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Marital Quality and Cardiovascular Risk in Women During the Menopausal Transition

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During the Menopausal Transition

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ABSTRACT

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Marital quality is linked to health benefits for men and women. Although women have less risk factors than men for cardiovascular disease prior to menopause, their risk increases substantially after menopause. The purpose of this study was to assess the impact of marital quality and vasomotor symptoms on cardiovascular risk factors including C-reactive protein (CRP) and carotid intima-media thickness (cIMT) in women before, during, and after the menopausal transition. The final sample consisted of 92 married women between the ages of 40 and 60 years. Hypotheses were tested using hierarchical regression and general linear modeling. Results suggest that greater marital quality reduces the negative effect of a lower level of vasomotor symptoms on cIMT but not CRP. Contrary to hypotheses, marital quality did not predict CRP or cIMT and vasomotor symptoms were not correlated with CRP or cIMT. While analyses did not support an interaction between menopausal status and lower marital quality on vasomotor symptoms or CRP, there was limited support for an interaction between menopausal status and lower marital quality on cIMT ($p = .057$) suggesting that for postmenopausal women higher marital quality is related to lower levels of cIMT. Overall, findings suggest that it is important to consider the impact of psychosocial aspects of a middle aged woman's life (i.e., marital quality) in conjunction with biological stressors when assessing cardiovascular risks in women during the menopausal transition.

Keywords: marital quality, menopause, vasomotor, cardiovascular disease, C-reactive protein, carotid intima-media thickness

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Cardiovascular disease (CVD) is responsible for more deaths worldwide than any other factor and in the United States CVD is the main cause of death in women 50 and older (Bonow, Smaha, Smith, Mensah, & Lenfant, 2002; Chida & Steptoe, 2010; Mendis et al., 2007; Michaud, Murray, & Bloom, 2001). In fact, a greater number of women die from CVD than men (Mosca, Benjamin, & Berra, 2011; Vitale et al., 2007). Lifestyle risk factors (e.g., poor diet, sedentary lifestyle, tobacco use, and alcohol consumption) account for a large proportion of the incidence of cardiovascular conditions. This is primarily due to elevations in blood pressure, cholesterol, and weight associated with these lifestyle factors (Bonow, et al., 2002; Mendis et al., 2007). Although prevalent in both sexes, women have fewer risk factors of CVD prior to menopause and symptoms associated with CVD typically develop about a decade later in women compared to men. The changes associated with the onset of perimenopause substantially increase the risk of CVD in women and this elevated risk continues through postmenopause. This has led many researchers to believe that estrogen is cardioprotective (Collins et al., 2007; Ouyang, Michos, & Karas, 2006; Sherwood et al., 2010; Vitale, Miceli, & Rosana, 2007) and based on this belief, many of the studies examining CVD in women have focused on hormonal fluctuations (Derry, 2008; Newhart, 2013).

Risk factors typically associated with CVD (i.e., age, obesity, high blood pressure, and high cholesterol levels) do not explain the prolific rates of CVD in the population (Everson-Rose & Lewis, 2005; House, Landis, & Umberson, 1988; Uchino, Cacioppo, & Kiecolt-Glaser, 1996; Wamala et al., 1999). Several studies suggest that psychosocial factors such as low levels of social support and high levels of stress contribute to the development and progression of the disease (Das & O'Keefe, 2008; Everson-Rose & Lewis, 2005; Uchino et al., 1996). The role of social relationships in health and illness has been studied extensively in the literature. The quality of social relationships has been associated with reduced rates of disease and increased longevity (Cohen, 2004; Holt-Lunstad, Smith, & Layton, 2010; House et al., 1988; Hughes & Howard, 2009). Social support has also been found to have a beneficial effect on traditional cardiovascular risk factors including blood pressure regulation (Bowen et al., 2014; Phillips, Gallagher, & Carroll, 2009; Uchino et al., 1996).

Research studies examining the association between psychosocial factors and cardiac risk factors have largely drawn their samples from male populations leaving gaps in the literature in regards to the cardiac health of females (Everson-Rose & Lewis, 2005; Hughes & Howard, 2009). With regards to social relationships, most research in this area focuses on younger populations with some studies suggesting that the benefits received from different types of social support depend upon the gender and age of the individual (Hughes, 2007). In some studies, women appear more physiologically reactive than men during marital disagreements with this reactivity predicting poorer health outcomes (Coyne et al., 2001; Gallo, Troxel, Matthews, & Kuller, 2003; Kiecolt-Glaser et al., 1997). This has led some researchers to conclude that marital quality plays an important health role in women (Coyne et al., 2001; Gallo, Troxel, Matthews, & Kuller, 2003). Additionally, Schwarzer & Rieckmann (2002) postulated that changing social roles and norms at different periods across the lifespan may impact the health benefit of perceived social support from various sources. For women, the period of midlife, which encompasses menopause, may involve several transitions in social, family, and vocational dynamics and roles (Dare, 2011; Degges-White & Myers, 2006). Therefore, it seems important to consider the impact of these changes on women during the menopausal transition in addition to the impact of the physiological changes. The present study examined the role of marital quality in CVD risk factors in women during the menopausal transition time period.

Menopausal Transition

The period of menopause is marked by emotional, social, and physiological changes in women (Dare, 2011; Degges-White & Myers, 2006; Newhart, 2013). Physiologically, women experience a decline in the number of ovarian follicles. These changes generally begin to occur between the ages of 45 and 55 and are thought to lead to reduced estrogen levels and eventually to the cessation of menses (Abernethy, 2008; Gracia et al., 2005). The transition period varies and may last for months or years and frequently includes physical changes (e.g., vasomotor symptoms [night sweats and hot flashes], mood changes, and vaginal dryness) that are perceived as distressing (Buckler, 2005; Pimenta, Leal, Maroco, & Ramos, 2012; Pitkin, 2010). In addition, elevations are seen in lipid levels and the distribution of adipose tissue changes becoming more prevalent in the chest and abdominal region (Carr, 2003; El Khoudary et al., 2012a; Vitale et al., 2007). Research suggests that lipid changes (i.e., elevations in low-density lipoprotein cholesterol) are associated with an increased risk of CVD (Prospective Studies Collaboration, Lewington et al., 2007).

The construct of menopause and the stages associated with it are not clearly defined making it difficult for researchers to apply their conclusions to comparative populations and to generalize their findings (Sherman, 2005; Utian, 2004). While there are changing patterns in reproductive hormones, there are currently no clear cut-offs to define menopausal status and currently no accurate biological markers (Sherman, 2005; Utian, 1999, 2004). Recently researchers have either assessed bleeding patterns or changes in cycle length to define menopausal status (Gracia et al., 2005; Weinstein et al., 2003). In the current study, menopausal status will be defined using criteria proposed from the Stages of Reproductive Aging Workshop (STRAW; Soules et al., 2001). This workshop is a recent attempt to define a standardized reproductive staging system for use in both research and clinical settings in which committee members encouraged investigators to use correct definitions for menopausal status and to properly describe study populations (Sherman, 2005; Soules et al., 2001; Utian, 2004). The International Menopause Society (retaining some of the World Health Organization's definitions) encourages global use of the following terms and definitions: Premenopause encompasses the "entire reproductive period before the final menstrual period," perimenopause consists of the time "immediately before the menopause and the first year after menopause," and menopause is defined as the "permanent cessation of menstruation resulting from the loss of ovarian follicular activity" occurring only after "12 consecutive months of amenorrhea" (Sherman, 2005, p. 4S). The current study defines premenopause as having consistent menstrual cycles in both length and time and postmenopause as not having a period for 12 consecutive months.

Menopause and Cardiovascular Disease Risk

Traditional cardiovascular risk factors increase in postmenopausal women (Gambacianni & Pepe, 2009; Matthews et al., 2009; Tehrani, Behboudi-Gandevani, Ghanbarian, & Azizi, 2014). Studies have shown that late perimenopausal and early postmenopausal women have increased levels of total cholesterol, low density lipoproteins, and triglycerides compared to their premenopausal counterparts (Bade, Shah, Nahar, & Vaidya, 2014; Carr et al., 2000; Derby et al., 2009; Matthews et al., 2009). In addition, the change in fat-distribution during the menopausal transition is associated with greater metabolic risk factors than are seen during the premenopause stage (Carr, 2003; Janssen, Powell, Crawford, Lasley, & Sutton-Tyrell, 2008; Kuh et al., 2005;

Vitale et al., 2007). In other studies, postmenopausal women have been shown to have higher ambulatory blood pressure levels (Migneco et al., 2008; Owens, Stoney, & Matthews, 1993; Staessen, Ginocchio, Thijs, & Fagard, 1997). The associations between increased blood pressure, hypertension, abdominal fat, and metabolic syndrome in postmenopausal women place them at greater risk of CVD than women in the premenopausal stage (Perez-Lopez, Chedraui, Gilbert, & Perez-Roncero, 2009; Rosano, Vitale, & Tulli, 2006).

In addition to an increase in traditional risk factors, postmenopausal women appear to be at a cardiovascular disadvantage both physiologically and in response to environmental demands compared to premenopausal women (Matthews, Kuller, Sutton-Tyrell, & Chang, 2001) showing more reactivity to laboratory stress, higher heart rates at rest, and less parasympathetic activity (Farag, Bardwell, Nelesen, Dimsdale, & Mills, 2003). Sherwood et al. (2010) found that in response to a laboratory stress task postmenopausal women exhibited greater systemic vascular resistance than premenopausal women. Studies utilizing carotid intima-media thickness (cIMT) to assess for atherosclerosis in postmenopausal women have found that postmenopausal hormone changes (including lower levels of estrogen) are associated with higher cIMT levels and suggest that atherosclerotic changes begin around the late-perimenopausal stage and progress at a faster rate in this population (El Khoudary et al., 2012a, 2012b; Sutton-Tyrell et al., 1998). Research conducted examining C-reactive protein (CRP), an inflammatory marker associated with CVD, has found higher levels in postmenopausal women, compared to premenopausal women, along with a positive association between CRP levels and arterial stiffening (Silva et al., 2014; Woodard et al., 2011;Yasmin et al., 2004).

Researchers have implicated decreased levels of endogenous estrogen in the increased CVD risks seen in postmenopausal women (Bittner, 2009; Derry, 2008; Hodis et al., 2001;

Mendelsohn & Karas, 1999; van Eickels et al., 2001). Studies suggest that decreased estrogen may trigger an inflammatory response due to increased release of cytokines and also increased arterial stiffening (Jonason, Henriksen, Kangro, Vessby, & Ringqvist, 1998; Pfeilschifter, Koditz, Pfohl, & Schatz, 2002; Thurston, Sutton-Tyrrell, Everson-Rose, Hess, & Matthews, 2008). Wander, Brindle, and O'Connor (2008) examined CRP levels in conjunction with hormone levels related to the menstrual cycle and found that elevated levels of estrogen were associated with decreased levels of CRP.

Estrogen levels have also been implicated in the distressing physical symptoms seen during the menopausal transition including vasomotor symptoms (Buckler, 2005). Although research has not fully elucidated the physiology of vasomotor symptoms, estrogen is implicated in their development because the onset of these symptoms occurs during the menopausal transition period in which women experience reproductive hormonal changes and also because estrogen is often used to treat these symptoms (Thurston & Joffee, 2011). There is also some support for a thermoregulatory model which posits that the body system responsible for temperature regulation is narrower in women who experience vasomotor symptoms and thus minor shifts in core body temperature can more easily trigger homeostatic mechanisms such as sweating (Thurston & Joffee, 2011). Several other body systems have been implicated in the development of vasomotor symptoms including noradrenergic and autonomic systems which makes sense given that some studies have found elevated sympathetic nervous system activity in postmenopausal women (Sherwood, 2010; Thurston & Joffee, 2011).

Although the physiology of vasomotor symptoms is not well understood, much research has been dedicated to studying the association between vasomotor symptoms and CVD. Vasomotor symptoms have been associated with increased lipid levels and increased cardiovascular reactivity to stressful situations seen in women during the menopausal transition (Gambacianni & Pepe, 2009; Gast, Grobbee, & Pop, 2008). Vasomotor symptoms are also associated with increased aortic calcification, increased blood pressure levels, hypertension, and cIMT (Brown et al., 2001; Gambacianni & Pepe, 2009; Gast et al., 2008; Gerber, Sievert, Warren, Pickering, & Schwartz, 2007; Ozkaya, et al., 2011; Thurston et al., 2011).

The prevalence of vasomotor symptoms increases during the perimenopausal stage, the time period associated with most physiological changes in the majority of women (Dennerstein, Dudley, Hopper, Guthrie, & Burger, 2000; Matthews & Bromberger, 2005; Newhart, 2013). These changes are associated with fluctuating estrogen levels and indicate that changes have begun to occur in female reproductive functioning (Overlie, Moen, Holte, & Finset, 2002). Vasomotor symptoms are therefore seen as markers of perimenopause in women that experience these symptoms (Soules et al., 2001). Vasomotor symptoms are the most common reason women seek medical attention (Gambacianni & Pepe, 2009; Keefer & Blanchard, 2005; MacLennan, Lester, & Moore, 2003) and synthetic hormones were developed to treat these symptoms. This biomedical treatment model encourages the belief that menopause is a "hormone deficiency disease" (Ballard, Kuh, & Wadsworth, 2001, p. 398; Newhart, 2013).

Research findings on the impact of hormone replacement therapy (HRT) have been ambiguous with some studies suggesting that HRT was cardioprotective, while other studies suggested the possibility of an increased risk of CVD and breast cancer with HRT (Anderson et al., 2004; Col et al., 1997; Rossouw et al., 2002). More recent studies have suggested that the timing and type of HRT is important. One study found that estrogen therapy initiated in 50-59 year olds was associated with decreased plaque in the coronary arteries and less evidence of subclinical atherosclerosis (Manson et al., 2007). Recently, a consensus statement from the

International Menopause Society cited support for the use of HRT in the treatment of vasomotor symptoms in women who are less than 60 years of age or who have not been postmenopausal for more than ten years (de Villiers et al., 2013) suggesting that the benefits of HRT in alleviating negative symptoms and restoring quality of life likely outweigh the risks in this population. However, they recommend that a risk-benefit analysis be made considering both symptom relief and risks of treatment. In light of the controversy related to hormone intervention for the negative symptoms associated with the menopausal transition, it is important to consider the role that other factors may play in elevating and reducing risks.

Social Support and Cardiovascular Risk

Research suggests that perceived social support is associated with better health outcomes including decreased risk of disease and quicker recovery from illness (Cohen, 2004). Social support refers to perceived availability of help or actual support from an individual's social network that increases their ability deal with various stressors. Subtypes of support that have been investigated include instrumental support, informational support, and emotional support (House & Kahn, 1985; Schwarzer & Rieckmann, 2002). Instrumental support refers to material assistance such as monetary support or help with a problem or task. Informational support comprises educating the individual in a way that increases their ability to cope with current challenges and usually involves advice about how to deal with one's problems. Emotional support consists of empathic expression, sympathy, or understanding as well as providing opportunities for the individual to express emotion (Cohen, 2004; Schwarzer & Rieckmann, 2002). Of these subtypes, emotional support has been the most commonly studied and relationships that are characterized by high levels of emotional support have been found to be cardioprotective (Uchino et al., 1996; Wong, Wu, Gregorich, & Pérez-Stable, 2014).

Recipients of social support show reduced physiological responses to stressful events, decreased risk factors for CVD, reduced risk of stroke, and decreased risk of morbidity and mortality (Cohen, 2004; Mezuk, Roux, & Seeman, 2010; Nagayoshi et al., 2014; Uchino, 2006; Uchino & Garvey, 1997). A meta-analysis assessing the association between social relationships and mortality risk found a "50% greater likelihood of survival" in individuals with sufficient social networks and suggested that the impact of this reduced risk of death is comparable with the health advantages of modifying other lifestyle risk factors (Holt-Lunstad et al., 2010, p. 14). Rozanski, Blumenthal, and Kaplan (1999) found that social support moderates levels of atherosclerosis. Other studies have found that social support moderates the impact of depression on cardiac mortality in men following myocardial infarction and also moderates functional recovery from stroke in those with greater impairment post-stroke (Frasure-Smith et al., 2000; Hughes & Howard, 2009; Tsouna-Hadjis, Vemmos, Zakopoulos, & Stamatelopoulos, 2000).

Perceptions of inadequate social support and feelings of isolation are associated with poorer health outcomes including increased risk of mortality and morbidity (Brummett et al., 2001, 2005; Orth-Gomer et al., 1998). A longitudinal study investigating the effects of loneliness in an older population found an association between reported feelings of loneliness and increased mortality risk (Cacioppo & Cacioppo, 2014; Luo, Hawkley, Waite, & Cacioppo, 2012). Loneliness is also associated with increased vascular resistance and blood pressure, metabolic syndrome, and decreased inflammatory control, all risk factors for CVD (Cacioppo et al., 2002; Cole et al., 2007; Whisman, 2010). Lower perception of social support was associated with increased epinephrine levels in response to stress in a hypertensive adult population (Grant, Hamer, & Steptoe, 2009; Wirtz et al., 2006). Grant et al. (2009) found that social isolation increased recovery times of systolic blood pressure in response to a stress task.

Gender differences may impact the health effects of social support (Grant et al., 2009; Hughes, 2007; Hughes & Howard, 2009). Hughes (2007) reported gender differences in cardiac responses with women high in social support showing decreased blood pressure reactivity to a stress task and men high in social support showing elevated blood pressure reactivity in response to the same task. Socially isolated men have been found to have higher cholesterol and leptin levels in response to stress (Grant et al., 2009). Elevated leptin levels are implicated in more rapid development of atherosclerosis. Research findings of gender differences between perceived social support and health outcomes highlight the need to consider gender when assessing heart disease risk.

Social Support via the Marital Relationship

Social support can be embedded within the marital relationship and when espoused, the support provided by one's partner is typically deemed to be of utmost importance (Gallo, Troxel, Matthews, et al., 2003; Holt-Lunstad, Birmingham, & Jones, 2008; Kiecolt-Glaser & Newton, 2001). Some studies have assessed whether certain types of support such as those provided through the marital relationship provide greater health benefits than other types. For example, marriage may provide a health advantage by protecting against social isolation and also by alleviating socioeconomic burden (Brummett et al., 2001; Johnson, Backlund, Sorlie, & Loveless, 2000; Robles & Kiecolt-Glaser, 2003). In addition, many researchers propose that marriage may provide a buffer from the deleterious effects of stress on an individual (Burman & Margolin, 1992; Cohen & Wills, 1985; Donoho, Crimmins, & Seeman, 2013).

Although prior research shows that marital status is associated with greater health benefits and reduced risk of CVD in men, there have been inconsistencies in research findings regarding the health benefits of marriage for women (Pienta, Hayward, & Jenkins, 2005; Ross, Mirowsky, & Goldsteen, 1990). Specifically, one limitation of prior studies is that only marital status was assessed and recent research suggests that for women, the quality of the marital relationship is an important predictor of cardiovascular outcomes (Gallo, Troxel, Matthews, et al., 2003; Holt-Lunstad et al., 2008; Umberson, Chen, House, Hopkins, & Slaten, 1996). Additionally, some studies suggest that women may be more negatively impacted health wise by marital challenges as they tend to show greater physiological reactivity (e.g., elevated blood pressure responses) to marital conflict (Gallo, Troxel, Matthews, et al., 2003; Kiecolt-Glaser et al., 1997; Smith et al., 2009). However, other studies suggest that there are no gender differences in cardiovascular reactivity in response to marital conflict (Robles, Slatcher, Trombello, & McGinn, 2014; Smith et al., 2009).

The finding that negative marital interactions are associated with larger cardiovascular responses has led researchers to propose that cardiovascular functioning is one pathway by which the marriage relationship negatively impacts health (Robles & Kiecolt-Glaser, 2003; Smith et al., 2009). It is hypothesized that repeated marital conflict overtime may promote sustained elevations in blood pressure owing to the increased heart rate and blood pressure responses that are associated with negative marital interactions (Smith et al., 2009). Further, cardiovascular reactivity is predictive of hypertension and atherosclerosis (Carroll et al., 2011; Chida & Steptoe, 2010; Matthews et al., 1998; Matthews, Woodall, & Allen, 1993; Stewart & France, 2001). One study examining relationship status and quality found that women who described their marital relationship as satisfying had less atherosclerosis in the carotid arteries when compared to women that described their relationship as less-satisfying (Gallo, Troxel, Kuller, et al., 2003). In addition, the women that reported their marriages were satisfying tended to show slower progression rates of atherosclerosis over a 3 year period. One study of middle aged healthy adults that looked at the association between the quality of marital interactions and cIMT found that daily marital interactions characterized by higher levels of negativity were associated with higher levels of cIMT which placed the individuals at greater risk for myocardial infarction and stroke (Joseph, Kamarck, Muldoon, & Manuck, 2014).

Other proposed physiological pathways by which the marital relationship impacts cardiovascular health includes negative lipid profiles, greater inflammation, and stress (Gallo, Troxel, Matthews, et al., 2003; Holt-Lunstad et al., 2008; Kiecolt-Glaser & Newton, 2001). Gallo, Troxel, Kuller, et al. (2003) found that in addition to greater psychosocial and lifestyle risk factors, middle aged women in less satisfying relationships also had greater biological risk profiles (i.e., elevated lipids) for atherosclerosis. The authors proposed that chronic exposure to biological and psychosocial risk factors experienced by women in less satisfying marriages may influence their risk of CVD overtime due to chronic exposure to stress hormones and inflammation (Kiecolt-Glaser, Gouin, & Hantsoo, 2010). Thus, the marital relationship in particular, especially when strained, appears to have the potential to lead to deleterious cardiovascular health consequences given the amount of time spent with one's partner and the repeated exposure to distressing interpersonal interactions.

Atherosclerosis is associated with low-grade inflammation (Libby, 2002; Stork, Bots, Grobbee, & van der Schouw, 2008). One indicator of inflammation that used in the literature is CRP (Donoho et al., 2013; Kiecolt-Glaser et al., 2010). Mezuk et al. (2010) found a protective role of social support against the impact of stress on CRP levels in women of middle age. Perceived marital support was found to be associated with lower levels of CRP in women (Donoho et al., 2013). One study found that couples who interacted in a hostile manner had elevated levels of interleukin-6, a proinflammatory cytokine that has been demonstrated to promote CRP production (Kiecolt-Glaser et al., 2005). Another study found that individuals with inflammatory arthritis who perceived their marriages as well adjusted had lower levels of CRP (Mustafa, Looper, Purden, Zelkowitz, & Baron, 2012). Shen, Farrell, Penedo, Schneiderman, and Orth-Gomer (2010) found an interaction between marital stress and waist circumference and CRP suggesting that women with more abdominal fat (which often occurs during the menopausal transition) may be more vulnerable to inflammation during periods of marital stress. Uchino et al. (2013) found that perceived ambivalence in a support context from one's spouse was associated with elevated levels of CRP.

Studies assessing cardiovascular risk factors during the menopausal transition have found mixed results regarding associations between hormone levels and CRP (Lakosi & Herrington, 2005). Some studies have found that CRP levels are elevated in postmenopausal women who generally have lower estrogen levels than their premenopausal counterparts (Bell, Davison, Papalia, McKenzie, & Davis, 2007; Silva et al., 2014). Other studies suggest that estrogen replacement therapy in oral form is associated with increased CRP levels in postmenopausal women (Bermudez, Rifai, Buring, Manson, & Ridker, 2002; Lakosi & Herrington, 2005; Ridker, Buring, Shih, Matias, & Hennekens, 1998). One study found that elevated levels of circulating endogenous estrogen was associated with elevated levels of CRP in postmenopausal women, a finding that seems to oppose the medical model of treating reductions in estrogen in postmenopausal women with HRT (Stork et al., 2008). However, other studies have shown that greater levels of endogenous estrogen are associated with lower levels of CRP (Blum et al., 2005). These inconsistent findings and other studies that suggest that HRT is associated with increased CVD risks, highlight the need to understand other possible contributors to the association between menopause and CVD.

Research findings suggest that psychosocial factors are prevalent during the menopausal transition and across the lifespan and play an important role in a woman's experience of transitional life periods and their experience of menopause (Dare, 2011; Degges-White & Meyers, 2006). Several studies have found that negative life events and stress predict greater prevalence of some menopausal symptoms (Binfa et al., 2004; Hardy & Kuh, 2002; Pimenta et al., 2012). Pimenta et al. (2012) explored the association between menopausal symptoms and life events and concluded that elevated negative symptoms might be more related to women's perception of stressful events than to hormonal changes. In addition, one study found a negative relationship between marital adjustment and menopausal symptoms (Sis & Pasinlioğlu, 2013). Other studies have found that women in unhappy or perceived low quality marriages, characterized by higher conflict and lower support, report more menopausal symptoms including vasomotor symptoms, than did women in higher quality marriages (Fielder & Robinson-Kurpius, 2005; Robinson-Kurpius, Foley-Nicpon, & Maresh, 2001). These findings support the idea that psychosocial variables play a role in menopausal symptoms and suggest that understanding the association between quality relationships and the experience of the menopausal transition is important to help women successfully manage this transition and improve health and well-being.

Several terms have been used in the literature to describe the marital relationship including marital satisfaction, marital quality, and marital adjustment (Sabatelli, 1988; Spanier & Lewis, 1980). Although there have been "conceptual and methodological criticisms" regarding inferences made from marital scales, given that the measures used to assess the constructs have been found to be highly correlated with one another, these terms are often used interchangeably with one another (Fincham & Bradbury, 1987; Sabatelli, 1988, p. 893; Trost, 1985). In addition, findings from a longitudinal study suggest that scales developed to measure marital adjustment (e.g., Dyadic Adjustment Scale) perform similarly to measures that assess marital satisfaction (Bradbury, Fincham, & Beach, 2000; Karney & Bradbury, 1997). Marital adjustment has been used to refer to a relationship in which the partners frequently spend time together interacting and communicating in ways that are conducive to strong relationship (Locke, 1951; Spanier, 1976). Satisfaction generally refers to how an individual feels about their spouse and the relationship (Roach, Frazier, & Bowden, 1981; Sabatelli, 1988). The construct of marital quality became more prolific in the marital research literature during the 1980's and reflects portions of both marital adjustment and marital satisfaction (Sabatelli, 1988). This combination is characterized by marital partners who are satisfied with their relationship and who endorse positive communication, minimal conflict, and the ability to respectfully resolve disagreements (Lewis & Spanier, 1979; Sabatelli, 1988). Consistent with prior research, I have used the term marital quality to reflect adjustment and satisfaction.

The Current Study

Although research supports the idea that adequate social relationships are linked to positive health outcomes, the majority of samples are Caucasian men. Research suggests that women have lower blood pressure and decreased risk of CVD prior to menopause, but indicates that these indices begin to rise around midlife eventually surpassing that of men. The majority of research during this time period examines biological risk factors including vasomotor symptoms and estrogen levels in women and proposes that estrogen deficiency accounts for the elevated CVD risk seen in women during the menopausal transition. Few studies have examined psychosocial factors such as marital quality and the role psychosocial factors play in the experience of the menopause transition and increased CVD risk. Although Gallo, Troxel, Kuller, et al. (2003) looked at marital quality and cIMT in women during the midlife transition they did not assess vasomotor symptoms or CRP levels in this population. Matthews et al. (2001) and (2009) looked at CRP and cIMT in women during the midlife transition, but did not assess vasomotor symptoms or psychosocial factors that may be associated with CVD changes. To this author's knowledge, studies assessing marital quality, menopausal status, and vasomotor symptoms, and their impact on CRP and cIMT levels have not been done.

Objectives. The primary objective of this study was to assess whether marital quality predicts vasomotor symptoms, CRP, and cIMT in pre-, peri- and postmenopausal women. In addition, I wanted to know whether this varied by menopausal status as increased vasomotor symptoms tend to occur during the late perimenopause and early postmenopausal stages and vasomotor symptoms have been found to be associated with cardiovascular risk factors (including cIMT) in prior studies. Data collected on peri- and postmenopausal women was assessed relative to premenopausal women.

Hypothesis 1a. Women who reported greater marital quality will show reduced CRP levels and less cIMT.

Hypothesis 1b. Women who reported increased vasomotor symptoms will have elevated CRP levels and greater cIMT.

Hypothesis 2. Marital quality and vasomotor symptoms will interact in predicting CRP and cIMT such that among those with higher marital quality vasomotor symptoms will have less impact on CRP and cIMT.

Hypothesis 3. Perimenopausal and postmenopausal women who reported lower marital quality will endorse greater vasomotor symptoms and show increased levels of CRP and increased cIMT.

Method

Participants

Participants included 107 women between ages 40 – 60 years, the ages typically associated with midlife, the menopause transition, and also the time period in which blood pressure begins to rise (Degges-White & Myers, 2006; Gambacciani & Pepe, 2009). The mean age of women in the study was 50.39 (*SD* = 5.45). The majority of women reported their primary ethnicity as White (96.7%), while 3.3% reported their primary ethnicity as Hispanic. Self-specified menopausal status among participants consisted of premenopausal (54.3%), perimenopausal (21.7%), and postmenopausal (23.9%). Almost half of the participants indicated that they were not experiencing vasomotor symptoms (46.7%). The average reported income was approximately $67,000 (*M* = 67.11, *SD* = 18.67). The majority of women (95.7%) reported being affiliated with the dominant religious group in the area (The Church of Jesus Christ of Latter Day Saints [LDS]).

Participants were recruited through fliers posted in medical and community settings, a university wellness program, and a women's website advertisement. Women were screened for eligibility criteria including age, never having had a heart attack or stroke, no surgical menopause, blood pressure < 160/100 mmHg, no use of HRT or birth control medication, no hypertensive or heart medication, and no reliance on medications known to influence biological risk factors or blood pressure. They were compensated fifty dollars for their participation in the study.

Prior to coming to the lab, participants signed a consent form and completed a packet of self-report questionnaires that have established reliability and validity to assess overall health and well-being. In addition, participants were assessed for carotid intima artery thickness using an ultrasound machine that directly assesses level of CVD. Carotid ultrasounds were conducted by Ron Hager, Ph.D., of the Exercise Science department at Brigham Young University. Dr. Hagar is trained and certified in the use of this machine. Women self-identified their menopausal status according to STRAW criteria by answering questions associated with stage of menopause. Women that indicated they menstruate on a regular basis and have predictable cycles are classified as premenopausal. Women that reported having missed periods, a change in the length of menstruation, or a change in the number of days between cycles are classified as perimenopausal. Women that indicated absence of menstrual cycles for over 12 months are classified as postmenopausal.

Procedure

Participants attended a lab visit where height, weight, waist, and hip measurements were taken. Two research assistants were required to run each session. Male research assistants sat behind a panel wall out of view from participants and monitored and recorded blood pressure measurements and other physiological data. Female research assistants took anthropometric measurements and administered the Trier Social Stress Task (TSST). Trained research assistants placed a blood pressure cuff on participant's left arm and instructed the participants to sit quietly without moving their arm during blood pressure measurements. Participants were administered a modification of the TSST (Kirschbaum, Pirke, & Hellhammer, 1993) by a trained research assistant. The TSST is a standardized method of inducing a stress response (e.g., elevated heart rate and blood pressure, cortisol responses) in participants in research settings. Although it may be modified, the TSST generally consists of a brief waiting period upon arrival where baseline stress measurements are taken (Birkett, 2011). The waiting period is typically followed by a speech preparation task, speech performance task, and verbal arithmetic performance period.

The performance tasks are then typically followed by a recovery period. In the current study, participants were seated in a comfortable chair and asked to sit quietly while they watched a 10 minute fish video (Harlin & Jensen, 2009) to relax. During this time period baseline blood pressure measurements were taken. Blood pressure readings were measured consistently throughout the laboratory procedure including at the beginning of each task. Participants were informed by the investigator that they would have three minutes to prepare for a job interview at an ideal job of their choice. They were instructed to think about why they would be a suitable candidate for the position. They were also informed that the investigator was trained to monitor nonverbal behavior and that their performance would be recorded for later analysis. After the three minute speech prep, participants were instructed to speak for five minutes about their qualifications for the job with the goal of convincing the research assistant that they were an ideal candidate. They were reminded that their voice would be recorded and that their performance would be evaluated at a later date and time. If the participants finished the speech task in less than five minutes standardized protocols were followed encouraging the participant to continue talking (e.g., there is still some time left, please continue). Following the speech task, participants were instructed to serially subtract the number 17 from 2,023. They were timed for five minutes and when they failed to give a correct response, they were asked by the examiner to stop and start over beginning with 2, 023. At the end of the serial subtraction task, participants were asked to complete online questionnaires about their experience during the lab visit. Following completion of the online questionnaires, participants were instructed to sit quietly and relax during a 20 recovery period. Following the 20 minute recovery period the investigator debriefed the participants by informing them that their performance was not recorded and would not be analyzed. All experimental sessions were run between 12:00 p.m. and 7:00 p.m.

Measures

Marital quality. All women completed a self-report questionnaire measuring overall marital quality (The Revised Dyadic Adjustment Scale; RDAS) through an online data collection company (Qualtrics.com) prior to their lab visit. The RDAS, a widely used measure of marital satisfaction and adjustment, is an improvement upon the Dyadic Adjustment Scale (DAS; Spanier, 1976). It is comprised of 14 questions that assess three facets of marital adjustment: dyadic consensus, dyadic satisfaction, and dyadic cohesion on a 6-point Likert type scale (Busby, Christensen, Crane, & Larson, 1995). Items on each of the three subscales were summed yielding scores ranging from 0 to 69, with higher scores indicating greater satisfaction. One limitation of using the RDAS in the past was that there were no established cutoff scores to differentiate distressed from non-distressed couples. A recent study that used conversion formulas based on the DAS cutoff score of 107 suggested that a score of 48 and above on the RDAS represents non-distressed marriages and a score of 47 and below indicates marital distress (Crane, Middleton, & Bean, 2000). Busby et al. (1995) reported a total scale internal consistency of .90 with correlations between the RDAS and the Locke-Wallace Marital Adjustment Test (MAT) of .68 ($p < .01$) and correlations between the RDAS and the DAS of .97 ($p < .01$) establishing construct validity. In addition, Busby et al. (1995) provided evidence for construct validity using confirmatory factor analysis. Discriminant analyses with the RDAS subscales provided evidence of criterion validity suggesting the possibility of discriminating between distressed and non-distressed couples (Busby et al., 1995). A reliability analysis performed on the RDAS measure in the current study showed $\alpha = .93$.

Vasomotor symptoms. Vasomotor symptoms were assessed using the Women's Health Questionnaire (WHQ; Hunter, 1992). The WHQ was developed to assess several areas of physical and emotional symptoms experienced by middle-aged women, including mood states, somatic symptoms, sleep disturbance, vasomotor symptoms, and sexual interest and satisfaction (Hunter, 2000). The WHQ has been shown to have good test-test reliability (ranging from .78 to .96) and good concurrent validity and is widely used in menopause research (Hunter, 2000; Wiklund, Karlberg, & Mattson, 1993). For the current study, only the vasomotor symptoms subscale was used. A reliability estimate for this subscale in the current study was $\alpha = .82$.

Inflammation assessment. An inflammatory biomarker, high-sensitivity CRP, was used to assess for the presence of inflammation. High-sensitivity CRP has been shown to predict several cardiovascular events (i.e., myocardial infarction, stroke) even after adjusting for traditional cardiovascular risk factors including age, diabetes, smoking, obesity, hypertension, and elevated lipids (Koenig, Lowel, Baumert, & Meisinger, 2004; Ridker, Hennekens, Buring, & Rifai, 2000). Based on the predictive power of CRP with regards to CVD, it is used frequently in the literature and guidelines have been published to inform the use of CRP as an adjunct to traditional cardiovascular risk factor screening methods. For the purpose of this study, CRP was measured via fasting blood draws that were collected by phlebotomists in the laboratory at the Brigham Young University Student Health Center. Samples were sent to Lab Corp for analysis. A high sensitivity assay was used for all CRP analyses.

Ultrasound protocol. Participants were given a brief explanation of the ultrasound procedure and were then asked to lie in a supine position on an exam table. Bilateral ultrasound scans of the right and left common carotid arteries (CCA) were then performed by a trained technician utilizing a SonoSite M-Turbo portable diagnostic ultrasound machine (Sonosite Inc., Bothell, WA). The Sonosite M-Turbo is an M-mode, high-resolution ultrasonograph equipped with a 5-MHz linear transducer. Once a suitable image was visualized, it was frozen and saved to memory. The CCA images included a single image from three different orientations (anterior, direct, and posterior) to ensure coverage of the entire circumference of the artery. The ultrasound technician attempted to capture images at a point 1 cm proximal to the common carotid bulb. The images were transferred to a laptop computer and analyzed by the same technician that performed the ultrasound examinations. A proprietary edge-detection software program was used to analyze all CCA images (SonoCalc, Sonosite Inc., Bothell, WA). The SonoCalc automated software analysis program has been described and validated previously (Fritz, Jutzy, Bansal, & Housten-Feenstra, 2005).

A total of six images (three from each CCA) were collected from each participant and averaged to determine the mean cIMT. The average cIMT was computed by combining and averaging measurements across a 10 millimeter segment of the far arterial wall. Utilizing far arterial wall measurements to assess cIMT ensures reliability (Linhart, Gariepy, Massonneau, & Dauzat, 2000). In certain instances where the carotid segment was unclear and the software was unable to identify the intima/lumen interface a manual analysis was performed using the same process described by Fritz et al.

Data Screening and Analytic Strategy

The software package SPSS Version 23 was used to conduct all statistical analyses. Primary study variables were examined for data accuracy and exploratory analyses were run to assess for missing values and outliers. Preliminary statistics were conducted to ensure that the distributions of the study variables complied with assumptions of multivariate analysis including normality, linearity, and homoscedasticity of the residuals.

Histograms, boxplots, and skewness and kurtosis statistics were used to assess for normality. Vasomotor symptoms and marital quality were negatively skewed and CRP was positively skewed. Log-transformations were applied to marital quality, CRP, and vasomotor symptoms to normalize the distributions. Bivariate correlations were used to explore linear associations between statistical control, independent, and dependent variables. Given that the associations between independent and statistical control variables entered into analyses were never greater than $r = .70$, issues of multicollinearity were determined to be minimal. Age was found to be related to vasomotor symptoms and was controlled for in analyses where vasomotor symptoms were used as a dependent variable. Additionally, curve estimations were used to assess for non-linear associations between the variables. Quadratic associations were found for statistical control variables including age with cIMT and BMI with CRP and were included in the appropriate analyses. Scatterplots of the predicted and residual values for each analysis were used to assess for homoscedasticity of the residuals and were without violation.

Regarding missing data diagnostics, individuals ($n = 2$) whose data on the dependent variables were missing were excluded from the analyses. Also, individuals ($n = 12$) were excluded if marital quality could not be assessed given that the woman was single, divorced, or widowed and not currently in a romantic relationship. A final participant was excluded due to equipment error in computing the individual's CRP score. Ninety two individuals comprised the final sample (see Figure 1 for Exclusion Flow Chart).

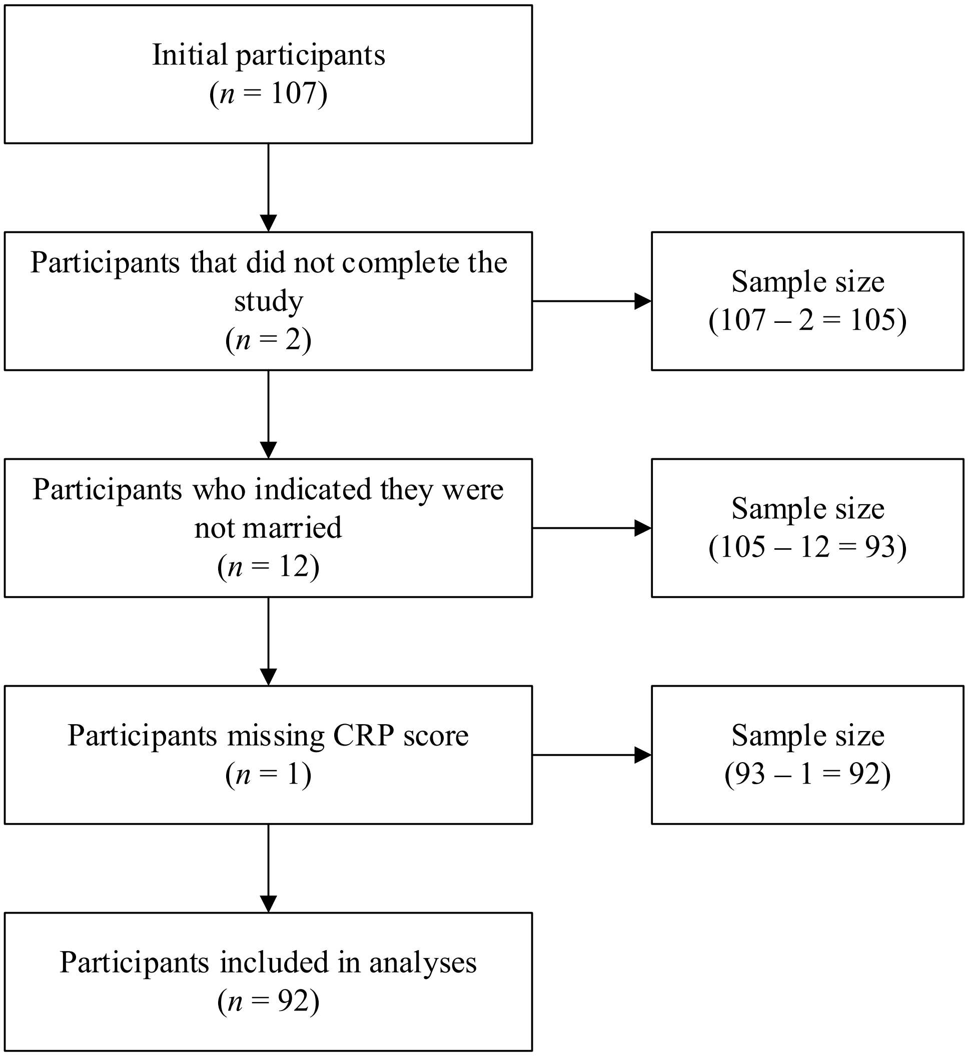

Figure 1. Participant Exclusion Flow Chart.

For hypothesis 1, separate hierarchical multiple regressions tested associations of marital quality with CRP levels and cIMT as well as vasomotor symptoms with CRP levels and cIMT. Statistical control variables (e.g., age, BMI) were entered in the first block and the independent variable of interest (e.g., marital quality) in the second block to estimate univariate hierarchical multiple regression models. In addition, a regression-based approach to testing moderation was used for hypothesis 2 to examine the joint effect of marital quality and vasomotor symptoms on CRP and cIMT. Independent variables were grand mean-centered to reduce non-essential multicollinearity. For hypothesis 3, general linear modeling was used to examine the effects of the interaction between menopausal status (e.g., pre, peri, and post) and marital quality on vasomotor symptoms, CRP, and cIMT.

Post hoc power analyses using G*Power (Faul, Erdfelder, Lang, & Buchner, 2007) indicated that the analyses were adequately powered to detect a moderate effect, though power was low and insufficient to detect a small effect in the current sample. Thus, there is increased potential risk of a Type II error.

Results

Sample characteristics are presented in Table 1. Sample characteristics based on menopausal status are presented in Table 2.

Table 1

Sample Characteristics

Variable	*n*	Minimum	Maximum	Mean (*SD*)	Range
Age	92	40	60	50.39 (5.45)	20
BMI	92	16.48	42.19	26.59 (5.69)	25.71
MQ	92	14.00	78.00	62.20 (11.28)	64.00
CRP	92	.13	12.99	2.20 (2.77)	12.86
cIMT	92	.47	.77	.63 (.07)	.30

Notes. BMI = body mass index; MQ = marital quality; CRP = C-reactive protein; cIMT = carotid intima-media thickness.

Table 2

Sample Characteristics Based on Menopausal Status

Variable	*n*	Minimum	Maximum	Mean (*SD*)	Range
Premenopausal	50				
Age		40	55	47.12 (4.23)	15
BMI		16.48	40.74	25.71 (4.82)	24.26
MQ		44	78	64.38 (7.58)	34
CRP		.13	12.62	2.17 (2.97)	12.49
cIMT		.47	.73	.61 (.06)	.26
Perimenopausal	20				
Age		41	58	52.75 (4.07)	17
BMI		20.07	41.29	28.18 (6.56)	21.23
MQ		14	78	62.70 (14.97)	64
CRP		.22	12.99	2.48 (2.94)	12.77
cIMT		.51	.74	.64 (.07)	.23
Postmenopausal	22				
Age		49	60	55.68 (3.41)	11
BMI		18.17	42.19	27.15 (6.50)	24.03
MQ		27	71	56.77 (13.13)	44
CRP		.22	7.27	2.02 (2.21)	7.05
cIMT		.56	.77	.65 (.07)	.21

Notes. BMI = body mass index; MQ = marital quality; CRP = C-reactive protein; cIMT = carotid intima-media thickness.

Marital Quality and CRP and cIMT

Hierarchical multiple regression was used to test the hypothesis that women who reported greater marital quality would have lower CRP levels than women who reported lower marital quality (see Table 3). For hypothesis 1a, the independent variables accounted for 18% of the variance in CRP ($R^2 = .18$, $F(2, 89) = 9.93$, $p < .001$). Preliminary analyses suggested BMI was correlated with CRP levels, therefore BMI was added as a covariate in block one and the analysis revealed that BMI significantly contributed to the amount of variance in CRP, $R^2 = .17$, $F(1, 90) = 18.70$, $p < .001$. The addition of the marital quality variable in the second block was not significant, $\Delta R^2 = .01$, $\Delta F(1, 89) = 1.14$, $p = .29$. Examining the standardized beta coefficients showed that marital quality did not predict CRP ($\beta = .10$, $t = 1.07$, $p = .29$). Our study results were consistent with prior literature showing that BMI predicted CRP levels ($\beta = .43$, $t = 4.42$, $p < .001$).

The hypothesis that women who reported greater marital quality would show reduced cIMT compared to women who reported lower marital quality was examined (see Table 3). The independent variables significantly contributed to the regression model, $F(2, 89) = 11.28$, $p < .001$ accounting for 20% of the variance in cIMT. In this analysis age was used as a covariate in block one of the hierarchical multiple regression as preliminary analyses suggested age was correlated with cIMT. Age significantly contributed to the amount of variance in cIMT, $R^2 = .18$, $F(1, 90) = 20.33$, $p < .001$. The marital quality variable was entered into the second block and was not significant, $\Delta R^2 = .02$, $\Delta F(1, 89) = 2.01$, $p = .16$. Examining the standardized beta coefficients indicated that there was no significant impact of marital quality on cIMT after controlling for age ($\beta = -.14$, $t = -1.42$, $p = .16$). Age was found to be significantly related to cIMT as prior literature suggests ($\beta = .40$, $t = 4.06$, $p < .001$).

Table 3

Main Effects of Marital Quality and CRP and Marital Quality and cIMT

Step	Variables Entered	*B* (*SE*)	β	*t*	Sig. (*p*)	R^2	ΔR^2	ΔF
CRP log								
1.	BMI	.08 (.02)	.42	4.32	.001			
2.	BMI	.08 (.02)	.43	4.42	.001	.18	.01	$F(1, 89) = 1.14$
	MQ	.43 (.40)	.10	1.07	.29			$p = .29$
cIMT								
1.	Age	.01 (.001)	.43	4.51	.001			
2.	Age	.01 (.001)	.40	4.06	.001	.20	.02	$F(1, 89) =2.01$
	MQ	-.04 (.03)	-.14	-1.42	.13			$p = .16$

Notes. CRP log = C-reactive protein log; cIMT = carotid intima-media thickness; MQ = marital quality; BMI = body mass index.

Vasomotor symptoms and CRP and cIMT. The hypothesis that women who reported increased vasomotor symptoms would have elevated CRP levels again controlling for BMI was tested (see Table 4). For hypothesis 1b, the overall regression model was significant, F (3, 88) = 6.32, $p < .001$ and accounted for 17.7% of the variance in CRP. The BMI and age variables were entered into the first block and significantly explained the variance in CRP, $R^2 = .17$, F (2, 89) = 9.25, $p < .001$. The vasomotor variable was entered into the second block and did not significantly contribute to the model, $\Delta R^2 = .01$, ΔF (1, 88) = .55, $p = .46$. Examination of the beta weight coefficients revealed no effect of vasomotor symptoms on CRP levels (β = .07, t = .74, $p = .46$).

The hypothesis that women who report increased vasomotor symptoms would have elevated cIMT was examined next (see Table 4). The overall model consisting of age and vasomotor symptoms significantly accounted for 20% of the variance in cIMT, F (2, 89) = 11.03, $p < .001$. Age was entered into the first block and the analysis revealed that age significantly explained the variance in cIMT, $R^2 = .18$, F (1, 90) = 20.33, $p < .001$. No significant additive effect was observed when the vasomotor symptoms variable was entered into the second block, $\Delta R^2 = .01$, ΔF (1, 89) = 1.59, $p = .21$. Examination of the beta weight coefficients revealed no significant effect of vasomotor symptoms on cIMT ($\beta = .12$, $t = 1.26$, $p = .21$).

Table 4

Main Effects of Vasomotor Symptoms and CRP and Vasomotor Symptoms and cIMT

Step	Variables Entered	*B* (*SE*)	*β*	*t*	Sig. (*p*)	R^2	ΔR^2	ΔF
CRP log								
1.	BMI	.08 (.02)	.42	4.30	.001			
	Age	.00 (.02)	.01	.08	.94			
2.	BMI	.07 (.02)	.41	4.25	.001	.18	.01	*F*(1, 88) = .55
	Age	-.00 (.02)	-.01	-.12	.90			*p* = .46
	VM	.04 (.06)	.07	.74	.46			
cIMT								
1.	Age	.01 (.001)	.43	4.51	.001			
2.	Age	.01 (.001)	.40	4.03	.001			*F*(1, 89) = 1.59
	VM	.004 (.003)	.12	1.26	.11	.20	.01	*p* = .21

Notes. CRP log = C-reactive protein log; cIMT = carotid intima-media thickness; VM = vasomotor; BMI = body mass index.

Marital Quality, Vasomotor Symptoms, and CRP and cIMT

Hierarchical linear regression was used to test the hypothesis that marital quality and vasomotor symptoms would interact in predicting CRP and cIMT. It was proposed that among those with higher marital quality vasomotor symptoms would have less impact on CRP (see Table 5). Log transformations were applied to both the marital quality and vasomotor symptoms predictor variables used in the interaction. These variables were mean-centered to reduce the risk of multicollinearity and to assist with the interpretation of the interaction effect. For hypothesis 2, the combination of BMI, vasomotor symptoms, marital quality, and the interaction between vasomotor symptoms and marital quality significantly accounted for 20.4% of the variance in CRP, F (4, 87) = 5.57, $p < .001$. Analyses revealed that BMI significantly explained 17.2% of the variance in CRP, F (1, 90) = 18.70, $p < .001$. The addition of the marital quality and vasomotor symptoms variables did not significantly explain additional variance in CRP, $\Delta R^2 = .02$, ΔF (2, 88) = .80, $p = .45$. The interaction of marital quality and vasomotor symptoms did not explain additional variance in CRP, $\Delta R^2 = .02$, ΔF (1, 87) = 1.88, $p = .17$.

Next, the impact of the interaction between marital quality and vasomotor symptoms on cIMT was examined. It was hypothesized that marital quality would reduce the negative effect of vasomotor symptoms on cIMT (see Table 5). The four independent variables significantly accounted for 25.8% of the variance in cIMT, F (4, 87) = 7.57, $p < .001$. Age was a confounding variable in this analysis and entered into the first block. Analyses revealed that age explained 18.4% of the variance in cIMT, F (1, 90) = 20.33, $p < .001$. The addition of the marital quality and vasomotor symptoms variables in the second block did not significantly contribute to the variance in cIMT, $\Delta R^2 = .03$, ΔF (2, 88) = 1.76, $p = .18$. The interaction of marital quality and vasomotor symptoms significantly explained an additional 4% of the amount of variance in cIMT, Δ*F* (1, 87) = 4.97, *p* = .03. Examination of the beta weight coefficients indicated that the interaction predicted cIMT, (β = .22, *t* = 2.23, *p* = .03; see Figure 2).

Table 5

Interaction of Marital Quality and Vasomotor Symptoms on CRP and cIMT

Step	Variables Entered	*B* (*SE*)	*β*	*t*	Sig. (*p*)	R^2	ΔR^2	Δ*F*
CRP log								
1.	BMI	.08 (.02)	.42	4.32	.001			
2.	BMI	.08 (.02)	.42	4.36	.001	.19	.02	$F(2, 88) = .80$
	MQ	.44 (.41)	.11	1.09	.28			$p = .45$
	VM	.14 (.21)	.07	.68	.50			
3.	BMI	.08 (.02)	.43	4.41	.001	.20	.02	$F(1, 87) = 1.88$
	MQ	.27 (.42)	.07	.65	.52			$p = .17$
	VM	.13 (.21)	.06	.65	.52			
	MQxVM	1.32 (.96)	.14	1.37	.17			
cIMT								
1.	Age	.01 (.001)	.42	4.51	.001			
2.	Age	.004 (.001)	.36	3.50	.001	.22	.03	$F(2, 88) = 1.76$
	MQ	-.04 (.03)	-.14	-1.47	.15			$p = .18$
	VM	.02 (.01)	.12	1.23	.22			
3.	Age	.004 (.001)	.33	3.28	.001	.26	.04	$F(1, 87) = 4.97$
	MQ	-.06 (.03)	-.21	-2.12	.04			$p = .03$
	VM	.02 (.01)	.12	1.28	.21			
	MQxVM	.13 (.06)	.22	2.23	.03			

Notes. CRP log = C-reactive protein log; cIMT = carotid intima-media thickness; MQ = marital quality; VM = vasomotor; BMI = body mass index.

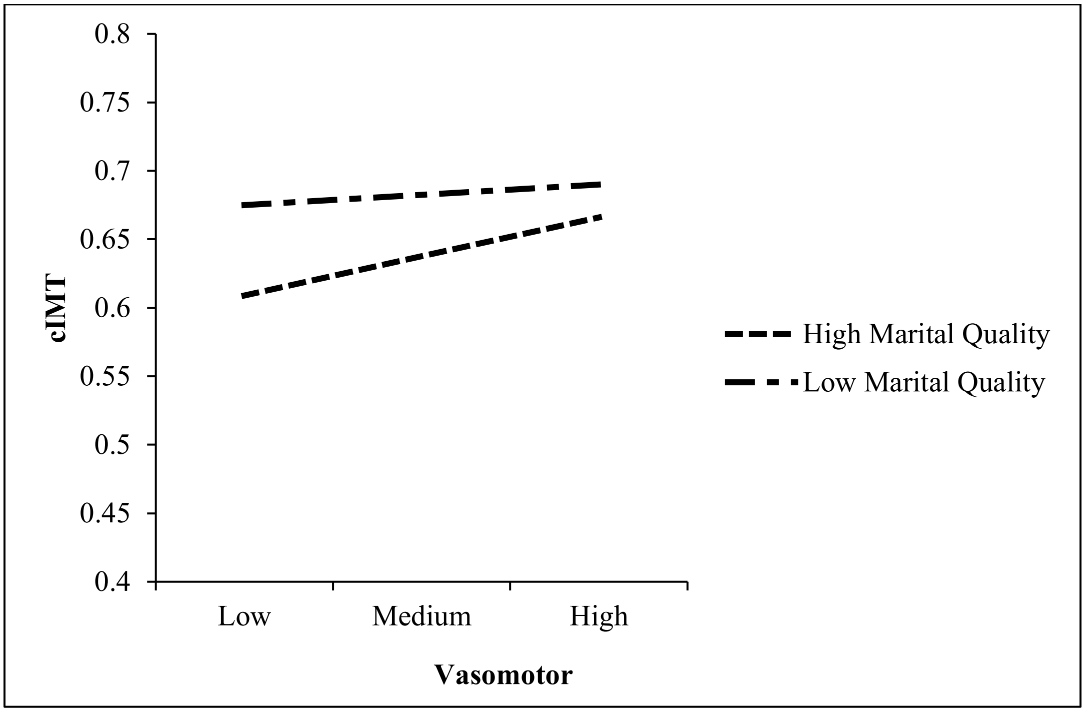

Figure 2. The Effect of Marital Quality on Vasomotor Symptoms.

Menopausal Status, Marital Quality, Vasomotor Symptoms, CRP, and cIMT

General linear modeling (GLM) was used to examine the hypothesis that perimenopausal and postmenopausal women who reported lower marital quality would endorse greater vasomotor symptoms and show increased levels of CRP and increased cIMT. Three groups were assessed based on menopausal status (premenopausal, perimenopausal, and postmenopausal). The premenopausal group was used as the reference group. Analyses revealed no interaction effects on vasomotor symptoms, $F(2, 85) = .95$, $p = .39$ (see Table 6). Additionally, no interaction effects on CRP were noted, $F(2, 85) = 2.61$, $p = .08$ (see Table 7). The interaction of menopausal status and marital quality on cIMT approached the significance level of .05, $F(2, 85) = 2.97$, $p = .06$ (see Table 8; see Figure 3).

Table 6

Interaction of Menopausal Status and Marital Quality in Predicting Vasomotor Symptoms

Source	Type III Sum of Squares	*df*	Mean Square	*F*	Significance
Intercept	.06	1	.06	.02	.88
Age	5.48	1	5.48	1.96	.17
Marital Quality (MQ)	4.84	1	4.84	1.73	.19
Menopausal status (MS)	16.62	2	8.31	2.97	.06
Interaction (MQxMS)	5.32	2	2.66	.95	.39

Notes. [a]R^2 = .241 (adjusted R^2 = .19); dependent variable: vasomotor symptoms

Table 7

Interaction of Menopausal Status and Marital Quality in Predicting CRP

Source	Type III Sum of Squares	*df*	Mean Square	*F*	Significance
Intercept	.35	1	.35	.40	.53
Age	15.17	1	15.17	17.26	.00
Marital Quality (MQ)	.07	1	.07	.08	.79
Menopausal status (MS)	2.38	2	1.19	1.35	.26
Interaction (MQxMS)	4.59	2	2.30	2.61	.08

Notes. [a]R^2 = .22 (adjusted R^2 = .17); dependent variable: CRP

Table 8

Interaction of Menopausal Status and Marital Quality in Predicting cIMT

Source	Type III Sum of Squares	*df*	Mean Square	*F*	Significance
Intercept	.07	1	.07	21.57	.00
Age	.04	1	.04	12.07	.00
Marital Quality (MQ)	.01	1	.01	3.14	.08
Menopausal status (MS)	.01	2	.00	.74	.48
Interaction (MQxMS)	.02	2	.01	2.97	.06

Notes. [a]R^2 = .28 (adjusted R^2 = .23); dependent variable: cIMT

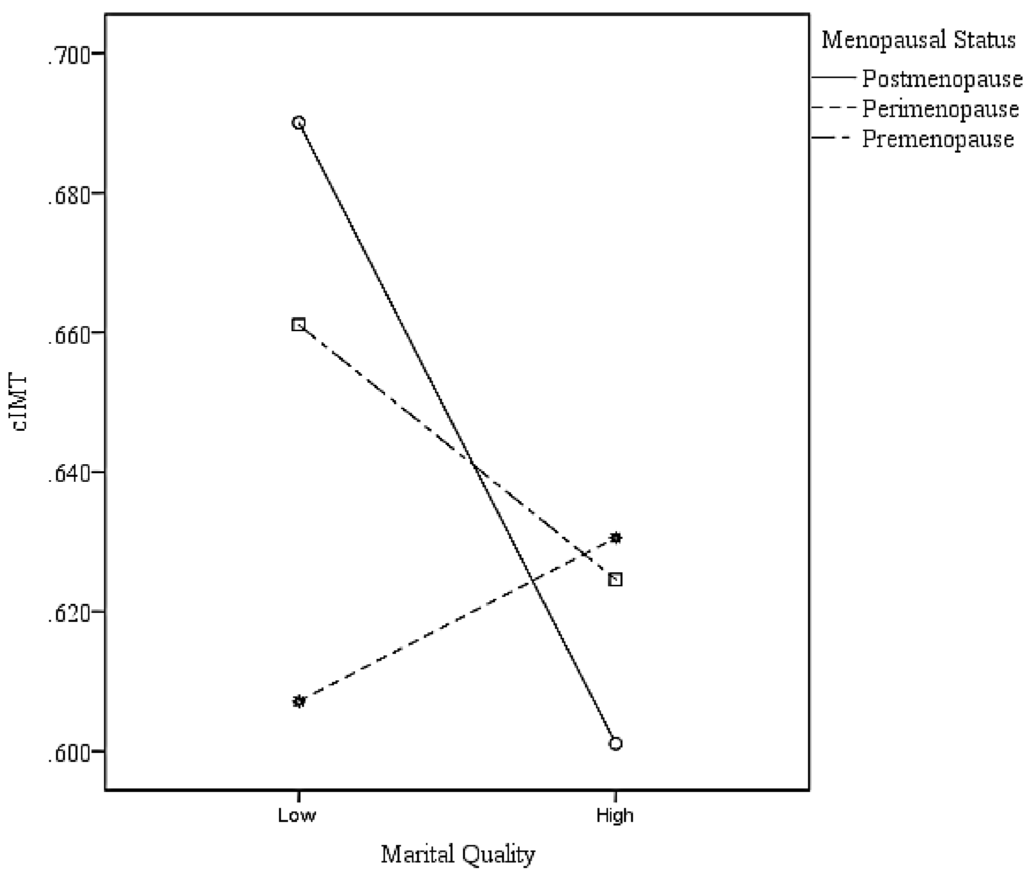

Figure 3. The Joint Effect of Marital Quality and Menopausal Status on cIMT.

Discussion

Physiological changes that occur during the menopausal transition period are postulated to have important implications with regards to women's cardiovascular health. In addition, research findings suggest that the quality of the marital relationship impacts the cardiovascular risk and outcomes of women (Coyne et al., 2001; Donoho et al., 2013; Gallo, Troxel, Matthews, et al., 2003; Holt-Lunstad et al., 2008; Orth-Gomer et al., 2000). The objective of this study was to examine whether marital quality was predictive of menopausal (vasomotor) symptoms and cardiovascular risks in middle-aged women, specifically, the time period surrounding the menopausal transition. While there are studies that have assessed marital quality and cardiovascular risk factors as well as studies that have assessed menopausal symptoms and cardiovascular risk factors, to this author's knowledge, no published studies to date have assessed these constructs in combination. Hypothesis 1a stated that greater marital quality would be associated with a reduction in CRP and cIMT. Findings did not support this hypothesis, yielding a non-significant effect of marital quality on these outcomes. Hypothesis 1b predicted that vasomotor symptoms would increase CRP and cIMT. Again, findings did not support this hypothesis.

Hypothesis 2 stated that there would be an interaction between marital quality and vasomotor symptoms on CRP and cIMT. It was predicted that in women who reported higher marital quality, vasomotor symptoms would have less impact on CRP and cIMT. Analyses revealed that greater marital quality buffers against the negative effect of vasomotor symptoms on cIMT but not CRP. In women whose relationships were characterized as higher in quality, lower levels of vasomotor symptoms did not significantly impact cIMT (i.e., only high levels of vasomotor symptoms increased cIMT). However, in women in lower quality marriages, the presence of vasomotor motor symptoms at any level increased cIMT levels.

Hypothesis 3 postulated that perimenopausal women who reported lower marital quality would endorse greater vasomotor symptoms and have higher levels of CRP and greater cIMT than premenopausal women. Contrary to prediction, analyses did not support an interaction between menopausal status and lower marital quality on vasomotor symptoms or CRP. There was limited support for the interaction between menopausal status and lower marital quality on cIMT ($p = .06$).

Interpretation of Study Findings

The integrated model tested in Hypothesis 2 analyses, which moved beyond simple main effects testing conducted in Hypothesis 1 analyses, provides an integrative perspective on how relationship factors may work in concert with biological factors to alter cardiovascular risk. In the current study, when assessed independently neither marital quality nor vasomotor symptoms were predictive of cardiovascular risk factors (CRP and cIMT). However, when observing the joint effect, women in lower quality marriages who were experiencing vasomotor symptoms showed similar elevations in cIMT in the presence of both infrequent and frequent vasomotor symptoms. Conversely, women in higher quality marriages only showed higher elevations in cIMT in the presence of a greater number of vasomotor symptoms. This suggests that both small and large changes in biological factors (i.e., vasomotor symptoms) may impact cardiovascular risk in women who perceive their marriages to be lower in quality while heightened levels of marital quality (i.e., relationships characterized by greater consensus on matters of importance, higher levels of satisfaction, and more cohesion between partners) appear to buffer against the negative impact of minor/less frequent vasomotor symptoms on cardiovascular risk factors (i.e., cIMT). Overall, women in lower quality marriages had higher levels of cIMT than women in higher quality marriages regardless of the reported frequency of vasomotor symptoms although the differences were not significant when reported frequency of vasomotor symptoms was greater.

While study findings did not provide evidence that menopausal status interacts with marital quality as predicted in Hypothesis 3, there was limited support for the interaction of marital quality and menopausal status on cIMT which merits mentioning. Past research suggests that cardiovascular risk factors increase in women during the late peri- and early postmenopausal stages of transition (El Khoudary et al., 2012a, 2012b; Gambacianni & Pepe, 2009; Matthews et al., 2009; Tehrani, et al., 2014). Researchers postulate that the increased risk in CVD is related to declining estrogen levels (Bittner, 2009; Derry, 2008; Hodis et al., 2001; Mendelsohn & Karas, 1999; van Eickels et al., 2001). The current study's findings of non-significant main effects in conjunction with the limited support found for the impact of the interaction between marital quality and menopausal status suggests that a biology by environment interaction may be more important in the elevated CVD risks seen in peri- and postmenopausal women as opposed to a biological (i.e., declining estrogen levels) or environmental factor alone.

Overall, study findings emphasize the importance of considering the impact of psychosocial aspects of a middle aged woman's life (i.e., marital quality) in conjunction with biological stressors when assessing cardiovascular risks in women during the menopausal transition. Specific details of how current findings compare to the literature and potential factors accounting for the lack of evidence of main effects in the current study are discussed below.

Marital quality, vasomotor symptoms, and CRP and cIMT. The lack of main effect findings for Hypothesis 1a suggesting marital quality was not related to CRP and cIMT were counter to hypotheses and inconsistent with literature that supports main effect models of social support (Donoho et al., 2013; Holt-Lunstad et al., 2010; House et al., 1988; Kiecolt-Glaser & Newton, 2001) and suggests that greater marital quality reduces cardiovascular risk including cIMT (Gallo, Troxel, Kuller, et al., 2003; Gallo, Troxel, Matthews, et al., 2003) as well as literature that suggests that individuals in discordant marriages show greater biological and lifestyle risk factors (Coyne et al., 2001; Orth-Gomer, et al., 2000). For example, Donoho et al. (2013) found an association between lower marital quality and higher CRP among women. In addition, Gallo, Troxel, Matthews, et al. (2003) found that women who described their marriages as lower in quality had higher lipid levels (a contributor to cIMT and CVD), were more likely to have greater plaque buildup in their carotid arteries, and were more likely to smoke than women who described their marriages as higher in quality. Of note, reported effect sizes from the Gallo study were small explaining somewhere between 2% to 4% of the variance in outcomes studied.

Gallo, Troxel, Matthews, et al. (2003) noted that perhaps the increased cardiovascular risk seen in women who were less satisfied with their marriages was due to chronic exposure to various risk factors (i.e., biological [e.g., elevations in blood pressure, cholesterol, triglycerides], lifestyle [e.g., smoking, obesity], and psychosocial [e.g., stress, depression, anxiety, low levels or perceived support]) during the middle age years. The authors proposed that the chronic exposure to cardiovascular risk factors was related to the perceived distress women experienced in the marital relationship. In contrast to the study participants in the Gallo study, the majority of individuals in the current study did not report smoking habits. Further, vasomotor symptoms (biological risk factors for CVD) were not found to be related to cIMT or CRP when assessed independent of psychosocial factors. Instead, cardiovascular risk elevations were only seen when observing the joint effect of biological and psychosocial factors.

It is important to note that while the studies performed by Gallo, Troxel, Kuller, et al. (2003) and Gallo, Troxel, Matthews, et al. (2003) assessed marital quality and biological risk factors for CVD (e.g., cIMT, lipids, blood pressure), these studies did not assess vasomotor symptoms. In addition, these studies were longitudinal assessing the impact of marital quality on other risk factors at different time points including the postmenopausal period. However, one limitation of the Gallo studies is that the authors did not assess marital quality at each time point and noted that marital quality was assessed "years before" the actual carotid ultrasounds were performed (Gallo, Troxel, Kuller et al., 2003, p. 960). The fact that marital quality was not assessed around the time the carotid ultrasounds were performed is of concern as it does not allow the reader to know whether marital quality improved, remained stable, or worsened.

Assessing the relationship between vasomotor symptoms and cardiovascular risk factors (i.e., CRP and cIMT) in Hypothesis 1b was intended to provide more insight into biological factors that might mediate or moderate the relationship. The lack of a significant relationship between vasomotor symptoms and CRP and cIMT was unexpected and inconsistent with the literature. Multiple studies have found an association between vasomotor symptoms and CVD risk factors (Franco et al., 2015; Gast et al., 2008; Muka et al., 2016) including increased cIMT (Ozkaya, et al., 2011; Thurston et al., 2011).

Present findings are more consistent with the moderator effect model which poses that social support becomes influential in the presence of elevated levels of stress (Cohen & Wills, 1985). While main effects were not found in the current study, marital quality (a type of social support) moderated the effect of vasomotor symptoms on cIMT. The current study results might also be viewed from an allostatic load model. The concept of allostasis introduced by Sterling and Eyer (1988) is that the body attempts to adapt to environmental and physiological stressors to reduce the negative impact on regulatory systems (e.g., cardiovascular, immune, neuroendocrine). The cumulative effect of the body's attempts to respond to chronic stressors outside of normal bodily functioning creates a burden on the regulatory systems that has been defined as allostatic load (Juster, McEwen, & Lupien 2010; McEwen & Stellar 1993; Seeman, Singer, Rowe, Horwitz, & McEwen, 1997). In the current sample, it appears that the cumulative effect consists of biological vulnerabilities (vasomotor symptoms) interacting with life stressors (marital distress) thus creating a burden on the cardiovascular system that is associated with elevations in cIMT. Thus, it is the additive (or joint) effect of multiple stressors that impact the cardiovascular system.

Current findings add to the current literature base assessing the association between the marital relationship and CVD by supporting the importance of managing the number of stressors during the menopausal transition including negative dyadic interactions. Reducing the number of lifestyle risk factors (e.g., smoking, alcohol use) and psychosocial stressors (e.g., marital discord) and adhering to healthier lifestyle practices (e.g., exercise, maintain healthy weight) may slow down the atherosclerotic process and improve cardiovascular health in women during the menopausal transition. These findings, in conjunction with the research findings from the limited research base assessing the association between midlife changes and psychosocial factors on cardiovascular risk, illustrate the need for continued research efforts in this population. A greater understanding of the link between biological, lifestyle, and psychosocial factors that impact cardiovascular risk during the menopausal transition would aid health psychologists in assisting middle-aged women to modify risk behaviors and to lead healthier, more fulfilling lives.

Menopausal status and marital quality as predictors of vasomotor symptoms, CRP, and cIMT. Lack of significance for Hypothesis 3 with respect to the interaction of menopausal stage and marital quality on cardiovascular risk factors is inconsistent with research findings by El Khoudary et al. (2012b) who found that increases in cIMT were more substantial during the late perimenopasual stage than during the premenopausal or early perimenopausal stages. Additionally, one study observed greater cIMT in postmenopausal women who were five to eight years past their last menstrual period when compared to premenopausal women (Matthews et al., 2001; Sutton-Tyrell et al., 1998). The author's suggest that risk of developing cIMT increases as women progress from premenopausal status to postmenopausal status and indicate that premenopausal risk factors (e.g., elevated lipid levels, increases in blood pressure) predict postmenopausal levels of cIMT. One limitation of the current study is that stage of perimenopause or length of time past menopause was not assessed in the sample. Lack of significant findings is at least in part due to the small sample size of the perimenopausal groups. Future studies would benefit from increasing the sample size of the perimenopausal group drawn from a population similar to the current study's sample. In addition, it may be important to differentiate between early and late perimenopause as well as to obtain the number of years that have passed since menopause.

Interpretation of non-significant main effects. The sample characteristics of the current study may play a role in the nonsignificant main effect findings as 96.2% of the sample endorsed being affiliated with a religious group (LDS) that practices health behaviors that reduce stress and impact cardiovascular health. Therefore, research in religion and health may provide a lens with which to interpret the current study findings. Anyfantakis et al. (2013) found that religiosity/spirituality was associated with decreased levels of cIMT. Several studies suggest that religious attendance is associated with positive health behaviors and improved social relationships (Hill, Burdette, Ellison, & Music, 2006; Strawbridge, Shema, Cohen, & Kaplan, 2001). Further, research findings suggest an association between religious attendance (once a week or more) and lower prevalence of risk factors for CVD including decreased rates of hypertension and lower blood pressure (Bell, Bowie, & Thorpe, 2012; Gillum and Ingram, 2006) as well as a reduction in cardiovascular mortality (Chida, Steptoe, & Powell, 2009). A large proportion of the current sample indicated they attend church services or activities weekly (61.9%) or several times a week (32.4%). If religiosity is associated with decreased risk factors for CVD, it follows that cardiovascular health is less variable among religious populations who practice positive health behaviors. Therefore it may be that the tenets of the LDS faith (i.e., church attendance and abstinence from nicotine and alcohol) serve to reduce the number of traditional lifestyle cardiovascular risk factors in this population thus reducing the variability of cardiovascular health in the current sample.

Another psychosocial factor found in religious communities that may contribute to nonsignificant findings related to relationship quality and cardiovascular health is perceived social support. Some studies have found that individuals who are involved with a religious organization perceive elevated levels of support (Chen & Contrada, 2007; Koenig, McCullough, & Larson, 2001). There is a prolific literature base describing the positive impact of social support on health including reduced risk of mortality (Cohen, 2004; Holt-Lunstad et al., 2010; Mezuk et al., 2010; Nagayoshi et al., 2014; Uchino, 2006; Wong et al., 2014). Additionally, some research suggests that support provided via the marital relationship may serve to reduce negative appraisals of potentially stressful events thereby decreasing the likelihood of a physiological stress response and the subsequent negative impact on body systems (Cohen &

Wills, 1985; Donoho et al., 2013). It may be that women in the current study have elevated perceptions of support related to church activity (i.e., attending services weekly or more) that fosters implementation of coping strategies and promotes reductions in negative appraisal of both distressing marital interactions as well as other potentially stressful events in their life.

The lack of variability in scores on the marital measure is another potential contributor to nonsignificant findings in the current study. The majority of participants rated their marriages as higher in quality as only 11 out of 93 scored below the cutoff. While in general, it is difficult to obtain an adequate sample comprised of equal sized distressed and non-distressed couple groups (Robles et al., 2014), comparing the marital quality scores in the current sample with prior research suggests much less variability in scores on the marital measure used in the current study (cf. Alves et al., 2015; Gallo, Troxel, Matthews, et al., 2003; Reid, Carpenter, & Draper, 2011). In addition, mean scores in the current sample were several points higher than mean scores in previous research studies suggesting that individuals in the current study perceived their marriages as less distressing and more satisfying on average than individuals in other studies (cf. Johnson et al., 2015; Reid, Carpenter, Draper, & Manning, 2010; Seedall, Butler, Zamora, & Yang, 2016). The reduction in variability based on sample reporting limits the ability to observe and compare findings related to the impact of marital quality on cardiovascular risk factors to prior research. Further, truncation in range may have impacted the analysis of marital distress on cardiovascular risk factors.

It is difficult to ascertain why there is less variability on the marital measure in the current study. It may be that women in the LDS religion are hesitant to report dissatisfaction in their marriage in an effort to present their marriages in a positive light. This type of socially desirable responding (SDR) is of concern in the literature when self-report measures are used to gather data (Paulhus, 1991). Paulhus (1984) proposed that SDR consists of both impression management and self-deception. Impression management is seen as an effort to consciously appear more favorable in the eyes of others. Self-deception on the other hand is considered to be an unconscious process and purports that the individual believes their enhanced self-reports are true although these reports may not be accurate.

It is also possible that individuals affiliated with the LDS religion may find their spousal relationships more satisfying than individuals not affiliated with the LDS religion. It is well known that a major LDS tenet is to place high priority on family relationships. Additionally, members of the LDS faith are asked by church leaders to practice principles such as forgiveness and long-suffering in their relationships. Heaton, Bar, and Jacobsen (2004) suggested that in general, members of the LDS church have lower divorce rates than the national average. Further, Strawbridge et al. (2001) found an association between religious attendance and marital stability. Therefore, with the high church attendance endorsed by the current sample, it is plausible that the sampling group believes their marriages are stable and therefore rated them higher in quality. Further, religious individuals, specifically those of the LDS faith who perceive their marriages to be of utmost importance and to extend beyond mortality may be more committed to making their marriage work than others whose beliefs about marriage differ. This commitment, combined with their belief that practicing religious principles such as service and forgiveness may benefit their marriage, may serve to reduce the negative impact of marital discord. In short, they are in the marriage for the long haul and willing to forego temporary discomfort for eternal rewards.

Finally, research suggests that both CRP and obesity are associated with an increased risk of CVD (Kuller, Tracy, Shaten, & Meilahn, 1996; Poirier et al., 2006; Ridker et al., 2000; Van

Gaal, Mertens, & De Block, 2006). In addition, although the research literature assessing the association between BMI and CRP is limited, several studies suggest that BMI/obesity is positively associated with elevated levels of CRP (Brooks, Blaha, & Blumenthal, 2010; Kao et al., 2009; Visser, Bouter, McQuillan, Wener, & Harris, 1999). Therefore, it may be that one pathway by which obesity contributes to CVD and other chronic diseases is through elevated levels of CRP (Kao et al., 2009). This may partially explain the nonsignificant main effect findings in the current study with regards to CRP as when BMI was controlled for there was no association between marital quality and CRP. When comparing the average BMI of women in the current study (26.53) to the average BMI of participants in several other studies (range 23.74 to 28.45), the average BMI of women in the current study fell somewhere in the middle (cf. Donoho et al., 2013; El Khoudary et al., 2012b; Gallo, Troxel, Kuller, et al., 2003; Gallo, Troxel, Matthews, et al., 2003; Hughes & Howard, 2009; Orth-Gomer et al., 2000). Of note, the average BMI of women in the study done by Donoho et al. (2013) who found that lower levels of support were associated with higher CRP levels in women was 28.3, which is somewhat higher than the average BMI of women in the current study.

Study Limitations/Future Directions

There are a number of limitations to the present study to consider when interpreting findings. First, the cross sectional design of the study prohibits the ability to assess relationships across time. Prior research suggests that premenopausal risk factors (e.g., lipids, elevated systolic blood pressure, BMI) are closely associated with cIMT measurements and CVD outcomes in postmenopausal women (Gallo, Troxel, Kuller, et al., 2003; Matthews et al., 2001). These studies suggest that the effects of premenopausal status lifestyle and psychosocial risk factors on cIMT may not be seen until the later stages of menopause or during the postmenopausal period when atherosclerotic plaques have developed and can be measured. Further, research suggests that various dimensions of dyadic relationship interactions (e.g., conflictual, supportive) have different implications in regards to cardiovascular health outcomes (Burman & Margolin, 1992; Gallo, Troxel, Matthews et al., 2003; Orth-Gomer et al., 2000; Smith et al., 2009). The specific characteristics of dyadic relationships that impact cardiovascular health may change, evolve, or diminish as the relationship progresses across various developmental stages of life and depending upon the number of other biological, psychological, and social stressors individuals in the dyadic relationship may be experiencing during specific time periods. Future studies may benefit from assessing the impact of biological risk factors in conjunction with lifestyle risk factors (e.g., health behaviors) and marital quality across time points allowing researchers to test trends and trajectories associated with risk factors and changes in risk factors. Additionally, Robles et al. (2014) noted the importance of uncovering moderating factors that play a role in the association between marital quality and various health outcomes.

Second, significantly fewer participants identified as being in lower quality marriages or currently experiencing vasomotor symptoms producing truncation in range of those variables. Further, there were fewer women who identified as peri- or postmenopausal in the sample. Restricted range limits the ability to find correlations in regression analyses (Alexander, 1988; Pearson, 1903). Future studies would benefit from increasing the sample size of the lower quality marriage group and the peri- and postmenopausal groups.

A third limitation to the present study is that a major portion of participants reported income levels associated with the middle class. Research findings suggest that individuals who are higher in socioeconomic status have access to more resources and are generally healthier than individuals of lower socioeconomic status (Adler et al., 1994; Marmot, 2006). Fourth, post hoc power analyses were indicative of power to find a moderate effect, but suggested that sample size was not sufficient to detect a small effect. Future studies may benefit from increased sample size and greater socioeconomic status heterogeneity.

Fifth, the homogenous ethnic and religious nature of the study sample based on the sampling context is limiting. The majority of participants were highly religious individuals that follow a health code of conduct which includes abstinence from traditional addictive substances (e.g., alcohol and nicotine) that are associated with increased cardiovascular risk. While the studies that were reviewed to develop the current research questions assessed women during the menopausal transition, their samples were drawn from a population whose demographic factors varied from the current sample (e.g., a greater number of individuals smoked and consumed alcohol). This precludes our ability to make accurate comparisons. Further, to attempt to generalize these findings considering the unique sample characteristics beyond the current context is not warranted. Despite generalization limitations, it is important to note that the literature that examines marital relationship factors, vasomotor symptoms, and specific health outcomes (e.g., cardiovascular health as measured by CRP and cIMT) in women during the menopausal transition is quite limited. Therefore, perhaps the current study can be used to inform this research base which requires replication and continued research across more diverse settings. Future studies may want to evaluate comparisons between samples that implement positive health behaviors and abstain from addictive substances and samples that do not.

Conclusions

Chronic health conditions are more common in aging populations. Prior research suggests that cardiovascular risk increases in women during late peri- and early postmenopausal stages. While some studies have found an association between marital quality and cardiovascular risk, the results of the current study demonstrate an association between marital quality and cardiovascular risk (i.e., cIMT) only in the presence of a moderating factor (i.e., vasomotor symptoms). This suggests that healthier populations may not receive the same benefit from a supportive spousal relationship as populations with greater lifestyle and psychosocial risk factors. However, study results suggest that a higher quality marriage may provide a buffer from the detrimental effects of vasomotor symptoms on the cardiovascular system in relation to cIMT. It appears that the additive effect of biological and psychosocial factors may negatively impact cardiovascular risk in populations that adhere to healthier lifestyle behaviors as opposed to one biological or psychosocial factor alone.

References

Abernethy, K. (2008). How the menopause affects the cardiovascular health of women. *Primary Health Care, 18,* 41–47.

Adler, N. E., Boyce, T., Chesney, M. A., Cohen, S., Folkman, S., Kahn, R. L., & Syme, S. L. (1994). Socioeconomic status and health: The challenge of the gradient. *The American Psychologist, 49*(1), 15–24.

Alexander, R. A. (1988). Group homogeneity, range restriction, and range enhancement effects on correlations. *Personnel Psychology, 41*, 773–777.

Alves, S., Janeiro, C., Narciso, I., Canavarro, M. C., Datallio, F. M., & Pereira, M. (2015). Attachment in patients with mental health disorders: Associations with patient and partner's adjustment. *Couple and Family Psychology: Research and Practice, 4*(4), 252–269.

Anderson, G. L., Limacher, M., Assaf, A. R., Bassford, T., Beresford, S. A., Black, H., . . . Wassertheil-Smoller, S. (2004). Effects of conjugated equine estrogen in postmenopausal women with hysterectomy: The Women's Health Initiative randomized controlled trial. *Journal of the American Medical Association, 291*(14), 1701-1712.

Anyfantakis, D., Symvoulakis, E. K., Panagiotakos, D. B., Tsetis, D., Castanas, E., Shea, S., . . . Lionis, C. (2013). Impact of religiosity/spirituality on biological and preclinical markers related to cardiovascular disease: Results from the SPILI III study. *Hormones, 12*(3), 386-396.

Bade, G., Shah, S., Nahar, P., & Vaidya, S. (2014). Effect of menopause on lipid profile in relation to body mass index. *Chronicles of Young Scientists, 5*(1), 20-24. doi: 10.4103/2229-5186.129331

Ballard, K. D., Kuh, D. J., & Wadsworth, M. E. J. (2001). The role of the menopause in women's experiences of the 'change of life'. *Sociology of Health and Illness, 23*(4), 397–424.

Bell, C. N., Bowie, J. V., & Thorpe, R. J. (2012). The interrelationship between hypertension and blood pressure, attendance at religious services, and race/ethnicity. *Journal of Religion and Health, 51*, 310–322. doi: 10.1007/s10943-010-9346-7

Bell, R. J., Davison, S. L., Papalia, M. A., McKenzie, D. P., & Davis, S. R. (2007). Endogenous androgen levels and cardiovascular risk profile in women across the adult lifespan. *Menopause, 14*, 630–638.

Bermudez, E. A., Rifai, N., Buring, J., Manson, J. E., & Ridker, P. M. (2002). Interrelationships among circulating interleukin-6, C-reactive protein, and traditional cardiovascular risk factors in women. *Arteriosclerosis Thrombosis Vascular Biology, 22*, 1668-1673.

Binfa, L., Castelo-Branco, C., Blümel, J. E., Cancelo, M. J., Bonilla, H., Muñoz, I., . . . Ríos, R. V. (2004). Influence of psycho-social factors on climacteric symptoms. *Maturitas, 48*, 425–431.

Birkett, M. A. (2011). The Trier Social Stress Test protocol for inducing psychological stress. *Journal of Visualized Experiments, 56*, e3238. doi: 10.3791/3238

Bittner, V. (2009). Menopause, age, and cardiovascular risk: A complex relationship. *Journal of American College of Cardiology, 54*, 2374–2375.

Blum, C. A., Muller, B., Huber, P., Kraenzlin, M., Schindler, C., DeGeyter, C., . . . Puder, J. J. (2005). Low-grade inflammation and estimates of insulin resistance during the menstrual cycle in lean and overweight women. *Journal of Clinical Endocrinology Metabolism, 90*, 3230–3235.

Bonow, R. O., Smaha, L. A., Smith, S. C., Mensah, G. A., & Lenfant, C. (2002). The international burden of cardiovascular disease: Responding to the emerging global epidemic. *Circulation*, *106*, 1602-1605. doi: 10.1161/01.CIR.0000035036.22612.2B

Bowen, K. S., Uchino, B. N., Birmingham, W., Carlisle, M., Smith, T. W., & Light, K. C. (2014). The stress-buffering effects of functional social support on ambulatory blood pressure. *Health Psychology, 33*(11), 1440-1443.

Bradbury, T. N., Fincham, F. D., & Beach, S. R. H. (2000). Research on the nature and determinants of marital satisfaction: A decade in review. *Journal of Marriage and the Family, 62*, 964-980.

Brooks, G. C., Blaha, M. J., & Blumenthal, R. S. (2010). Relation of C-reactive protein to abdominal adiposity. *American Journal of Cardiology, 106*, 56–61.

Brown, D., Sievert, L. L., Aki, S.L., Mills, P. S., Etrata, M. B., Paopao, R. N., & James, G. D. (2001). Effects of age, ethnicity and menopause on ambulatory blood pressure: Japanese-American and Caucasian school teachers in Hawaii. *American Journal of Human Biology, 13*(4), 486–493.

Brummett, B. H., Barefoot, J. C., Siegler, I. C., Clapp-Channing, N. E., Lytle, B. L., Bosworth, H. B., . . . Mark, D. B. (2001). Characteristics of socially isolated patients with coronary artery disease who are at elevated risk for mortality. *Psychosomatic Medicine, 63*, 267–72.

Brummett, B. H., Mark, B. B., Siegler, I. C., Williams, R. B., Babyak, M. A., Clapp-Channing, N. E., & Barefoot, J. C. (2005). Perceived social support as a predictor of mortality in coronary patients: Effects of smoking, sedentary behavior, and depressive symptoms. *Psychosomatic Medicine 67*, 40 – 45.

Buckler, H. (2005). The menopause transition: Endocrine changes and clinical symptoms. *Journal of the British Menopause Society, 11*(2), 61-65.

Burman, B., & Margolin, G. (1992). Analysis of the association between marital relationships and health problems: An interactional perspective. *Psychological Bulletin, 112*, 39–63.

Busby, D. M., Christensen, C., Crane, D. R., & Larson, J. H. (1995). A revision of the Dyadic Adjustment Scale for use with distressed and nondistressed couples: Construct hierarchy and multidimensional scales. *Journal of Marital and Family Therapy. 21*(3), 289-308.

Cacioppo, J. T. & Cacioppo, S. (2014). Social relationships and health: The toxic effects of social isolation. *Social and Personality Psychology Compass, 8*(2), 58–72. doi: 10.1111/spc3.12087

Cacioppo, J. T., Hawkley, L. C., Crawford, L. E., Ernst, J. M., Burleson, M. H., Kowalewski, R. B., . . . Berntson, G. G. (2002). Loneliness and health: Potential mechanisms. *Psychosomatic Medicine, 64*(3), 407-417.

Carr, M. C., Kim, K. H., Zambon, A., Mitchell, E. S., Woods, N. F., Casazza, C. P., . . . Schwartz, R. S. (2000). Changes in LDL density across the menopausal transition. *Journal of Investigative Medicine, 48*(4), 245-250.

Carr, M. C. (2003). The emergence of the metabolic syndrome. *The Journal of Clinical Endocrinology & Metabolism, 88*(6), 2404–2411. doi: 10.1210/jc.2003-030242

Carroll, D., Smith, G. D., Shipley, M. J., Steptoe, A., Brunner, E. J., & Marmot, M. G. (2011). Blood pressure reactions to acute mental stress and future blood pressure status: Data from the 12-year follow-up of the West of Scotland Study. *Psychosomatic Medicine, 63*(5), 737-743.

Chen, Y. Y. & Contrada, R. J. (2007). Religious involvement and perceived social support: Interactive effects on cardiovascular reactivity to laboratory stressors. *Journal of Applied Biobehavioral Research, 12(*1), 1–12.

Chida, Y. & Steptoe, A. (2010). Greater cardiovascular responses to laboratory mental stress are associated with poor subsequent cardiovascular risk status: A meta-analysis of prospective evidence. *Hypertension, 55*, 1026-1032. doi: 10.1161/HYPERTENSIONAHA.109.146621

Chida, Y., Steptoe, A., & Powell, L. H. (2009). Religiosity/spirituality and mortality. *Psychotherapy and Psychosomatics, 78*, 81–90. doi:10.1159/000190791

Cohen, S. (2004). Social relationships and health. *American Psychologist, 59*, 676-684.

Cohen, S., & Wills, T. A. (1985). Stress, social support, and the buffering hypothesis. *Psychological Bulletin, 98,* 310 – 357.

Col, N., Eckman, M., Karas, R., Pauker, S., Goldberg, R., Ross, E., . . . Wong, J. B. (1997). Patient-specific decisions about hormone replacement therapy in postmenopausal women. *Journal of the American Medical Association, 277*, 1140–1147.

Cole, S. W., Hawkley, L. C., Arevalo, J. M., Sung, C. Y., Rose, R. M., & Cacioppo, J. T. (2007). Social regulation of gene expression in human leukocytes. *Genome Biology, 8*, R189. doi:10.1186/gb-2007-8-9-r189

Collins, P., Rosano, G., Casey, C., Daly, C., Gambacciani, M., Hadji, P., . . . Stramba-Badiale, M. (2007). Management of cardiovascular risk in the perimenopausal woman: A consensus statement of European cardiologists and gynaecologists. *European Heart Journal, 28*, 2028–2040. doi:10.1093/eurheartj/ehm296

Coyne, J. C., Rohrbaugh, M. J., Shoham, V., Sonnega, J. S., Nicklas, J. M., & Cranford J. A. (2001). Prognostic importance of marital quality for survival of congestive heart failure. *American Journal of Cardiology, 88*, 526–529.

Crane, D. R., Middleton, K. C., & Bean, R. A. (2000). Establishing criterion scores for the Kansas Marital Satisfaction Scale and the Revised Dyadic Adjustment Scale. *American Journal of Family Therapy, 28*(1), 53-60.

Dare, J. S. (2011). Transitions in midlife women's lives: Contemporary experiences. *Health Care for Women International, 32*, 111–133. doi: 10.1080/07399332.2010.500753

Das, F. & O'Keefe, J. H. (2008). Behavioral cardiology: Recognizing and addressing the profound impact of psychosocial stress on cardiovascular health. *Current Hypertension Reports, 10*, 374 – 381.

de Villiers, T. J., Gass, M. L., Haines, C. J., Hall, J. E., Lobo, R. A., Pierroz, D. D. & Rees, M. (2013). Global consensus statement on menopausal hormone therapy. *Climacteric, 16*(2), 203-204.

Degges-White, S. & Myers, J. E. (2006). Transitions, wellness, and life satisfaction: Implications for counseling midlife women. *Journal of Mental Health Counseling, 28*(2), 133–150.

Dennerstein, L., Dudley, E. C., Hopper, J. L., Guthrie, J. R., & Burger, H. G. (2000). A prospective population-based study of menopausal symptoms. *Obstetrics and Gynecology, 96*(3), 351–358.

Derby, C. A., Crawford, S. L., Pasternak, R. C., Sowers, M. F., Sternfeld, B., & Matthews, K. A. (2009). Change in lipids during the menopause transition in relation to age and weight: The Study of Women's Health across the Nation (SWAN). *American Journal of Epidemiology, 169*, 1352– 1361.

Derry, P. S. (2008). Update on hormones, menopause, and heart disease: Evaluating professional responses to the Women's Health Initiative. *Health Care for Women International, 29*, 720–737. doi: 10.1080/07399330802179122

Donoho, C. J., Crimmins, E. M., & Seeman, T. E. (2013). Marital quality, gender, and markers of inflammation in the MIDUS cohort. *Journal of Marriage and Family, 75*, 127-141. doi:10.1111/j.1741-3737.2012.01023.x

El Khoudary, S. R., Wildman, R. P., Matthews, K., Thurston, R. C., Bromberger, J. T., & Sutton-Tyrrell, K. (2012a). Endogenous sex hormones impact the progression of subclinical atherosclerosis in women during the menopausal transition. *Atherosclerosis, 225*, 180-186.

El Khoudary, S. R., Wildman, R. P., Matthews, K., Thurston, R. C., Bromberger, J. T., & Sutton-Tyrrell, K. (2012b). Progression rates of carotid intima-media thickness and adventitial diameter during the menopausal transition. *Menopause: The Journal of the North American Menopause Society, 20*(1), 8-14. doi: 10.1097/gme.0b013e3182611787

Everson-Rose, S. A. & Lewis, T. T. (2005). Psychosocial factors and cardiovascular diseases. *Annual Reviews in Public Health, 26*, 469–500. doi: 10.1146/annurev.publhealth.26.021304.144542

Farag, N. H., Bardwell, W. A., Nelesen, R. A., Dimsdale, J. E., & Mills, P. J. (2003). Autonomic responses to psychological stress: The influence of menopausal status. *The Society of Behavioral Medicine, 26*(2), 134-138.

Faul, F., Erdfelder, E., Lang, A.G., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods, 39*, 175-191.

Fielder, V. K., & Robinson-Kurpius, S. E. (2005). Marriage, stress and menopause: Midlife challenges and joys. *Psicologia, 19*, 87-106.

Fincham, F. D., & Bradbury, T. N. (1987). The assessment of marital quality: A reevaluation. *Journal of Marriage and the Family, 49*, 797–809.

Franco, O. H., Muka, T., Colpani, V., Kunutsor, S., Chowdhury, S., Chowdhury, R., & Kavousi, M. (2015). Vasomotor symptoms in women and cardiovascular risk markers: Systematic review and meta-analysis. *Maturitas, 81*, 353–361.

Frasure-Smith, N., Lespe´rance, F., Gravel, G., Masson, A., Juneau, M., Talajic, M., & Bourassa, M. G. (2000). Social support, depression, and mortality during the first year after myocardial infarction. *Circulation, 101*, 1919-1924.

Fritz, H., Jutzy, R., Bansal, R., & Housten-Feenstra, L. (2005). Validation of an automated computerized analyzing system for measuring common carotid artery intima–media thickness by brightness mode. *Journal of Vascular Ultrasound, 29*, 21–26.

Gallo, L. C., Troxel, W. M., Kuller, L. H., Sutton-Tyrrell, K., Edmundowicz, D. & Matthews, K. A. (2003). Marital status, marital quality, and atherosclerotic burden in postmenopausal women. *Psychosomatic Medicine, 65*, 952–962. doi: 10.1097/01.PSY.0000097350.95305.FE

Gallo, L. C., Troxel, W. M., Matthews, K. A., & Kuller, L. H. (2003). Marital status and quality in middle-aged women: Associations with levels and trajectories of cardiovascular risk factors. *Health Psychology, 22*(5), 453–463.

Gambacciani, M. & Pepe, A. (2009). Vasomotor symptoms and cardiovascular risk. *Climacteric, 12,* 32–35.

Gast, G. C., Grobbee, D. E., & Pop, V. J., (2008). Menopausal complaints are associated with cardiovascular risk factors. *Hypertension, 51*, 1492–1498.

Gerber, L. M., Sievert, L. L., Warren, K., Pickering, T. G., & Schwartz, J. E. (2007). Hot flashes are associated with increased ambulatory systolic blood pressure. *Menopause: The Journal of the North American Menopause Society, 14(*2), 308-315. doi: 10.1097/01.gme.0000236938.74195.c6

Gillum, R. F., & Ingram, D. D. (2006). Frequency of attendance at religious services, hypertension, and blood pressure: The third national health and nutrition examination survey. *Psychosomatic Medicine Issue, 68*(3), 382-385. doi: 10.1097/01.psy.0000221253.90559.dd

Gracia, C. R., Sammel, M. D., Freeman, E. W., Lin, H., Langan, E., Kapoor, S., & Nelson, D. B. (2005). Defining menopause status: Creation of a new definition to identify the early changes of the menopausal transition. *Menopause: The Journal of the North American Menopause Society, 12*(2), 128-135. doi: 10.1097/01.GME.0000141980.81159.B2

Grant, N., Hamer, M., & Steptoe, A. (2009). Social isolation and stress-related cardiovascular, lipid, and cortisol responses. *Annals of Behavioral Medicine, 37*, 29–37. doi: 10.1007/s12160-009-9081-z

Hardy, R., & Kuh, D. (2002). Change in psychological and vasomotor symptom reporting during the menopause. *Social Science and Medicine, 55*, 1975–1988.

Harlin, L. (Producer) & Jensen, J. (Director). (2009). *Reefscapes: Nature's aquarium* (DVD). Australia: Undersea Productions.

Heaton, T. B., Bahr, S. J., & Jacobson, C. K. (2004). *A statistical profile of Mormons: Health, wealth, and social life*. Lewiston, NY: Edwin Mellen Press.

Hill, T., Burdette, A., Ellison, C., & Music, M. (2006). Religious attendance and the health behaviors of Texas adults. *Preventive Medicine, 42*, 309-312.

Hodis, H. N., Mack, W. J., Lobo, R. A., Shoupe, D., Sevanian, A., Mahrer, P. R., . . . Azen, S. P. (2001). Estrogen in the prevention of atherosclerosis. *Annals of Internal Medicine, 135*, 939-953.

Holt-Lunstad, J., Birmingham, W., & Jones, B. Q. (2008). Is there something unique about marriage? The relative impact of marital status, relationship quality, and network social support on ambulatory blood pressure and mental health. *Annals of Behavioral Medicine, 35*, 239–244. doi 10.1007/s12160-008-9018-y

Holt-Lunstad, J., Smith, T. B., & Layton, J. B. (2010). Social relationships and mortality risk: A meta-analytic review. *PLoS Medicine 7*(7), e1000316. doi:10.1371/journal.pmed.1000316

House, J. S., & Kahn, R. L. (1985). Measures and concepts of social support. In S. Cohen & S. L. Syme (Eds.), *Social support and health* (pp. 83–108). New York: Academic Press.

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

Hughes, B.M. (2007). Social support in ordinary life and laboratory measures of cardiovascular reactivity: Gender differences in habituation-sensitization. *Annals of Behavioral Medicine, 34,* 166-176.

Hughes, B. M. & Howard, S. (2009). Social support reduces resting cardiovascular function in women. *Anxiety, Stress, & Coping, (22)*5, 537-548.

Hunter, M. S. (1992). The Women’s Health Questionnaire: A measure of mid-aged women’s perceptions of their emotional and physical health. *Psychology and Health, 7*, 45–54.

Hunter, M. S. (2000). The Women's Health Questionnaire (WHQ): The development, standardization and application of a measure of mid-aged women's emotional and physical health. *Quality of Life Research*, *9*(6), 733-738.

Janssen, I., Powell, L. H., Crawford, S., Lasley, B., & Sutton-Tyrrell, K. (2008). Menopause and the metabolic syndrome: The Study of Women’s Health Across the Nation. *Archives of Internal Medicine, 168*, 1568-1575.

Jonason, T., Henriksen, E., Kangro, T., Vessby, B., & Ringqvist, I. (1998). Menopause is associated with the stiffness of the common carotid artery in 50-year-old women. *Clinical Physiology, 18*(2), 149–155.

Johnson, N. J., Backlund, E., Sorlie, P. D., & Loveless, C. A. (2000). Marital status and mortality: The national longitudinal mortality study. *Annals of Epidemiology, 10*, 224–238.

Johnson, L. N., Tambling, R. B., Mennenga, K. B., Ketring, S. A., Oka, M., Anderson, S. R., . . . Miller, R. B. (2015). Examining attachment avoidance and attachment anxiety across eight sessions of couple therapy. *Journal of Marital and Family Therapy, 42*(2), 195–212. doi: 10.1111/jmft.12136

Joseph, N. T., Kamarck, T. W., Muldoon, M. F., & Manuck, S. B. (2014). Daily marital interaction quality and carotid artery intima-medial thickness in healthy middle-aged adults. *Psychosomatic Medicine, 76*(5), 347-354. doi: 10.1097/PSY.0000000000000071

Juster, R. P., McEwen, B. S., & Lupien, S. J. (2010). Allostatic load biomarkers of chronic stress and impact on health and cognition. *Neuroscience Biobehavioral Reviews, 35*, 2-16.

Kao, T. W., Lu, I. S., Liao, K. C., Lai, H. Y., Loh, C. H., & Kuo, H. K. (2009). Associations between body mass index and serum levels of C-reactive protein. *South African Medical Journal, 99*(5), 326-330.

Karney, B. R. & Bradbury, T. N. (1997). Neuroticism, marital interaction, and the trajectory of marital satisfaction. *Journal of Personality and Social Psychology, 72*, 1075-1092.

Keefer, L., & Blanchard, E. B. (2005). A behavioral group treatment program for menopausal hot flashes: Results of a pilot study. *Applied Psychophysiology and Biofeedback, 30*(1), 21-30. doi: 10.1007/s10484-005-2171-1

Kiecolt-Glaser, J. K., Glaser, R., Cacioppo, J. T., MacCallum, R. C., Snydersmith, M., Kim, C., & Malarkey, W. B. (1997). Marital conflict in older adults: Endocrinological and immunological correlates. *Psychosomatic Medicine, 59*, 339–349.

Kiecolt-Glaser, J. K., Gouin, J. P., & Hantsoo, L. (2010). Close relationships, inflammation, and health. *Neuroscience and Biobehavioral Reviews, 35*, 33–38.

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

Kiecolt-Glaser, J. K., & Newton, N. (2001). Marriage and health: His and hers. *Psychological Bulletin, 127*, 472–503.

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

Koenig, W., Lowel, H., Baumert, J., & Meisinger, C. (2004). C-reactive protein modulates risk prediction based on the Framingham Score: Implications for future risk assessment: Results from a large cohort study in southern Germany. *Circulation, 109*, 1349-1353.

Koenig, H. G., McCullough, M. E., & Larson, D. B. (2001). *Handbook of religion and health*. New York: Oxford University Press.

Kuh, D., Langenberg, C., Hardy, R., Kok, H., Cooper, R., Butterworth, S., & Wadsworth, M. E. (2005). Cardiovascular risk at age 53 years in relation to the menopause transition and use of hormone replacement therapy: A prospective British birth cohort study. *British Journal of Obstetrics & Gynecology, 112*, 476–485.

Kuller, L. H., Tracy, R. P., Shaten, J., & Meilahn, E. N. (1996). Relationship of C-reactive protein and coronary heart disease in the MRFIT nested case-control study: Multiple Risk Factor Intervention Trial. *American Journal of Epidemiology, 144*, 537-547.

Lakosi, S. G. & Herrington, D. M. (2005). Effects of hormone therapy on C-reactive protein and IL-6 in postmenopausal women: A review article. *Climacteric, 8,* 3l7-326.

Lewis, R. A., & Spanier, G. B. (1979). Theorizing about the quality and stability of marriage. In Wesley R. Burr, Reuben Hill, F. Ivan Nye, & Ira L. Reiss (Eds.), *Contemporary Theories about the Family* (pp. 268-294). New York: Free Press.

Libby, P. (2002). Inflammation in atherosclerosis. *Nature, 420*, 868–874.

Linhart, A., Gariepy, J., Massonneau, M., & Dauzat, M. (2000). Carotid intima-media thickness: The ultimate surrogate end-point of cardiovascular involvement in atherosclerosis. *Applied Radiology, 29*, 25–39.

Locke, H. J. (1951). *Predicting adjustment in marriage: A comparison of a divorced and happily married group*. New York: Holt.

Luo, Y., Hawkley, L. C., Waite, L. J., & Cacioppo, J.T. (2012). Loneliness, health, and mortality in old age: A national longitudinal study. *Social Science Medicine, 74*(6), 907-14. doi: 10.1016/j.socscimed.2011.11.028

MacLennan, A., Lester, S., & Moore, V. (2003). Oral oestrogen replacement therapy versus placebo for hot flushes (Cochrane Review). In: The Cochrane Library, Issue 1. Oxford: Update Software.

Manson, J., Allison, M., Rossouw, J., Carr, J., Langer, R., Hsia, J., . . . Stefanick, M. L. (2007). Estrogen therapy and coronary-artery calcification. *New England Journal of Medicine, 356*, 2591–2602.

Marmot, M. G. (2006). Status syndrome: A challenge to medicine. *Journal of the American Medical Association, 295*(11), 1304–1307.

Matthews, K. A., & Bromberger, J. T. (2005). Does the menopausal transition affect health-related quality of life? *The American Journal of Medicine, 118*(12), 25–36.

Matthews, K. A., Crawford, S. L., Chae, C. U., Everson-Rose, S. A., Sowers, M. F., Sternfeld, B., & Sutton-Tyrrell, K. (2009). Are changes in cardiovascular disease risk factors in midlife women due to chronological aging or to the menopausal transition? *Journal of the American College of Cardiology, 54*(25), 2366-2373. doi:10.1016/j.jacc.2009.10.009

Matthews, K. A., Kuller, L. H., Sutton-Tyrrell, K., & Chang, Y. (2001). Changes in cardiovascular risk factors during the perimenopause and postmenopause and carotid artery atherosclerosis in healthy women. *Stroke, 32*, 1104-1111. doi: 10.1161/01.STR.32.5.1104

Matthews, K. A., Owens, J. F., Kuller, L. H., Sutton-Tyrrell, K., Lassila, H. C., & Wolfson, S. K. (1998). Stress-induced pulse pressure change predicts women's carotid atherosclerosis. *Stroke, 29*, 1525–1530.

Matthews, K. A., Woodall, K. L., & Allen, M. T. (1993). Cardiovascular reactivity to stress predicts future blood pressure status. *Hypertension, 22*, 479–485.

McEwen, B. S. & Stellar, E. (1993). Stress and the individual: Mechanisms leading to disease. *Archives of Internal Medicine, 153*, 2093-2101.

Mendis, S., Lindholm, L. H., Mancia, G., Whitworth, J., Adlerman, M., Lim, S., & Heagerty, T. (2007). World Health Organization (WHO) and International Society of Hypertension (ISH) risk prediction charts: Assessment of cardiovascular risk for prevention and control of cardiovascular disease in low and middle-income countries. *Journal of Hypertension, 25*, 1578–1582.

Mendelsohn, M. E., & Karas, R. H. (1999). The protective effects of estrogen on the cardiovascular system. *New England Journal of Medicine, 340*(23), 1801–1811.

Mezuk, B., Roux, A. V. D., & Seeman, T. (2010). Evaluating the buffering vs. direct effects hypotheses of emotional social support on inflammatory markers: The Multi-Ethnic Study of Atherosclerosis. *Brain, Behavior, and Immunity, 24*, 1294–1300.

Michaud, C. M., Murray, C. J. L., & Bloom, B. R. (2001). Burden of disease: Implications for future research. *Journal of the American Medical Association, 285*, 535–539.

Migneco, A., Ojetti, V., Covino, M., Mettimano, M., Montebelli, M. R., Leone, A., . . . Savi, L. (2008). Increased blood pressure variability in menopause. *European Review for Medical and Pharmacological Sciences, 12*(2), 89-95.

Mosca, L., Benjamin, E. J., & Berra, K. (2011). Effectiveness-based guidelines for the prevention of cardiovascular disease in women – 2011 update: A guideline from the American Heart Association. *Circulation, 123*, 1243–1262.

Muka, T., Oliver-Williams, C., Colpani, V., Kunutsor, S., Chowdhury, S., Chowdhury, R., . . . Franco, O. H. (2016). Association of vasomotor and other menopausal symptoms with risk of cardiovascular disease: A systematic review and meta-analysis. *PLoS ONE 11*(6), 1-15. doi:10.1371/journal.pone.0157417

Mustafa, S. S., Looper, K. J., Purden, M., Zelkowitz, P., & Baron, M. (2012). Better marital adjustment is associated with lower disease activity in early inflammatory arthritis. *Turkish Journal of Medical Sciences, 42*(1), 1180-1185. doi:10.3906/sag-1112-43

Nagayoshi, M., Everson-Rose, S. A., Iso, H., Mosley, T. H., Rose, K. M., & Lutsey, P. L., (2014). Social network, social support, and risk of incident stroke: Atherosclerosis risk in communities study. *Stroke, 45*(10), 2868-2873.

Newhart, M. R. (2013). Menopause matters: The implications of menopause research for studies of midlife health. *Health Sociology Review, 22*(4), 365–376.

Orth-Gomer, K., Horsten, M., Wamala, S. P., Mittleman, M. A., Kirkeeide R, Svane, B., . . . Schenck-Gustafsson, K. (1998). Social relations and extent and severity of coronary artery disease: The Stockholm Female Coronary Risk Study. *European Heart Journal, 19*, 1648–1656.

Orth-Gomer, K., Wamala, S. P., Horsten, M., Schenck-Gustafsson, K., Schneiderman, N., & Mittleman, M. A. (2000). Marital stress worsens prognosis in women with coronary heart disease: The Stockholm Female Coronary Risk Study. *Journal of the American Medical Association, 284*, 3008–3014.

Ouyang, P., Michos, E. D., & Karas, R. H. (2006). Hormone replacement therapy and the cardiovascular system: Lessons learned and unanswered questions. *Journal of the American College of Cardiology, 47*, 1741–1753.

Øverlie, I., Moen, M. H., Holte, A., & Finset, A. (2002). Androgens and estrogens in relation to hot flushes during the menopausal transition. *Maturitas, 41*(1), 69–77.

Owens, J. F., Stoney, C. M., & Matthews, K. A. (1993). Menopausal status influences ambulatory blood pressure levels and blood pressure changes during mental stress. *Circulation, 88*, 2794-2802.

Ozkaya, E., Cakir, E., Kara, F., Okuyan, E., Cakir, C., Ustun, G., & Jucukozkan, T. (2011). Impact of hot flashes and night sweats on carotid intima-media thickness and bone mineral density among postmenopausal women. *International Journal of Gynecology & Obstetrics, 113* (3), 235-238.

Pearson, K. (1903). Mathematical contributions to the theory of evolution—XI. On the influence of natural selection on the variability and correlation of organs. *Philosophical Transactions, 321*, 1–66.

Paulhus, D. L. (1984). Two-component models of socially desirable responding. *Journal of Personality and Social Psychology*, *46*, 598-609. doi:10.1037/0022-3514.46.3.598

Paulhus, D. L. (1991). Balanced Inventory of Desirable Responding (BIDR). In J. P. Robinson, P. R. Shaver, & L. S. Wrightsman (Eds.), *Measures of personality and social psychological attitudes* (pp. 37-41). San Diego, CA: Academic Press.

Perez-Lopez, F. P., Chedraui, P., Gilbert, J. J., & Perez-Roncero, G. (2009). Cardiovascular risk in menopausal women and prevalent related co-morbid conditions: Facing the post-Women's Health Initiative era. *Fertility and Sterility, 92*(4), 1171-1186. doi:10.1016/j.fertnstert.2009.06.032

Pfeilschifter, J., Konitz, R., Pfohl, M., & Schatz, H. (2002). Changes in proinflammatory cytokine activity after menopause. *Endocrinology Review, 23*(1), 90–119.

Phillips, A. C., Gallagher, S., & Carroll, D. (2009). Social support, social intimacy, and cardiovascular reactions to acute psychological stress. *Annals of Behavioral Medicine, 37*, 38–45. doi: 10.1007/s12160-008-9077-0

Pienta, A. M., Hayward, M. D., & Jenkins, K R. (2005). Health consequences of marriage for the retirement years. *Journal of Family Issues, 21*, 559-586. doi: 10.1177/019251300021005003

Pimenta, F., Leal, I., Maroco, J., & Ramos, C. (2012). Menopausal symptoms: Do life events predict severity of symptoms in peri- and postmenopause? *Maturitas, 72*, 324– 331.

Pitken, J. (2010). Cultural issues and the menopause. *Menopause International, 16*(4), 156-161.

Poirier, P., Giles, T. D., Bray, G. A., Hong, Y., Stern, J. S., Pi-Sunyer, F. X., & Eckel, R. H. (2006). Obesity and cardiovascular disease: Pathophysiology, evaluation, and effect of weight loss. *Circulation, 113*, 898–918.

Prospective Studies Collaboration, Lewington, S., Whitlock, G., Clarke, R., Sherliker, P., Emberson, J., Halsey, J., . . . Collins, R. (2007). Blood cholesterol and vascular mortality by age, sex, and blood pressure: A meta-analysis of individual data from 61 prospective studies with 55,000 vascular deaths. *Lancet, 370*, 1829–1839.

Reid, R. C., Carpenter, B. N., & Draper, E. D. (2011). Disputing the notion of psychopathology among women married to hypersexual men using the MMPI-2-RF. *Journal of Sex & Marital Therapy, 37*, 45–55. doi: 10.1080/0092623X.2011.533585

Reid, R. C., Carpenter, B. N., Draper, E. D., & Manning, J. C. (2010). Exploring psychopathology, personality traits, and marital distress among women married to hypersexual men. *Journal of Couple & Relationship Therapy, 9*(3), 203-222. doi: 10.1080/15332691.2010.491782

Ridker, P. M., Buring, J. E., Shih, J., Matias, M., & Hennekens, C. H. (1998). Prospective study of C-reactive protein and the risk of future cardiovascular events among apparently healthy women. *Circulation, 98*, 731-733.

Ridker, P. M., Hennekens, C. H., Buring, J. E., & Rifai, R. (2000). C-reactive protein and other markers of inflammation in the prediction of cardiovascular disease in women. *The New England Journal of Medicine, 342*, 836-843.

Roach, A. J., Frazier, L. P. & Bowden, S. R. (1981). The Marital Satisfaction Scale: Development of a measure for intervention research. *Journal of Marriage and the Family 43*, 537-546.

Robinson-Kurpius, S. E., Foley-Nicpon, M. F., & Maresh, S. E. (2001). Mood, marriage, and menopause. *Journal of Counseling Psychology, 48*, 77-84.

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

Robles, T. F., Slatcher, R. B., Trombello, J. M., McGinn, M. M. (2014). Marital quality and health: A meta-analytic review. *Psychological Bulletin, 140*(1), 140-187. doi: 10.1037/a0031859

Rosano, G. M. D., Vitale, C., & Tulli, A. (2006). Managing cardiovascular risk in menopausal women. *Climacteric, 9*(1), 19–27.

Ross, C. E., Mirowsky, J., & Goldsteen, K. (1990). The impact of the family on health: The decade in review. *Journal of Marriage and the Family, 52*, 1059–1078.

Rossouw, J. E., Anderson, G. L., Prentice, R. L., LaCroix, A. Z., Kooperberg, C., Stefanick, M. L., . . . Ockene, J. (2002). Risks and benefits of estrogen plus progestin in healthy postmenopausal women: Principal results From the Women's Health Initiative randomized controlled trial. *Journal of the American Medical Association, 288*(3), 321-333.

Rozanski, A., Blumenthal, J. A., & Kaplan, J. (1999). Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy. *Circulation, 99*(16), 2192-2217.

Sabatelli, R. M. (1988). Measurement issues in marital research: A review and critique of contemporary survey instruments. *Journal of Marriage and Family, 50*(4), 891-915.

Schwarzer, R. & Rieckmann, N. (2002). Social support, cardiovascular disease, and mortality. In G. Weidner, M. Kopp, & M. Kristenson (Eds.), *Heart disease: Environment, stress, and gender* (pp. 185-197). Amsterdam, Netherlands: IOS Press.

Seedall, R. B., Butler, M. H., Zamora, J. P., & Yang, C. (2016). Attachment change in the beginning stages of therapy: Examining change trajectories for avoidance and anxiety. *Journal of Marital and Family Therapy, 42*(2), 217–230. doi: 10.1111/jmft.12146

Seeman, T. E., Singer, B. H., Rowe, J. W., Horwitz, R. I., & McEwen, B. S. (1997). Price of adaptation - allostatic load and its health consequences. MacArthur studies of successful aging. *Archives of Internal Medicine, 157*, 2259-2268.

Shen, B. J., Farrell, K. A., Penedo, F. J., Schneiderman, N., & Orth-Gomer, K. (2010). Waist circumference moderates the association between marital stress and C-reactive protein in middle-aged healthy women. *Annals of Behavioral Medicine, 40*, 258–264. doi: 10.1007/s12160-010-9211-7

Sherman, S. (2005). Defining the menopausal transition. *The American Journal of Medicine, 118* (12B), 3S–7S.

Sherwood, A., Park, S. B., Hughes, J. W., Blumenthal, J. A., Hinderliter, A., Trivedi, R., & McFetridge-Durdle, J. (2010). Cardiovascular hemodynamics during stress in premenopausal versus postmenopausal women. *Menopause, 17*(2), 403-409. doi: 10.1097/gme.0b013e3181b9b061

Silva, D. C., Costa, L. O., Vasconcelos, A. A., Cerqueira, J. C., Fantato, D., Torres, D. C., . . . Costa, H. F. (2014). Waist circumference and menopausal status are independent predictors of endothelial low-grade inflammation. *Endocrinology Research, 39*(1), 22–25.

Sis, A. & Pasinlioğlu, T. (2013). Determining the association between Turkish women's menopausal symptoms and their marital adjustment. *Turkish Journal of Medical Science, 43,* 928-938. doi:10.3906/sag-1208-7

Smith, T. W., Uchino, B. N., Berg, C. A., Florsheim, P., Pearce, G., Hawkins, M., . . . Olsen-Cerny, C. (2009). Conflict and collaboration in middle-aged and older couples: II. Cardiovascular reactivity during marital interaction. *Psychology & Aging, 24*(2), 274-286. doi: 10.1037/a0016067

Soules, M. R., Sherman, S., Parrott, E., Rebar, R., Santoro, N., Utian, W., & Woods, N. (2001). Executive summary: Stages of Reproductive Aging Workshop (STRAW). *Menopause: The Journal of the North American Menopause Society, 8*(6), 402–407.

Spanier, G. B. (1976). Measuring dyadic adjustment: New scales for assessing the quality of marriage and similar dyads. *Journal of Marriage and the Family, 38*, 15-28.

Spanier, G. B., & Lewis, R. A. (1980). Marital quality: A review of the seventies. *Journal of Marriage and the Family, 42*, 825-839.

Staessen, J. A., Ginocchio, G., Thijs, L., & Fagard, R. (1997). Conventional and ambulatory blood pressure and menopause in a prospective population study. *Journal of Human Hypertension, 11*, 507-514.

Sterling, P. & Eyer, J. (1988). Allostasis: A new paradigm to explain arousal pathology. In S. Fisher& J. Reason (Eds.), *Handbook of Life Stress, Cognition, and Health* (pp. 631-651). New York, NY: Wiley.

Stewart, J. C., & France, C. R. (2001). Cardiovascular recovery from stress predicts longitudinal changes in blood pressure. *Biological Psychology, 58*, 105–120.

Stork, S., Bots, M. L., Grobbee, D. E., & van der Schouw, Y. T. (2008). Endogenous sex hormones and C-reactive protein in healthy postmenopausal women. *Journal of Internal Medicine, 264*, 245–253. doi: 10.1111/j.1365-2796.2008.01946.x

Strawbridge, W. J., Shema, S. J., Cohen, R. D., & Kaplan, G. A. (2001). Religious attendance increases survival by improving and maintaining good health behaviors, mental health, and social relationships. *Annals of Behavioral Medicine, 23*(1), 68–74.

Sutton-Tyrrell, K., Lassila, H. C., Meilahn, E., Bunker, C., Matthews, K. A., & Kuller, L. H. (1998). Carotid atherosclerosis in premenopausal and postmenopausal women and its association with risk factors measured after menopause. *Stroke, 29*, 1116-1121.

Tehrani, F. R., Behboudi-Gandevani, S., Ghanbarian, A., & Azizi, F. (2014). Effect of menopause on cardiovascular disease and its risk factors: A 9-year follow-up study. *Climacteric, 17*, 164-172.

Thurston, R. C. & Joffee, H. (2011). Vasomotor symptoms and menopause: Findings from the Study of Women's Health Across the Nation. *Obstetrics & Gynecology Clinics of North America, 38*(3), 489-501. doi:10.1016/j.ogc.2011.05.006.

Thurston, R. C., Sutton-Tyrrell, K., Everson-Rose, S. A., Hess, R., & Matthews, K. A. (2008). Hot flashes and subclinical cardiovascular disease: Findings from the Study of Women's Health Across the Nation Heart Study. *Circulation, 118*, 1234–1240.

Thurston, R. C., Sutton-Tyrrell, K., Everson-Rose, S. A., Hess, R., Powell, L. H., & Matthews, K. A. (2011). Hot flashes and carotid intima media thickness among midlife women. *Menopause, 18*(4), 352-8. doi: 10.1097/gme.0b013e3181fa27fd

Trost, J. E. (1985). Abandon adjustment! *Journal of Marriage and the Family, 47*, 1072-1073.

Tsouna-Hadjis, E., Vemmos, K.N., Zakopoulos, N., & Stamatelopoulos, S. (2000). First-stroke recovery process: The role of family social support. *Archives of Physical Medicine and Rehabilitation, 81*, 881-887.

Uchino, B. N. (2006). Social support and health: A review of physiological processes potentially underlying links to disease outcomes. *Journal of Behavioral Medicine, 29*(4), 377-387. doi: 10.1007/s10865-006-9056-5

Uchino, B. N., Bosch, J. A., Smith, T. W., Carlisle, M., Birmingham, W., Bowen, K. S., . . . O'Hartaigh, B. (2013). Relationships and cardiovascular risk: Perceived spousal ambivalence in specific relationship contexts and its links to inflammation. *Health Psychology, 32* (10), 1067-1075. doi: 10.1037/a0033515

Uchino, B. N., Cacioppo, J. T., & Kiecolt-Glaser, J. K. (1996). The relationship between social support and physiological processes: A review with emphasis on underlying mechanisms and implications for health. *Psychological Bulletin, 119*, 488–531.

Uchino, B. N. & Garvey, T. S. (1997). The availability of social support reduces cardiovascular reactivity to acute psychological stress. *Journal of Behavioral Medicine, 20*(1), 15-27.

Umberson, D., Chen, M. D., House, J. S., Hopkins, K., & Slaten, E. (1996). The effect of social relationships on psychological well-being: Are men and women really so different? *American Sociological Review, 61,* 837–57.

Utian, W. H. (1999). The International Menopause Society menopause-related terminology definitions. *Climacteric 2*, 284–286.

Utian, W. H. (2004). Menopause-related definitions. *International Congress Series, 1266*, 133–138.

van Eickels, M, Grohe, C., Cleutjens, J. P., Janssen, B. J., Wellens, H. J., & Doevendans, P. A. (2001). Beta-estradiol attenuates the development of pressure-overload hypertrophy. *Circulation 104,* 1419–1423.

Van Gaal, L. F., Mertens, I. L., & De Block, C. E. (2006). Mechanisms linking obesity with cardiovascular disease. *Nature, 444*, 875–880.

Visser, M., Bouter, L. M., McQuillan, G. M., Wener, M. H., & Harris, T. B. (1999). Elevated C-reactive protein levels in overweight and obese adults. *Journal of the American Medical Association, 282*, 2131-2135.

Vitale, C., Miceli, M., & Rosano, G. M. C. (2007). Gender-specific characteristics of atherosclerosis in menopausal women: Risk factors, clinical course and strategies for prevention. *Climacteric, 10*(2), 16–20.

Wamala, S. P., Murray, M. A., Horsten, M., Eriksson, M., Schenck-Gustafsson, K., Hamsten, A., . . . Orth-Gomér, K. (1999). Socioeconomic status and determinants of hemostatic function in healthy women. *Arteriosclerosis Thrombosis and Vascular Biology, 19*(3), 485-92.

Wander, K., Brindle, E., & O'Connor, K. A. (2008). C-reactive protein across the menstrual cycle. *American Journal of Physical Anthropology, 136*, 138–146.

Weinstein, M., Gorrindo, T., Riley, A., Mormino, J., Niedfeldt, J., Singer, B., . . . Pincus, S. (2003). Timing of menopause and patterns of menstrual bleeding. *American Journal of Epidemiology, 158*, 782-791.

Whisman, M. S. (2010). Loneliness and the metabolic syndrome in a population-based sample of middle-aged and older adults. *Health Psychology, 29*(5), 550-554. doi: 10.1037/a0020760

Wiklund, I., Karlberg, J., & Mattson, L. A. (1993). Quality of life of postmenopausal women on a regimen of transdermal estradiol therapy: A double-blind placebo-controlled study. *American Journal of Obstetrics and Gynecology, 168*, 824–830.

Wirtz, P. H., von Kanel, R., Mohiyeddini, C., Emini, L., Ruedisueli, K., Groessbauer, S., & Ehlert, U. (2006). Low social support and poor emotional regulation are associated with increased stress hormone reactivity to mental stress in systemic hypertension. *Journal of Clinical Endocrinology and Metabolism, 91*, 3857–3865.

Wong, S.T., Wu, A., Gregorich, S., & Pérez-Stable, E. J. (2014). What type of social support influences self-reported physical and mental health among older women? *Journal of Aging & Health, 26*(4), 663-678. doi: 10.1177/0898264314527478

Woodard, G. A., Mehta, V. G., Mackey, R. H., Tepper, P., Kelsey, S. F., Newman, A. B., & Sutton-Tyrrell, K. (2011). C-reactive protein is associated with aortic stiffness in a cohort of African American and white women transitioning through menopause. *Menopause, 18*(12) 1291–1297. doi:10.1097/gme.0b013e31821f81c2

Yasmin, McEniery, C. M., Wallace, S., Mackenzie, I.S., Cockcroft, J. R., & Wilkinson, I.B. (2004). C-reactive protein is associated with arterial stiffness in apparently healthy individuals. *Arteriosclerosis, Thrombosis, and Vascular Biology, 24*(5), 969–974. doi.org/10.1161/01.ATV.zhq0504.0173