Bad Marriage, Bad Heart?

Marital Quality and Cardiovascular Risks among Older Adults

Hui Liu
Department of Sociology
Michigan State University

Linda Waite
Department of Sociology
University of Chicago

Address correspondence to Hui Liu, Department of Sociology, Michigan State University, Berkey Hall, 509 E. Circle Drive 316, East Lansing, MI, 48824. Telephone: (517) 353-3265; Fax: (517) 432-2856; and Email: liuhu@msu.edu. We are grateful for comments and suggestions from participants at the conference of Integrated Research in Health and Aging: Early Results from NSHAP Wave 2. This research was supported by the National Institute On Aging under Award Number K01AG043417 (PI: Hui Liu).

Bad Marriage, Bad Heart?

Marital Quality and Cardiovascular Risks among Older Adults

Abstract

This study is among the first to assess how marital quality is related to cardiovascular risk—an important pathogenic mechanism involved in a host of age-related health conditions—at the population level. Working from a life course perspective, we identify several reasons to expect age and gender differences in the link between marital quality and cardiovascular risk. Using nationally representative longitudinal data from the first two waves of the National Social Life, Health, and Aging Project, we analyze four cardiovascular risk outcomes: hypertension, rapid heart rate, C-reactive protein, and general cardiovascular events. Results suggest that marital quality and cardiovascular risk are generally more closely related for older married people than for their younger counterparts; and that the link between marital quality and cardiovascular risk is more pronounced among women than among men at older ages. These findings fit with the gendered life course perspective and cumulative disadvantage framework.

Cardiovascular disease (CVD) is the leading cause of death and disability of both men and women in the United States, and the problem continues to grow (National Academy on An Aging Society 2000; Ostchega et al. 2007). According to the US Centers for Disease Control (CDC), about 600,000 Americans die of CVDs every year; that's one in every four deaths. This health problem is even more serious in the older population (Lakatta and Levy 2003). In the United States, more than 35% of adults aged 75 and older suffer from some type of CVD (Schiller, Lucas and Peregoy 2012). Because most CVD can be prevented, identifying relevant risk factors is extremely important in designing effective prevention strategies and programs. Many personal, social, and behavioral factors contribute to the risk of CVD over individuals' lifetime (Lakatta and Levy 2003; McFarland, Hayward, and Brown 2013). The present study focuses specifically on marital quality as a social factor that may shape the risks of CVD during later adulthood. This is an area that has been understudied, in spite of its importance.

Marital quality—broadly defined as spouses' subjective appraisals of their marital relationships, including in terms of satisfaction, happiness, strain, and conflict—is increasingly highlighted as a key aspect of adult life that has the potential to affect health by intervening in a person's life environment (Umberson et al. 2006). Cardiovascular risk refers to the presence of any physiological or functional state that is a step on the way to CVD. These risk factors constitute important pathogenic mechanisms involved in a host of age-related conditions, and they are directly affected by social and behavioral factors such as marital quality (Everson-Rose and Lewis 2005; Izzo and Black 2003; Poirier et al. 2006). To date, most empirical evidence for the impact of marital quality on cardiovascular risk comes from lab-based cross-sectional studies and clinical or community samples (Robles and Kiecolt-Glaser 2003). It is difficult to generalize conclusions from this body of research to the population as a whole or to make casual inferences.

Moreover, although hundreds of studies have examined gender differences in the effects of marital quality on health, empirical evidence is quite mixed. Some studies suggest a stronger cardiovascular response to marital quality for women, some report stronger effects on men, and still other studies find no significant gender differences (Ewart et al. 1996; Umberson et al. 1996; also see a review in Kiecolt-Glaser and Newton 2001). Finally, although both marital quality and cardiovascular risk have been shown, separately, to vary by age (Lakatta and Levy 2003; Umberson et al. 2005), the potential age variation in the relationship between marital quality and cardiovascular risk has been virtually ignored.

The present study is among the first to assess the association of cardiovascular risk with marital quality during the later stages of the life course. Data are from the first two waves of the National Social Life, Health, and Aging Project (NSHAP), a nationally representative longitudinal data set on older adults. We work from a life course perspective to address two major research questions: (1) how is marital quality related to cardiovascular risk among older adults and (2) does this relationship vary by gender and/or age. The importance of this study is highlighted by the continued very high prevalence of CVD among older adults in the United States. Major obstacles to our scientific understanding of the relationship between marital quality and cardiovascular risk include (1) a lack of nationally representative evidence, (2) a consistent failure to consider age variations in key relationships, and (3) mixed evidence on the gender patterns. The present study addresses these obstacles, and its findings have important implications for health policy and practice.

Marital Quality and Cardiovascular Risk: Clinical and Community Evidence

To date, most empirical evidence on the effects of marital quality on cardiovascular function comes from lab-based clinical samples that focus on psychobiological responses to

marital stress. The evidence from such studies suggests limited cardiovascular responses to marital support (Robles and Kiecolt-Glaser 2003). The common premise is that excessive cardiovascular reactivity to marital stress is a risk factor for hypertension, rapid heart rate, and CVD. Exposure to marital stress causes the sympathetic nervous system to metabolize glucose and to induce the release of stress hormones (e.g., catecholamines, cortisol). The release of stress hormones in turn increases blood pressure and heart rate, accelerates breathing, and constricts blood vessels. This "fight or flight" process may increase allostatic load, cause wear and tear on the regulatory mechanisms of the human body, and lead to chronic conditions, such as hypertension and CVD (Taylor et al. 2000; Everson-Rose and Lewis 2005).

A number of community-based clinical studies support this hypothesis and find that marital strain and hostile interactions produce clinically significant increases in systolic and diastolic blood pressure and heart rates (Ewart et al. 1991; Everson-Rose and Lewis 2005; Taylor et al. 2000). This effect is more pronounced for women than for men (Ewart et al. 1991). For example, Ewart et al. (1991) studied 24 female and 19 male patients (aged 32–73 years) who had been diagnosed with hypertension; and they find that hostile interaction and marital dissatisfaction are associated with increased blood pressure for women but not for men. In following up on 292 female patients who had experienced an acute coronary event during the period 1991 to 1994, Orth-Gomer et al. (2000) find that women who reported moderate to severe marital strain at baseline were almost three times more likely to experience a recurrent coronary event (including cardiac death, acute myocardial infarction, and revascularization procedures) than women who reported lower levels of marital strain. Another clinical study followed 189 congestive heart failure patients; its findings suggest that marital quality is a strong predictor of mortality rates for such patients over the next four years following the event (Coyne et al. 2001).

Clinical evidence on the association between cardiovascular risk and the positive aspects of marital quality is mixed. For example, Gallo et al. (2003) studied 493 middle-aged women in Allegheny County, Pennsylvania, and did not find evidence of a relationship between marriage satisfaction and either systolic or diastolic blood pressure. However, when compared with women who expressed higher levels of marriage satisfaction, women with lower levels of marriage satisfaction had lower high-density-lipoprotein cholesterol and higher low-density-lipoprotein cholesterol and triglycerides—both of which are cardiovascular risk factors. Findings of no evidence for supportive marital experience related to blood pressure fluctuations are also confirmed in other clinical studies (e.g., Ewart et al. 1991).

Clinical contributions represent a significant step forward in identifying the links between cardiovascular health and marital quality. However, the nonrepresentative samples in these community-based studies limit the generalizability of findings (Burman and Margolin 1992). One of the very few national studies relevant to this topic was recently conducted by Donoho, Crimmins, and Seeman (2013). Based on a national representative sample from the Survey of Midlife in the United States (MIDUS) Biomarker Study (n = 553), Donoho et al. assessed how marital quality was related to the level of C-reactive protein (CRP). Although CRP is used as a measure of inflammation in Donoho's study, CRP is also shown to be a strong predictor of CVD (Ridker 2003). Results suggest that women with higher marital support tend to have lower CRP but that marital support is only minimally related to the CRP level of men (Seeman et al. 2013). The Donoho study does not find evidence for marital strain in relation to CRP. Given this line of literature, we expect to find the following:

Hypothesis 1: Higher levels of negative marital quality are strongly related to higher cardiovascular risk, while higher levels of positive marital quality are only modestly related to lower cardiovascular risk *at the population level*.

A Gendered Life Course Perspective on Cumulative Advantage/Disadvantage Processes

Increasingly, scholars have been applying a life course approach to guide their analyses of marriage and health (Williams and Umberson 2004). According to the life course perspective, an individual undergoes life course stages within unique social and personal contexts; and marital quality is one of the key factors to define an individual's life context during adulthood (Umberson et al. 2006). Both the stress and support processes associated with marital relationships shape an individual's life context, which in turn may lead to health outcomes over the life course (Carr and Springer 2010). For example, involvement in a distressed marriage exposes an individual to stressful interactions that may lead to depression. Depression, in turn, affects health either indirectly, by promoting unhealthy behaviors (e.g., smoking, drinking), or directly, by stimulating the production of stress hormones, evoking physiological responses, and triggering chronic arousal (Everson-Rose and Lewis 2005; Graham, Christian, and Kiecolt-Glaser 2006). In contrast, involvement in a happy marriage may provide a person with social support, which may buffer the negative consequences of stressful life events and in turn enhance physical health (Robles and Kiecolt-Glaser 2003; Umberson et al. 2006; Williams 2003). In this study, we work from this life course perspective to understand how marital quality is related to cardiovascular risk among older men and women during later life course stages.

Age. "Life course theory is temporal and contextual in locating people in history through birth years and in the life course through the social meanings of age-graded events and activities." (Elder and O'Rand 1995:454). The life course is composed of multiple age-related

social pathways (i.e., sequences of roles and experience) that shape the course of human development and aging. Because age is the most important proxy closely tied to individuals' occupancy and transitions of experiences, roles, and statuses (e.g., school, work, marital and parental roles) throughout life (Settersten and Mayer 1997), life course scholars often adopt age as a key indicator for life course stages.

Although empirical evidence on potential age variations in links between marital quality and cardiovascular risk is rare, age is considered important in gaining a fuller understanding of this relationship (Liu 2012). According to the life course theory, the effect of marital quality on cardiovascular health may be contingent upon age-graded life course position, because advantage or disadvantage may have cumulative effects on an individual's life over life course stages (Dannefer 2003). This "cumulative-advantage/disadvantage" argument suggests that marital quality will take a toll on health with advancing age and that, therefore, the health impacts of marital quality will become more salient at older ages. For example, some detrimental effects of marital stress, such as an increased risk of smoking, may require longer durations to produce negative health outcomes at older ages. Moreover, human biological response varies by age. Marital stress at older ages may stimulate more, and more intense, cardiovascular responses because of the declining immune function and increasing frailty that typically develops in aging individuals. One recent national longitudinal study on *self-rated health* suggests that the effect of negative marital quality on self-rated overall health tends to be stronger at older ages (Umberson et al. 2006). This line of literature leads us to expect the following:

Hypothesis 2: The association between marital quality and cardiovascular risk is stronger at older ages.

Gender. Gender is another fundamental factor that can be used to define the life course context, and gender has been a central focus of research on marriage and health (Pinnelli, Racioppi and Rettaroli 2007). While married men on average receive more health promotion benefits (e.g., emotional support, regulation of health behaviors) from their marriages, married women's health seems to be more vulnerable to marital stress (Kiecolt-Glaser and Newton 2001). According to the life course perspective on cumulative advantage/disadvantage, the marital experience may take a cumulative toll on men and women's health in different ways because age-graded life course experiences and roles often unfold differently for men and women. We expect that women's cardiovascular function is more responsive to marital strain as women age: women are generally more sensitive to the quality of a relationship than are men, and therefore women in strained marriages are more likely to have a greater number of symptoms of metabolic syndrome and/or a greater incidence of depression (Wu and DeMaris 1996). Depression is one of the most often proposed mediating mechanisms linking marital relationship and physical health (Burman and Margolin 1992; Graham et al. 2006). This link may be related to the different hormone levels and profiles that exist between men and women. Indeed, a handful of clinical studies confirm this view and conclude that marital conflict tends to evoke greater and more persistent physiological changes (e.g., increases in systolic blood pressure, decreases in phytohemagglutinin, changes in hormones) in women than in men (Ewart et al. 1991; Kiecolt-Glaser and Newton 2001). A few studies show that men appear to have greater cortisol and catecholamine responses to a range of laboratory stressors, compared with women (Kiecolt-Glaser and Newton 2001). Other studies find no gender difference in the links between general health (e.g., overall self-rated health) and marital quality (Umberson et al. 1996). The mixed gender evidence may reflect gender differences in the specific physiological

mechanisms and health outcomes measured in the studies. To the extent that chronic conditions develop slowly over time and that women appear to be both physiologically and psychologically more reactive to marital stress than men (Donoho et al. 2013), we expect that the relationships between cardiovascular risk factors and age are stronger for women than for men. Taken together, we expect the following:

Hypothesis 3: The association between marital quality and cardiovascular risk, as well as its age-graded pattern, is stronger for women than for men.

Potential Reversal Relationship between Marital Quality and Cardiovascular Risk among Older Adults

Although many scholars emphasize the fact that different levels of marital quality produce different health outcomes (this is also the primary conceptual framework adopted by the present study), it is likely that poor health increases negative marital quality and that good health promotes positive martial quality (Galinsky and Waite forthcoming). This is because spouses with better health may have more energy to provide both emotional and economic support to each other and to carry out their respective roles (Ivaniuk et al. 2014). In contrast, poor health is often associated with reduced energy and less time spent with other family members. Additionally, having a sick spouse is often accompanied by increased economic, physical, and emotional burdens on the healthy spouse (Galinsky and Waite forthcoming). This may increase the psychological distress of the spouse who serves as the primary caregiver. Stress associated with the long-term care-giving role may reduce both the quantity and the quality of the communication and interactions between spouses, and this in turn may lead to a strained marital relationship (Yorgason, Booth, and Johnson 2008).

Although the reverse causality described here is plausible and also documented in some previous studies (e.g., Booth and Johnson 1994), a more recent national longitudinal study conducted by Umberson et al. (2006) investigated the temporal ordering of marital quality and health following 1,049 respondents over an eight-year period and concluded: "[T]he present data suggest a stronger association of marital quality with subsequent health than vice versa." (p. 13). This conclusion echoed another longitudinal study conducted even earlier among an Iowa sample (Wickrama et al. 1997). In addition, other previous studies suggest that marital quality tends to decline with age but that the rate of decline tends to slow over time (Umberson et al. 2005). Although some studies suggest that there might be a modest increase in marital quality at very old ages, researchers tend to agree that marital quality generally remains relatively stable and at a low level among older adults. Therefore, marital quality may be less sensitive to other factors such as health at older ages. In contrast, health change is more rapid at older ages, and there is also substantial biological heterogeneity within the elderly population (Kaplan 1992; Rowe and Kahn 1987). Therefore, the health of older adults may be more likely to vary with marital quality. Taken together, we hypothesize the following:

Hypothesis 4: The association between marital quality and cardiovascular risk is mostly accounted for by the effect of marital quality on cardiovascular risks rather than vice versa.

DATA

We use the first two waves of national longitudinal data, which lie at the foundation of a life course perspective, from the National Social Life, Health and Aging Project (NSHAP). NSHAP, the first national-scale population-based study of health and intimate relationships, was conducted by the National Opinion Research Center (NORC) at the University of Chicago. A nationally representative probability sample of community-dwelling individuals aged 57–85

years was selected from households across the United States and screened in 2004. African Americans, Latinos, men, and those 75–84 years old at the time of screening were over-sampled. All analyses are weighted. We use the survey data analysis commands in Stata (StataCorp 2012) to account for clustering and stratification of the complex sampling design.

The first wave of the NSHAP (Wave 1) included a sample of 3,005 adults aged 57–85 years who were interviewed during 2005 and 2006. Both in-home interviews and lab tests and assays were conducted. Wave 2 consisted of 2,422 Wave 1 respondents who were re-interviewed during 2010–2011. We restricted the analysis to the 1,250 respondents who had remained married and who had been interviewed in both waves. Missing values on key independent variables, including Wave 1 marital quality and Wave 1 cardiovascular risk, were deleted in a list-wise fashion. Thus, we obtained a total sample of 1,198 respondents (459 women and 739 men). In the final models, we further excluded missing values on the specific dependent variable analyzed (i.e., specific cardiovascular risk outcomes at Wave 2 or marital quality at Wave 2). The final analyzed sample size varies across dependent variables.

MEASURES

Cardiovascular Risk Outcomes. We include four measures of cardiovascular risk: hypertension, rapid heart rate, C-reactive protein (CRP), and general CVD events.

Hypertension is a long-demonstrated risk factor for CVD. Elevated blood pressure is associated with an increased risk of hypertensive heart disease, stroke, heart attack, and heart failure (Izzo and Black 2003). Hypertension increases the pressure on blood vessels and the heart and can lead to atherosclerosis and clogging, narrowing, and damaging of the blood vessels, all of which create risks for many CVDs (Izzo and Black 2003). To measure hypertension, we combine both the biological and self-reported measures collected by the NSHAP (Cornwell and

Waite 2012). The NSHAP measured twice the blood pressure of each respondent by using a LifeSource digital blood pressure monitor (model UA-767PVL). Hypertension is identified when the mean of the two readings is greater than 140 mm Hg systolic or 90 mm Hg diastolic. For respondents who report to have been diagnosed with diabetes, we use lower cutoffs of either 130 mm Hg systolic or 90 mm Hg diastolic for hypertension (National Heart, Lung, and Blood Institute 2003). In addition, respondents were asked whether they had ever been told by any medical doctors that they had high blood pressure or hypertension. Based on this self-reported information, along with the biological measures of blood pressure, we categorize respondents into four groups: (1) normal blood pressure reading and no reported hypertension (referred as "normal" blood pressure group in the remainder of the article), (2) normal blood pressure reading but reported hypertension (referred as "controlled" hypertensive group), (3) high blood pressure reading but no reported hypertension (referred as "undiagnosed" hypertensive group), and (4) high blood pressure reading and reported hypertension (referred as "uncontrolled" hypertensive group). The normal blood pressure group is the reference group.

The second measure of cardiovascular risk is *rapid heart rate*. Heart rate indicates the number of times the heart beats per minute. When the heart cannot effectively deliver blood and oxygen to meet the need of human body, it will beat abnormally fast. A long-term elevated resting heart rate greater than 80 beats per minute is linked to a significant risk of hypertension, heart disease, and mortality (Palatini 2011). Heart rate was measured twice for the NSHAP respondents. Rapid heart rate (coded as 1) is identified if the mean reading is greater than 80 beats per minute, and all others are identified as normal heart rate (coded as 0).

The third measure of cardiovascular risk is *C-reactive protein* (CRP). CRP is often used as a marker for systemic inflammation, and it has emerged as an important predictor of the future

onset of CVD (Nallanathan et al. 2008; Ridker 2003). Ridker (2003) suggests that CRP is a stronger predictor of cardiovascular events than are other traditional markers, such as low-density-lipoprotein cholesterol. During the home interviews, blood was obtained via a single finger-stick using a retractable-tip, single-use disposable lancet and then applied to filter paper. The filter paper was allowed to dry for the remainder of the interview before being placed in a plastic bag with desiccant for transport and storage. High-sensitivity CRP (mg/L) was derived from the dried blood. Details about the procedures of NSHAP dried blood spot CRP measurement are described by Nallanathan et al. 2008. We follow the recommendations of the CDC with respect to classification of cardiovascular risk when interpreting CRP values: low and normal risks exist when CRP <= 3.0 mg/L (reference), and high risk exists when CRP > 3.0 (Pearson et al. 2003). Observations in which CRP > 10 mg/L are excluded from the analysis because of the apparent presence of acute, active infection (Pearson et al. 2003). Because the blood spot was only randomly collected for 5/6 of the total NSHAP sample, we flag those respondents whose blood sample was not collected, and thus the CRP measure is not available at Wave 1. Respondents whose blood sample was not collected at Wave 2 are excluded from the analysis for CRP.

Finally, we include a measure for cardiovascular outcomes using self-reported *general CVD events*. During the home interviews, all NSHAP respondents were asked whether they had ever been told by a medical doctor that they had a heart attack, heart failure, or stroke. Heart attack, heart failure, and stroke are the most common forms of CVD among older adults. Respondents who reported any of these CVD events are coded as 1, and others are coded as 0.

Marital Quality. Family scholars have long recognized different dimensions of marital quality (Umberson et al. 2006). To measure marital quality, we follow the methods that Galinsky

and Waite (forthcoming) developed to calculate marital quality scales using the NSHAP data. Our measures of marital quality are composed of nine items, which are recoded in order to obtain consistent response categories across all items. First, respondents were asked how close they felt their relationship with the spouse was (item 1). Responses range from (1) not very close or somewhat close, (2) very close, to (3) extremely close. Respondents were also asked how happy they were in their spousal relationship (item 2: (1) very unhappy, to (7) very happy) and how emotionally satisfied they feel with their spousal relationship (item 3: (0) not at all, to (4) extremely). Because these two items (i.e., items 2 and 3) were highly skewed, we collapsed the categories. For relationship happiness we collapsed the values to: 1 = Unhappy (1, 2, 3, 4), 2 = Happy (5, 6), and 3 = Very Happy (7). For emotional satisfaction we collapsed the values to 1 = Not Satisfied (0, 1, 2), 2 = Satisfied (3), and 3 = Very Satisfied (4) (see Galinsky and Waite forthcoming, Warner and Kelley-Moore 2012).

Additionally, respondents were asked whether they preferred to spend their free time doing things with their spouse or apart from them (item 4). Responses ranged from (1) mostly together, (2) some together and some apart, to (3) mostly apart. We reversely recoded this item so that higher values indicate better marital quality. Finally, respondents were asked four questions about their spouse: how often they could open up to the spouse if they needed to talk about their worries (item 5), how often they could rely on the spouse for help if they had a problem (item 6), how often the spouse made too many demands on them (item 7), and how often the spouse criticized them (item 8). In Wave 2, NHSAP added an additional question: "How often does spouse gets on your nerves?" (item 9, not available in Wave 1). Responses to each question (items 5–9) are (1) never, hardly ever, or rarely, (2) some of the time, and (3) often.

Results from exploratory factor analyses suggest that these nine items form two different dimensions, which we refer to as positive and negative marital quality, respectively (Galinsky and Waite forthcoming; Warner and Kelley-Moore 2012). We create two factor scores for positive and negative marital quality based on the iterated principle factor method and an oblique rotation. To make full use of the marital quality information offered by the measures available in NSHAP, we measure marital quality by using all available items in each wave. Our additional analysis (not shown but available upon request) using only the items shared in both waves suggested similar results as we report here. However, when we consider change in marital quality between waves, we use only the items available in both waves in order to make them comparable.[1] Table 1 shows the factor loadings of each item used to generate the factor scores for positive and negative marital quality, respectively.

Table 1 about here

Other Covariates. We include three types of covariates (all measured at Wave 1) that are related to both marital quality and cardiovascular risks: socio-demographic covariates, health-behavior-related covariates, and psychological distress.

Socio-Demographic Covariates. Age is categorized into three groups: aged 57–64 years (young-old, reference), aged 65–74 years (middle-old), and aged 75–85 years (old-old). *Race-ethnicity* includes non-Hispanic white (reference), non-Hispanic black, Hispanic, and other. *Education* is grouped into four categories: no diploma (reference), high school graduate, some college, and college graduate. *Family income* is derived from the question that asked respondents to self-assess their family income levels compared with other American families. Responses range from below average (reference), average, to above average. There are 182 (out of 1,198,

the total analyzed sample) respondents have missing reports on family income. We include them as a separate missing category for family income.

Health-Behavior-Related Covariates. Because respondents may take medications for hypertension, we include an indicator for *taking any antihypertensive medicine* (1 = Yes, 0 = No). We also control for obesity as indexed by body mass, because obesity has been identified as a significant predictor of CVD (Poirier et al. 2006). The Body Mass Index (BMI) is calculated based on the measured height and weight of the respondents and then grouped into four categories: normal or underweight (BMI < 25), overweight (25 <= BMI < 30), obese (30 <= BMI < 40), and morbidly obese (BMI >= 40). There are 53 (out of 1,198, the total analyzed sample) missing cases on BMI that are imputed with the mean. Additional analysis (not shown) suggested that excluding the missing cases on BMI did not reveal different results from what we report here. In addition, we control indicators for *currently smoke* (1 = Yes, 0 = No), *currently drink alcohol* (1 = Yes, 0 = No), and *physical exercise* (1= vigorous physical activity or exercise three times or more per week, 0 = others).

Psychological Distress. Finally, we control for *depression*, which is measured by an 11-question subset of the Center for Epidemiological Studies Depression Scale (CES-D). Respondents were asked how often in the past week they experienced any of the following: (1) I did not feel like eating; (2) I felt depressed; (3) I felt that everything I did was an effort; (4) My sleep was restless; (5) I was happy; (6) I felt lonely; (7) People were unfriendly; (8) I enjoyed life; (9) I felt sad; (10) I felt that people disliked me; and (11) I could not get "going". Response categories ranges from (0), rarely or none of the time, to (3), most of the time. The items are recoded such that higher values indicate higher levels of depression. The final scale is the sum of the 11 scores. There are 13 (out of 1,198, the total analyzed sample) missing cases on depression,

and we impute them with the mean. Tables 2–3 show the weighted descriptive statistics of all analyzed variables for women and men separately.

Tables 2 and 3 about here

ANALYTIC APPROACH

We conduct four sets of analyses for the four cardiovascular risk outcomes separately. We use the lagged dependent variable approach to analyze the two waves of data. Specifically, we use Wave 1 marital quality to predict Wave 2 cardiovascular risks controlling for Wave 1 cardiovascular risks along with all other covariates. We also include change in marital quality between Waves 1 and 2 as a predictor of cardiovascular risk at Wave 2 if its effect is significant. In the case that the effect of change in marital quality between Waves 1 and 2 was not significant in the preliminary analysis, we did not include it in the final reported models for the purpose of model parsimony. The specific models we estimate vary depending on the measures of the cardiovascular risk outcomes. We estimate multinomial logistic regression models to predict hypertension, and we estimate binary logistic regression models to predict rapid heart rate, high CRP, and general CVD events. Because the longstanding literature on marriage and health emphasizes the fundamental differences between men and women, we stratify all analyses by gender. Our additional analyses (not shown but available upon request), which included gender interaction terms, revealed significant gender differences in the relationships between marital quality and cardiovascular risk outcomes.

For both men and women, we estimate a sequence of models. In the first model, we examine the general relationship between marital quality and cardiovascular risk controlling for only the basic socio-demographic covariates. Next, we add health-behavior-related covariates and psychological distress separately as additional covariates to understand whether such factors

explain the relationship between marital quality and cardiovascular risks. Because our preliminary results (not shown but available upon request) suggested that adding health-behavior-related covariates and psychological distress did not significantly change the results, we only report the final full model in which all covariates are controlled—including socio-demographic, health-behavior-related covariates and psychological distress. In order to assess potential age differences in the relationship between marital quality and cardiovascular risk, we add interaction terms of age by marital quality in the full model.

To assess the possibility of a reversal relationship between marital quality and cardiovascular risk, we estimate Ordinary Least Squares regression models to predict Wave 2 marital quality using Wave 1 cardiovascular risk outcomes controlling for Wave 1 marital quality along with all other covariates. Because positive and negative marital quality are correlated with each other ($r = -0.64$ in Wave 1 and -0.68 in Wave 2), we analyze them in separate models.

Correction for Sample Selection Bias. Because our analyses are restricted to married respondents in both waves, the samples are selective of those with relatively better marital quality. This is because those with worse marital quality are more likely to become divorced or widowed. Moreover, sample attrition between waves that is due to mortality will lead to another source of selection bias because of nonrandom selectivity. We want to emphasize that although our conclusions in the present study may only apply to a selected population of older adults who are healthier, less likely to die, and more likely to stay married, this is a random sample from this segment of the population. Indeed, those who died between waves and those who were not married in both waves were also more likely to suffer higher cardiovascular risk (McFarland et al. 2013). In this sense, our estimates of cardiovascular risk should be seen as conservative.

Moreover, we apply the approach, developed by Heckman (Heckman 1979) to adjust the sample selection biases that are due to selection processes through both marriage and mortality. This approach consists of modeling the probability that a respondent would die between Waves 1 and 2 and modeling the probability that a respondent would remain married at both waves, using logistic regression models, conditional on a set of predictors measured at Wave 1. Then, for individuals who did not die and who remained married at both waves, cardiovascular risk outcomes are modeled as a function of a set of independent variables, including the estimated probabilities of dying and of being married at both waves. Following this Heckman-type correction, estimates on cardiovascular risk should be interpreted as being adjusted for factors that may affect that risk, as well as for the tendency to die and the tendency to remain married. This approach has been well used in previous studies to correct sample selection bias (e.g., Umberson et al. 2006; Liu 2012).

RESULTS

Results that predict hypertension from multinomial logistic regression models and predict rapid heart rate, high CRP, and general CVD events from binary logistic regression models are shown in Tables 4–7. We report results from both the main effects model (Model 1) and the age interaction model (Model 2) with all covariates controlled. Estimated regression coefficients are reported in tables. For interpretation, the relative risk ratios and odds ratios can be derived from the reported coefficients by exponentiation.

Table 4 about here

We first discuss results from Table 4, which shows the estimated regression coefficients for negative marital quality predicting cardiovascular risk outcomes from logistic regression models for women. Results from Model 1 of Table 4 suggest that negative marital quality at

Wave 1 is not significantly associated with any of the Wave 2 cardiovascular risk outcomes among women after controlling for Wave 1 cardiovascular risks and all other covariates. However, an increase in negative marital quality between Wave 1 and Wave 2 is significantly associated with higher risks of controlled and uncontrolled hypertension for women (Model 1 of Table 4). Specifically, for women, the relative risk of controlled and uncontrolled hypertension relative to normal blood pressure increases by 1.68 (i.e., exp(0.52)) and 1.57 times, respectively, for every one unit increase of negative marital quality between Waves 1 and 2. An increase in negative marital quality between Waves 1 and 2 is also related to higher odds of having high risk CRP levels for women ($b = 0.52$, $p < 0.05$). Moreover, the relationship between Wave 1 negative marital quality and Wave 2 cardiovascular risks for women is contingent upon age, as is indicated by the significant age interaction effects in Model 2 of Table 4. Results from Model 2 of Table 4 suggest that for women aged 57–65 years, negative marital quality at Wave 1 is not significantly associated with any of the cardiovascular risk outcomes at Wave 2. The associations of negative marital quality with controlled hypertension, uncontrolled hypertension, high CRP and CVD events are not significantly different between women aged 65–75 years and their younger counterparts (i.e., women aged 57–65). However, in comparison with women aged 57–65 years, for women aged 65–74 years, higher levels of negative marital quality are more likely to be related to both lower risks of undiagnosed hypertension ($b = -0.05$-1.25) and lower odds of rapid heart rate ($b = 0.20$-1.97). More importantly, we see a consistent and strong age pattern in the relationship between negative marital quality and cardiovascular risks among women at the oldest age group (i.e., aged 75–85 years), as is indicated by the significant positive age interaction effects with negative marital quality at Wave 1 in Model 2 of Table 4. Results from Model 2 of Table 4 suggest that in comparison with women aged 57–65 years, women aged 75–

85 years are more likely to have a positive association between negative marital quality and cardiovascular risk, especially in terms of hypertension, rapid heart rate, and CVD events. Specifically, among women aged 75–85 years, for each one unit higher of negative marital quality at Wave 1, the relative risk of controlled, undiagnosed, and uncontrolled hypertension increases by about 13.74 (i.e., exp(−0.12 + 2.74)), 9.49, and 9.87 times, respectively; and the odds of rapid heart rate and CVD events increase by 3.46 and 9.39 times, respectively. Our additional analyses suggest that these relationships among women aged 75–85 years are all significant ($p < 0.05$). Although the age interaction effects with negative marital quality at Wave 1 are not statistically significant when predicting odds of high CRP, the direction of the interaction effect of ages 75–85 years by negative marital quality at Wave 1 ($b = 0.13$) is consistent with the pattern for other cardiovascular outcomes.

Table 5 about here

Table 5 shows the results for positive marital quality that predicts cardiovascular risk outcomes from logistic regression models for women. Results from Model 1 of Table 5 suggest that positive marital quality at Wave 1 is not significantly related to any of the cardiovascular risk outcomes at Wave 2 for women. However, this relationship varies by age, in particular for hypertension and CVD events. Results from Model 2 of Table 5 suggest that positive marital quality at Wave 1 is not significantly associated with the risks of hypertension or CVD events at Wave 2 among women aged 57–64 years, and this pattern does not vary among women aged 65–75 years. However, among women aged 75–85 years, positive marital quality at Wave 1 is negatively associated with hypertension and CVD events by Wave 2. Specifically, among women aged 75–85 years, for every one unit higher in positive marital quality at Wave 1, the relative risk of controlled, undiagnosed, and uncontrolled hypertension at Wave 2 decreases by

about 67.37 (i.e., (1-exp(0.08-1.20))X100), 70.48, and 74.84 percent, respectively; and the odds of reporting any CVD events at Wave 2 decrease by 52.29 percent. Moreover, an increase of positive marital quality between Waves 1 and 2 tends to be associated with lower odds of CVD events at Wave 2 among women aged 75–85 years (b = 1.35-2.10), although this relationship does not appear among women aged 57–64 years or 65–74 years (Model 2). Our additional analyses suggest that these relationships among women aged 75–85 years are all significant ($p < 0.05$).

Table 6 about here

Next we discuss the results for men. Table 6 shows the results for negative marital quality that predicts cardiovascular risk outcomes from logistic regression models for men. Results from Model 1 of Table 6 suggest that an increase in negative marital quality between Waves 1 and 2 is related to a lower relative risk of controlled hypertension at Wave 2 ($b = -0.57$, $p < 0.05$). However, this relationship tends to decrease among men at older ages (Model 2 of Table 6). Indeed, our additional analyses suggest that a change in negative marital quality between Waves 1 and 2 is not significantly ($p > 0.05$) associated with hypertension at Wave 2 among men aged 65–74 years or 75–85 years. Results from Table 6 also suggest that men with higher levels of negative martial quality at Wave 1 tend to have higher odds of high CRP ($b = 0.33$, $p < 0.05$, Model 1), and this relationship does not vary by age (Model 2). Negative marital quality is not significantly related to the odds of reporting either general CVD events or rapid heart rate for men at all older ages.

Table 7 about here

Table 7 shows results for positive marital quality that predict cardiovascular risk outcomes among men. These results suggest that positive marital quality is basically not related

to cardiovascular risk outcomes for men, with only one exception: an increase in positive marital quality between Waves 1 and 2 is more likely to relate to lower odds of having high CRP at Wave 2 among men aged 75–85 years than among men aged 57–64 years, as is indicated by the significant age interaction effect for CRP in Model 2. However, our additional analysis (not shown but available upon request) suggested that this relationship for men aged 75–85 years (b = 0.39-1.09) was not statistically significant.

Table 8 about here

Cardiovascular risks may also affect later marital quality. To assess this possibility, we use cardiovascular risks at Wave 1 to predict marital quality at Wave 2 controlling for Wave 1 marital quality and other covariates. We present these results in Table 8. Because the age interaction effects are basically not significant, we only present the main effects models in Table 8. Results from Table 8 suggest that both heart rate and CVD events at Wave 1 are not significant predictors of marital quality at Wave 2. However, both controlled hypertension and CRP at Wave 1 are significantly related to marital quality at Wave 2, and the patterns vary by gender. Men who had controlled hypertension at Wave 1 have lower levels of negative marital quality at Wave 2 than do men with normal blood pressure at Wave 1 ($b = -0.22$, $p < 0.05$). In contrast, women who had controlled hypertension at Wave 1 have lower levels of positive marital quality at Wave 2 than do women with normal blood pressure at Wave 1 ($b = -0.18$, $p < 0.05$). Additionally, women with high CRP at Wave 1 tend to report higher levels of negative marital quality ($b = 0.28$, $p < 0.01$) and lower levels of positive marital quality ($b = -0.23$, $p < 0.05$) at Wave 2 than do women with low or normal CRP at Wave 1.

DISCUSSION

While marriage has long been argued to promote health (Robles and Kiecolt-Glaser 2003; Waite and Gallagher 2000), it is not the case that any marriage is better than none in terms of health benefits (Williams 2003). This study highlights the importance of marital quality in relation to cardiovascular risk—the central mechanism in the pathogenesis of many acute and chronic critical conditions—among older adults in the United States. Our current scientific understanding about this relationship comes primarily from laboratory-based clinical studies. For the first time, we assess cardiovascular risk associated with marital quality among older adults using population-based, nationally representative longitudinal data. We further extend this line of literature by highlighting the importance of age along with gender in moderating these relationships. Below, we outline our major findings and implications from this study in relation to each research hypothesis.

Marital Quality and Cardiovascular Risk. A life course approach emphasizes the importance of life context when considering the links between health and the social world (Dannefer 2003). Marital quality is one of the key factors that define life course contexts. We consider both positive and negative aspects of marital quality, which intervene in life contexts in different ways. Based on previous lab-based clinical evidence, we hypothesize that negative marital quality will be positively related to cardiovascular risk while positive marital quality will be modestly and negatively related to cardiovascular risk *at the population level* (Hypothesis 1). Our results support this hypothesis, and we find evidence for the association between marital quality and each examined cardiovascular risk outcome. Although many of these relationships are only present among the oldest age group, we do find some general patterns across all ages. For example, we find that for women at all older ages, an increase in negative marital quality leads to higher risks of hypertension. Previous literature suggests that marital quality is relatively

low but stable at older ages (Umberson et al. 2005). It may be that women experience low, but relatively stable, marital quality during their later lives. Therefore, in the case of a fluctuation in the quality of a marital relationship (especially in a negative direction), a woman's emotions may become very sensitive to the change, and this may provoke a reaction in her body, particularly in her cardiovascular system.

Additionally, we find that negative martial quality tends to increase the risk of high CRP for both men and women at all older ages. This finding is different from that of a recent study, which found minimal evidence for the association between negative marital quality and CRP levels for both men and women (Donoho et al. 2013). The difference may be due to different measures of CRP, as well as to different age ranges of the study samples. We want to emphasize that our results are indeed more consistent with previous clinical studies that suggest a stronger effect of negative marital quality than positive marital quality in affecting health (Ewart et al. 1991; Robles and Kiecolt-Glaser 2003).

Moreover, although we find associations of both negative and positive marital quality with cardiovascular risk (especially at older ages), negative marital quality is associated with more types of cardiovascular risk than is positive marital quality. This is consistent with previous clinical evidence that suggests that a strained martial relationship is more profound in affecting cardiovascular risk than a happy marriage (Robles and Kiecolt-Glaser 2003). Family and health researchers argue that both the support and stress processes related to marriage produce health outcomes (Burman and Margolin 1992). Our results suggest that the stress process related to marriage may be more important than the supportive aspect of marriage in shaping individuals' cardiovascular health.

Age. Age has been identified as the strongest predictor of cardiovascular risk (Izzo and Black 2003; Sidney et al. 2013). Family scholars also emphasize that marital quality fluctuates with age (Umberson et al. 2005). Surprisingly, none of previous studies has considered whether the relationship between marital quality and cardiovascular risk varies by age. The life course theory and cumulative advantage/disadvantage perspective lay a solid theoretical foundation for us to expect that marital quality would take a toll on individuals' health and that the association between marital quality and cardiovascular risk would be stronger at older ages (Hypothesis 2). Consistent with this hypothesis, we find that both the detrimental effect of negative marital quality and the protective effect of positive marital quality on the cardiovascular system are more apparent among the oldest group (especially for women) than among their younger counterparts. This age pattern is very strong and appears across all of the cardiovascular risk outcomes examined. These results fit with the cumulative advantage/disadvantage argument and suggest that the advantages and disadvantages associated with marriage may have greater impacts on individuals' health as they get older. It is likely that social networks and contacts with friends tend to decrease at later life course (Turner and Marino 1994). Therefore, the marital relationship becomes increasingly important for life at very old ages. Additionally, the development of chronic diseases is a long-term process. Therefore, the effects of marital quality may be strongest at the oldest ages because of the long time required in developing CVDs. Younger men and women are being stressed by negative marital quality, but the effects on their hearts may take longer to emerge. Moreover, aging is a process often accompanied by a decline of the immune system and an increase in frailty. This suggests that older individuals, especially those at very old ages, may be more vulnerable to a negative relationship (Uchino, Kiecolt-Glaser and Glaser 2000) and that negative marital quality may stimulate more cardiovascular responses at very old

ages as a result of changes in physical fitness, body fat percentage, and health status (e.g., the onset of illness). The determination of which specific mechanisms work to foster the relationship between marital quality and cardiovascular risk at very old ages is a subject that clearly warrants future research attention.

Gender. Our results also point to important gender variations in the processes that link marital quality with cardiovascular risk. Consistent with our hypothesis on gender (Hypothesis 3), we find that the expected association between marital quality and cardiovascular risk is primarily present among women; it is less so among men. This finding is in line with previous clinical literature that suggests that marital strain tends to evoke greater and more persistent physiological and cardiovascular changes in women than in men. A long sociological tenet is that marriage promotes the health of men more than of women. However, conversely, a handful of clinical studies suggest that marital quality tends to be more pertinent to women's health than to men's. Using a nationally representative longitudinal data set, our study adds further evidence to the existing mixed literature on gender difference. Our results suggest that in particular, with respect to cardiovascular health, marital quality seems to be more important for women than for men. It may be that women are more likely to internalize their emotions and feelings about marital strain and thus are more likely to feel depressed than are men (Rosenfield 1999). Depression associated with marital strain may impose a variety of health threats by altering cardiovascular function for women (Everson-Rose and Lewis 2005; Graham et al. 2006; Kiecolt-Glaser and Newton 2001). However, our results suggest that the association between negative marital quality and cardiovascular risk for women, especially among the oldest group, remains very robust even after depression is controlled. Physiological adjustment to social relationship is different for men and women (Yang et al. 2013). Future research should explore whether gender

differences in physiological responses to stress (such as hormone levels, metabolic system, and immune function) may explain the identified gender differences in the association between martial quality and cardiovascular risk.

Reversal Relationships. Although we use a causal framework to build our hypotheses on how marital quality affects cardiovascular risk, it is also likely that cardiovascular risk shapes marital quality. Given that marital quality is relatively more stable and cardiovascular function is more likely to fluctuate among older adults, we expected that the association between marital quality and cardiovascular risk would be mostly driven by the effect of marital quality on cardiovascular risk rather than vice versa (Hypothesis 4). Our results reveal some mixed evidence on this hypothesis, depending on gender and specific cardiovascular risk outcomes. Consistent with this hypothesis, our results on heart rate and CVD events suggest that marital quality may affect heart rate and CVD events, especially for women at older ages, but that experiences of rapid heart rate and CVD events seem to have little impact on later changes of marital quality. The results on hypertension and CRP vary by gender. For men, we find a *negative reversal* process: men who had controlled hypertension at Wave 1 actually experience lower levels of negative marital quality at Wave 2 than do men whose blood pressure was normal at Wave 1. This result does not vary by age. This finding is unexpected from our hypotheses. We suspect that this may be related to our measure of controlled hypertension. On one hand, the controlled hypertension reflects a higher cardiovascular risk in comparison with normal blood pressure; on the other hand, it also reflects a more regular use of medicine and the adoption of a routine life style in order to control the high blood pressure. It is likely that for men with controlled hypertension, wives (who usually take the role of caregiver) are more cautious about

their husbands' health problems and therefore try to avoid conflicts in the marriage. In this sense, such men may experience lower levels of negative martial quality.

For women, we find some evidence for a potential reversal process ongoing between marital quality and cardiovascular risk. We find that uncontrolled hypertension and high CRP are both significant predictors of subsequent poor marital quality; at the same time, marital quality also affects the subsequent risk of hypertension (especially among older women) and high CRP. This reversal process among women is consistent with the long-standing observation that husbands are less likely than wives to provide support and healthcare to a sick spouse. Husbands are also less likely than wives to take the initiative to maintain a good marital relationship with a sick spouse (Iveniuk et al. 2014). In this way, a wife's poor health may affect her subjective assessment of the marital relationship.

Limitations. Several study limitations should be considered. First, our study is based on two waves of longitudinal data. Although we attempt to tease out some selectivity issues and a causal relationship, we are not fully able to deal with such issues with the current data. To fully explore how different selection and causal processes contribute to the links between marital quality and cardiovascular risk, future studies should employ longitudinal data with more waves of follow-up. The NSHAP is currently collecting the third wave of data, which will provide opportunities for researchers to further untangle the causality issues. Second, our samples are restricted to respondents who survived and were married in both waves. Therefore, conclusions in the present study may only apply to a selected population of older adults—those who are both healthier and more likely to stay married. Third, cardiovascular function has multiple dimensions, and future studies should consider a broader range of cardiovascular risk measures. Finally, various social, biological, psychological, and behavioral mechanisms work together to

determine the association between marital quality and cardiovascular risk. Future studies should seek to identify the precise mechanisms and processes through which marital quality and cardiovascular risk are linked, and try to address how those mechanisms and processes vary across gender and age.

CONCLUSION

Leading scientists have established a strong linkage between social relationships and health among the elderly population (Waite, Iveniuk and Laumann, forthcoming). Much of this work considers different types of social relationships and highlights marital relationships as holding the greatest significance for health in older adulthood (Robles and Kiecolt-Glaser 2003; Umberson et al. 2006; Waite and Gallagher 2000). This study extends current clinical evidence on the importance of marital quality in relation to cardiovascular health by using a nationally representative longitudinal data set. More importantly, results from this study demonstrate that the effect of marital quality on cardiovascular risk is highly contingent upon age; previous studies, mostly clinically based, either ignored age or controlled for it as a covariate. Indeed, we find that marital quality becomes increasingly important in shaping cardiovascular risk at older ages. Our results further suggest that gender as a fundamental factor to define social context is a significant moderator in this relationship and that women's cardiovascular health is more sensitive to marital quality than is men's. Given the fact that CVD continues to be the leading cause of death among the elderly population, implementation of public policies and programs designed to promote marital quality should also reduce the risks of CVD and thus promote longevity, especially for women at very old ages. Finally, this study highlights the importance of further applying an interdisciplinary approach to advance our understanding of the interactions of social and biological processes, as they affect cardiovascular health.

[1] We list-wise deleted all missing values on Wave 1 marital quality. There are 44 cases (out of the total sample of 1,198) with missing data on change of marital quality between Waves 1 and 2 that is due to missing values on marital quality of Wave 2. Because in the final models we only include change of marital quality to predict cardiovascular risks if the effect is significant, we retain the 44 missing cases in our study sample to improve the statistical power, and we impute them with the mean. Additional analysis excluding those missing cases suggested similar results as we reported in the paper, although statistical power was reduced because of the smaller sample size.

REFERENCES

Booth, Alan and David R. Johnson. 1994. "Declining Health and Marital Quality." *Journal of Marriage and the Family* 56(1):218-23.

Burman, Bonnie and Gayla Margolin. 1992. "Analysis of the Association between Marital Relationships and Health Problems: An Interactional Perspective." *Psychological Bulletin* 112(1):39–63.

Carr, Deborah and Kristen W. Springer. 2010. "Advances in Families and Health Research in the 21st Century." *Journal of Marriage and Family* 72(3):743–61.

Cornwell, Erin York and Linda J. Waite. 2012. "Social Network Resources and Management of Hypertension." *Journal of Health and Social Behavior* 53(2):215-31.

Coyne, James C., Michael J. Rohrbaugh, Varda Shoham, John S. Sonnega, John M. Nicklas, and James A. Cranford. 2001. "Prognostic Importance of Marital Quality for Survival of Congestive Heart Failure." *The American Journal of Cardiology* 88(5):526–9.

Dannefer, Dale. 2003. "Cumulative Advantage/Disadvantage, and the Life Course: Cross-Fertilizing Age and Social Science Theory". *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences* 58(6):S327–37.

Donoho, Carrie J., Eileen M. Crimmins, and Teresa E. Seeman. 2013. "Marital Quality, Gender, and Markers of Inflammation in the MIDUS Cohort." *Journal of Marriage and Family* 75(1):127-41.

Elder, Glen H., Jr., and Angela M. O'Rand. 1995. "Adult Lives in a Changing Society." Pp. 452–75 in *Sociological Perspectives on Social Psychology*, edited by K. S. Cook, G. A. Fine, and J. S. House. Needham Heights, MA: Allyn and Bacon.

Everson-Rose, Susan A. and Tené T. Lewis. 2005. "Psychosocial Factors and Cardiovascular Diseases." *Annual Review of Public Health* 26:469–500.

Ewart, Craig K., Barr C. Taylor, Helena C. Kraemer, and Stewart W. Agras. 1991. "High Blood Pressure and Marital Discord: Not Being Nasty Matters More than Being Nice." *Health Psychology* 10(3):155–63.

Galinsky, Adena M. and Linda J. Waite. Forthcoming. "Sexual Activity and Psychological Health as Mediators of the Relationship Between Physical Health and Marital Quality." *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences*.

Gallo, Linda C., Wendy M. Troxel, Karen A. Matthews, and Lewis H. Kuler. 2003. "Marital Status and Quality in Middle-Aged Women: Associations with Levels and Trajectories of Cardiovascular Risk Factors." *Health Psychology* 22(5):453–63.

Graham, Jennifer E., Lisa M. Christian, and Janice K. Kiecolt-Glaser. 2006. "Marriage, Health, and Immune Function." Pp. 61–76 in *Relational Processes and DSM-V: Neuroscience, Assessment, Prevention, and Treatment*, Vol. 11, edited by S. R. H. Beach, M. Z. Wamboldt, N. J. Kaslow, R. E. Heyman, M. B. First, J. G. Underwood, and D. Reiss. Arlington, VA: American Psychiatric Publishing, Inc.

Heckman, James J. 1979. "Sample Selection Bias as a Specification Error." *Econometrica* 47(1):153–61.

Iveniuk, James, Linda J. Waite, Edward O. Laumann, Martha K. McClintock, and Andrew D. Tiedt. 2014. "Marital Conflict in Older Couples: Positivity, Personality and Health." *Journal of Marriage and Family* 76(1):130–44.

Izzo, Joseph L., Jr. and Henry R. Black, eds. 2003. *Hypertension Primer: The Essentials of High Blood Pressure*. 3rd ed. Baltimore: Lippincott Williams & Wilkins.

Kaplan, George. A. 1992. "Health and Aging in the Alameda County Study." Pp. 69–88 in *Aging, Health Behaviors, and Health Outcomes*, edited by K.W. Schaie, D. G. Blazer and J.S. House. Hillsdale, NJ: Lawrence Erlbaum Associates.

Kiecolt-Glaser, Janice K. and Tamara L. Newton. 2001. "Marriage and Health: His and Hers." *Psychological Bulletin* 1274):472–503.

Lakatta, Edward.G. and Daniel Levy. 2003. "Arterial and Cardiac Aging: Major Shareholders in Cardiovascular Disease Enterprises: Part I: Aging Arteries: A "Set Up" for Vascular Disease" *Circulation* 107(1):139–46.

Liu, Hui. 2012. "Marital Dissolution and Self-Rated Health: Age Trajectories and Birth Cohort Variations." *Social Science & Medicine* 74(7):1107–16.

Malarkey, William B., Janice K. Kiecolt-Glaser, Dennis Pearl, and Ronald Glaser. 1994. "Hostile Behavior during Marital Conflict Alters Pituitary and Adrenal Hormones." *Psychosomatic Medicine* 56(1):41–51.

McFarland, Michael J., Mark D. Hayward, and Dustin Brown. 2013. "I've Got You under My Skin: Marital Biography and Biological Risk." *Journal of Marriage and Family* 75(2):363–80.

Nallanathan, Bhairavi, Thomas McDade, Sharon Williams, and Stacy Tessler Lindau. 2008. "Dried Blood Spot Measurement of C-reactive Protein in Wave I of the National Social Life, Health & Aging Project (NSHAP)." NORC and the University of Chicago. (http://biomarkers.uchicago.edu/pdfs/TR-CRP.pdf).

National Academy on an Aging Society. 2000. "Hypertension: A Common Condition for Older Americans." In Challenges for the 21st Century: Chronic and Disabling Conditions.

National Heart, Lung, and Blood Institute. 2003. "The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure." Bethesda, MD: U.S. Department of Health and Human Services.

Orth-Gomér, Kristina, Sarah P. Wamala, Myriam Horsten, Karin Schenck-Gustafsson, Neil Schneiderman, and Muarry A. Mittleman. 2000. "Marital Stress Worsens Prognosis in Women with Coronary Heart Disease." *Journal of the American Medical Association* 284(23):3008–14.

Ostchega, Yechiam, Charles F. Dillon, Jeffery P. Hughes, Margaret Carroll, and Sarah Yoon. 2007. "Trends in Hypertension Prevalence, Awareness, Treatment, and Control in Older U.S. Adults: Data from the National Health and Nutrition Examination Survey 1988 to 2004." *Journal of the American Geriatrics Society* 55(7):1056–65.

Palatini, Paolo. 2011. "Role of Elevated Heart Rate in the Development of Cardiovascular Disease in Hypertension." *Hypertension* 58(5):745–750.

Pearson, Thomas A., George A. Mensah, R. Wayne Alexander, Jeffrey L. Anderson, Richard O. Cannon III, Michael Criqui, Yazid Y. Fadl, Stephen P. Fortmann, Yuling Hong, Gary L. Myers, Nader Rifai, Sidney C. Smith, Jr., Kathryn Taubert, Russell P. Tracy, and Frank Vinicor. 2003. "Markers of Inflammation and Cardiovascular Disease: Application to Clinical and Public Health Practice: A Statement for Healthcare Professionals from the Centers for Disease Control and Prevention and the American Heart Association." *Circulation* 107(3):499–511.

Pinnelli, Antonella, Filomena Racioppi, and Rosella Rettaroli, eds. 2007. *Genders in the Life Course: Demographic Issues*. Dordrecht: Springer.

Poirier, Paul, Thomas D. Giles, George A. Bray, Yuling Hong, Judith S. Stern, F. Xavier Pi-Sunyer, and Robert H. Eckel, 2006. "Obesity and Cardiovascular Disease: Pathophysiology, Evaluation, and Effect of Weight Loss: An Update of the 1997 American Heart Association Scientific Statement on Obesity and Heart Disease from the Obesity Committee of the Council on Nutrition, Physical Activity, and Metabolism." *Circulation* 113(6):898–918.

Ridker, Paul M. 2003. "Clinical Application of C-reactive Protein for Cardiovascular Disease Detection and Prevention." *Circulation* 107(3):363–69.

Robles, Theodore F. and Janice K. Kiecolt-Glaser. 2003. "The Physiology of Marriage: Pathways to Health." *Physiology & Behavior* 79(3):409–16.

Rosenfield, Sarah. 1999. "Gender and Mental Health: Do Women Have more Psychopathology, Men More, or Both the Same (and Why)?" Pp. 348–60 in *A Handbook for the Study of Mental Health: Social Contexts, Theories, and Systems,* edited by A. V. Horwitz and T. L. Scheid. New York: Cambridge University Press.

Rowe, John W. and Robert L. Kahn. 1987. "Human Aging: Usual and Successful." *Science* 237(4811):143–9.

Schiller, Jeannine S., Jacqueline W. Lucas, and Jennifer A. Peregoy. 2012. "Summary Health Statistics for U.S. Adults: National Health Interview Survey, 2011." National Vital and Health Statistics Reports, Series 10, No 256. Hyattsville, MD: National Center for Health Statistics.

Settersten, Richard A., Jr. and Karl Ulrich Mayer. 1997. "The Measurement of Age, Age Structuring, and the Life Course." *Annual Review of Sociology* 23:233–61.

Sidney, Stephen, Wayne D. Rosamond, Virginia J. Howard, and Russell V. Luepker, and the National Forum for Heart Disease and Stroke Prevention. 2013. "The 'Heart Disease and Stroke Statistics—2013 Update' and the Need for a National Cardiovascular Surveillance System. *Circulation* 127(1):21–3.

StataCorp LP. 2012. *Stata 10 User's Guide.* College Station, TX: StataCorp LP.

Taylor, Shelley E., Laura Cousino Klein, Brian P. Lewis, Tara L. Gruenewald, Regan A. R. Gurung, and John A . Updegraff. 2000. "Biobehavioral Responses to Stress in Females: Tend-and-Befriend, not Fight-or-Flight." *Psychological Review* 107(3):411–29.

Turner, R. Jay and Franco Marino. 1994. "Social Support and Social Structure: A Descriptive Epidemiology." *Journal of Health and Social Behavior* 35(3):193–212.

Uchino, Bert N., Janice K. Keicolt-Glaser, and Ronald Glaser. 2000. "Psychological Modulation of Cellular Immunity." Pp. 397-424 in *Handbook of Psychophysiology*, 2nd ed, edited by J. T. Cacioppo, L. G. Tassinary, and G. G. Berntson. New York: Cambridge University Press.

Umberson, Debra, Kristi Williams, Daniel A. Powers, and Hui Liu. 2006. "You Make Me Sick: Marital Quality and Health Over the Life Course." *Journal of Health and Social Behavior* 47(1):1–16.

Umberson, Debra, Kristi Williams, Daniel A. Powers, Meichu D. Chen, and Anna M. Campbell. 2005. "As Good As It Gets? A Life Course Perspective on Marital Quality." *Social Forces* 84(1):493–511.

Umberson, Debra, Meichu D. Chen, James S. House, Kristine Hopkins and Ellen Slaten. 1996. "The Effect of Social Relationships on Psychological Well-Being: Are Men and Women Really So Different?" *American Sociological Review* 61(5):836–56.

Waite, Linda J., James Iveniuk, and Edward O. Laumann. Forthcoming. "Social Connectedness at Older Ages, and Implications for Health and Well-Being." In *Interpersonal Relationships and Health: Social and Clinical Psychological Mechanisms*, edited by C.R. Agnew and S. C. South. New York: Oxford University Press.

Waite, Linda J. and Maggie Gallagher. 2000. *The Case for Marriage: Why Married People Are Happier, Healthier, and Better Off Financially*. New York: Doubleday.

Warner, David F. and Jessica Kelley-Moore. 2012. "The Social Context of Disablement among Older Adults: Does Marital Quaity Matter for Loneliness?" *Journal of Health and Social Behavior* 53(1):50–66.

Wickrama, K. A. S., Frederick O. Lorenz, Rand D. Conger, and Glen H. Elder, Jr. 1997. "Marital Quality and Physical Illness: A Latent Growth Curve Analysis." *Journal of Marriage and the Family*59(1):143-55.

Williams, Kristi. 2003. "Has the Future of Marriage Arrived? A Contemporary Examination of Gender, Marriage, and Psychological Well-Being." *Journal of Health and Social Behavior* 44(4):470–87.

Williams, Kristi and Debra Umberson. 2004. "Marital Status, Marital Transitions, and Health: A Gendered Life Course Perspective." *Journal of Health and Social Behavior* 45(1):81–98.

Wu, Xiaoqin and Alfred DeMaris. 1996. "Gender and Marital Status Differences in Depression: The Effects of Chronic Strains." *Sex Roles* 34(5-6):299–319.

Yang, Yang Claire, Martha K. McClintock, Michael Kozloski, and Ting Li. 2013. "Social Isolation and Adult Mortality: The Role of Chronic Inflammation and Sex Differences." *Journal of Health and Social Behavior* 54(2):182–203.

Yorgason, Jeremy B., Alan Booth, and David Johnson. 2008. "Health, Disability, and Marital Quality: Is the Association Different for Younger Versus Older Cohorts?" *Research on Aging* 30(6):623–48.

Table 1. Factor Loadings For Marital Quality

	Wave 1		Wave 2	
	PMQ	NMQ	PMQ	NMQ
How close do you feel is your relationship with spouse?	**0.58**	-0.11	**0.64**	0.03
How would you describe your marriage in terms of happiness?	**0.57**	-0.15	**0.60**	-0.10
How emotionally satisfying do you find your relationship with spouse?	**0.63**	-0.08	**0.56**	-0.08
Do you and spouse spend free time together or apart?	**0.38**	-0.02	**0.42**	0.03
How often can you open up to spouse?	**0.60**	0.08	**0.61**	-0.01
How often can you rely on spouse?	**0.61**	0.09	**0.51**	0.07
How often does spouse make too many demands on you?	-0.01	**0.64**	0.07	**0.60**
How often does spouse criticize you?	0.03	**0.71**	0.01	**0.71**
How often does spouse get on your nerves?	---	---	-0.31	**0.36**

--- Item not available at the specific wave.
PMQ: positive marital quality. NMQ: negative marital quality.

Table 2. Weighted Descriptive Statistics for Women (N=459)

Marital quality	Mean	S.D.	Min	Max
Positive marital quality W1	-0.11	0.95	-3.75	0.95
Negative marital quality e W1	-0.10	0.78	-0.92	2.52
Positive marital quality W2	-0.09	0.93	-3.62	1.00
Negative marital quality W2	-0.07	0.79	-0.91	2.71

Cardiovascular risk outcomes	Percent /mean(SD)		Percent /mean(SD)
Hypertension W1		Hypertension W2	
Normal	38.8	Normal	30.98
Controlled hypertension W1	20.17	Controlled hypertension W1	25.13
Undiagnosed hypertension W1	15.62	Undiagnosed hypertension W1	20.85
Uncontrolled hypertension W1	25.41	Uncontrolled hypertension W1	22.63
		Missing	0.41
Rapid heart rate W1		Rapid heart rate W2	
No	81.13	No	78.83
Yes	18.87	Yes	18.72
		Missing	2.45
CRP W1		CRP W2	
<=3	42.74	<=3	50.56
>3 and <10	18.64	>3 and <10	30.99
Not applicable	38.62	Missing or >10	18.45
CVD events W1		CVD events W2	
No	89.36	No	83.62
Yes	10.64	Yes	15.67
		Missing	0.71

Covariates			
Age group		Smoke W1	
57-64	53.6	No	89.5
65-74	32.93	Yes	10.5
75-85	13.47	Drink W1	
Race-ethnicity		No	38.47
Non-Hispanic white	86.94	Yes	61.53
Non-Hispanic black	5.57	BMI W1	
Hispanic	5.33	Normal or underweight	27.33
Others	2.16	Overweight	41.85
Education		Obesity	28.01
No diploma	10.13	Morbidly obese	2.81
High school graduate	26.13	Physical activity W1	
Some college	38.17	< 3 times per week	36.15
College graduate	25.57	>= 3 times per week	63.85
Relative family income		Antihypertensive medication use W1	
Below average	15.85	No	48.05
Average	39.79	Yes	51.95
Above average	32.24		
Missing	12.12	Probability of death at W2	0.06(0.06)
		Probability of remaining married	
Psychological distress W1	4.51(4.32)	in both waves	0.44(0.18)

W1: Wave 1. W2: Wave 2.

Table 3. Weighted Descriptive Statistics for Men (N=739)

Marital quality	Mean	S.D.	Min	Max
Positive marital quality W1	0.14	0.79	-3.52	0.95
Negative marital quality W1	-0.01	0.80	-0.96	2.56
Positive marital quality W2	0.15	0.77	-3.73	1.00
Negative marital quality W2	-0.03	0.80	-0.91	2.85

Cardiovascular risk outcomes	Percent /mean(SD)		Percent /mean(SD)
Hypertension W1		Hypertension W2	
Normal	31.49	Normal	25.25
Controlled hypertension W1	20.41	Controlled hypertension W1	26.1
Undiagnosed hypertension W1	17.88	Undiagnosed hypertension W1	19.23
Uncontrolled hypertension W1	30.22	Uncontrolled hypertension W1	29.32
		Missing	0.1
Rapid heart rate W1		Rapid heart rate W2	
No	82.33	No	78.11
Yes	17.67	Yes	16.1
		Missing	5.79
CRP W1		CRP W2	
<=3	55.79	<=3	62.6
>3 and <10	12.93	>3 and <10	22.63
Not applicable	31.28	Missing or >10	14.77
CVD events W1		CVD events W2	
No	79.75	No	70.25
Yes	20.25	Yes	29.21
		Missing	0.54

Covariates			
Age group		Smoke W1	
57-64	54.54	No	84.53
65-74	30.43	Yes	15.47
75-85	15.03	Drink W1	
Race-ethnicity		No	31.22
Non-Hispanic white	83.41	Yes	68.78
Non-Hispanic black	6.12	BMI W1	
Hispanic	8	Normal or underweight	18.4
Others	2.47	Overweight	41.79
Education		Obesity	36.04
No diploma	13.2	Morbidly obese	3.77
High school graduate	21.8	Physical activity W1	
Some college	29.21	< 3 times per week	30.14
College graduate	35.79	>= 3 times per week	69.86
Relative family income		Antihypertensive medication use W1	
Below average	17.16	No	46.44
Average	33.04	Yes	53.56
Above average	34.07		
Missing	15.73	Probability of death at W2	0.11(0.08)
		Probability of remaining married in	
Psychological distress W1	4.07(4.23)	both waves	0.63(0.18)

W1: Wave 1. W2: Wave 2.

Table 4. Regression Coefficients for Negative Marital Quality Predicting Cardiovascular Risks from Logistic Regression Models for Women

	Hypertension W2 (N=456)						Rapid heart rate W2 (N=443)		High CRP W2 (N=374)		CVD events W2 (N=454)	
	Model 1			Model 2			Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
	Controlled	Undiagnosed	Uncontrolled	Controlled	Undiagnosed	Uncontrolled						
Age W1 (ref=57-64)												
65-74	0.48	0.81*	0.89	0.56	0.62	1.13*	0.97*	0.56	-0.54	-0.58	1.71	1.53
	(0.43)	(0.39)	(0.48)	(0.51)	(0.47)	(0.54)	(0.47)	(0.49)	(0.31)	(0.31)	(0.96)	(0.81)
75-85	-0.31	1.68*	0.65	0.42	2.41**	1.53*	1.91*	1.93*	-1.52*	-1.44*	2.76**	2.72**
	(0.81)	(0.76)	(0.77)	(0.65)	(0.78)	(0.74)	(0.84)	(0.87)	(0.66)	(0.67)	(0.97)	(0.91)
NMQ W1	0.06	-0.13	0.41	-0.12	-0.05	-0.10	-0.09	0.20	0.02	0.13	0.11	-0.24
	(0.21)	(0.23)	(0.23)	(0.27)	(0.31)	(0.34)	(0.19)	(0.24)	(0.16)	(0.20)	(0.30)	(0.67)
NMQ W1 X age 65-74				-0.52	-1.25*	0.61		-1.97***		-0.36		-0.68
				(0.54)	(0.61)	(0.62)		(0.53)		(0.30)		(0.89)
NMQ W1 X age 75-85				2.74***	2.30**	2.39**		1.04*		0.13		2.48*
				(0.73)	(0.78)	(0.79)		(0.46)		(0.41)		(1.04)
Change of NMQ W2-W1	0.52*	0.45	0.45*	0.62	0.27	0.45			0.52*	0.61*		
	(0.25)	(0.24)	(0.21)	(0.35)	(0.42)	(0.35)			(0.19)	(0.29)		
Change of NMQ W2-W1 X age 65-74				-0.66	0.23	0.30				-0.18		
				(0.56)	(0.70)	(0.51)				(0.43)		
Change of NMQ W2-W1 X age 75-85				0.66	0.53	-0.22				-0.30		
				(0.91)	(1.01)	(1.01)				(0.63)		
Constant	0.40	0.09	0.21	0.43	0.33	0.03	-1.86	-1.71	-0.61	-0.51	-4.06	-4.57*
	(1.42)	(1.30)	(1.47)	(1.38)	(1.43)	(1.47)	(1.18)	(1.04)	(1.19)	(1.15)	(2.10)	(2.00)

*** p<0.001, ** p<0.01, * p<0.05.

Standard errors in parentheses.

NMQ: negative marital quality. W1: Wave 1. W2: Wave 2.

In all models, we control for race-ethnicity, education, relative family income, smoking, drinking, BMI physical activity, antihypertensive medication use, and psychological distress (all measured at Wave 1) as well probability of death at W2 and probability of remaining married in both waves. Wave 1 measures of hypertension, rapid heart rate, high CRP and CVD events are also controlled in all models.

Our preliminary analyses suggested that the effects of change of negative marital quality between W1-W2 were not statistically significant (p > 0.05) when predicting rapid heart rate and CVD events for women, and are thus not included in the final models.

Table 5. Regression Coefficients for Positive Marital Quality Predicting Cardiovascular Risks from Logistic Regression Models for Women

	Hypertension W2 (N=456)						Rapid heart rate W2 (N=443)		High CRP W2 (N=374)		CVD events W2 (N=454)	
	Model 1			Model 2			Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
	Controlled	Undiagnosed	Uncontrolled	Controlled	Undiagnosed	Uncontrolled						
Age W1 (ref=57-64)												
65-74	0.42	0.80*	0.84	0.53	0.90*	0.95	0.99*	1.03*	-0.58	-0.55	1.78	2.13*
	(0.41)	(0.39)	(0.46)	(0.45)	(0.43)	(0.49)	(0.47)	(0.45)	(0.32)	(0.32)	(0.98)	(0.98)
75-85	-0.25	1.86*	0.67	-0.22	1.77*	0.67	1.93*	1.63*	-1.36*	-1.35	2.81**	2.76**
	(0.79)	(0.73)	(0.76)	(0.68)	(0.70)	(0.76)	(0.84)	(0.80)	(0.66)	(0.68)	(0.99)	(1.00)
PMQ W1	-0.00	0.06	-0.16	0.08	0.03	0.17	0.09	0.12	-0.04	-0.19	-0.01	0.93
	(0.20)	(0.18)	(0.17)	(0.28)	(0.23)	(0.25)	(0.19)	(0.26)	(0.17)	(0.20)	(0.28)	(0.81)
PMQ W1 X age 65-74				0.14	0.53	-0.25		0.51		0.39		-0.86
				(0.45)	(0.41)	(0.45)		(0.41)		(0.23)		(0.87)
PMQ W1 X age 75-85				-1.20*	-1.25*	-1.55**		-0.83		0.21		-1.67*
				(0.57)	(0.60)	(0.57)		(0.55)		(0.34)		(0.80)
Change of PMQ W2-W1											-0.44	1.35
											(0.57)	(0.77)
Change of PMQ W2-W1 X age 65-74												-2.20
												(1.16)
Change of PMQ W2-W1 X age 75-85												-2.10*
												(1.03)
Constant	0.12	-0.09	-0.12	0.01	-0.12	-0.31	-1.86	-1.91	-0.77	-0.63	-4.28*	-4.80*
	(1.41)	(1.25)	(1.48)	(1.31)	(1.28)	(1.42)	(1.15)	(1.05)	(1.18)	(1.16)	(1.97)	(1.82)

*** p<0.001, ** p<0.01, * p<0.05.

Standard errors in parentheses.

PMQ: positive marital quality. W1: Wave 1. W2: Wave 2.

In all models, we control for race-ethnicity, education, relative family income, smoking, drinking, BMI physical activity, antihypertensive medication use, and psychological distress (all measured at Wave 1) as well probability of death at W2 and probability of remaining married in both waves. Wave 1 measures of hypertension, rapid heart rate, high CRP and CVD events are also controlled in all models.

Our preliminary analyses suggested that the effects of change of positive marital quality between W1-W2 were not statistically significant ($p > 0.05$) when predicting hypertension, rapid heart rate and high CRP for women, and are thus not included in the final models.

Table 6. Regression Coefficients for Negative Marital Quality Predicting Cardiovascular Risks from Logistic Regression Models for Men

	Hypertension W2 (N=738)						Rapid heart rate W2 (N=700)		High CRP W2 (N=631)		CVD events W2 (N=736)	
	Model 1			Model 2			Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
	Controlled	Undiagnosed	Uncontrolled	Controlled	Undiagnosed	Uncontrolled						
Age W1 (ref=57-64)												
65-74	0.45	0.77	0.15	0.43	0.74	0.10	-0.13	-0.13	-0.51	-0.57*	0.82*	0.83*
	(0.46)	(0.41)	(0.37)	(0.45)	(0.41)	(0.37)	(0.34)	(0.34)	(0.27)	(0.26)	(0.33)	(0.33)
75-85	-0.88	0.19	-1.14*	-0.98	0.09	-1.23*	0.44	0.22	0.03	0.04	1.15	1.27*
	(0.60)	(0.68)	(0.53)	(0.60)	(0.71)	(0.55)	(0.49)	(0.55)	(0.42)	(0.41)	(0.62)	(0.60)
NMQ W1	-0.16	0.03	-0.18	-0.36	0.17	-0.24	0.01	-0.03	0.33*	0.22	-0.02	0.04
	(0.19)	(0.20)	(0.21)	(0.29)	(0.30)	(0.34)	(0.15)	(0.19)	(0.13)	(0.15)	(0.21)	(0.32)
NMQ W1 X age 65-74				0.24	-0.35	0.02		-0.15		0.38		0.05
				(0.51)	(0.46)	(0.53)		(0.43)		(0.30)		(0.38)
NMQ W1 X age 75-85				0.61	-0.31	0.28		0.62		-0.03		-0.45
				(0.75)	(0.61)	(0.74)		(0.51)		(0.36)		(0.52)
Change of NMQ W2-W1	-0.57*	-0.33	-0.16	-1.07**	-0.26	-0.49						
	(0.25)	(0.20)	(0.21)	(0.31)	(0.27)	(0.25)						
Change of NMQ W2-W1 X age 65-74				1.30*	0.16	0.99*						
				(0.51)	(0.49)	(0.45)						
Change of NMQ W2-W1 X age 75-85				0.66	-0.44	0.45						
				(0.68)	(0.68)	(0.71)						
Constant	-0.46	2.92	1.86*	-0.59	2.96*	1.76	-2.20	-2.26	-1.35	-1.33	-2.23	-2.27
	(1.11)	(1.47)	(0.92)	(1.07)	(1.45)	(0.91)	(1.40)	(1.38)	(0.97)	(0.96)	(1.51)	(1.52)

*** p<0.001, ** p<0.01, * p<0.05.

Standard errors in parentheses.

NMQ: negative marital quality. W1: Wave 1. W2: Wave 2.

In all models, we control for race-ethnicity, education, relative family income, smoking, drinking, BMI physical activity, antihypertensive medication use, and psychological distress (all measured at Wave 1) as well probability of death at W2 and probability of remaining married in both waves. Wave 1 measures of hypertension, rapid heart rate, high CRP and CVD events are also controlled in all models.

Our preliminary analyses suggested that the effects of change of negative marital quality between W1-W2 were not statistically significant ($p > 0.05$) when predicting rapid heart rate, high CRP and CVD events for men, and are thus not included in the final models.

Table 7. Regression Coefficients for Positive Marital Quality Predicting Cardiovascular Risks from Logistic Regression Models for Men

	Hypertension W2 (N=738)						Rapid heart rate W2 (N=700)		High CRP W2 (N=631)		CVD events W2 (N=736)	
	Model 1			Model 2			Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
	Controlled	Undiagnosed	Uncontrolled	Controlled	Undiagnosed	Uncontrolled						
Age W1 (ref=57-64)												
65-74	0.42	0.78	0.17	0.38	0.78	0.14	-0.14	-0.13	-0.50	-0.43	0.84*	0.81*
	(0.45)	(0.41)	(0.37)	(0.44)	(0.40)	(0.37)	(0.35)	(0.34)	(0.27)	(0.27)	(0.32)	(0.32)
75-85	-0.78	0.26	-1.09*	-0.80	0.21	-1.08	0.41	0.32	0.03	0.25	1.19	1.18
	(0.59)	(0.69)	(0.52)	(0.58)	(0.70)	(0.55)	(0.49)	(0.51)	(0.43)	(0.45)	(0.61)	(0.61)
PMQ W1	-0.02	0.00	0.10	-0.13	0.00	0.02	-0.09	0.02	-0.17	0.10	0.22	0.14
	(0.16)	(0.19)	(0.18)	(0.22)	(0.26)	(0.25)	(0.17)	(0.22)	(0.14)	(0.19)	(0.17)	(0.24)
PMQ W1 X age 65-74				0.38	-0.13	0.32		-0.07		-0.60		0.18
				(0.37)	(0.37)	(0.38)		(0.33)		(0.36)		(0.34)
PMQ W1 X age 75-85				-0.12	0.41	-0.19		-0.61		-0.55		0.13
				(0.62)	(0.63)	(0.73)		(0.55)		(0.51)		(0.52)
Change of PMQ W2-W1									0.11	0.39		
									(0.14)	(0.27)		
Change of PMQ W2-W1 X age 65-74										-0.23		
										(0.38)		
Change of PMQ W2-W1 X age 75-85										-1.09*		
										(0.48)		
Constant	-0.57	2.85	1.80	-0.55	2.80	1.83	-2.15	-2.08	-1.26	-1.28	-2.31	-2.28
	(1.09)	(1.47)	(0.95)	(1.10)	(1.44)	(0.96)	(1.43)	(1.40)	(1.03)	(1.01)	(1.51)	(1.50)

*** p<0.001, ** p<0.01, * p<0.05.

Standard errors in parentheses.

PMQ: positive marital quality. W1: Wave 1. W2: Wave 2.

In all models, we control for race-ethnicity, education, relative family income, smoking, drinking, BMI physical activity, antihypertensive medication use, and psychological distress (all measured at Wave 1) as well probability of death at W2 and probability of remaining married in both waves. Wave 1 measures of hypertension, rapid heart rate, high CRP, and CVD events are also controlled in all models.

Our preliminary analyses suggested that the effects of change of positive marital quality between W1-W2 were not statistically significant ($p > 0.05$) when predicting hypertension, rapid heart rate and CVD events for men, and are thus not included in the final models.

Table 8. Regression Coefficients for Cardiovascular Risks Predicting Marital Quality from Logistic Regression Models

	NMQ W2		PMQ W2	
	Men	Women	Men	Women
Hypertension W1 (ref=normal)				
Controlled hypertension W1	-0.22*	0.15	0.04	-0.18*
	(0.10)	(0.09)	(0.08)	(0.08)
Undiagnosed hypertension W1	-0.00	0.02	0.00	0.00
	(0.10)	(0.09)	(0.09)	(0.10)
Uncontrolled hypertension W1	-0.13	0.07	0.05	0.06
	(0.09)	(0.09)	(0.09)	(0.08)
Rapid heart rate W1	-0.13	0.02	0.02	-0.09
	(0.07)	(0.08)	(0.06)	(0.06)
CRP W1 (ref: CRP<=3)				
CRP>3	0.22	0.28**	-0.16	-0.23*
	(0.13)	(0.09)	(0.10)	(0.09)
Not applicable	-0.01	-0.03	-0.04	0.12
	(0.07)	(0.06)	(0.05)	(0.08)
CVD W1	0.02	0.07	-0.07	-0.13
	(0.07)	(0.09)	(0.05)	(0.11)
NMQ/PMQ W1	0.54***	0.71***	0.59***	0.71***
	(0.04)	(0.04)	(0.04)	(0.04)
Constant	-0.12	-0.57**	-0.05	0.24
	(0.33)	(0.20)	(0.25)	(0.22)
N	613	388	613	388
R-squared	0.369	0.532	0.452	0.584

*** p<0.001, ** p<0.01, * p<0.05.
Standard errors in parentheses.
NMQ: negative marital quality. PMQ: positive marital quality W1: Wave 1. W2: Wave 2.
In all models, we control for race-ethnicity, education, relative family income, smoking, drinking, BMI physical activity, antihypertensive medication use, and psychological distress (all measured at Wave 1) as well probability of death at W2 and probability of remaining married in both waves.