine, R&D Systems, Minneapolis, MN), with a lower sensitivity of detection at 0.16 pg/mL. All samples were tested in duplicate. Duplicate test values that varied by more than 5% were subject to repeat testing and the average of the duplicate tests was reported. Any value over 10 pg/mL was re-run in diluted sera to fall on the standard curve. The laboratory intra-assay coefficient of variance (CV) was 4.1%, and the inter-assay CV was 12.9% (generated by inclusion of a low and high IL-6 serum pool in each assay).

**Serum IL-6r.** Serum soluble IL-6 receptor levels were determined by sandwich ELISA kits (Quantikine, R&D Systems, Minneapolis, MN). The serum samples were diluted 1:100 so values would fall on the standard reference curve from 7 to 2000 pg/mL. The intra-assay CV was 2.0% and inter-assay CV was 6.9%.

**Serum CRP.** The serum levels of CRP were measured by a particle-enhanced immunonephelometric assay at Dr. Tracy’s laboratory in University of Vermont College of Medicine (Macy, Hayes, & Tracy, 1997). Polystyrene particles are coated with monoclonal antibodies to CRP, which in the presence of CRP results in an antigen agglutinate and increased light intensity that can be measured on a BNII nephelometer (Seimans Healthcare Diagnostics, Deerfield, IL). The assay range is 0.16–1100. The intra-assay CV is 2.3 to 4.4%, and the inter-assay CV ranged from 2.1% to 5.7%.

**Statistical analyses**

Structural equation modeling (SEM) was used for testing the associations between depression, marital relationship quality, and inflammatory markers. SEM has been widely used in psychology research because it can enhance the traditional way of estimation of coefficients by separating the measurement errors from estimation errors (Hox & Bechger, 1998). The SEM analyses of this study were conducted by four steps. First, a measurement model for latent variables was defined by running factor
analyses on depression, marital risk, and, spousal strain (Hox & Bechger, 1998). If a clear factor structure of each scale emerged, the structure was applied to set up a measurement model of a latent variable. If not, random selection of items was used to parcel out the items to create multiple measurement variables. For sleep quality and marital disagreement scales, subscales of each questionnaire were used to divide items for each measurement variable.

Although it is not necessary to have significant direct pathways to assume mediational pathways (MacKinnon, Fairchild, & Fritz, 2007), the direct pathways between depression at Time 1 and physical health/sleep outcomes at Time 3 were then examined. It was also explored whether depression at Time 2, which is a much more proximal time point than Time 1, is more closely related to the examined health outcomes at time 3. Next, the mediating pathways were estimated in the model as depicted by Figure 4.2 below. There are pathways from depression to three mediators (the indicators of negativity in the marital relationship), as well as pathways from the mediators to outcome variables of observed inflammatory markers, self-reported physical symptoms and chronic conditions, and sleep quality. Finally, the mediation effect was then tested by ‘the joint significance test’ (MacKinnon et al., 2007). The test examines the significance of both coefficients of α and β pathways to determine the mediation effect.

Additionally, bootstrapping analyses were conducted to confirm the bias corrected 95% confidence intervals of α and β coefficients. The model fit indices including root mean squared error of approximation (RMSEA) and Tucker-Lewis Index (TLI) are reported to show the fit between the estimated values from the SEM model and the actual data points (Allen, Markovitz, Jacobs, & Knox, 2001). The cutoff scores of RMSEA > .05 and TLI < .90 were used to evaluate the model (Allen et al., 2001). Finally, gender and age moderation effects for the significant mediational pathways were examined by multi-group structural equation modeling technique. After removing gender (or age) from the model, all of the paths of each mediation model were constrained to be equal for women and men (or
young and older adults) and the model was then compared to an otherwise identical model in which all paths were not constrained to be equal. A $\chi^2$ difference test was then applied to determine if the fit of the constrained model became significantly worse than the one without constraints. The age of 65 years old was used to divide the young adult group who are younger than 65 years old ($n = 339$) and older adult group who are over 65 years old ($n = 113$). SPSS 20 and AMOS 20 were used for the analyses.

**Results**

**Preliminary analyses**

As shown in Table 4.2, a depressive episode of two weeks or longer was reported by 23.7% and 19.7% of the participants of this study at Time 1 and 2 respectively. The scores for marital disagreement on financial, recreational, and household matters were relatively higher than the scores for spousal emotional strain and marital risk that measured broader perceptions on levels of negative interactions with a partner and the risk of eventually being separated with a partner. In terms of health outcomes, participants reported to have 3 to 4 physical symptoms and chronic conditions on the average at the baseline. A majority (53.8%) were taking some form of anti-inflammatory medicine (including ibuprofen) and 25.9% taking statins. The mean global sleep quality ($M = 5.82, SD = 3.44$) reflects the varied levels of sleep disturbances among participants.

Table 4.3 presents the correlations among measured indicators, using total scores of the marital relationship variables and sleep. Depression indicators at Time 1 were associated with higher marital risk ($r = .16, p < .01$ for duration of sadness and $r = .15, p < .01$ for depressive episode) at Time 2 and were also associated with higher CRP ($r = .14, p < .01$ for depressive symptoms), worse sleep quality ($r = .14, p < .01$ for duration of sadness and $r = .13, p < .01$ for depressive episode) at Time 3. All of the depression indicators at Time 2 were moderately associated with depression at Time 1 ($r's = .15-.25, p's < .01$) as
expected. Depression at Time 2 was also associated with all the three negative marital relationship indicators at Time 2 \((r's = .12-.19, p's < .01)\), and higher CRP \((r = .09, p < .05\) for duration of sad mood), sIL-6r \((r = .10, p < .05\) for depressive episode), greater number of physical symptoms \((r's = .13-.19, p's < .01)\) at Time 3. In contrary to expectations, none of the three indicators for negative marital relationships were significantly associated with sleep quality, number of physical symptoms, or any of markers of inflammation. Greater numbers of physical symptoms were significantly associated with worse sleep quality \((r = .19, p < .01)\), higher CRP \((r = .20, p < .01)\) and higher IL-6 \((r = .25, p < .01)\) but not with sIL-6r.

**Measurement model evaluation**

**Depressive symptoms.** Using the variables for depressive episode, duration of sad mood, and depressive symptoms at Times 1 and 2, the measurement model for depressive symptoms was a good fit to the data. Next, the variances of depressive episode and duration of sad mood were restricted to be the same as only those who reported to have any depressive episode were asked about how long the depressive mood lasted during the episode. Also, the error variance for depressive symptoms was allowed to be covaried with the error variance of depressive episode. This final model was an excellent fit to the data, \(\chi^2(8) = 9.97, p = .27,\) TLI = .99, CFI = .99, and RMSEA = .02 (90% CI = .00-.06). Each of the factor loading related three measured indicators to depression 1 and 2 was significant \((B's between .77-.99, p's <.001)\).

**Negativity in marital relationship.** Using measured items for marital disagreement, spousal strain, and marital risk, the three latent variables for each of the negative marital relationship indicators were established. The three marital relationship indicators were allowed to correlated with each other, and the final measurement model with the three indicators was excellent fit to the data, \(\chi^2(17) = 17.62, p = .41,\) TLI = .99, CFI = 1.00, and RMSEA = .01 (90% CI = .00-.04). All factor loadings of the measured components of sleep to the latent variable were significant at \(p < .001, B's between .63 and .87.\)
Sleep quality. Using the seven component scores of PSQI, the latent model for sleep was established. The three pairs of components were allowed to be correlated with each other due to their close relationships with each other; sleep duration and habitual sleep efficiency, sleep latency and sleep disturbances, and sleep latency and use of sleep medicine. The final model showed a good fit to the data with $\chi^2(11) = 24.54$, $p = .01$, TLI = .94, CFI = .98, and RMSEA = .05 (90% CI = .02 -.08). All factor loadings of the measured components of sleep to the latent variable were significant at $p < .001$, B's between .29 and .88.

Central analyses with structural models

Model with direct pathways between depressive symptoms and health outcomes. The results showed a significant direct pathway from depressive symptoms at Time 1 to sleep quality at Time 3 ($B = .18$, $p < .01$) and a marginally significant pathway between depression at Time 1 and the levels of CRP at Time 3 ($B = .08$, $p = .08$). Either the other two inflammatory markers (e.g. IL-6 and sIL-6r) or number of physical symptoms at Time 3 was not directly associated with depression at Time 1.

Model with mediation of marital relationships. There was significant mediation of marital relationships only for the association between depressive symptoms at Time 1 and the number of physical symptoms and chronic conditions at Time 3. Specifically, the $\alpha$ pathway between depression at Time 1 and marital risk at Time 2 was significant ($B = .16$, $p < .01$). Next, the $\beta$ pathway between marital risk at Time 2 and the number of physical symptoms at Time 3 was significant ($B = .21$, $p < .05$). The bootstrap analyses showed that the 95% bias corrected confidence intervals for the beta coefficients of the $\alpha$ and $\beta$ pathways did not include 0 (CI = .04 -.40 for $\alpha$ and CI = .03 -.60 for $\beta$), thus further confirmed the validity of the beta coefficients. There were no mediations by marital relationships that accounted for the association between depression and all the other health outcomes, including the three inflammatory markers and sleep.
Model with mediation of sleep quality. Additional exploratory analyses suggested that sleep quality played a mediating role in terms of explaining the association between depression at both Time 1 and Time 2 with certain health outcomes. Sleep quality accounted for the association between depression at Time 1 and the number of physical symptoms and chronic conditions at Time 3; the pathway between depressive symptoms and sleep ($B = .13, p < .05$) and the pathway between sleep and number of physical symptoms/chronic conditions were significant ($B = .26, p < .001$). Sleep quality also accounted for the associations between depressive symptoms at Time 2 and the number of physical symptoms and chronic conditions at Time 3.

The final model with depression, marital relationships, sleep, the number of physical symptoms, and markers of inflammations. The final model showing integration of all significant pathways is presented in Figure 5. Model fit indices showed the good fit of the model to the data with CFI = .95, TLI = .93, RMSEA = .04. In this model, greater depression at time 1 was associated with greater marital risk, more spousal strain, and worse sleep quality. Marital risk and sleep quality were also associated with self-reported physical symptoms at time 3, which confirms the mediational pathways suggested in the previous model. Marital disagreement was associated with physical symptoms in an unexpected direction: Higher levels of marital disagreement were associated with less physical symptoms. Having more physical symptoms was significantly associated with higher levels of serum CRP and IL-6, but not with sIL-6.

Moderation by age and gender. The final model in Figure 4.3 was significantly different by different age and gender groups, as presented in Figure 4.4 (a) for gender moderation and (b) for age moderation. First, the age moderation effect was confirmed by the significant $\chi^2$ difference test ($\chi^2_{\text{diff}} (6) = 20.21, p < .01$) that compares the model with constraints for the two significant mediational pathways to be equal with the model without any constraint. Overall, the model without constraints showed better model fit
indices ($\chi^2$ (858) = 1183.13, $p < .001$, CFI = .94, TLI = .92, RMSEA = .03 with 90% CI = 02-.03) than the model with constraints ($\chi^2$ (864) = 1203.34, $p < .001$, CFI = .93, TLI = .92, RMSEA = .03 with 90% CI = .02 - .03). When younger adults aged 34-64 (n = 339) were compared with older adults aged over 65 years old (n = 113), the mediation of sleep for the association between depression at Time 1 and number of physical symptoms at Time 3 only significant for young adults ($B = .17, p < .01$ for the pathway between depression and sleep; $B = .19, p < .01$ for the pathway between sleep and physical symptoms).

Also, older and young adults were different in the associations between depression and negativity in their marital relationships. Only among older adults were levels of depression at Time 1 significantly associated with marital risk at Time 2 ($B = .36, p < .001$). However, age did not moderate the effect of marital risk on the association between depressive symptoms and physical health; The mediating effect of marital risk did not hold significant when the two age groups were examined separately.

The key pathways in the final model were also different between women and men ($\chi^2_{\text{diff}}$ (6) = 14.18, $p < .05$). The model without constraints to have equal pathways between women and men showed better model fit indices ($\chi^2$ (748) = 999.85, $p < .001$, CFI = .95, TLI = .94, RMSEA = .03 with 90% CI = 02-.03) than the model with constraints ($\chi^2$ (754) = 1014.04, $p < .001$, CFI = .93, TLI = .92, RMSEA = .03 with 90% CI = .02 - .03). Only for men was sleep quality a mediator of the association between depression and physical symptoms ($B = .21, p < .01$ for the pathway between depression and sleep; $B = .25, p < .01$ for the pathway between sleep and physical symptoms). There were gender differences for the association between depressive symptoms at Time 1 and marital risk at Time 2. Only men but not women showed the significant pathway from depression at Time 1 and marital risk at Time 2 ($B = .16, p < .05$) whereas only women but not men showed the path between marital risk and number of physical symptoms and chronic conditions to be marginally significant ($B = .42, p = .08$). However, gender did not moderate the effect of marital risk on the association between depression at Time 1 and the number of
physical symptoms and chronic conditions at Time 3: The mediating effect of marital risk did not stay significant when the effect was separately examined among men and women.

Discussion

Based on the two waves of a nationally representative MIDUS data that were collected over a 10 years period, this study examined the depression, sleep, and three different indicators of negative marital relationships on several physical health indicators: number of physical symptoms and chronic conditions, and levels of several inflammatory markers. Findings revealed that marital risk and sleep quality partially accounted for the association between depression at Time 1 (baseline) and number of physical symptoms and chronic conditions among these participants; these results were independent of the levels of age, gender, BMI, anti-inflammatory anti-lipids medication use, existence of chronic conditions at Time 1, and levels of depression at Time 2. Overall, this study thus provides support for the theoretical model (Figure 1.2) of how social interactions may partially mediate the degree to which mood disorders affect health. A major strength of this study was that the longitudinal nature of the data allowed analyses that could speak to the temporal relationship between depression, impaired marital relationships, and health, suggesting that depression can lead to both marital and sleep problems, which in turn are associated with health problems. Additionally, age and gender were found to affect results: Depressive symptoms were particularly damaging for later marital relationships and sleep among men and older adults. In contrast to expectation, having disagreement in activities appeared to be associated with better physical health outcomes.

The role of marital risk in linking depressive symptoms with health problems

As predicted in $H_1$ and $H_2$, depressive symptoms at baseline were associated with marital risk and physical symptoms and chronic conditions across a 10 year period depressive symptoms at Time 1 were
associated with self-reported physical symptoms and chronic conditions at Time 3, ten years later. Further, as hypothesized in H₄, the perception of risk in the marriage (e.g., the perceived chance of being eventual separation) at Time 2 accounted for the association between depressive symptoms at Time 1 and self-reported physical symptoms and chronic conditions at Time 3, which were in turn also associated with circulating levels of CRP and IL-6. These results are consistent with previous literature on the impact of marital discord on physical health as reviewed earlier (for reviews see, Kiecolt-Glaser et al., 2010; Kiecolt-Glaser & Newton, 2001) and also consistent with the longitudinal association between depressed symptomatology and later impairment in relationship with a romantic partner (Gustavson et al., 2012; Hammen, 1991; Vujeva & Furman, 2011). The long-term effects of depression are striking but consistent with previous research which suggests that individuals who have interpersonal problems tend to have recurrent types of depression that are maintained over a long time and are resistant to treatments (Slavich et al., 2010). Depressive symptoms that were observed in this study were linked with increases in perceived serious marital troubles and greater chance of separation in the marital relationship. Although negativity in relationships did not directly predict inflammatory markers, marital negativity was associated with greater numbers of physical symptoms and chronic conditions, thus providing partial support for H₃. Thus, the results suggest that depressive symptoms can have a particularly long lasting impact on physical health if the symptoms generate distress in a marital relationship.

As described earlier, there are theoretical explanations for why depressive symptoms would be associated with aggravation in close interpersonal relationships and how those relationship dynamics, in turn, may lead to negative physical health outcomes. As reviewed in chapter 1, studies have shown that depressed individuals tend to exhibit certain cognitive and behavioral patterns that may generate more interpersonal stress. For example, depressed individuals are likely to repeatedly engage in seeking feedback to confirm their negative self-view that put a burden on their partner for repeatedly denying such
negative self-view (Joiner & Metalsky, 2001). The negative feedback seeking behaviors can lead to relationship dissatisfaction by the partner of depressed ones and later rejection by the partner, which would further aggravate the existing depressed symptoms (Coyne, 1976). In addition, depressed individuals are known to be less effective at providing and eliciting support for their partner (O’ Mahen, Beach, & Banawan, 2001). In a study by Davila and colleagues, compared to their non-depressed counterparts depressed husbands and wives exhibited more negative support behaviors such as blaming and exaggerating a problem—when they were asked to engage in a 10 min discussion with their partner about something to change in their marital relationship (Davila, Bradbury, Cohan, & Tochluk, 1997). These negative support behaviors can generate negative social interactions in face of problems that require couple’s collective efforts and thus aggravate marital relationships. An aggravated marital relationship itself serves as a source of constant interpersonal stress, thus negating the social support that a good marital relationship usually provides. The lack of social support and frequent interpersonal stress in a marital relationship can directly exacerbate physical health and disturb homeostasis of the inflammatory system of the body (Burman & Margolin, 1992; Kiecolt-Glaser et al., 2010; Kiecolt-Glaser & Newton, 2001).

**Spousal emotional strain and marital disagreement: unexpected findings**

Interestingly, among the three indicators of negative marital relationships (marital risk, spousal emotional strain, and marital disagreement), spousal emotional strain was not directly associated with later physical symptoms and chronic conditions, whereas marital disagreement and marital risk were. Also, the higher levels of marital disagreement were associated with less physical symptoms and chronic conditions, which again means that H$_3$ was only partially supported. Some levels of emotional strain and disagreement is ordinary in marital relationships, and the sample overall in the present research did not have clinically diagnosed depression and reported overall good marital functioning (Donoho, Crimmins,
& Seeman, 2013). However, perceptions of troubles in marital relationship and high chance of being eventually separated with the partner may indicate the existence of more severe and long-term problems in the marriage. Perhaps in keeping with this, previous studies on the adverse effects of marital relationship on health seem to commonly used the global negative evaluations on marital relationships, similar to the marital risk construct of this study, as the indicator of negative marital relationships rather than emotional strain or cognitive agreement between couple (Hawkins & Booth, 2005; Umberson, Williams, Powers, Liu, & Needham, 2006).

It was also unexpected that past studies using the MIDUS data have reported somewhat divergent results in terms of the specific indicators of negative marital relationships that are associated with health indicators. For example, a previous study used MIDUS baseline survey data to examine the effects of spousal emotional strain and marital disagreement on physical health and found a significant effect of spousal emotional strain on numbers of physical symptoms and chronic health conditions but that marital disagreement was not associated with physical health outcomes (Bookwala, 2005). The results of Bookwala’s study are not consistent with the present study that found a non-significant effect of spousal emotional strain and a significant effect of marital disagreement on physical symptoms and chronic conditions one year later. The present results may be more reliable than the contrasting results in Bookwala for two reasons: the results hold their significance over and above baseline chronic conditions and were obtained by examining longitudinal associations across the three time points. Another MIDUS study using the wave 2 main survey and physical health outcomes at 1 year follow-up also found inconsistent results from present study: The present study found no direct association between spousal emotional strain and IL-6 whereas Donoho and colleagues found that spousal emotional strain was associated with higher IL-6 in married men and women (Donoho et al., 2013). However, Donoho and colleagues acknowledged that the association between spousal emotional strain and IL-6 is inconsistent
and weak and diminished after addition of psychosocial variables including depressed mood. The present study included depressive symptoms at baseline and simultaneously examined the longitudinal association between depressive symptoms, spousal emotional strain, and IL-6. The fact that baseline depressive symptoms were associated with greater spousal emotional strain at 9 year follow-up may explain the divergent results between present study and Donoho and colleagues. Finally, marital risk was often not included as indicators of marital relationship in the previous MIDUS studies, but the results of the present study redirects attention to the important role marital risk plays in the association between depressive symptoms and later physical health outcomes. Thus, the present study advances the understanding of previous investigations using the MIDUS data by utilizing all of the available three measurement time points and testing the integrative model that included depressive symptoms, marital relationship indicators, and more extensive lists of biomedical covariates that influence physical health outcomes.

**How does sleep explains the association between depressive symptoms and health problems?**

In addition to dynamics related to marital quality, sleep quality appears to be a behavioral factor that also account for the association between depression at Time 1 and physical symptoms and chronic conditions at Time 3. Sleep was initially expected to be indirectly connected to depression via aggravated marital relationship, as hypothesized in H₄, but results of this study suggest a more direct role of sleep. The results suggest that individual with more depressed symptoms have worse sleep quality later on, outside of negative marital relationship factors, and that levels of sleep quality are in turn associated with aggravated physical symptoms and chronic conditions.

The significant pathway between depression and sleep observed in the present research is consistent with previous studies on the adverse impact of depressive symptoms on sleep quality (for a review, see Tsuno, Besset, & Ritchie, 2005). Epidemiological studies estimate that 50-90% of depressed
individuals complain about poor sleep quality, and sleep disturbances seem to be core somatic symptoms of clinical depression (Hetta, Rimón, & Almqvist, 1985; Riemann, Berger, & Voderholzer, 2001). The second part of the mediational pathway between sleep quality and physical symptoms and chronic conditions is also supported by previous literature. In the “Sleep in America” survey with 1506 middle aged and older adults, those with life history of depression were 2 times more likely to have difficulty falling asleep, waking too early, and waking unrefreshed, and the quality of sleep was significantly associated with the number of medical conditions (Foley, Ancoli-Israel, Britz, & Walsh, 2004). There are many different physiological mechanisms underlying sleep problems and physical symptoms and chronic disease conditions. For example, previous sleep laboratory studies showed that modest sleep restriction from 8 to 6 hours induced increases sleepiness, impairment of psychomotor performance, and increases in 24 hour secretion of IL-6 among healthy young men and women (Vgontzas et al., 2004). Also, the Sleep Heart Health Study revealed that levels of sleep disordered breathing, a type of common sleep disorder, predicts increased risk for cardiovascular disease incidents at 3 years follow up among nationally representative sample of adults over 40 years old (Newman et al., 2001).

**Inflammation linked with physical symptoms and chronic conditions**

Although inflammatory markers were expected to be directly influenced by depressive symptoms and indicators of marital relationship, inflammatory markers appear to be associated with them via the number of physical symptoms and chronic conditions. The physical symptoms and chronic conditions that were measured in this study involve many inflammation-related health conditions, such as circulation problems, coronary artery disease, and arthritis. The final SEM model demonstrated that the physical symptoms and chronic conditions were associated with higher levels of both CRP and IL-6. Thus, the results of the present study suggest that depressive symptoms and aggravated marital relationship may
generate more physical symptoms and chronic conditions that are related to elevated markers of inflammation.

**Gender moderation**

There were differences by gender on effects of marital risk and sleep on the association between depressive symptoms and health problems. The mediation of depression and health problems by marital risk did not retain significance when men and women were examined separately. Depression was associated with increased marital risk only among men and not women, whereas marital risk was marginally associated with physical symptoms and chronic conditions only among women. This contrasts with the expectation put forth in H5, which was that depression among women would have a stronger overall effect on their inflammation and health. Results suggest that heterosexual men seem to be particularly vulnerable to heightened risk in their marital relationship if they have depressed symptoms. Men in our society may feel pressure to maintain active and assertive gender roles in their marital relationship; according to gender socialization theory (Bussey & Bandura, 1999; Fagot, Rodgers, & Leinbach, 2000), stress and dissatisfaction in marital relationship can occur when individuals feel incapable of maintaining such socially prescribed gender roles due to their depressive symptoms. Men may also be particularly likely to withdraw from interpersonal conflicts when they are depressed, which may generate more frustration and stress in their marriage (O’ Mahen et al., 2001). Further, depressed symptoms of men may be particularly likely to intensify existing risk factors of marital relationships because men are more likely to heavily rely on marital relationship as a source of social support and do not have many alternative sources of social support than women (Carr, 2004).

In general, previous research suggests that men experience more health benefits than women when they are married, whereas women have benefits from marriage only if the marital relationship is satisfying; further, men seem to experience greater health problems after the dissolution of marriage.
At the same time, several studies indicate that marital conflict in existing relationships is associated with greater physiological responses to stress among women (Kiecolt-Glaser, Glaser, Cacioppo, & Malarkey, 1998; Kiecolt-Glaser et al., 2005). The present results also lend tentative support for the possibility that women in U.S. culture may be more at risk of having physical symptoms result from marital conflict than their male counterparts. However, while the results of the present research are in line with this type of research, they must be interpreted with caution, as neither men nor women in the present study showed significantly greater health outcome differences based on their marital risk status when examined separately, either due to a lack of power or non-robust effects.

Gender also appeared to moderate the effects of sleep; sleep quality accounted for the effect of depression on physical symptoms and chronic conditions only among men, when they were examined separately. Although poor sleep quality was associated with more physical symptoms and chronic conditions in both men and women, only in males were depressive symptoms associated with worse sleep. The results are in contrast to previous work suggesting that men with Major Depressive Disorders (MDD) have a unique vulnerability for sleep dysregulation. In past research, men and women with MDD had comparably severe sleep problems, but men were more likely to have suppressed regulation of slow wave sleep, which is inversely associated with REM sleep duration than women (For a review, see Armitage & Hoffmann, 2001). It is possible that men tend to underreport their depressive symptoms and may present different symptoms of depression – such as being angry about oneself and being irritated by others – which may contribute to men being under-diagnosed with clinical depression (Shaw, Kennedy, & Joffe, 1995). As a result, men tend not to seek out treatment until the symptoms are severe (Shaw et al., 1995).

Thus, the report of depressive episode and depressive symptoms among men that was utilized in this study may indicate more substantial symptoms, which thus have the long-term effect on sleep quality 9 years later.
Age moderation

Age also moderated the effects of marital risk and sleep on the association between depressive symptoms and health problems. The mediation by marital risk for the association between depression and physical symptoms was not significant when the young adult group (34-65 years old) and older adult groups (over 65 years old) were examined separately. Only older adults over 65 years old showed a significant association between depression and marital risk. Interestingly, the levels of depression among this group were associated with increases in all three indicators of negative marital relationship. Previous studies have shown that marital quality generally declines as people age and also as marital duration is increased over time, despite the slight improvement in quality reported in many marriages after any children in the household become independent (Umberson, Williams, Powers, Chen, & Campbell, 2005; VanLaningham, Johnson, & Amato, 2001). Thus, marital relationships in the older adults over 65 years old in this study may have been more vulnerable to be negatively affected by depressive symptoms their partner than younger age group with age range from 34-65 years old.

Another age moderation effect was found on in the degree to which sleep quality mediated the association between depression and physical symptoms and chronic conditions. It was only the younger adults (age 36-64) who showed the significant association between depressive symptoms and later sleep quality, and that association between depressive symptoms and sleep was not significant among older adults over 65 years old. Although there is general age-related sleep deterioration (Floyd, Medler, Ager, & Janisse, 2000; Miles & Dement, 1980), there are mixed results about what age group is particularly vulnerable to have an adverse effect of depression on sleep. One study showed that the differences in REM sleep latency between depressed patients and healthy controls were not present among adults in the 18 to 44 year old range, but were present for adults in the 45-65 year old group (Lauer, Riemann, Wiegand, & Berger, 1991). Another study showed that patients with depression from age 15-64 did not
show differences in their patterns of sleep across different age groups compared to age matched controls except in early morning awakening; in an older age group (ages 55-64) early morning awakening was increased to a greater degree among those with depression than those without depression (Gillin et al., 1981). However, another study that examined the link between depressive feelings and subjective report of sleep disturbances among older adults age over 63 years old showed that their depressive feelings were associated with later sleep disturbances, although this study did not compare whether strength of the associations was different for younger adults (Rodin, McAvay, & Timko, 1988). Overall, findings from previous studies suggest that those who are in their 40-60s are likely to show greater adverse impact of depression on sleep than those who are younger than 40s or older than 60s. Indeed, the younger adult group of present study includes the age range from 34 to 65, and it might be those in the middle age who drove the stronger association between depressive symptoms at Time 1 and sleep quality at Time 3, rather than the older adult group over 65 years old. Results of the study indicate that younger adults under 65 years old, particularly those who are in the middle age, are prone to have impaired sleep quality if they are depressed.

Limitation of the study

There are several limitations of the present research. First, there are limitations in the extent to which results can be generalized to the general population due to some of the sample characteristics: The sample was predominantly Caucasian and well-educated. Also, in order to examine the influence of initial depression on later marital relationship functioning and health sequelae, this study did not include those who were unmarried at baseline or those who did not stay married between the 9 years of the study period. This may have increased selection bias toward people who chose to be married or who had better relationships, which is known to be related to better health status (Kiecolt-Glaser & Newton, 2001). Also, there is a possibility that present sample includes individuals who were divorced after the first main
survey and were married with a different person before the second main survey. Finally, the three time points that were utilized for this study did not have equal time intervals between them: The average time interval between Time 1 and Time 2 was 9 years, whereas the time interval between Times 2 and 3 was one year. Differences from the first time interval to one of the other time intervals indicates a relatively long-lasting impact of depression on marital relationships and health, whereas differences from the second time interval imply a relatively short-term change in physical health status due to negativity in marital relationship; thus, comparisons between findings related to the first and second time intervals should be interpreted accordingly.

**Conclusion**

In sum, the findings of this study suggest that marital risk and sleep are important interpersonal and behavioral factors that explain how depressed symptoms of individuals adversely affect later physical health using a large sample of adults in the U.S. As expected, depressive symptoms were associated with higher levels of CRP and lower quality of sleep at 10 year follow-up, which supported the pathway ‘h’ that proposes the pathway between depression and physical health outcomes in Figure 4.1. Also, depression predicted increased risk in marital relationship functioning 9 years later, and the heightened risk within the marital relationship predicted greater physical symptoms and chronic conditions the following year, including major chronic disease conditions such as cardiovascular disease and arthritis. Therefore, the findings of this study provide evidence to support the pathways ‘i’ and ‘j’ that propose the link between depression and physical health status is explained by negativity in marital relationship that generate social stress. Also, sleep quality accounted for the relationship between depression and physical symptoms and chronic conditions: Depression predicted lower sleep quality 10 years later, and the deteriorated sleep was associated with the number of physiological symptoms and chronic conditions. Thus, the findings of this research support the stress generation model of depression, which posits that the
role of depressive symptoms generate more mental and interpersonal stress, which in turn may make individuals vulnerable to developing physical symptoms and chronic disease conditions by exacerbating close social relationships or sleep quality. Objective inflammatory markers were closely associated with the number of physical symptoms and chronic conditions, which validates the actual health implication of depression-marital risk and depression-disturbed sleep connection. Finally, the findings also suggest that men and younger adults may be more prone to experience an adverse impact of depression on their later marital risk and sleep quality. Overall, this study suggests that managing the negative consequences of subclinical depression on marital relationship and sleep is important for maintaining physical health of married adults.
Table 4.1 Demographic characteristics of participants (N = 456)

<table>
<thead>
<tr>
<th>Variable</th>
<th>M (SD) or %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>56.7 (11.94)</td>
</tr>
<tr>
<td>Gender = Women</td>
<td>49.3%</td>
</tr>
<tr>
<td>Education: some college and beyond</td>
<td>50.2%</td>
</tr>
<tr>
<td>Race = Caucasian</td>
<td>93.4%</td>
</tr>
<tr>
<td>Household income</td>
<td>$38,000 - 42,000</td>
</tr>
<tr>
<td>Body mass index</td>
<td>29.5 (6.0)</td>
</tr>
</tbody>
</table>
Table 4.2 Sample characteristics on the key study variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean (SD) or %</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Depression at T1 and T2</strong></td>
<td></td>
</tr>
<tr>
<td>Felt depressed 2 weeks or longer at T1</td>
<td>23.7%</td>
</tr>
<tr>
<td>Duration of depressed mood at T1</td>
<td>.54 (1.10)</td>
</tr>
<tr>
<td>Depressed symptoms at T1</td>
<td>.45 (1.53)</td>
</tr>
<tr>
<td>Felt depressed 2 weeks or longer at T2</td>
<td>19.7%</td>
</tr>
<tr>
<td>Duration of depressed mood at T2</td>
<td>.49 (1.11)</td>
</tr>
<tr>
<td>Depressed symptoms at T2</td>
<td>.48 (1.53)</td>
</tr>
<tr>
<td><strong>Indicators of negativity in marital</strong></td>
<td></td>
</tr>
<tr>
<td>relationship</td>
<td></td>
</tr>
<tr>
<td>Marital disagreement</td>
<td>5.76 (2.05)</td>
</tr>
<tr>
<td>Spousal emotional strain</td>
<td>2.15 (0.62)</td>
</tr>
<tr>
<td>Marital risk</td>
<td>3.05 (1.51)</td>
</tr>
<tr>
<td><strong>Inflammatory markers</strong></td>
<td></td>
</tr>
<tr>
<td>CRP (μg/mL)</td>
<td>2.54 (3.86)</td>
</tr>
<tr>
<td>IL-6 (pg/mL)</td>
<td>2.71 (2.79)</td>
</tr>
<tr>
<td>sIL-6r (ng/mL)</td>
<td>36.30 (9.96)</td>
</tr>
<tr>
<td><strong>Sleep quality</strong></td>
<td>5.82 (3.44)</td>
</tr>
<tr>
<td>No. of physical symptoms and chronic conditions</td>
<td>3.91 (2.75)</td>
</tr>
</tbody>
</table>
Medication use

<table>
<thead>
<tr>
<th>Medication</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anti-inflammatory drugs</td>
<td>53.8%</td>
</tr>
<tr>
<td>Statins</td>
<td>25.9%</td>
</tr>
<tr>
<td>Estrogen</td>
<td>1.8%</td>
</tr>
</tbody>
</table>

Note.  

\(^a\) Lower scores are indicative of greater sleep quality

\(^b\) Although natural transformed values were used in all analyses, raw values were reported here.
|   | 2    | 3    | 4    | 5    | 6    | 7    | 8    | 9    | 10   | 11   | 12   | 13   | 14   | 15   | 16   | 17   | 18   | 19   | 20   |
|---|------|------|------|------|------|------|------|------|------|------|------|------|------|------|------|------|------|------|
| 1 | CRP  | .47**| .16**| .11* | .20**| .14**| .07  | .07  | .05  | .08  | .09* | .03  | -.06 | .10* | .13**| .41**| .09  | -.05 | .13**|
| 2 | IL-6 | —    | .16**| .06  | .25**| .02  | -.01 | -.03 | .003 | -.01 | -.003| .03  | -.06 | .28**| -.06 | .27**| -.03 | .13**| .04  |
| 3 | sIL-6r| —    | .01  | .07  | .01  | .02  | .04  | .10* | .09  | -.02 | -.01 | -.02 | .01  | .05  | .08  | .001 | -.01 | .04  |
| 4 | Sleep| —    | .19**| .09  | .14**| .13**| .20**| .24**| .23**| .08  | .05  | -.10*| .13* | .09  | .01  | -.02 | .04  | —    | —    |
| 5 | Physical symptoms| —    | .00  | .07  | .02  | .19**| .13**| .14**| -.07 | -.002| -.01 | .42**| .06  | .13**| .22**| .22**| -.002| —    | —    |
| 6 | T1 Depre. symptoms| —    | .53**| .76**| .15**| .15**| .19**| -.002| .003 | .02  | -.09*| .08  | .04  | .001 | -.06 | .04  | .004 | .004 | —    | —    |
| 7 | T1 Sad over 2 weeks| —    | .87**| .16* | .22**| .23**| .08  | .09  | .16**| -.15**| .14**| .06  | -.04 | .004 | .004 | —    | —    | —    | —    | —    |
| 8 | T1 duration of sad mood| —    | .19**| .21**| .25**| .07  | .08  | .15**| -.16**| .11* | .07  | -.05 |-.02 | .01  | —    | —    | —    | —    | —    | —    |
| 9 | T2 Depre. symptoms| —    | .63**| .82**| .12**| .16**| .19**| -.07*| .14**| .03  | -.01 |-.05 | .06  | —    | —    | —    | —    | —    | —    |
|10 | T2 Sad over 2 weeks| —    | .90**| .13**| .13**| .14**| -.11*| .14**| .10* | .02  | .04  | .02  | —    | —    | —    | —    | —    | —    | —    |
|11 | T2 duration of sad mood| —    | .14**| .13**| .16**| -.13**| .14**| .09  | .01  | -.01 | .01  | .05  | —    | —    | —    | —    | —    | —    | —    |
|12 | Marital disagreement| —    | .64**| .57**| -.19**| .05  | .04  | -.07 | -.08 | .05  | —    | —    | —    | —    | —    | —    | —    | —    | —    |
|13 | Spousal strain| —    | .66**| -.12*| .07  | .03  | -.06 | -.01 | .07  | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    |
|14 | Marital risk| —    | -.23**| .06  | -.01 | -.04 | -.09 | .10* | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    |
|15 | Age  | —    | -.14**| -.03 | .27**| .28**| -.07 | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    |
|16 | Gender| —    | -.09 | -.02 | -.15**| .15**| —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    |
|17 | BMI  | —    | .001 | .10* | .004 | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    |
|18 | Anti-inflammatory | —    | .19**| .02  | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    |
|19 | Statins| —    | -.04 | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    |
|20 | Estrogen| —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    | —    |
Note. T1, time 1; T2, time 2; Depre. symptoms, depressive symptoms; Sleep, low sleep quality; BMI, Body Mass Index; Anti-inflammatory, anti-inflammatory drugs; Estrogen, estrogen use.

* $p < .05$

** $p < .01$
Table 4.4 Regression coefficients for mediation paths, indirect effects, and bias corrected confidence intervals for mediation by marital risk and sleep quality that account for the association between depression and the number of physical symptoms

<table>
<thead>
<tr>
<th>Mediating variable</th>
<th>(α) Depression effect on mediator</th>
<th>(β) Mediator effects on physical symptoms</th>
<th>(αβ) Indirect effect</th>
<th>Upper 95% CI</th>
<th>Lower 95% CI</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marital risk</td>
<td>.16(.08)**</td>
<td>.21(.51)*</td>
<td>.03</td>
<td>.19</td>
<td>.95</td>
<td>.001</td>
</tr>
<tr>
<td>Sleep quality</td>
<td>.14(.05)*</td>
<td>.20(.38)**</td>
<td>.03</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. *p < .05, **p < .01, ***p < .001
Note. H₁: Depression at Time 1 will be associated with levels of inflammatory markers, sleep quality, and number of physical symptoms and chronic conditions at Time 3.

H₂: Depression at Time 1 will be associated with negativity in marital/partner relationships at Time 2.

H₃: The negativity in marital relationships at Time 2 will predict inflammatory markers, sleep quality, and number of physical symptoms and chronic conditions at Time 3.
Figure 4.2 The structural equation model showing measurement models and proposed mediation pathways.
Figure 4.3 The final SEM model

Note. Model fit indices $\chi^2 (428) = 701.86, \ p < .01$, CFI = .95, TLI = .93, RMSEA = .04 (90% CI = .03-.04). Key paths and variables are shown in boldface. All coefficients significant at $p < .05$ or better. For all variables, higher values indicate higher levels of the construct except the sleep quality, in which higher values indicate poorer sleep quality. The lack of path indicates lack of significant associations. The latent constructs were represented as ovals and the measured variables as rectangles.
Figure 4.4 The gender (a) and age moderation (b) for the key mediating pathways

(a)

Depression at T1

Marital risk at T2

Sleep quality at T3

Physical symptoms & chronic conditions at T3

Women

0.13

0.07

0.42†

0.15*

Depression at T1

Marital risk at T2

Sleep quality at T3

Physical symptoms & chronic conditions at T3

Men

0.16*

0.21**

0.13

0.25***

(b)

Depression at T1

Marital risk at T2

Sleep quality at T3

Physical symptoms & chronic conditions at T3

Younger adults
(34-64 yrs)

0.09

0.17**

0.20

0.19**

Depression at T1

Marital risk at T2

Sleep quality at T3

Physical symptoms & chronic conditions at T3

Older adults
(+65 yrs)

0.36***

0.02

0.18

0.22*

Note. † .05 < p < .10; * p < .05; ** p < .01
Chapter 5  General Discussion

Prior research on emotion, social relationships, and health suggests that there is a robust influence of emotional and social factors on physical health. However, the specific pathways by which emotional states/trait/disorders and social relationships dynamically influence physical health have not been identified. This dissertation presented two theoretical models (Figure 1.1 and Figure 1.2) which illustrate how I propose emotional traits, states, and symptoms of depression may interact with various aspects of close relationships to affect physical health. The first figure demonstrated the pathways by which short-term experiences of positive and negative emotional states affect physical health outcomes and how ongoing social interactions may further aggravate or mitigate the impact of emotional states on physical health outcomes. The second figure presented how long-lasting emotional disturbances such as depression may affect social relationships in ways that may generate stressful or supportive social interactions and how social relationships may explain the association between emotional disturbances on physical health. Different pathways in the two figures were tested throughout three studies. Figure 5.1 and 5.2 present the original theoretical models with highlighted pathways that were examined in the three studies.

The pathways ‘H_{2,1,\& 2}’ and ‘H_{2,3,4,\& 5}’ in the first theoretical (moderational) model (see Figure 5.1) were tested by hypotheses in the second study in chapter 3 that examined the ongoing dynamic interaction between state emotion and partner responses to pain behaviors on sleep quality by utilizing daily diary measurements within the context of individuals with chronic pain and their partners. The results presented in chapter 3 advance the previous literature by examining how different kinds of social interactions may change the degree to which negative and positive emotional states influence sleep quality of older adults with chronic pain. Both the first and third
studies (in chapter 2 and 4) examined different pathways in the second (mediational) theoretical model as it is presented in Figure 5.2. The first study in chapter 2 examined whether negative social relationships are associated with inflammatory responses to stress (the pathway ‘H1.1’ in Figure 5.2) and whether negative social relationships interact with trait hostility to influence inflammatory responses to stress (the pathway ‘H1.4’ in Figure 5.2). This study involved an experimental protocol to induce physiological responses to stress in a lab setting and provided the chance to observe the effects of negative social relationships and trait hostility on inflammatory responses to stress over and above the levels of depressed mood. The results presented in chapter 2 advance the previous social relationship literature by examining social conflicts in four different relationship areas and by investigating whether multiple social conflicts interact with trait hostility to affect physiological responses to stress. The third study in chapter 4 tested the mediational pathways between depression, social relationships, and inflammation by utilizing longitudinal data from a nationally representative data set. The results of this study clarified pathways ‘H3.1’, ‘H3.2’, and ‘H3.3’ of Figure 5.2 that proposed the social relationship processes by which depression is related to diverse chronic disease outcomes.

In this final overall discussion, the results of all three studies will be integrated by four different topic areas that are relevant to the theoretical review in chapter 1: a) how the three studies reveal important aspects related to diverse dimensions of emotion; b) the degree to which the three studies support the stress amplifying model of social relationships; c) the dynamic interaction between emotion and social relationship via the stress generation model; d) the role of gender and age on the joint effects of emotional disturbances and negative social relationships on physical health.

**Different dimensions of emotion: emotional states, traits, and disorders**

The three studies in this dissertation examined the effects of emotional traits, states, and symptoms of affective disorders on various indicators of physical health. Cohen and Rodriguez
suggested the term ‘affective disturbances’ to refer to both subclinical negative emotion and affective disorders. In their framework, emotional state refers to “transient fluctuations in emotion” whereas emotional traits refer to “stable individual differences in affective level that are often associated with cognitive and behavioral style”; clinical mood disorders are “episodes of extreme affect that occur in conjunction with specified behavioral and cognitive dysfunctions”. The first study examined two emotional traits – trait hostility and depression – whereas the day to day fluctuations in negative and positive emotional states and depressive symptoms were examined in the second and third study.

Although it is difficult to compare emotional traits, states, and disorders across individuals with different health conditions and age groups, previous literature suggests two possible explanations for whether emotional states, traits, and disorders may or may not have distinguishable effects on health. First, negative state mood, negative emotional traits like trait depression, and depressive disorders may have similar adverse effects on physical health but with different degree and severity. For example, both depressed emotional states and clinical depressive disorders are associated with suppressed immunity, but clinical depressive disorder to a greater degree (for a review, see Herbert & Cohen, 1993). Second, emotional states, traits, and disorders may have qualitatively distinctive effects on physical health. Another study by Cohen and colleagues that compare state and trait negative affect suggests that only state negative affect was associated with objective markers of severity in respiratory viral infection (Cohen, Doyle, Skoner, Gwaltney, & Newsom, 1995). Other studies suggest that negative emotional traits but not emotional states are associated with other cognitive traits such as low self-esteem that are associated with symptoms of chronic disease conditions including heart diseases (Costa & McCrae, 1980). This explanation stipulates that emotional states may reflect acute illness, whereas emotional traits and disorders may be associated more with other biological and behavioral changes involved in pathogenesis of chronic disease conditions.
The results of three studies in this dissertation generally support the idea that negative emotional states, trait hostility, and symptoms of depression generally work in the same direction to negatively affect inflammation, sleep quality, and general physical health status. In the first study, trait hostility and depression were examined in terms of their effects on baseline levels of inflammatory markers as well as inflammatory responses to a laboratory stressor. The results of the first study showed that both trait hostility and depression were associated with higher baseline IL-6 among healthy young adults. In the second study, the effects of day to day fluctuations in negative and positive state emotion on the same night’s sleep quality were examined in older adults with chronic pain condition. The results of the study demonstrated that higher levels of daily negative emotional state were associated with poorer sleep quality and lower levels of daily positive emotional state were associated with feeling less refreshed after sleep within the same individual across different days. Finally, the third study examined whether depressive symptoms were associated with physical health status at 10 years follow up among middle aged and older adults. The results for direct effects of depression on physical health indicators suggest that depressive symptoms at baseline were marginally associated with higher levels of CRP and significantly associated with lower sleep quality 10 years later. Therefore, the three studies in the dissertation confirm that negative emotional states, negative emotional traits, and mood disorders have the effect on physical health in the same direction and also provide evidence in the short-term impact of emotional states and long-lasting impact of depressive symptoms on physical health outcomes.

**The stress amplifying model of social relationships – different aspects of social relationships that aggravate psychological distress**

The three studies primarily examined negative aspects of social relationships with the exception of the second study, which included both negative and positive spouse responses to pain behaviors. The stress amplifying model of social relationship suggests that close
interpersonal relationships may generate stressors if the relationships involve frequent negative interactions, conflicts, inequity, and lack of commitment (Burman & Margolin, 1992; Cohen, 2004; Guerrero & Anderson, 2000); even while they can also provide support when things are going well, all three studies in this dissertation indicate that multiple negative social relationships, mismatched supportive behaviors, and perception of risk in a close relationship may generate greater chronic or acute stress and lead to adverse physical health outcomes. Different pathways in the theoretical models represented in Figures 5.1 and 5.2 were established based on the stress amplifying model and were tested via the three studies of this dissertation. The first study examined the pathways between social stress (as generated by multiple negative relationships) and inflammatory response to stress, and whether trait hostility moderated that relationship, as illustrated by the ‘H1,1’ and ‘H1,4’ in Figure 5.2. The findings of the first study demonstrated that individuals who have negativity across multiple relationship areas showed increases in the levels of IL-6 and TNF-α after stress; in terms of the stress amplifying model, the study thus suggests that having multiple negative relationships is a detrimental chronic stressor that may trigger acute over-production of cytokines in face of an acute psychosocial stressor. The second study tested the pathways ‘H2,1 & 2’ and ‘H2,3,4, & 5’ in Figure 5.1 for the amplifying or mitigating effects of social relationships on the association between emotional states and sleep. The results of the second study revealed that even seemingly supportive helping behaviors of a partner (i.e., solicitous responses) can have adverse effects on sleep among older adults with a chronic pain condition; the detrimental effects of solicitous response of a partner were pronounced when those with chronic pain had more than usual levels of negative emotional state or less than usual levels of positive emotional state. These results underscore the potential aggravation of the existing emotional distress of those with chronic pain when they receive support from their partner that is not desired or which violates their sense of autonomy. Finally, the third study tested the pathways from depression to later social relationship processes that generate social stress (pathway ‘H3,2’).
and the pathways from social stress to physical health outcomes (pathway ‘H₃’) in Figure 5.2. The results of the third study exhibited that perceiving risk and troubles in one’s marital relationships is associated with more numbers of physical symptoms and chronic conditions among married adults, which in turn are associated with higher levels of inflammatory markers. Perceiving risk and troubles in one’s marital relationship generates substantial psychological distress via the threat of relationship termination and also by taking away the support and security that social bonding with a partner can provide.

The supportive side of social relationships was not the focus of this dissertation but was examined to some extent via the daily empathizing response of partners in the second study (chapter 3). The stress buffering model of social relationships proposes that individuals with positive social relationships experience less impact from ongoing stressors because positive social relationships provide additional resources to cope with stress (Cohen, 2004; Uchino et al., 1996). Based on that model, receiving empathic responses from a partner was expected to reduce the impact of negative emotional states on sleep of chronic pain patients in study 2. However, the results of this study showed that receiving more or less empathic responses from a partner than the usual for the person did not change the degree by which negative or positive emotional state affect sleep quality or feeling refreshed after sleep among older adults with chronic pain. Thus, this finding indicates that individuals with chronic pain may not be sensitive to daily fluctuations of supportive interactions with their spouse; rather they are influenced by the overall quality of marital relationship and typical levels of empathy from the spouse (Leonard et al., 2006).

**Stress generation model of depression for explaining how emotional states, traits, and mood disorders interact with close interpersonal relationships to affect physical health**

The stress generation model of depression provides a comprehensive framework for how emotional traits and mood disorder and interpersonal relationships interact with each other in generating stress and produce negative consequences on physical health (Coyne, 1976; Davila et
al., 1997; Hammen, 1991). The model suggests that individuals with negative emotional traits, such as trait depression and hostility or mood disorders, tend to have negative reactions in their social interactions (e.g., feedback seeking for their negative self-view or aggressive reactions) and show decreased functioning in their social roles in their family or workplace. The negative reactions and decreased role functioning can further generate stress in the interpersonal relationship, stress which may have direct negative impact on physical health or indirect impact on health via deteriorating coping resources via social support (Coyne, 1976). The stress generation model of depression was supported by the results of third study but was not confirmed by the first study.

The third study (chapter 4) strongly supported the stress generation model by examining the longitudinal association between depressive symptoms, and subsequent aggravation of the marital relationship and physical health. The significant pathways between depressive symptoms and greater marital risk at 9 years follow up are in concordance with the pathways in Figure 5.2 that were established based on the stress generational model. The greater marital risk in turn resulted in more numbers of physical symptoms and chronic conditions that were linked with higher levels of inflammatory markers.

The first study (chapter 2) does not fully support the stress generation model because there was a lack of interaction between trait hostility and negative social relationships in multiple social areas. Trait hostility was associated with elevated baseline levels of IL-6, whereas having multiple negative social relationships was associated with acute increases in IL-6 and TNF-α in response to a laboratory stressor. Elevated baseline levels of IL-6 can be viewed as an indicator of underlying dysregulation of the inflammatory response system in the body. If the inflammatory response system of individuals is already compromised, they are likely to respond to an acute stressor with over production of pro-inflammatory cytokines. Although the pattern of association did not play out in this particular study exactly as expected, based on this and previous research it
seems clear that hostility and negative relationships are related to inflammation; the effects of high hostility and negative social relationships may well be additive over time, leading to long-term health problems such as development of inflammation related chronic disease conditions.

**Negativity and positivity in emotion and social relationship: is bad stronger than good?**

There is some evidence in the literature that negative emotion and negativity in close relationships has more powerful impact on general adaptations (including better physical health), than does positivity in emotional and interpersonal experiences (Baumeister, Bratslavsky, Finkenauer, & Vohs, 2001; Gable & Reis, 2001; Taylor, 1991). The results of this dissertation provide mixed evidence to add to this the literature. In the second study (chapter 3), positive state emotion was associated with feeling refreshed after sleep, over and above the levels of negative state emotion. However, negative state emotion was associated with overall sleep quality, which suggests that fluctuations in either negative or positive state emotion predict slightly different perceptions of sleep quality. Also in the second study, neither the partner’s punishing nor did empathic responses to pain behaviors predict sleep quality, whereas solicitous responses aggravated the adverse impacts of daily state emotion on sleep quality. Thus, in this study using within person comparisons over time, negative state emotion and negative partner responses do not necessarily have a more powerful impact on sleep than positive emotion and partner responses.

The first and third studies examined only the negative emotional traits and disorder and negative aspects of close relationships, and are thus of limited utility in providing direct evidence for whether ‘bad is stronger than good’ in terms of the effects of emotion on health. However, these studies provide additional evidence that negative emotional traits (like hostility and depression) and negativity in close relationships do have an adverse impact on markers of inflammation and physical health status. Although it was not a direct goal of the current dissertation to examine this, all of the three datasets suggest that positive emotional experiences
and supportive social interactions were much more prevalent and common than negative emotion and negative social interactions across the three samples. As Baumeister and colleagues suggested in their review, negative emotion and social relationships are associated with more immediate psychological, behavioral, and physiological reactions and consequences that are directly related to survival in one’s environment but “good may prevail over bad by superior force of numbers: “Many good events can overcome the psychological effects of a single bad one” (Baumeister et al., 2001). Thus, studies comparing emotional and social negativity with positivity suggest that any levels of negativity in emotion and social relationships would have more pronounced impact on physical health than positivity, but positivity would have detrimental effects on health when it loses relative prevalence over negativity in emotion and social relationships (e.g., anhedonia or social isolation).

**Moderation by gender and age**

The three studies utilized three different data sets that involved women and men with different age ranges. Although examining the role of gender and age was not the main goal of this dissertation, gender moderation was examined on an exploratory basis in the first and third studies, and age moderation was examined in the third study.

Previous studies suggest certain gender differences in the emotional, social, and physiological factors that are examined in this dissertation. For example, in U.S. culture women are more likely to experience intense emotion and express their emotion to others (Fujita, Diener, & Sandvik, 1991). Women are twice more likely to develop major depressive disorders than men (Fujita, Diener, & Sandvik, 1991; Kring & Gordon, 1998; Nolen-Hoeksema, 2001). Several studies also suggest that women are more likely to seek out social support and engage in emotional focused coping with stress, take on the role of maintaining in social relationships, and exhibit different physiological responses to stress from men (Tamres, Janicki, & Helgeson, 2002; Taylor et al., 2000).
The results of the third study suggest that the link between depression and marital risk at follow-up was significant only among men. Also, only men in the sample of the third study appear to evidence a negative impact of depression on their sleep quality. Gender socialization theory suggests that the feminine and masculine characteristics in emotional and social behaviors are learned as girls and boys are socialized to play out given gender roles. Specifically, parents and other members of society provide gender biased feedback to women to play the traditional gender roles of warm, emotional, and sensitive females and to men to emphasize assertive and action oriented qualities, from infancy throughout the lifespan (Tamres et al., 2002). This theory suggests that depressed men may have particular difficulty when they cannot maintain the active and assertive gender role in their marital relationship. Men’s tendency to withdraw from interpersonal conflict may be aggravated when men are depressed, which may generate more frustration and stress in their marriage (O’ Mahen et al., 2001). Also, given the previous research that men tend to underreport their symptoms of depression (Shaw et al., 1995), the depressive symptoms reported by men may have represented more severe conditions than those reported by women, and thus explaining their more negative impact on men’s sleep disturbances. Thus, the gender moderation in the third study suggests that men may have extra vulnerability to negative consequences of depression on their social relationship and health and imply that more gender role tailored interventions for diagnosing and intervening with depression in men may be beneficial (Kilmartin, 2005).

In the first study, only men but not women showed the increases in IL-6 response to stress and also showed a slight elevation in baseline levels of TNF-α. The tend and befriend theory provides an evolutionary explanation for why women and men may show different physiological reactions to stressful situations and why women often take the primary roles to create, maintain, and utilize social groups (Taylor et al., 2000). The theory suggests that in order to enhance survival of both self and offspring, women developed more calming responses and
support seeking ways of coping with stress to supplement flight and flight responses. Taylor and colleagues suggest that oxytocin and endogenous opioid mechanisms may play an important role in generating the gender differences in physiological responses to stress (Taylor et al., 2000). Although the gender differences in stress response may depend on the characteristics of stressors (Stroud, Salovey, & Epel, 2002), women exhibit less stress reactivity in blood pressure and catecholamine production but more reactivity in producing glucocorticoids (Arthur, Katkin, & Mezzacappa, 2004; Gallucci et al., 1993). It is known that glucocorticoids, which are assumed to be activated at a larger degree in women under acute stress, play a role in suppressing the production of pro-inflammatory cytokines in the body (Chrousos & Gold, 1992). Thus, tend and befriend theory may provide possible explanations for why women with multiple negative relationships did not (but men did) show reactivity in cytokine responses to the acute psychosocial stressor.

As people age, they typically experience deteriorations in the quality of their sleep and develop more severe physical health problems (Floyd et al., 2000; Miles & Dement, 1980). Also, people engage in different goals throughout lifespan that create different social contexts, and are known to experience emotions with different intensity (Pearlin & Skaff, 1996; Williams & Umberson, 2004). Although it was not the purpose of this dissertation to focus on aging and thus to recruit diverse age groups, the three studies of this dissertation involved different age groups with different physical health status, from healthy young adults to older adults with knee osteoarthritis and middle aged and older adults with several chronic conditions. Only the third study enabled the examination of age moderation of the significant findings, which was discussed in depth in the previous chapter. For young and middle aged adults who are less than 64 years old, depression was a significant predictor for sleep quality 9 years later. Depression predicted greater marital risk only among older adults who are over 65 years old. Thus, the results indicated that
individuals may have different vulnerabilities for having impact of depression on sleep and marital risk across life span.

Overall, the gender and age moderation findings in this dissertation suggest the potential differences in gender and age groups in impact of social relationship problems on health and the degree to which interaction between emotional disturbances and social conflicts affect later physical health outcomes. Thus, the dissertation reveals the necessity of having gender balanced sample and of considering age related socioemotional and physiological changes when designing a study to examine the joint effects of emotional and social factors for physical health.

Clinical application and public health implication

Given the critical role of sustained inflammation and sleep problems in the development of major chronic disease conditions, the results of this dissertation underscore the importance of informing people who are at risk for developing such disease conditions to manage their on-going emotional disturbances and social relationship conflicts. In addition to the primary physical examinations and biomedical treatments for patients, healthcare professionals should utilize brief checklists for screening out individuals with substantial emotional disturbances and unresolved social conflicts in close relationship areas. As occurrence of short-term and long-term emotional disturbances and social relationship problems can be the underlying factor for aggravated physical health status, healthcare professionals should consider providing integrated treatment plans that involve both biomedical treatments and psychosocial interventions. There are well validated psychotherapy options for treating chronic experience of emotional disturbance (e.g., depressive symptoms), such as cognitive behavioral therapy and interpersonal therapy (Beck & Alford, 2009). As the results of this dissertation suggest that the adverse effects of emotional disturbances on physical health status often are exacerbated by problems in social relationships, the interpersonal therapy that involves not only a patient with physical illness but also a spouse,
family, and close others might be better effective at tackling the negative impact of emotional and social problems on physical illness.

From public health stand point, a psychotherapy type of approach might not be the most cost effective option that can be provided to everyone; a simple and standardized intervention that can be provided by any healthcare professional may seem preferable. However, previous studies with clinical trials have not been successful at developing standardized interventions that are effective at providing social support and resolving emotional difficulties for individuals with chronic disease conditions (e.g., ENRICHED study for improving depression and perceived social support for the patients who experienced cardiac events; Berkman et al., 2003). One likely reason for the ineffectiveness of these trials is that they focused on broadly trying to improve patient social skills without involving their partners or helping find ways to improve existing social relationships. Social conflicts in naturally existing social relationships can involve different issues that may not be handled well by a single solution of a standardized intervention. Thus, some degree of tailoring of an intervention to meet individual’s unique issues in their existing social relationships and emotional vulnerability would be necessary, despite the increased costs and efforts that come with this type of approach.

**Conclusion**

Overall, the three studies in the dissertation bridged the emotion and social relationship literatures by examining many of the moderating and mediating roles of social relationships on the association between emotional characteristics and physical health outcomes that are specified in the two theoretical models (Figure 5.1 and 5.2). Also, the study advances the social relationship and health literatures by examining the characteristics of social relationships in a detailed fashion and by taking the distinctive roles of state/trait emotion/and mood disorders into consideration. Thus, the findings and theoretical models of this dissertation demonstrated the importance of considering the dynamic interactions between individual’s emotional
characteristics and social processes that may generate additional stress or mitigate the existing stress when examining stress-related physical health status and disease outcomes.

Individual’s inner experiences of negative or positive emotion and individual differences in vulnerability to affective disorders do not act in solo to influence physical health. Rather, emotional states can be amplified or reduced in their strength by concurrent social interactions; further, symptoms of affective disorders can also modify the typical way people engage in social relationships. Thus, this dissertation captures interesting points where emotional factors and social relationships become interdependent with one another to affect molecular markers like inflammatory cytokines in the body as well as perceptions of sleep quality and physical symptoms of individuals. The findings of this dissertation imply that interventions for individuals who are vulnerable to the negative impact of emotional disturbances on their health should expand the scope of interventions from each individual to the not only them but also their close others. Specifically, it will be beneficial for both those with emotional vulnerability and their close others to acknowledge that their daily patterns of interaction involving emotional states/traits and social behaviors may be detrimental and to encourage them to focus resources toward changing maladaptive patterns that generate psychological and physical stress with the goal of improving their physical health.
Figure 5.1 The first theoretical model with tested pathways

Note. Yellow pathways were tested by hypotheses 1-5 in Study 2 of this dissertation.
Figure 5.2 The second theoretical model with tested pathways

Note. Green pathways were tested by hypotheses 1-5 in Study 1, and skyblue pathways were examined by hypothesis 1-3 in Study 3 of this dissertation.
References


Howren, M. B., Lamkin, D. M., & Suls, J. (2009). Associations of depression with C-reactive protein, IL-1, and IL-6: A meta-analysis. *Psychosomatic Medicine, 71*, 171-186. doi: [10.1097/PSY.0b013e3181907c1b](10.1097/PSY.0b013e3181907c1b)


medically treated patients with angiographically documented coronary artery disease.


Appendix A.

Center for Epidemiologic Studies Depression Scale (CES-D; Study 1)

INSTRUCTION: Below is a list of the ways you might have felt or behaved. Please tell me how often you have felt this way during the past week, using the scale below.

<table>
<thead>
<tr>
<th>During the Past Week</th>
<th>Rarely or none of the time (less than 1 day)</th>
<th>Some or a little of the time (1-2 days)</th>
<th>Occasionally or a moderate amount of time (3-4 days)</th>
<th>Most or all of the time (5-7 days)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

1. I was bothered by things that usually don’t bother me.
2. I did not feel like eating; my appetite was poor.
3. I felt that I could not shake off the blues even with help from my family or friends.
4. I felt I was just as good as other people.
5. I had trouble keeping my mind on what I was doing.
6. I felt depressed.
7. I felt that everything I did was an effort.
8. I felt hopeful about the future.
9. I thought my life had been a failure.
10. I felt fearful.
11. My sleep was restless.
12. I was happy.
13. I talked less than usual.
15. People were unfriendly.
16. I enjoyed life.
17. I had crying spells.
18. I felt sad.
19. I felt that people dislike me.
20. I could not get “going.”

SCORING: zero for answers in the first column, 1 for answers in the second column, 2 for answers in the third column, 3 for answers in the fourth column. The scoring of positive items is reversed. Possible range of scores is zero to 60, with the higher scores indicating the presence of more symptomatology.
Appendix B. Cook-Medley Hostility Scale (Study 1)

INSTRUCTION: This inventory consists of numbered statements. Read each statement and decide whether it is true as applied to you or false as applied to you. Please circle the appropriate letter (T-true, F-false) directly to the right of each statement. If a statement is TRUE or MOSTLY TRUE as applied to you, circle the letter “T”. If a statement is FALSE or NOT USUALLY TRUE, as applied to you, circle the letter “F”. Remember to give YOUR OWN opinion of yourself. Do not leave any blank spaces if you can avoid it.

<table>
<thead>
<tr>
<th>Statement</th>
<th>T</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. When I take a new job, I like to be tipped off on who should be gotten next to.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>2. When someone does me a wrong, I feel I should pay him back if I can just for the principle of the thing.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>3. 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.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>4. I think a great many people exaggerate their misfortunes in order to gain the sympathy ad help of others.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>5. I take a lot of argument to convince most people of the truth.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>6. I think most people would lie to get ahead.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>7. Someone has it in for me.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>8. Most people are honest chiefly through fear of being caught.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>9. Most people will use somewhat unfair means to gain profit for an advantage rather than to lose it.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>10. I commonly wonder what hidden reason another.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>11. It makes me impatient to have people ask my advice or otherwise interrupt me when I am working on something important.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>12. I feel that I have often been punished without cause.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>13. I am against giving money to beggars.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>14. Some of my family have habits that bother and annoy me very much.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>15. My relatives are nearly all in sympathy with me.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>16. My way of doing things is apt to be misunderstood by others.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>17. I don’t blame anyone for trying to grab everything he can get in this world.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>18. No one cares much what happens to you.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>19. I can be friendly with people who do things I consider wrong.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>20. It is safer to trust people.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>21. I do not blame a person for taking advantage of someone who lays himself open to it.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>22. I have often felt that strangers were looking at me critically.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>23. Most people make friends because friends are likely to be useful to them.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>24. I am sure I am being talked about.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>25. I am likely not to speak to people until they speak to me.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>26. Most people inwardly dislike putting themselves out to help other people.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>27. I tend to be on my guard with people who are somewhat friendlier than I had expected.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>28. I have sometimes stayed away from another person because I feared doing something that I might regret afterwards.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>29. People often disappoint me.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>30. I like to keep people guessing what I’m going to do next.</td>
<td>T</td>
<td>F</td>
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<tr>
<td>---</td>
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</tr>
<tr>
<td>31. I frequently ask for advice.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>32. I am not easily angered.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>33. I have often met people who were supposed to be experts who were not better than I.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>34. I would certainly enjoy beating a crook at his own game.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>35. It makes me feel like a failure when I hear of the success of someone I know well.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>36. I have at times had to be rough with people who were rude or annoying.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>37. People generally demand more respect for their own rights than they are willing to allow for others.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>38. There are certain people whom I dislike so much that I am inwardly pleased when they are catching it for something they have done.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>39. I am often inclined to go out of my way to win a point with someone who has opposed me.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>40. I am quite often not in on the gossip and talk of the group I belong to.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>41. The man who had most to do with me when I was a child (such as my father, stepfather, etc.).</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>42. I have often found other people jealous of my good ideas, just because they had not thought of them first.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>43. When a man is with a woman he is usually thinking about things related to her sex.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>44. I do not try to cover up my poor opinion or pity of a person so that he won’t know how I feel.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>45. 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 mistakes onto those under them.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>46. I strongly defend my own opinions as a rule.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>47. People can pretty easily change me even though I thought my mind was already make up on a subject.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>48. Sometimes I am sure that other people can tell what I am thinking.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>49. A large number of people are guilty of bad sexual conduct.</td>
<td>T</td>
<td>F</td>
</tr>
<tr>
<td>50. I have often had to take orders from someone who did not know as much as I did.</td>
<td>T</td>
<td>F</td>
</tr>
</tbody>
</table>
Appendix C. Questionnaire for Negative Social Relationships (Study 1)

Please describe your current living conditions (check all that apply):

___ I live alone
___ I live with roommates on campus
___ I live with roommates off campus
___ I live with my spouse/partner or partner
___ Other, please describe:

A. ROOMMATES

This section asks about your relationship with your roommates who are not your significant other (i.e., girlfriend, boyfriend, spouse). If you do not have roommates, then skip to section B (Closest Friend).

1. Describe the number of roommates that you have: ____ male, ____ female

2. Using a scale from 1 to 5 where 1 means “the worst possible relationship” and 5 means “the best possible relationship”, how would you rate your overall relationship with your roommate(s) these days? ____

1 2 3 4 5
Worst Best

3. How much do(es) your roommate(s) understand the way you feel about things? ____

1 2 3 4 5
Not at all Not at all Very much

4. How much can you depend on your roommate(s) to be there when you really need them? ____

1 2 3 4 5
Not at all Very much

5. How much do(es) your roommate(s) show concern for your feelings and problems? ____

1 2 3 4 5
Not at all Very much

6. How much can you trust your roommate(s) to keep their promises to you? ____

1 2 3 4 5
Not at all Very much

7. How much can you open up to your roommate(s) about things that are really important to you? ____
1 2 3 4 5
Not at all Very much

8. How much tension is there between you and your roommate(s)? ___

1 2 3 4 5
Not at all Very much

9. How often do you have an unpleasant disagreement with your roommate(s)? ___

1 2 3 4 5
Never Very often

10. How often do things become tense when you and your roommates(s) disagree? ___

1 2 3 4 5
Never Very often

11. How often do(es) your roommate(s) say cruel or angry things during a disagreement? ___

1 2 3 4 5
Never Very often

12. How often do you and your roommate(s) both refuse to compromise during disagreements?

1 2 3 4 5
Never Very often

B. CLOSEST FRIEND

This section asks about your relationship with your closest friend who is not your significant other (i.e., boyfriend, girlfriend, or spouse).

1. Describe your closest friend: ___male ___female

2. Using a scale from 1 to 5 where 1 means "the worst possible relationship" and 5 means "the best possible relationship," how would you rate your overall relationship with your closest friend these days? ___

1 2 3 4 5
Worst Best

3. How much does your closest friend understand the way you feel about things? ___

1 2 3 4 5
Not at all Very much

4. How much can you depend on your closest friend to be there when you really need them? ___
5. How much does your closest friend show concern for your feelings and problems? ___

Not at all           Very much

6. How much can you trust your closest friend to keep their promises to you? ___

Not at all           Very much

7. How much can you open up to your closest friend about things that are really important to you? ___

Not at all           Very much

8. How much tension is there between you and your closest friend? ___

None               A lot

9. How often do you have an unpleasant disagreement with your closest friend? ___

Never             Very often

10. How often do things become tense when the two of you disagree? ___

Never             Very often

11. How often does your closest friend say cruel or angry things during a disagreement? ___

Never             Very often

12. How often do the two of you both refuse to compromise during disagreements? ___

Never             Very often

C. FAMILY

This section asks about your relationship with your family (e.g., parents and siblings).
1. Using a scale from 1 to 5 where 1 means “the worst possible relationship” and 5 means “the best possible relationship”, how would you rate your overall relationship with your family these days? ____

1  2  3  4  5
Never          Very often

2. How much does your family understand the way you feel about things? ____

1  2  3  4  5
Not at all      Very much

3. How much can you depend on your family to be there when you really need them? ____

1  2  3  4  5
Not at all      Very much

4. How much does your family show concern for your feelings and problems? ____

1  2  3  4  5
Not at all      Very much

5. How much can you trust your family to keep their promises to you? ____

1  2  3  4  5
Not at all      Very much

6. How much can you open up to your family about things that are really important to you? ____

1  2  3  4  5
Not at all      Very much

7. How much tension is there between you and your family? ____

1  2  3  4  5
None           A lot

8. How often do you have an unpleasant disagreement with your family? ____

1  2  3  4  5
Never          Very often

9. How often do things become tense when you and your family disagree? ____

1  2  3  4  5
Never          Very often

10. How often does your family say cruel or angry things during a disagreement? ____
11. How often do you and your family both refuse to compromise during disagreements? ___

1  2  3  4  5
Not at all  Very much

D. SIGNIFICANT OTHER/PARTNER/SPouse.

This section asks about your relationship with your significant other (e.g., boyfriend, girlfriend, spouse). The questions are not inquiring about your sexual activity with your significant other. Rather, they are inquiring about affectionate contact (which may or may not include sexual activity). Please feel free to skip any question that you do not feel comfortable answering.

1. Using a scale from 1 to 5 where 1 means "the worst possible relationship" and 5 means "the best possible relationship," how would you rate your overall relationship these days? ___

1  2  3  4  5
Worst  Best

2. How much does your significant other understand the way you feel about things? ___

1  2  3  4  5
Not at all  Very much

3. How much can you depend on your significant other to be there when you really need them? ___

1  2  3  4  5
Not at all  Very much

4. How much does your significant other show concern for your feelings and problems?

1  2  3  4  5
Not at all  Very much

5. How much can you trust your significant other to keep their promises to you? ___

1  2  3  4  5
Not at all  Very much

6. How much can you open up to your significant other about things that are really important to you? ___

1  2  3  4  5
Not at all  Very much

7. How much tension is there between you and your significant other? ___
1  2  3  4  5  
Not at all  Very much

8. How often do you have an unpleasant disagreement with your significant other? ___
1  2  3  4  5  
Not at all  Very much

9. How often do things become tense when the two of you disagree? ___
1  2  3  4  5  
Not at all  Very much

10. How often does your significant other say cruel or angry things during a disagreement? ___
1  2  3  4  5  
Not at all  Very much

11. How often do the two of you both refuse to compromise during disagreements? ___
1  2  3  4  5  
Not at all  Very much

E. CHILDREN

This section asks about your relationship with your children. If you do not have children, then move on to the next questionnaire.

Describe the number of children that you have: __ male, __ female.

2. Using a scale from 1 to 5 where 1 means "the worst possible relationship" and 5 means “best possible relationship”, how would you rate your overall relationship with your child(ren) these days? ___
1  2  3  4  5  
Worst  Best

3. How much do(es) your child(ren) understand the way you feel about things? ___
1  2  3  4  5  
Not at all  Very much

4. How much can you depend on your do(es) you child(ren) to be there when you really need them? ___
1  2  3  4  5  
Not at all  Very much
5. How much do(es) your child(ren) show concern for your feelings and problems? ___

1 2 3 4 5
Not at all Very much

6. How much can you trust your child(ren) to keep their promises to you? ___

1 2 3 4 5
Not at all Very much

7. How much can you open up to your child(ren) about things that are really important to you? ___

1 2 3 4 5
Not at all Very much

8. How much tension is there between you and your child(ren)? ___

1 2 3 4 5
None A lot

9. How often do you have an unpleasant disagreement with your child(ren)? ___

1 2 3 4 5
Never Very much

10. How often do things become tense when you and your child(ren) disagree? ___

1 2 3 4 5
Never Very much

11. How often do(es) your child(ren) say cruel or angry things during a disagreement? ___

1 2 3 4 5
Never Very much

12. How often do you and your child(ren) both refuse to compromise during disagreements? ___

1 2 3 4 5
Not at all Very much
Appendix D. Mood Assessment (Study 1)

Please indicate below how you are feeling at this moment. Answer quickly & honestly.

<table>
<thead>
<tr>
<th>Mood Description</th>
<th>Not at all</th>
<th>Very much</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Nervous, tense, worried</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>2. Happy</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>3. Thirsty</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>4. Frustrated</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>5. Impatient</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>6. Tired, low energy</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>7. Nauseous or queasy</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>8. Angry</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>9. Sad</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>10. Hungry</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>11. Restless</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>12. Focused, attentive</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>13. Irritable</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>14. Curious, interested</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>15. Disorganized</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>16. Relaxed, comfortable</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>17. Stressed</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>18. Light-headed</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
</tbody>
</table>
Appendix E

Questionnaire for Daily Negative and Positive Mood (Study 2)

INSTRUCTION: Please answer the following questions by circling your response.

<table>
<thead>
<tr>
<th></th>
<th>not at all</th>
<th>slightly</th>
<th>moderately</th>
<th>extremely</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. To what extent have you felt <strong>depressed</strong> or blue over the past 30 minutes?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>2. To what extent have you felt <strong>happy</strong> over the past 30 minutes?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>3. To what extent have you felt <strong>frustrated</strong> over the past 30 minutes?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>4. To what extent have you felt <strong>joyful</strong> over the past 30 minutes?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>5. To what extent have you felt <strong>angry or hostile</strong> over the past 30 minutes?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>6. To what extent have you felt <strong>pleased</strong> over the past 30 minutes?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>7. To what extent have you felt <strong>enjoyment</strong> over the past 30 minutes?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>8. To what extent have you felt <strong>unhappy</strong> over the past 30 minutes?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>9. To what extent have you felt <strong>worried or anxious</strong> over the past 30 minutes?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>
Appendix F

Questionnaire for Partner Responses to Patients’ Pain Behaviors (Study 2)

<table>
<thead>
<tr>
<th></th>
<th>Question</th>
<th>Scale</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Today, you took over your spouse’s jobs or duties to help him/her avoid pain.</td>
<td>1, 2, 3</td>
</tr>
<tr>
<td>2</td>
<td>Today, you ignored your spouse when he/she seemed to be in pain.</td>
<td>1, 2, 3</td>
</tr>
<tr>
<td>3</td>
<td>Today, you tried to get your spouse to rest when he/she seemed to be in pain.</td>
<td>1, 2, 3</td>
</tr>
<tr>
<td>4</td>
<td>Today, you showed your spouse affection in order to give comfort when he/she seemed to be in pain.</td>
<td>1, 2, 3</td>
</tr>
<tr>
<td>5</td>
<td>Today, you got your spouse something to eat or drink when he/she seemed to be in pain.</td>
<td>1, 2, 3</td>
</tr>
<tr>
<td>6</td>
<td>Today, you got frustrated with your spouse when he/she seemed to be in pain.</td>
<td>1, 2, 3</td>
</tr>
<tr>
<td>7</td>
<td>Today, you showed your spouse that you knew how the pain made him/her feel.</td>
<td>1, 2, 3</td>
</tr>
<tr>
<td>8</td>
<td>Today, you tried to just be there for your spouse when he/she seemed to be in pain, by giving your undivided attention.</td>
<td>1, 2, 3</td>
</tr>
<tr>
<td>9</td>
<td>Today, you became irritated with your spouse when he/she seemed to be in pain.</td>
<td>1, 2, 3</td>
</tr>
</tbody>
</table>

- Items for solicitous responses to pain: 1, 3, 5
- Items for punishing responses to pain: 2, 6, 9
- Items for empathic responses to pain: 4, 7, 8
Appendix G. Questionnaire for Daily Sleep Quality (Study 2)

Good Morning! Please answer the following questions before going about your day, by circling your response.

1. How would you rate the overall quality of your sleep last night?
   - 0  very good
   - 1  fairly good
   - 2  fairly bad
   - 3  very bad

<table>
<thead>
<tr>
<th>not at all</th>
<th>slightly</th>
<th>moderately</th>
<th>extremely</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

2. How refreshed or rested do you feel after last night's sleep?

<table>
<thead>
<tr>
<th>not at all</th>
<th>slightly</th>
<th>moderately</th>
<th>extremely</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>
Appendix H

Physical Health: Self-Reported Health & Comorbidities (Study 2)

We will end this interview with several sets of questions about your physical health.

1. In general, would you say your health is:
   1. Excellent
   2. Very good
   3. Good
   4. Fair, or
   5. Poor?

Please answer the following yes or no questions.

2. Do you currently have high blood pressure?
   1. No
   2. Yes

3. Do you currently have problems with an irregular heart beat or chest pain?
   1. No
   2. Yes

4. Have you ever been told that you have coronary heart disease or coronary artery disease?
   1. No
   2. Yes

5. Have you ever had a heart attack?
   1. No
   2. Yes

6. Have you ever been treated for congestive heart failure?
   1. No
   2. Yes

7. Have you ever had a stroke?
   1. No
   2. Yes

8. Do you currently have fibromyalgia, osteoporosis, or any other serious muscular or bone problems in addition to osteoarthritis?
   1. No
   2. Yes

9. Do you currently have asthma, emphysema, chronic bronchitis, chronic obstructive lung disease, or any other serious respiratory problems?
   1. No
2. Yes

10. Do you currently have stomach ulcers, irritable bowel syndrome, or any other serious problems with your stomach or bowels?
   1. No
   2. Yes

11. Do you have diabetes?
   1. No
   2. Yes

12. Do you currently have problems with your kidneys?
   1. No
   2. Yes

13. Do you have cirrhosis or any other serious liver problems?
   1. No
   2. Yes

14. Do you currently have cancer?
   1. No
   2. Yes

15. Do you currently have rheumatoid arthritis, lupus, acquired immune deficiency syndrome, multiple sclerosis, scleroderma, or any other autoimmune problem?
   1. No
   2. Yes

16. Do you currently have problems with blood circulation in your legs, hemophilia, or any other blood-related problems?
   1. No
   2. Yes

17. Do you have epilepsy or any other neurological problems?
   1. No
   2. Yes

18. Do you currently have an overactive or underactive thyroid, or any other thyroid problems?
   1. No
   2. Yes

19. (FOR WOMEN ONLY:) Do you currently have any serious gynecological problems?
   1. No
   2. Yes

20. Do you currently have any problems with your vision or hearing?
   1. No
   2. Yes

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Appendix I. Marital Risk Scale (Study 3)

SECTION L: MARRIAGE OR CLOSE RELATIONSHIP

If you are married, or living with a partner in a marriage-like relationship, please answer the questions in this section. If you do not currently have a spouse or partner, please go to Section M on page 42.

1. During the past year, how often have you thought your relationship might be in trouble?

___ Never
___ Once
___ A few times
___ Most of the time
___ All of the time

2. It is always difficult to predict what will happen in a relationship, but realistically, what do you think the chances are that you and your partner will eventually separate?

___ Very likely
___ Somewhat likely
___ Not very likely
___ Not likely at all
Appendix J. Marital Disagreement Scale (Study 3)

INSTRUCTION: Couples often disagree about a lot of issues in life. How much do you and your spouse or partner disagree on the following issues?

<table>
<thead>
<tr>
<th></th>
<th>A lot</th>
<th>Some</th>
<th>A little</th>
<th>Not at all</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Money matters, such as how much to spend, save or invest.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>b. Household tasks, such as what needs doing and who does it.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>c. Leisure time activities, such as what to do and with whom.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>
Appendix K. Spouse Strain (Study 3)

INSTRUCTION: Answer how much for each of these items.

<table>
<thead>
<tr>
<th>Question</th>
<th>Often</th>
<th>Sometimes</th>
<th>Rarely</th>
<th>Never</th>
</tr>
</thead>
<tbody>
<tr>
<td>g. How often does your spouse or partner make too many demands on you?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>h. How often does he or she make you feel tense?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>i. How often does he or she argue with you?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>j. How often does he or she criticize you?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>k. How often does he or she let you down when you are counting on him or her?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>l. How often does he or she get on your nerves?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>
Appendix L

The Pittsburgh Sleep Quality Index, PSQI (Study 3)

Instructions: The following questions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. Please answer all questions.

During the past month,
1. When have you usually gone to bed? ___________________
2. How long (in minutes) has it taken you to fall asleep each night? ___________________
3. When have you usually gotten up in the morning? ___________________
4. How many hours of actual sleep do you get at night? (This may be different than the number of hours you spend in bed) ___________________

<table>
<thead>
<tr>
<th>5. During the past month, how often have you had trouble sleeping because you…</th>
<th>Not during the past month (0)</th>
<th>Less than once a week (1)</th>
<th>Once or twice a week (2)</th>
<th>Three or more times a week (3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Cannot get to sleep within 30 minutes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>b. Wake up in the middle of the night or early morning</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>c. Have to get up to use the bathroom</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>d. Cannot breathe comfortably</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>e. Cough or snore loudly</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>f. Feel too cold</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>g. Feel too hot</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>h. Have bad dreams</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>i. Have pain</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>j. Other reason(s), please describe, including how often you have had trouble sleeping because of this reason(s):</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

6. During the past month, how often have you taken medicine (prescribed or “over the counter”) to help you sleep?

7. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?

8. During the past month, how much of a problem has it been for you to keep up enthusiasm to get things done?

9. During the past month, how would you rate your sleep quality overall

<table>
<thead>
<tr>
<th>Very good (0)</th>
<th>Fairly good (1)</th>
<th>Fairly bad (2)</th>
<th>Very bad (3)</th>
</tr>
</thead>
</table>
Appendix M.

Physical symptoms and chronic conditions (Study 3)

Have you ever had any of the following conditions/illnesses?

<table>
<thead>
<tr>
<th>Symptoms and conditions</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>High blood pressure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Circulation problems</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blood clots</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart murmur</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Transient Ischemic Attack (TIA) or stroke</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anemia or other blood disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cholesterol problems</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asthma</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Emphysema/Chronic obstructive pulmonary disease (COPD)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tuberculosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive TB skin test</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thyroid disease</td>
<td></td>
<td></td>
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<tr>
<td>Peptic ulcer disease</td>
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<tr>
<td>Cancer</td>
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<td></td>
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<tr>
<td>Colon polyp</td>
<td></td>
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<tr>
<td>Arthritis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Condition</td>
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<td></td>
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<tr>
<td>-----------------------------------------------</td>
<td>--------</td>
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</tr>
<tr>
<td>Glaucoma</td>
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<tr>
<td>Cirrhosis/liver disease</td>
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<tr>
<td>Alcoholism</td>
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<tr>
<td>Depression</td>
<td></td>
<td></td>
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<tr>
<td>Ever had blood transfusion before 1993</td>
<td></td>
<td></td>
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<tr>
<td>Ever had other conditions</td>
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</tr>
</tbody>
</table>
Appendix N. Sample SAS codes for Study 2

- The sample SAS code with negative and positive mood predicting sleep quality (main effects) with unstructured covariance structure

```sas
proc mixed data=Das.Pt_depr_daily5c noclprint covtest method=reml;
    class dyad assess_date ;
    where flag3=0 and flag4=0 and gap=.;
    model sleep1 = pt_lag_EOD_NA_c pt_lag_EOD_PA_c opgender pain_c opage_c oppci_c opyrseduc_c opbmi_c opsleepted pt_BOD_NA_c pt_BOD_PA_c/solution ddfm=bw;
       random intercept/subject=dyad type=un;
    run;
```

- The same main effect model with autoregressive covariance structure

```sas
proc mixed data=Das.Pt_depr_daily5c noclprint covtest method=reml;
    class dyad assess_date ;
    where flag3=0 and flag4=0 and gap=.;
    model sleep1 = pt_lag_EOD_NA_c pt_lag_EOD_PA_c opgender pain_c opage_c oppci_c opyrseduc_c opbmi_c opsleepted pt_BOD_NA_c pt_BOD_PA_c/solution ddfm=bw;
       random intercept/subject=dyad type=un;
```
repeated / subject = dyad*studyday type = AR(1);
run;

- The sample SAS code with the interaction term between negative mood and spouse report of solicitous response

```sas
proc mixed data=Das.Pt_depr_daily5c noclprint covtest method=reml;
class dyad assess_date ;
where flag3=0 and flag4=0 and sp_flag4=0 and gap=.;
model sleep1 =pt_lag_EOD_NA_c|pt_so_resp_c opgender pain_c opage_c oppci_c opyrseduc_c opbmi_c opsleepmed pt_BOD_NA_c pt_BOD_PA_c/solution ddfm=bw;
random intercept /subject=dyad type=un;
run;
```

- The sample SAS code with ESTIMATE statements to show the patterns of interaction

```sas
proc mixed data=Das.Pt_depr_daily5d noclprint covtest method=reml;
class dyad assess_date ;
where flag3=0 and flag4=0 and sp_flag4=0 and gap=.;
model sleep1 =pt_lag_EOD_NA_c|sp_so_resp_c opgender pain_c opage_c oppci_c opyrseduc_c opbmi_c opmed pt_BOD_NA_c pt_BOD_PA_c/solution ddfm=bw;
```
random intercept /subject=dyad type=un;

estimate "Mean sleep1 for Average individual" intercept 1
pt_lag_EOD_NA_c 0 sp_so_resp_c 0 pt_lag_EOD_NA_c*sp_so_resp_c 0
opgender 0 pain_c 0 opage_c 0 oppci_c 0 opyrseduc_c 0
opbmi_c 0 opmed 0 pt_BOD_NA_c 0 pt_BOD_PA_c 0;

estimate "Mean sleep1 for DAYS with average NA (Average
daily so resp couples)" intercept 1 pt_lag_EOD_NA_c 0
sp_so_resp_c 0 pt_lag_EOD_NA_c*sp_so_resp_c 0
opgender 0 pain_c 0 opage_c 0 oppci_c 0 opyrseduc_c 0
opbmi_c 0 opmed 0 pt_BOD_NA_c 0 pt_BOD_PA_c 0;

estimate "Mean sleep1 for DAYS with average NA (Higher
daily so resp couples)" intercept 1 pt_lag_EOD_NA_c 0
sp_so_resp_c 0.9 pt_lag_EOD_NA_c*sp_so_resp_c 0
opgender 0 pain_c 0 opage_c 0 oppci_c 0 opyrseduc_c 0
opbmi_c 0 opmed 0 pt_BOD_NA_c 0 pt_BOD_PA_c 0;

estimate "Mean sleep1 for DAYS with average NA (Lower daily
so resp couples)" intercept 1 pt_lag_EOD_NA_c 0 sp_so_resp_c -0.9
pt_lag_EOD_NA_c*sp_so_resp_c 0 opgender 0 pain_c 0 opage_c 0
oppci_c 0 opyrseduc_c 0 opbmi_c 0 opmed 0 pt_BOD_NA_c 0
pt_BOD_PA_c 0;

run;
VITA

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2013 Ph.D. Candidate in Biobehavioral Health, The Pennsylvania State University, University Park, PA
2007 M.A. Psychology, Department of Psychology, Yonsei University, Seoul, South Korea
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PROFESSIONAL EXPERIENCE
2008-2013 Graduate Research and Teaching Assistant, Department of Biobehavioral Health, The Pennsylvania State University, University Park, PA
2005-2007 Graduate Research and Teaching Assistant, Department of Psychology, Yonsei University, Seoul, South Korea

SELECTED PRESENTATIONS
Song, S., Whetzel, C., Klein, L. C., & Graham, J. E. (2010). Effects of state positive affect on cortisol area under the curve among rheumatoid arthritis patients. The Annual Meeting of the American Psychosomatic Society, Portland, OR.


SELECTED PUBLICATIONS


HONORS AND AWARDS
2013-2014 Kligman Graduate Fellowship Award
2012-2013 Hintz Graduate Education Enhancement Fellowship
2012 Biobehavioral Health Outstanding Graduate Teaching Award
2011 Trainee Scholar Award, Psychoneuroimmunology Research Society (PNIRS) 2011 annual convention
2008-2009 University Graduate Fellowship at Penn State University
2006-2007 Brain Korea 21 scholarship (A national scholarship for training the next generation scholars with creativity)
2005-2006 Scholarship for Outstanding Academic Performance for Graduate Students at Yonsei University
2005 Honors award at Yonsei University