

**Marital Satisfaction and Inflammatory Functioning: A Biopsychosocial Pathway to
Health
A Meta-Analytic Review**

by

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Abstract

Marriage is a dominant relationship among adults and has been shown to impact physical and mental health. The exact pathway to describe its impact remains unclear, but recent advances in the literature focus on a biopsychosocial approach integrating affective, behavioral, and physiological correlates. Combined, these pathways can be best illustrated through a stress buffering model. Using theoretical and methodological conceptualizations of the physiological impact of acute and chronic stress, we review how marital satisfaction, a descriptor of marital functioning, impacts health through altered inflammatory functioning. Using a meta-analytic design, seven published empirical articles spanning over the last 30 years in 2,349 individuals were reviewed, evaluating the association between marital satisfaction and immunological biomarkers associated with inflammation: C-reactive protein, interleukin-6, interleukin-1 beta, and tumor necrosis factor-alpha. Marital satisfaction was not significantly related to inflammation, and effect sizes remained insignificant after accounting for moderators such as year of publication and inflammatory marker. The conclusions are limited by sample size, and exclusion of conflict variables, and the use of a statistical design that limits the interpretation of causal inferences. Future implications of these findings, highlighting a need to focus on moderating factors among marital satisfaction, inflammatory functioning, and health are discussed.

Keywords: marriage, marital satisfaction, stress, inflammation, health

Chapter 1

Introduction

As early as 1970, both the quality and quantity of one's social relationships have consistently shown to be associated with physical health (Cassel, 1976; Cobb, 1976; Berkman & Syme, 1979; Blazer, 1982; Smith & Christakis, 2008). Evidence illustrating that stronger social networks are associated with the promotion of a higher life expectancy have led many to conclude that social relationships, or the lack of, constitute a major risk factor for health alongside cigarette smoking and fluctuations in blood pressure (House, Landis, & Umberson, 1998; Umberson et al., 2006). Moreover, establishing and identifying how social relationships influence health outcomes is not as consistent. Through identifying factors that define intimate, satisfying, and high-quality relationships, theoretical and empirical work proposes that social relationships affect health through behavioral, psychosocial, and physiological mechanisms (Cohen, 1988, Pietromonaco, Uchino, & Dunkel Schetter, 2013; Uchino et al., 1996; Slatcher & Seluk, 2007).

Although much of this key evidence to support such mechanistic frameworks are designed and rooted in an individual's broad social network(s) such as their friends, colleagues, and acquaintances, recent advances in the literature suggest that close relationships such as marriage and romantic, intimate relationships have particularly strong effects on health (Kiecolt-

Glaser, Gouin, Hantsoo, 2010). By understanding the development of these close relationships, one can further illustrate how social relationships impact health through identifying potential mediators and moderators that influence health through behavioral, psychosocial, and physiological pathways.

One physiological pathway is that of inflammation. Although a more comprehensive review of this literature is provided below, inflammation has been associated with numerous health issues such as cardiovascular disease, psoriasis, and rheumatoid arthritis (Steptoe, Hamer, & Chida, 2007). Moreover, there has been a link between couples relationships, the evaluation of their relationship, and inflammatory processes (Fagundes et al, 2011; Kiecolt-Glaser, Gouin, & Hantsoo, 2010). It should be noted, however, that the literature in this area often involves unique, study specific methodology and thorough, intricate assessment (Kiecolt-Glaser, Gouin, & Hantsoo, 2010). Therefore, the aim of this manuscript is to quantify the relationships between perceived marital satisfaction and inflammatory processing to provide a foundation of understanding the association of the two. Drawing from theoretical and conceptual models (Berkman et al, 2000; Cohen, 1985; Pietromonaco, Uchino, & Dunkel Schetter, 2013; Mezuk, Roux, & Seeman, 2010) from the last 30 years, a meta-analytic design is used to illustrate how inflammatory processing in married couples may aid in understanding how close social relationships impact health. Further, the implications of this model for mechanistic understanding of social relationships and health, and its practicality for future research and effective intervention is outlined.

What is a Social Relationship?

Before understanding how a social relationship can impact health, it is important to understand what is meant by the term “social relationship.” A social relationship can be broadly

defined as any relationship or interaction between two or more individuals. A social relationship with multiple individuals forms a social network. Conceptually, social networks are comprised of several distinct features of being socially connected such as closeness, connectedness, and dynamic communication (Umberson and Montez, 2010).

Social network analyses in adults have displayed that social networks are individually characterized by one's perception of each member, their own closeness and popularity, and the extent to which each member serves to fulfill a role in aiding in the individual's personal and social development. Social networks are used to incite feelings of affiliation, provide emotional and informational support, and are ultimately for seeking pleasure and enjoyment (Gillath, Karantzas, & Lee, 2018). The structure of one's social network plays a large role in determining an individual's behavior(s) and beliefs by molding how they initiate, maintain, and dissolve the resources that result from being engaged socially (Berkman et al, 2000).

One's social network often mirrors their attachment style. Attachment Theory is broadly conceptualized as an understanding of the functions of a close bond with an attachment figure, for example, a parent or romantic partner. As it is theorized, an attachment behavioral system is innate and biologically based that serves to protect an individual when they are threatened, such as from the experience of pain or stress, by introducing behaviors that keep the individual close to their attachment figure.

Not only does this fused psychobiological system prevent individuals from enduring physical harm, it also serves to regulate one's affective state which can be altered as a result of the distress. This component of the system introduces a regulatory function that is essential for one's well-being, aiding in the restoration of one's emotions (Bowlby, 1969, 1973). This is important to highlight as emotional dysregulation can increase physiological responding through

hypothalamic-pituitary-adrenal (HPA) axis activity, which can lead to negative health consequences such as immunodeficiencies and cardiovascular disease (Bowly, 1969, 1973; Mikulincer & Shaver, 2007; Beckman et al, 2000; Gross, 2002 Pietromonaco, Uchino, & Dunkel Schetter, 2013).

Individual differences in this emotional and behavioral responding exist and reflect experiences from prior relationships (Shaver & Mikulincer, 2007; Schoebi & Randall, 2015). These differences can be separated into two components, insecure, characterized by high levels of anxiety and avoidance and secure characterized by low levels of anxiety and avoidance. Often those with secure attachment form strong, stable social relationships with more positive affect, and are more likely to form romantic relationships (Simpson, Collins, Tran, & Haydon, 2007; Schoebi & Randall, 2015). These dimensions of attachment can serve to modulate the way one responds to stress. Insecure attachment induces increased levels of emotional and physiological reactions, indicative of a poor stress response. For example, when solving a lab-induced task with their partner, they have a greater cortisol reaction, a marker of the body's stress response (Powers, Pietromonaco, Gunlicks, & Sayer, 2006; Schoebi & Randall, 2015). Conversely, secure attachment provides resources to stabilize the stressor.

Moreover, ineffective emotion regulation decreases the expression of both positive and negative cues and behaviors that may block social signals that one's partner may need. Further, this component has strong implications for forming and maintaining successful intimate social relationships as the emotional suppression could make the individual less responsive to the emotional cues of their partner. This could lead to negative social consequences, increasing distress and isolation, triggering physiological responses that may be detrimental to one's health

(Bowlly, 1969, 1973; Mikulincer & Shaver, 2007; Beckman et al, 2000; Gross, 2002 Pietromonaco, Uchino, & Dunkel Schetter, 2013).

This ability to regulate not only one's behavior, but their behavior in relation to their partner, such as through providing care, reassurance, and comfort, promotes the development of a successful and long-term relationship. Awareness of this dyadic dynamic and its successful implementation allows couples to readily acknowledge when their partner is experiencing distress, resulting in behaviors that will protect and support the well-being of the distressed thus fostering the foundation of a strong, stable relationship (Bowlly, 1969, 1973; Mikulincer & Shaver, 2007; Beckman et al, 2000; Pietromonaco, Uchino, & Dunkel Schetter, 2013).

The relevance and strength of Attachment Theory is that it asserts that an individual has a biologically innate need for developing secure attachments within their social networks that may serve to mitigate the effects distress may have on their health. Such attachments promote a sense of safety and can enhance self-esteem, providing the grounds on which one will use to form strong, secure social relationships and effective emotion regulation techniques throughout adulthood. The development of one's psychosocial environment as described and implied in this theory asserts that emotional and cognitive components of social attachment and loss are dynamically intertwined within our biology (Berkman et al, 2000).

These relationship-oriented connections with attachment reflect that support is central to the development and maintenance of an individual's emotional dynamics. Social relationships may serve as regulators, providing resources for one to be equipped to respond to stress which can be beneficial for one's health. However, social relationships do not always provide social support nor do they guarantee that the support provided will be meet the individual's needs. If a relationship is not serving as a source of support, it may not positively influence stress, leading to

adverse health consequences. Thus, when investigating the effects social relationships have on health, it is important to assess the quality of the relationship to reflect what it is providing (Dohono et al, 2013).

How do social relationships impact health?

Prospective studies of mortality consistently display that those with high levels of engagement in social relationships are less likely to die (House, Landis, & Umberson, 1988; Umberson & Montez, 2010). More specifically, involvement in social relationships has been associated with both health conditions and biomarkers that indicate varying and increasing risk of preclinical conditions and complications. Low quantity and quality of social relationships have been associated with cardiovascular disease, myocardial infarction, atherosclerosis, high blood pressure, and cancer (Ertel, Glymour, & Berkman, 2009; Everson-Rose & Lewis, 2005; Robles and Kiecolt-Glaser, 2003; Uchino, 2006; Umberson & Montez, 2010). Further, poor quality and low quantity of social relationships have also been associated with inflammatory biomarkers and dysregulated immune functioning, both of which are significantly and highly associated with adverse health outcomes such as increased susceptibility to the common cold, depression, and mortality (Robles & Kiecolt-Glaser, 2013; Umberson & Montez, 2010).

Despite the clear evidence showing that social relationships impact health, the mechanisms by which they make their impact remains unclear. The literature among the last 30 years exploring this can be summarized by three broad explanations: behavioral, psychosocial, and physiological (Umberson & Montez, 2010).

Behavioral Explanation: Personal behaviors that influence health are better known as health behaviors. For example, exercise and eating a well-balanced diet can promote health and aid in preventing illness while smoking and drug abuse are detrimental to health. Being in a

social relationship impacts one's personal behaviors so it can be said that social relationships influence health behaviors (Umberson, Crosnoe, & Reczek, 2010; Umberson & Montez, 2010).

Social relationships can exert influence on health behavior by pressuring or forcing one to inhibit, regulate, facilitate, or take part in activities that may promote or undermine health. Excessive monitoring or control in this way can lead to long-term detrimental health habits. For example, measuring susceptibility to peer-pressure, self-esteem, and internal health locus of control has been shown to be significantly and negatively correlated with substance use and misuse (Dielman et al., 1987). Social relationships may provide an outlet for one to create and develop habits that in turn may affect their physical and mental health; therefore, mortality.

Psychosocial Explanation: The psychosocial explanation combines the influence of behavioral, psychological, and environmental factors on one's ability to physically and mentally function optimally. Such factors shape how one sees themselves in a social relationship, allowing for the development of one's social role which impacts one's desire, willingness, and need to socially interact. It is logical to posit that this may then translate into health behaviors.

Psychosocial mechanisms used to illustrate how social relationships impact health are what is provided by being involved in a social relationship such as support, engagement, connectedness, and coherence (Antonovsky, 1987; Umberson & Montez, 2010). While their influences can be impactful individually, they are conceptually intertwined; therefore, their cumulative effect as described as a mechanism may be a better descriptor than any single (Thotis, 1995; Umberson, 2010; Umberson & Montez, 2010).

As mentioned above, social support is a component of relationships that provides a sense that one is loved, cared for, listened to, and appreciated. This quality invokes positive emotionality and may indirectly influence health through promoting one's sense of self, reducing

stress, and giving one a sense of purpose (Cohen, 2004; Uchino, 2004; Umberson & Montez, 2010). Physiologically, social support's effects may reduce one's blood pressure, heart rate, and the release of circulatory stress hormones (Reblin & Uchino, 2008). All of which are advantageous for health.

Physiological Explanation: Being involved in supportive relationships with others has shown to benefit cardiovascular, endocrine, and immune functioning, and reduce allostatic load, a reflection of wear and tear on the body. Allostatic processes are multifaceted, but are ultimately the result of the physiological systems that engage in the body's stress response being chronically activated or overworked. As these systems mature and adapt to encounters of stress, they have significant benefits and consequences for health (Umberson and Montez, 2010; McEwen, 2005; Robles et al, 2014; Robles & Carroll, 2011; Robles and Kiecolt-Glaser, 2003).

While social relationships can provide the central source of emotional support for many people, they can also be incredibly stressful. Relationship stress undermines the regulatory activity of the body's physiological systems, leading to cumulative adverse effects on health. Interpersonal conflicts in social relationships evoke cardiovascular responses, greatly increasing one's risk for future cardiovascular disease, increasing blood pressure, heart rate, and rate of disease progression (Linden, Gerin, & Davidson, 2003; Trieber et al, 2003; Robles et al, 2014; Chrousos, 2009). Further, greater negative social interactions result in elevations of circulatory catecholamines such as epinephrine and norepinephrine, which have been shown to have significant effects on regulating cardiovascular, metabolic, and immune functioning, increasing one's risk for anxiety, depression, type 2 diabetes mellitus, and metabolic syndrome (Lovallo & Thomas, 2000; McEwen, 1988; Robles & Kiecolt-Glaser, 2003; Robles et al, 2014; Kiecolt-Glaser et al, 1993; Robles and Kiecolt-Glaser, 2003; Chrousos, 2009).

Evolutionarily, physiological responses to stressful situations have been described. The ability to respond to stressors enhanced survival. The physiological processes that supported this could be naturally selected for as increased delivery of oxygen and glucose to the heart and skeletal muscles is advantageous. Over time, these physiological support mechanisms became adaptive behaviors known as “fight-or-flight” responses (Segerstrom & Miller, 2004). Although modern day stressors are much different than those before who faced predominantly intense physical and environmental stressors such as predation, fight-or-flight responses remain.

From a biopsychosocial perspective, it can be said that our body functions optimally when its psychological and physiological systems are in balance. Walter Cannon (1929) described this as “homeostasis.” Conceptually, homeostasis is the result of the brain coordinating with the body’s biological systems, such as body temperature and blood glucose levels, to maintain equilibrium to ensure an optimal state of well-being. Stress acts as a threat to homeostasis, challenging and altering the body’s ability to maintain stability (Selye, 1956; Goldstein, 2007). This threat, whether it be actual or perceived, is called a “stressor” and the body’s response to said stressor is the “stress response” (Selye, 1956; Mohd, 2008; Schneiderman, Ironson, & Siegel, 2008; Segerstrom & Miller, 2004). Stress responses were originally seen as an adaptive response, posing little or no threat to one’s health. Though, responses that are prolonged have been shown to have long-term effects that are detrimental to health, increasing the risk of cardiovascular disease, diabetes, and the common cold (Schneiderman, Ironson, & Siegel, 2008; Chrousos, 2009).

Upon encountering a stressor, a decision must be made to determine if it is a threat in order for the body to appropriately respond. Appraisals are made based on the determination of the meaning and nature of the stressor and an assessment of the body’s physical and mental

ability to cope with the stressor (Lazarus & Folkman, 1984; Schneiderman, Ironson, & Siegel, 2008). When the stressor is appraised as a threat, the brain elicits a response activating the central nervous system (CNS) to initiate the “fight-or-flight” response (Cannon, 1929; Schneiderman, Ironson, & Siegel, 2008).

The “fight-or-flight” response involves an outpouring of norepinephrine, cortisol, and other hormones into the bloodstream that prepare the body for defense against any threat. When under stress, the brain interprets the sensory information that has been received and relays it first to the reticular formation and the thalamus where the limbic system, the cerebral cortex, and the hypothalamus interpret the meaning of the stressor. The hypothalamus releases corticotrophin-releasing hormone (CRH), which activates the pituitary gland to secrete adrenocorticotrophic hormone (ACTH) into the bloodstream. ACTH is taken up by receptors in the adrenal glands, the adrenal medulla, and the adrenal cortex. The adrenal medulla secretes norepinephrine (noradrenaline) and epinephrine (adrenaline) into the bloodstream, triggering the “fight-or-flight” response causing an increase in one’s heart rate, pupil dilation, and slowed digestion. Once the “threat” is over, the body works to return back to homeostasis, slowing the heart rate and increasing intestinal and gland activity (Cannon, 1929; Goldstein, 2007; Straub, 2014).

Not all threats require a physical response though they may still have physical consequences. Psychological stress, threats experienced in the absence of a physical stressor, trigger the same physiological fight-or-flight responses, specifically changes in the immune system (Seegerstrom & Miller, 2004). Characteristics of social relationships such as social integration, social support, social conflict, and social rejection, can act as psychological or psychosocial stressors, and have been associated with increased alterations in inflammatory processing, a component essential to regulatory immune functioning. Correlational,

observational, and experimental studies have suggested that social isolation and lack of social support are associated with higher levels of circulating systemic pro-inflammatory markers such as interleukin-6 (IL-6) and acute phase proteins such as c-reactive protein (CRP) (Ridker, 2009; Kiecolt-Glaser, Gouin, & Hantsoo, 2010; Slavich, O'Donovan, Epel, & Kemeny, 2010; Robles et al, 2014). Such elevations may serve as a predictor for mortality in adults, increasing the risk of chronic diseases such as diabetes, Alzheimer's Disease, and declines in physical and mental functioning (Ershier & Keller, 2000; Kiecolt-Glaser et al, 2010; Chrousos, 2009).

So how is it that psychosocial experiences and interactions are capable of modifying the immune system? Below is a consolidation of mechanistic and empirical explanations illustrating the complexity of psychosocial stress and the human immune system. An extensive review is outside the scope of this paper; therefore, the focus is on the concepts most frequently evidenced in the stress, immunity, and social relationship literature. The discussion begins with a brief overview of the components of the immune system.

Overview of the Immune System

Stress has complex effects on the immune system and influences both innate and acquired immunity (Chrousos, 2009; Segerstrom & Miller, 2004). Innate immunity provides the first line of defense when the body encounters an invader or "pathogen." The response is non-specific, meaning its cells and mechanisms can defend against multiple pathogens rapidly. When under attack, cells called granulocytes, phagocytic cells that engulf their target, such as neutrophils and macrophages, are released, triggering inflammation. The neutrophils and macrophages gather around the site of injury or infection and release additional secretions to fight off the invaders. Macrophages also release cells called cytokines. Cytokines are molecules that communicate with others and promote wound healing. Further, they induce traditional

sickness-like behaviors, such as fever and fatigue through inflammatory processing in an effort to disrupt and destroy the invader. Pro-inflammatory cytokines most frequently studied in psychosocial stress and health literature are Interleukin-1 beta (IL-1 beta), Interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-alpha), will be discussed in more detail below (Segerstrom & Miller, 2004).

Other cells involved in the innate immune response are granulocytes such as mast cells and eosinophils, which are responsible for parasite and allergic defense, and natural killer cells which are crucial in limiting the effects of the early phases of viral invaders. In addition, complement proteins bind, upregulate phagocytosis and inflammation, and aid in the orchestration of antibody responses to eradicate the invader (Segerstrom & Miller, 2004).

Acquired immunity on the other hand, is a more specified response and occurs more slowly than innate immunity. Cells called lymphocytes, such as T-helper, T-cytotoxic, and B cells, are signaled into action when the pathogen withstands the initial innate defense. The cells are antigen-specific, meaning they bind to specific cells like cancer cells or allergens for example. These cells mediate the acquired response by producing additional cytokines. T-cytotoxic cells puncture infected cells, expelling their contents, ultimately leading to the cell's death, and B cells produce antibodies that aid in combat (Segerstrom & Miller, 2004).

Inflammation: A Pathway Between Stress, Immunity, and Health

As complex as the physiology of the immune system is, how can something like stress impose a strong enough impact to induce alterations in its functioning? A series of bidirectional signals from the nervous, endocrine, and immune system allow the central nervous system (CNS) to modulate the immune response. A key pathway for immune dysregulation is inflammatory processing.

Inflammation is a conglomerate of biological processes that respond to pathogenic invasion or physical functioning. It typically accompanies traditional “sick-like” symptoms such as redness, swelling, fever, and pain. These symptoms are reflective of increased blood flow, the release of inflammatory mediators, and leukocyte migration and accumulation to the site if infection or injury. Communication among leukocytes, platelets, endothelial cells, and inflammatory moderators such as histamine, complement, chemokines, and cytokines, work efficiently together to resolve the infection or damage without overactivation of inflammatory processes. Overactive inflammatory functioning can lead to dysfunction of this regulatory system and a chronic state of inflammation which is significantly damaging to local tissue and can lead to a host of clinical pathologies such as arthritis, hypertension, and panic disorder (Hansel et al, 2010; Kiecolt-Glaser et al, 2010; Schneiderman, Ironson, & Siegel, 2008; Schneiderman, Ironson, & Siegel, 2008; Weiss, 2008; Chrousos, 2009).

Cytokines are suitable markers of inflammation since they are produced by an activated immune system and themselves activate other cells to produce more cytokines during inflammatory processing. When these cytokines are released, acute phase proteins like c-reactive protein (CRP), are released from the liver. Circulating levels of pro-inflammatory cytokines such as IL-6, IL-1 beta, and TNF-alpha, leukocytes, and acute phase proteins like CRP, have been frequently studied as biomarkers of between psychology and health, as they are associated with psychological and psychosocial states and behavior, specifically stress (Hansel et al, 2010).

For example, social isolation or loneliness has consistently shown to be an important psychosocial aspect of health since individuals who are engaged socially benefit from receiving support. Significant elevations in CRP and IL-6 have been associated with decreases in social ties. Both IL-6 and CRP are strong influences in the development of cardiovascular disease,

impairing vasodilation and accelerating arterial stiffening, in addition to osteoporosis, frailty, and functional decline (Hansel et al, 2010; Kiecolt-Glaser et al, 2010; Schneiderman, Ironson, & Siegel, 2008; Schneiderman, Ironson, & Siegel, 2008; Weiss, 2008). Therefore, dysregulated inflammatory processing can potentially provide evidence linking the quality and quantity of social relationships to health and disease.

Why are intimate relationships unique to stress-induced immune changes?

Despite the immune system's many efforts to combat and withstand alterations, as shown above, it can be compromised by a host of psychological factors that can arguably be cited as characteristics of diseased states. The immune system treats psychosocial stress as an invader and attacks it, leading to increased risk in clinical pathology. Different aspects of social relationships such as integration, support, conflict, and closeness can greatly increase one's vulnerability when in low quality and quantity. Further, one of the most salient vulnerabilities is the type of relationship, specifically, marriage. Over 60 million couples in the United States identify their relationship status as married and on average (US Census Bureau; Bureau of Labor Statistics 1960-2017). Moreover, there is robust literature that shows that this specific type of relationship has unique characteristics that link to health.

The literature shows, at the most general level, that married people have better mental and physical health when compared to unmarried people (Kiecolt-Glaser & Newton, 2001; Robles et al, 2014; Kiecolt-Glaser & Wilson, 2017). Further, their mortality and morbidity rates are significantly lowered among a variety of health conditions such as cancer and heart attacks (Kiecolt-Glaser & Wilson, 2017; Chandra, Szklo, Goldberg, & Tonascia, 1983; Goodwin, Hunt, Key, & Same, 1987; Gordon & Rosenthal, 1995; House et al, 1988; Kiecolt-Glaser & Newton, 2001).

An important advance in the literature has focused on experiences and perceptions of affective dynamics as key components to interactions and associations with health and disease, particularly regarding implications for altered immune functioning (Bradbury, Fincham, & Beach, 2000; Gottman, 1993; Reis & Shaker, 1988; Schoebi & Randall, 2015). Further, recent work has focused on examining the exchange of emotional interactions and behavior on immune functioning through measures of relationship satisfaction (Timmons, Margolin, & Saxbe, 2015). These measures demonstrate that affective and physiological linkage could be associated with positive factors (high levels of satisfaction) or negative factors (low levels of satisfaction), and can have either protective or detrimental effects on health (Timmons, Margolin, & Saxbe, 2015). Given this, studying relationship satisfaction among married couples provides a unique insight into understanding how social relationships influence health.

Defined broadly, marital satisfaction is a subjective measurement of an evaluation of the relationship and the behaviors within it on both positive (supportive) and negative (stressful) aspects (Robles et al, 2014; Robles, 2015). Marital satisfaction is often used synonymously with marital quality and marital adjustment. This can be measured through self-report ratings of the marriage such as the frequency of or acceptability of the partners' behaviors and attitudes or reports of interaction patterns. It is also common to measure quality observationally by recoding couples discussing issues or conflicts and coding for supportive and unsupportive or hostile behaviors, but methodological do exist (Robles, 2015). The impact of having a better or worse marriage ranges from high to low quality. High marital quality operationalized by high levels of satisfaction with the relationship is indicated by positive attitudes towards the spouse and low levels of negative, unsupportive or hostile behaviors. Low marital quality is operationalized by the opposite, low satisfaction with the relationship as indicated by negative attitudes towards the

spouse and high levels of negative, unsupportive or hostile behaviors (Robles et al, 2014; Robles, 2015; Kiecolt-Glaser & Wilson, 2017).

The associations between marital satisfaction and health demonstrate robust links to physiology as consistently as social relationships do. A meta-analysis (Robles et al, 2014) quantified the magnitude of marital quality's association with health and compared it to other known health-risk factors. The results presented effect sizes of marital quality with similar magnitudes to those of diet and exercise on health. Further, they found that there was a lower risk for mortality among those with high levels of marital quality. Most interestingly, the largest associations were among studies evaluating chronic illness conditions (Robles et al 2014; Kiecolt-Glaser & Wilson, 2017).

Marital relationships exert major influence in shaping one's emotional state daily as couples become dependent on their spouse for not only psychological needs, but material needs such as shelter and financial support (Pietromanaco, Uchino, & Schett, 2013; Robles et al, 2014; Schoebi & Randall, 2015). Theories on marital relationships conceptualize how these dependent interactions and their appraisals shape affective experiences and behaviors within the relationship. Spouses tend to anticipate their partner's behaviors and use that feedback to fulfill their needs. These appraisals can induce both positive and negative arousal and play a key role in the elicitation of positive or negative affective responding and overall levels of satisfaction. Experiences of negative affect, anger, or engagement in hostile interactions are often associated with higher levels of relational conflicts and perceptions of threat and neglect. This type of relationship strain appears to be not only an important emotion-eliciting theme in couples literature, but in health literature as well (Pietromanaco, Uchino, & Schett, 2013; Robles et al, 2014; Schoebi & Randall, 2015).

Empirical evidence suggests that even after controlling for demographics and disease-related variables, marital strain is associated with an increased risk of mortality, higher self-reported symptoms of physical illness, increased risk of cardiovascular complications (i.e. cardiac arrest, myocardial infarction), and disease severity (Wickrama, Lorenz, Conger, 1997; Kimmel et al, 2000; Orth-Gomer et al, 2000; Kiecolt-Glaser & Newton, 2001; Robles and Kiecolt-Glaser, 2003). This data suggests that marital strain is strikingly similar to the health consequences seen as a result of social isolation, as described earlier (Smith & Ruiz, 2002; Robles & Kiecolt-Glaser, 2003).

Going back to Attachment Theory, it can be said that relationships are regulators, aiding in the maintenance of a homeostatic state. Up until this point, it has been presented that marriage can have strong influences on health, greater than those shown by general social relationships. Given the interplay between the dynamic of emotional experiences in an intimate relationship, how can it be explained that its effects can be both protective and beneficial as opposed to just being one or the other? A spouse can act as a buffer against the consequences of major and minor occurrences of daily stress, minimizing the stress-related emotions and physiological responses. Stress-buffering refers to a model whereby the presence of another's support can buffer the body's stress response (Cohen & Willis, 1985; Schoebi & Randall, 2015). A spouse can serve as a buffer by intervening between the stressful event and the stress response by preventing the event from occurring, or by reducing the stressful reaction by intervening between the experience of stress and the onset of the psychophysiological distress by providing words or gestures of comfort and care. The proximity and availability of having a spouse can aid in the reduction or management of stress and can serve as a strong tool against negative emotions. This buffering effect can be indexed through relationship satisfaction (Cohen, 2004; Mezuk, Roux, & Seeman,

2010; Robles et al, 2014; Thotis, 2011). For example, when holding hands during the anticipation of a mild, laboratory induced shock, participants' stress responses were significantly lower when the person holding their hand was their partner in a satisfied relationship (Coan, Schaefer, & Davidson, 2006; Schoebi & Randall, 2015). The regulation of these emotional and physiological responses to stress through the experience of interpersonal connectedness as explained by stress-buffering and conflict, show that relationship satisfaction may serve as a key moderator in understanding the link between marriage and health.

How does marital satisfaction influence inflammatory processing?

As mentioned above, while marriage typically has health benefits, marital satisfaction has important implications for immune functioning, and marital interaction studies provide the evidence (Kiecolt-Glaser & Newton, 2001; Fagundes et al, 2011). For example, when couples engaged in both a supportive and problem discussion, couples with more hostile interactions produced more IL-6 than the supportive discussion. Couples with less hostile interactions had similar IL-6 productions among both discussions (Kiecolt-Glaser, 2005; Robles et al, 2014; Kiecolt-Glaser et al, 2010; Robles & Kiecolt-Glaser, 2003). Further, during marital disagreement, couples with greater cognitive engagement as measured through word choice, produced less IL-6 and TNF-alpha over the next 24 hours when compared to couples who were less cognitively engaged (Graham et al, 2009).

Hypothesis:

Assessing the published literature among the last 30 years, we explored the question: what is the strength of the association between marital quality and inflammation? We hypothesized that if buffering is occurring through marital satisfaction, we would expect to find a

strong and significant association across studies. Further, we explored potential moderators of this association.

Chapter II

Methods

Article Search Strategy

Our meta-analytic design was modeled off of a meta-analysis published in 2014, “Marital Quality and Health: A Meta-Analytic Review” by Theodore Robles and colleagues. Further methodological features, modeling, and analyses were guided by David B. Wilson and Mark W. Lipsy’s book, *Practical Meta-Analysis* (2001). Electronic searches were performed across four databases: PsychInfo, PsychArticles, MedSearch (which includes Medline, PreMedline, and International Pharmaceutical Abstracts), and PubMed. Searches were restricted to publications between 1988 and 2018. This window of 30 years was chosen because the field of psychoneuroimmunology, the field focused on examining the relationship between stress, the immune system, and health outcomes, was first being conceptualized and integrated into methodological designs at this time. Dissertations were reviewed for potential inclusion in the model. The query used combinations of keywords for relationship satisfaction and key markers of systemic inflammation. See Table 1.

Reference lists were manually searched from the publications to assess for eligibility for additional applicable studies. These reference list searches were conducted by the principle investigator, the thesis chair, and two undergraduate research assistants. Search results were checked for duplicates and reviewed for inclusion by the authors. For screening and inclusion details, see Figure 1.

Study Inclusion Variables

Criteria for inclusion were peer reviewed journal articles in English which reported circulatory pro-inflammatory cytokine (IL-1-beta, IL-6, or TNF-alpha) and/or CRP levels at baseline with a measure of marital satisfaction in married couples. Studies were excluded if circulating pro-inflammatory cytokine and/or CRP responses were in response to a psychological lab stress task, those that only reported relationships between marital status and inflammation, those that assessed the impact of a physical or mental disease diagnosis on marital quality, and those that assessed the relationship between medical intervention on marital quality. Participant populations therefore included healthy adults, free of any illness, chronic or otherwise.

Independent Variable: Marital Quality

Different approaches can be used to define marital quality. In the context of this study, marital satisfaction was defined as self- or other reported perceptions of the quality and stability of the relationship, the partner's assessment of the marital relations, and emotional development. Further, we focused only positive dimensions of such marital measures such as happiness, support, and satisfaction as opposed to negative dimensions such as conflict, tension, strain, and dissatisfaction (Bradbury et al., 2000; Fincham & Bradbury, 1987; Robles et al., 2015). The sample analyzed excluded data reporting inflammatory or acute phase protein levels resulting from marital conflict or a reactivity task, in an effort to dismiss the confounding effects of active marital hostility that may explain the alterations in circulating levels. For the purposes of this analysis, we are only interested in exploring the relationship between reported marital satisfaction and a baseline level of inflammatory processing.

Self-report measures included both widely-used measures and study specific measures. The well-established measures included: the Marital Adjustment Test (Locke & Wallace, 1959),

the Dyadic Adjustment Scale (Spanier, 1976), the Couples Satisfaction Index (Funk & Rogge, 2007), and the Positive and Negative Marital Qualities Scale (Fincham & Linfield, 1997). Study specific measures varied from scales and subscales distinguishing between positive and negative affectivity in marital relationships (Fincham & Linfield, 1997; Galinsky & Waite, 2014; Graham et al., 2009; Liu & Waite, 2015; Schuster, Kessler, & Aseltine, 1990; Turner, Frankel, & Levin, 1983; Shen et al., 2010; Whisman & Sbarra, 2012), and scales of spousal support and spousal strain (Dohono, Crimmins, & Seeman, 2013). For this case of standalone ratings of support and strain, ratings of support were included in the analysis as it was determined that a rating of strain may be a result of reactivity. All displayed satisfactory reliability of internal consistency per population.

Dependent Variables: Biomarkers of Systemic Inflammation

Biomarkers of systemic inflammation such as IL-1 beta, IL-6, TNF-alpha and CRP, were included as dependent variables. We focus on these biomarkers as for reasons mentioned above, that they have consistent, documented associations with acute and chronic psychosocial stress (Kiecolt-Glaser et al, 2003; Sin et al, 2015) and clinical significance for long term health consequences. All markers were collected through a venous blood draw. To control for diurnal influences on inflammation, some samples were obtained in morning blocks (Uchino et al., 2018), others were collected across multiple days prior to the start of data collection, or before, during, and after a laboratory-induced marital conflict task (Heyman, 2004; Knox, 1971; Holley & Guilford, 1964; Xu & Lorber, 2014; Kiecolt-Glaser et al, 2015). For the purposes of this analysis, we only used measures that occurred at baseline or prior to any laboratory-induced task for reasons of increased reactivity as previously mentioned.

Moderator Variables

To examine the contribution of moderating variables, we performed analyses assessing the author's affiliation, psychology or other (i.e. behavioral medicine, psychiatry, sociology, general medicine, etc.), year of publication, and inflammatory marker (CRP, IL-1 beta, IL-6, and TNF-alpha).

Data Coding and Extraction

A coding sheet was created to allow for efficient data extraction. Variables selected for coding included: study year, first author affiliation, marital quality measure, inflammatory biomarker, sample size, and test statistics. If the study's results were analyzed using multiple models, we used the values of the models that controlled for the covariates. Each study was rated and coded by the primary author with input and review by the second and third.

In some of the articles included in the analysis, the authors reported effect sizes from more than one dependent variable, such as an effect size for CRP and IL-6 and marital satisfaction. If it was possible for such variables to be separated, they were analyzed separately. Statistics from studies including relationships between marital satisfaction and the inflammatory markers that separated their results by gender were averaged to create an overall effect size.

Our search did result in multiple papers where data was drawn from a single source population. Studies using the same population were not all included in an effort to minimize the variability in an individual's baseline measure. When assessing the eligibility of such studies (Uchino et al., 2013; Dohono, Crimmins, & Seeman, 2013; Kiecolt-Glaser et al., 2015; Kiecolt-Glaser et al, 2005), we included those in the analysis that included relationships among marital satisfaction and multiple dependent variables.

Data Analysis

Seven studies (2,349 individuals) met the inclusion criteria. Statistics on association or data needed to calculate this association were pulled from each study based on baseline biomarker level and the marital satisfaction rating. Then, a standardized mean difference effect size (ES), d , was calculated. Each ES was weighed by sample size and an inverse variance weight was calculated to help control for the effects of sample size. When more than one biomarker was analyzed at baseline with a marital satisfaction rating, they were included as separate effect sizes. In studies that involved a comparison between men and women, the two were collapsed to calculate an overall ES for the sample.

A z-score was calculated for each weighted mean effect size. Heterogeneity for the ES was calculated using a chi square test. We performed random effects models to account for the amount of variance between studies and participants. Inverse variance weight was calculated for each ES to analyze excess between study variability using 1-way ANOVA and weighted multiple regression. All analyses were conducted using Mark Lipsey and David B. Wilson's meta-analysis macros for SPSS (Version 25, 2017).

Chapter III

Results

Overall, the analysis displayed that there did not appear to be a relationship between marital satisfaction and the four inflammatory markers. The mean effect size for the model was -0.1888, ranging from -.102 to .050, and did not appear to be significant ($p = .2923$). Further, the fixed effects ordinarily squared model was insignificant ($Q(9.0) = 2.6043, p = .9779$).

Despite the lack of significant analyses, in order to fully assess our model and its implications, moderator analyses were conducted to assess for any accounts of variance. The selection of moderators was based upon study descriptors that were believed to have influence on our hypothesis. Moderators that were chosen to be in the analyses included: author's affiliation, psychology or other (i.e. behavioral medicine, psychiatry, sociology, general medicine, etc.), year of publication, and inflammatory marker. Moderators of all the inflammatory markers or the marital satisfaction ratings were not assessed due to the variability of their measurement across the studies included.

Chapter IV

Discussion

The present meta-analysis assessing the links between marital quality and inflammatory processing combined findings from the last 30 years of published literature. Despite the theoretical links and empirical evidence between marital satisfaction and health, we did not find any significant association between marital satisfaction and levels of inflammatory markers across studies. Moreover, results remained insignificant regardless of the author's affiliation, the year of publication, and inflammatory marker. The lack of evidence for a significant association between marriage satisfaction and inflammation suggests that there may be components of the interaction between marital quality and inflammation that are not being accounted for in this model. Below we discuss both theoretical and methodological characteristics that could be lacking from the present model, highlighting their implications for future work.

Theoretical perspectives on relationship and marital compatibility have been presented as either intrapersonal or interpersonal (Kelly & Conley, 1987; Humbad, Donnellan, Iacono, & Burt, 2010). The intrapersonal perspective is distinguished by how relationship functioning is linked to personality and or psychopathology. The interpersonal perspective is distinguished behaviorally by the couple's interactions and how such interactions are related to the relationship quality and status. Although the two are necessary components for marital compatibility, they are not mutually exclusive, but rather complementary. The present study focused on the interpersonal component, and not the latter, potentially underestimating the influence of the intrapersonal component.

Personality traits have been associated with partner selection, relationship satisfaction, and marital stability (Donnellan, Conger, & Bryant, 2004; Claxton et al, 2011). Characteristics that are considered to be positive such as agreeableness and conscientiousness have been associated with higher levels of stability and marital satisfaction (Lester, Haig, & Monello, 1989; Claxton et al, 2011). While negative traits, such as neuroticism, have been shown to be associated with lower levels of stability and marital satisfaction (Kelly & Conley, 1987; Claxton et al, 2011). Whether positive or negative, personality traits are linked to affect, variable mood inductions, and differences in emotion regulation strategies (Ormal & Wohlforth, 1991; Gross, Sutton, & Keteloor, 1998; Watson & Hubbard, 1996; Claxton et al, 2011), all of which can be shared amongst the couple. Specifically, having or sharing these negative personality traits could influence the amount of conflict that is experienced within the marriage, increasing the amount of stress in the relationship and therefore the couples' risk of experiencing health consequences. By excluding personality as a dependent variable or modifier, we could have overestimated how poor low scores of marital satisfaction were.

Further, the associations between psychopathology and marital distress are robust. Low levels of marital satisfaction have been linked to major depressive disorder, anxiety disorders, substance use disorders, and personality disorders (Whisman & Uebelacker, 2003; Markowitz, Weissman, Ovellette, Lish, & Klerman, 1989; McLeod, 1994; Homish, Leonard, & Cornelius, 2008; Newcomb, 1994; South, Turkheimer, & Oltmanns, 2008; Whisman & Schonbrun, 2009). Evidence indicates that marital satisfaction can be predicted by one's level of anxiety or depression and or his or her spouse's depression (Whisman, Uebelaker, & Weinstock, 2004). Depression is often used as a model to illustrate how marriage can impact one's health, initiating the dysregulation of the HPA axis and initiating the release of pro-inflammatory cytokines and

CRP (Kiecolt-Glaser et al, 2010). Although clinical psychopathologies have varying rates of comorbidity with physical illness, it often puts one at more risk for developing physical health issues or problems. In addition, partners tend to adjust their relationship behaviors in an effort to manage the harsh, negativity of their partner (Murray, Holmes, & Collins, 2006; Schoebi & Randall, 2015). This model has been shown in spouses who have felt less positively regarded by their spouse and were more reactive to their mood and behaviors, increasing their anger towards them, thus increasing their levels of anxiety (Murray et al., 2003; Schoebi & Randall, 2015). By excluding existing psychopathology as a dependent variable or modifier, we could not have known for certain if the lower satisfaction scores were accurately reflective of the relationship or of the responder's mental well-being.

Although these two theoretical perspectives may display criterion that we have failed to account for in our present model, there may also be methodological issues that may have been missed as well. Firstly, the operationalization of marital quality. Operationalizing marital quality in this way may underestimate the relationship of marital stability and marital compatibility. The differences in approaches and measures that the included studies used may have prevented a true moderator analysis; therefore, the importance of moderating variables within this framework may not have been appropriately emphasized or accounted for. Additional modifiers that were not accounted for included: age, gender, type of relationship, length of the relationship, the context of the relationship, and the presence of children. All of which could be important in modeling this relationship in addition to personality and psychopathology as mentioned above. Additionally, potential moderators of the inflammatory stress response such as age, gender, and disease status of the participants could influence this interaction as they have been shown to have unique and different impacts on stress (Kiecolt-Glaser, Gouin, Hantsoo, 2010).

Limitations and Future Directions

Regardless of the significance of our results, the lack of empirical studies on this topic is indeed noteworthy. This paper further emphasizes the need to study marriage and close relationships more thoroughly through biopsychosocial means as such pathways have great impact on health, specifically in regards to inflammatory processing. The design described here was limited in scope and sample size and failed to account for personality and existing physical illness and psychopathology. Our inclusion and exclusion criteria could have significantly limited our data range and sample size, and the scores of reported marital satisfaction were relatively high for majority of the studies included. Further, meta-analytic designs integrate and aggregate findings to provide overarching summaries so the interpretations and conclusions made must be approached with caution.

Despite the results of this analysis and these vast limitations, the literature clearly suggests that low marital satisfaction is a strong correlate and risk factor for health and disease. Thus far, no study has quantified a baseline association between marital satisfaction and inflammatory processing, and our meta-analytic model indicates that the mechanisms through which the interaction occurs remain to question. We suggest future studies on this topic address the individual differences in stress responses and design their models to emphasize the effects of moderators.

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Appendix: Tables and Figures

Table 1: Search Terms

<i>Independent Variables</i>	<i>Dependent Variables</i>
<i>Marriage Satisfaction</i>	C-Reactive Protein (CRP)
<i>Marital Adjustment</i>	Interleukin 1-Beta (IL-1B)
<i>Marriage Quality</i>	Interleukin Six (IL-6)
<i>Marital Relationship</i>	Tumor Necrosis Factor (TMF)
<i>Marital Satisfaction</i>	Inflammation
<i>Marital Quality</i>	Inflammatory Response
<i>Dyadic Satisfaction</i>	Immune Response
<i>Relationship Satisfaction Index</i>	Inflammatory Functioning
<i>Dyadic Quality</i>	Immune Functioning
<i>Dyadic Relationship</i>	Systemic Inflammation
<i>Couples Satisfaction</i>	Systemic Inflammatory Response
<i>Marital Functioning</i>	Systemic Inflammatory Functioning
<i>Relationship Quality</i>	
<i>Dyadic Adjustment</i>	
<i>Relationship Quality Index</i>	
<i>Couples Satisfaction Index</i>	

Figure 1: Screening and Inclusion Search Strategy

