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Bad Marriage, Broken Heart? Age and Gender Differences in the Link between Marital Quality and Cardiovascular Risks among Older Adults

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Abstract

Working from a life course perspective, we develop hypotheses about age and gender differences in the link between marital quality and cardiovascular risk and test them using data from the first two waves of the National Social Life, Health, and Aging Project. The analytic sample includes 459 married women and 739 married men (aged 57–85 in the first wave) who were interviewed in both waves. We apply Heckman-type corrections for selection bias due to mortality and marriage. Cardiovascular risk is measured as hypertension, rapid heart rate, C-reactive protein, and general cardiovascular events. Results suggest that changes in marital quality and cardiovascular risk are 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 in the U.S. (National Academy on An Aging Society 2000). According to the US Centers for Disease Control (CDC), about 600,000 Americans die of CVD every year or one in every four deaths. This problem becomes increasingly prevalent with advancing age (Lakatta and Levy 2003). In 2007–2010, 40.0% of men and 34.4% of women ages 40–59 suffered from CVD in the U.S., and these numbers rise to 70.2% and 70.9% for ages 60–79 and 83.0% and 87.1% for ages 80 and above (Go et. al. 2014). Because the onset of most CVD can be delayed and the disease treated, identifying relevant risk factors is extremely important in designing effective prevention strategies and treatment programs. Many personal, social, and behavioral factors contribute to the risk of CVD over the lifetime (Lakatta and Levy 2003).

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The present study focuses specifically on marital quality as a social factor that may shape the risks of CVD during later adulthood.

It has long been recognized that married people show better health than the unmarried, including better cardiovascular health and better outcomes following heart attack (Coyne et al. 2001; Zhang and Hayward 2006). Recent research points to marital quality—broadly defined as spouses' subjective appraisals of their marital relationship—as more important than marital status for health (Umberson et al. 2006). Cardiovascular risk (i.e., the presence of any physiological or functional state that is a step on the way to CVD) offers a model for assessing marital quality links with disease processes in specific dimensions of physical health. To date, most empirical evidence for the impact of marital quality on cardiovascular risk comes from lab-based studies, usually cross-sectional, and clinical or community samples (Robles and Kiecolt-Glaser 2003). Thus, these studies provide very limited evidence on causal direction, and the findings cannot be generalized to the population level. 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 makes significant contribution to the literature by overcoming these limitations.

We work from a life course perspective to address two major research questions: (1) how is marital quality related to cardiovascular risk over time among older adults? and (2) does this relationship vary by gender and/or age? We analyze data from the first two waves of the National Social Life, Health, and Aging Project (NSHAP), a nationally representative longitudinal data set on older adults. Our findings have important implications for health policy and practice given that cardiovascular risks 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 (Izzo and Black 2003).

Marital Quality and Cardiovascular Risk: Previous Empirical Evidence

Empirical evidence on the effects of marital quality on cardiovascular function is limited in both quantity and quality. The few studies examining this relationship primarily come from unrepresentative, lab-based clinical samples. These clinical studies are mostly cross-sectional and tend to focus on psychobiological responses to marital stress (Robles and Kiecolt-Glaser 2003). For example, several community-based clinical studies find that marital strain and hostile interactions produce clinically significant increases in systolic and diastolic blood pressure and heart rates (Ewart et al. 1991; Taylor et al. 2000). This effect is more pronounced for women than for men (Ewart et al. 1991). Following 292 female patients who experienced an acute coronary event, 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 than women who reported lower levels

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of marital strain. Another clinical study followed 189 congestive heart failure patients; its findings suggest that marital strain 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 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. Although clinical contributions represent a step forward in identifying the links between cardiovascular health and marital quality, these studies provide a starting point only due to their fundamental methodological weaknesses (e.g., small, unrepresentative samples, cross-sectional designs, and lacks of control covariates). Therefore, clinical research has failed to reliably demonstrate basic population patterns of the association between marital quality and cardiovascular risk (Robles and Kiecolt-Glaser 2003).

One of the very few national studies on 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 the relationship between marital quality and levels of C-reactive protein (CRP). This study used CRP as a marker for inflammation, but CRP is also 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 (Donoho et al. 2013). Surprisingly, the Donoho et al. study does not find evidence for marital strain in relation to CRP. Another study based on data from the first wave of the National Social Life, Health, and Aging Project found that partner relationship quality was a significant moderator in the link between stress and blood pressure, although there was no evidence of significant main effect of partner relationship quality on blood pressure (Birditt, Newton and Hope 2014).

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). Given this line of literature, we expect to find the following:

Hypothesis 1 Those with high levels of negative marital quality show greater cardiovascular risk subsequently than those with low levels of negative marital quality. Those with high levels of positive marital quality show lower cardiovascular risk subsequently than those with

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low levels of positive marital quality, but this difference is relatively modest.

A Gendered Life Course Perspective on Cumulative Advantage/ Disadvantage Processes

Recent research builds on a life course perspective on the link between marital quality and health (Umberson et al. 2006). According to the life course perspective, the developmental tasks and challenges of life change from childhood through young adulthood, middle and older ages. Each person meets these challenges within a specific social, cultural and economic context; the presence or absence of a marital partner as well as the relationship quality with the partner is an important component of the context in which a person moves through the life course (Umberson et al. 2006). Both the stress and support processes that flow from marital relationships shape an individual's life context, which in turn affect health at every stage (Robles and Kiecolt-Glaser 2003). 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 such as smoking and drinking, or directly, by stimulating the production of stress hormones, evoking physiological responses, and triggering chronic arousal (Robles and Kiecolt-Glaser 2003). In contrast, involvement in a happy marriage provides social support and a safe haven, which reduce exposure to stress and help buffer stressful life events, enhancing emotional well-being and physical health (Robles and Kiecolt-Glaser 2003; Waite and Gallagher 2000). 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, and how this relationship varies by age and gender.

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, experienced as sequences of roles and experiences, which shape the course of human development and aging. Because age is the most important proxy for individuals' occupancy of and transitions in experiences, roles, and statuses, especially school, employment, marital and parental roles, life course scholars often adopt age as a key indicator for life course stages (Elder and O'Rand 1995).

According to the life course theory, the effect of marital quality on cardiovascular health may be contingent upon life course position, because advantage or disadvantage may have cumulative effects (Dannefer 2003). This cumulative-advantage/disadvantage argument suggests that marital quality will take a toll on health with advancing age. Since most chronic conditions develop slowly over time, the costs and benefits from one's marriage will take some time to show up in better or worse health. Thus, we expect that the association between marital quality and health will become more pronounced with age. 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

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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 determinant of life course context, and has been a central focus of research on the benefits of marriage for health (Bernard 1972; Zhang and Hayward 2006). While, on average, men receive more health promotion benefits such as emotional support and regulation of health behaviors from marriage than do women, they also seem to be less vulnerable to physiological effects of marital stress (Kiecolt-Glaser and Newton 2001). The long-run implications of these differences may also differ by gender. 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.

Evidence to date on gender differences in marriage and health links is quite mixed. On the one hand, a handful of clinical studies conclude that marital conflict tends to evoke greater and more persistent physiological changes in women than in men, including increases in systolic blood pressure, decreases in phytohemagglutinin, and changes in hormones (Ewart et al. 1991; Kiecolt-Glaser and Newton 2001). A national study of older adults also finds that women with a marital loss face higher risks of CVD at midlife than do women who have been continuously married, but men show no difference (Zhang and Hayward 2006). To the extent that marital loss reflects poor marital quality, Zhang and Hayward's results suggest that women's health may be more sensitive to marital quality than men's. However, other studies suggest the opposite direction of the gender difference. For example, based on a national representative dataset, Sbarra (2009) finds that married men have lower levels of CRP than divorced and widowed men, but married women do not differ from divorced and widowed women, or from divorced or widowed men. He interprets these results to suggest that marital loss (often preceded by high marital conflict) is a much bigger stressor for men than for women, with consequences for levels of inflammation. A few clinical studies also 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). Yet, 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.

We expect that in comparison to men, women's cardiovascular function becomes more responsive to marital strain as they 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 symptoms of metabolic syndrome and significant depressive symptoms than those in happy marriages (Kiecolt-Glaser and Newton 2001). Depression is one of the most often-

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proposed mediating mechanisms linking marital quality and physical health (Robles and Kiecolt-Glaser 2003). This link may follow from gender differences in hormone levels, genetic profiles, and physiological response to stress (Taylor et al. 2000). 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 do men (Donoho et al. 2013), we hypothesize 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

Although a sizeable body of literature shows that different levels of marital quality produce different health outcomes—which is also the primary conceptual framework adopted by the present study, there is some evidence suggesting that poor health may in turn undermine later marital quality while good health may promote it (Booth and Johnson 1994; Galinsky and Waite 2014). Spouses in good 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 accompanied by low energy levels, which may limit time spent with other family members (Booth and Johnson 1994). Additionally, a sick spouse may cause economic, physical, and emotional burdens on the healthy spouse, who often serves as the primary caregiver (Galinsky and Waite 2014). This may increase the psychological distress of the caregiver spouse. 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 among older adults (Booth and Johnson 1994). Therefore, we expect that:

Hypothesis 4 Higher levels of cardiovascular risk at one point are related to higher negative marital quality and lower positive marital quality at a later point.

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, one the first national-scale population-based studies of health and intimate relationships at older ages, was conducted by NORC at the University of Chicago. A nationally representative probability sample of community-dwelling individuals ages 57–85 was selected from households across the U.S. and screened in 2004. African Americans, Latinos, men, and those 75–84 years old were over-sampled. All analyses are weighted and further adjusted for clustering and stratification of the complex sampling design using the survey data analysis commands in Stata (StataCorp 2012).

The first wave of the NSHAP (Wave 1) included a sample of 3,005 adults ages 57–85 who were interviewed during 2005–2006 (Waite, Laumann, et al., 2014). 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 (Waite, Cagney, et al., 2014). We

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restricted the analysis to the 1,250 respondents who had remained married and who had been interviewed in both waves. Cases with 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 also excluded cases with missing values on the dependent variable analyzed in that model. As a result, the sample size in various models differs slightly 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 key 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 inflammation of arteries, 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 the blood pressure of each respondent twice, 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 have been diagnosed with diabetes, we use the recommended lower cutoff of either 130 mm Hg systolic or 80 mm Hg diastolic (National Heart, Lung, and Blood Institute 2003). In addition, respondents were asked whether they had ever been told by a medical doctor that they had high blood pressure or hypertension. Based on responses to this question, along with the measures of blood pressure, we categorize respondents into four groups: (1) normal blood pressure reading and no diagnosis of hypertension (referred hereafter as the “normal” blood pressure group), (2) normal blood pressure reading but diagnosed with hypertension (referred as “controlled” hypertensive group), (3) high blood pressure reading but no diagnosis of hypertension (referred as “undiagnosed” hypertensive group), and (4) high blood pressure reading and diagnosed 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 is measured as the number of times the heart beats per minute. When the heart cannot effectively deliver blood and oxygen to meet the need of the body it will beat abnormally fast. A long-term resting heart rate greater than 80 beats per minute is linked to a significant risk of hypertension, heart disease, and mortality (Izzo and Black 2003). 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).

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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 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, since this suggests the presence of acute infection (Pearson et al. 2003). Because the blood spot was collected from a randomly-selected 5/6 of the total NSHAP sample, we flag those respondents for whom 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 of 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 had a heart attack, heart failure, or stroke. These events 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

Marital quality consists of both positive and negative dimensions, which are not opposite ends of a single dimension but distinct constructs. A marriage may be high, for example, on both positive and negative dimensions (Umberson et al. 2006). We follow Galinsky and Waite (2014) to calculate marital quality scales using the NSHAP data. These scales 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 2014).

Additionally, respondents were asked the extent to which they preferred to spend their free time doing things with their spouse (item 4). Responses ranged from (1) mostly together, (2) some together and some apart, to (3) mostly apart. We reverse-coded this item so that higher

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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, NSHAP 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. 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, analyses of change in marital quality between waves use only the items available in both waves for comparability. Table 1 shows the factor loadings of each item used to generate the factor scores for positive and negative marital quality, respectively.

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

We stratify all analyses by *gender*. *Age* is categorized into three groups: 57–64 (young-old, reference), 65–74 (middle-old), and 75–85 (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. We create a "missing" indicator category for the 15% of the analytic sample without valid values on 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 include Body Mass Index, a significant predictor of CVD, as an indicator of obesity (Izzo and Black 2003). Body Mass Index (BMI) is calculated from measured height and weight, grouped into four categories: normal or underweight (BMI < 25), overweight (25 <= BMI < 30), obese (30 <= BMI < 40), and morbidly obese (BMI >= 40) (World Health Organization 1995). Missing values on BMI (n=53) were imputed with the mean[1]. Excluding these cases showed similar results. In addition, we control indicators for *currently smoke* (1 = Yes, 0 = No), *currently drink*

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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) (Radloff 1977). 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. Cases missing on these items (n=13) were imputed with the mean. Tables 2–3 show the weighted descriptive statistics of all analyzed variables for women and men separately.

ANALYTIC APPROACH

We assess each of the four cardiovascular risk outcomes as predicted by negative marital quality and, separately, positive marital quality, since these two predictors are highly correlated (r = −0.64 in Wave 1 and −0.68 in Wave 2). We use the lagged dependent variable approach[2] to analyze the two waves of data. Specifically, we use Wave 1 marital quality along with change in marital quality between waves to predict Wave 2 cardiovascular risks controlling for Wave 1 cardiovascular risks and all other covariates. For parsimony we dropped change in marital quality from the final model if it was not significant in preliminary analyses[3]. The specific analytic techniques we use depend on the outcomes being modeled. We estimate multinomial logistic regression models to predict hypertension, and binary logistic regression models to predict rapid heart rate, high CRP, and general CVD events. We stratify all analyses by gender, and assess the statistical significance of gender differences using Wald tests (Agresti and Finley 2009).

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 socio-demographic covariates only. Next, we add measures of health behaviors and psychological distress separately. This allows us to test the idea that health behaviors and psychological distress may mediate the relationship between marital quality and cardiovascular risks. Since preliminary results (available upon request) showed no evidence of mediation, we only report the final full model (Model 1) in which all covariates are

[1]Other imputation techniques such as multiple imputation are more sophisticated than the simple approaches we applied in this study to handle the missing data. However, due to the relatively smaller number of missing cases in our sample, Allison (2001) suggests that the simple missing data handling approaches are more efficient.

[2]We have also estimated cross-lagged models to estimate the effects of wave 1 marital quality on wave 2 cardiovascular risks and the effects of wave 1 cardiovascular risks on wave 2 marital quality simultaneously. The results from the cross-lag models (not shown but available upon request) showed similar patterns as reported in the paper.

[3]Although adding all of those non-significant covariates of change in marital quality as well as their age interaction effects in the models did not significantly change our results, it reduced the statistical power of some of our key findings. For the purpose of model parsimony, we only include changes in marital quality between Wave 1 and Wave 2 as well as their age interaction effects in the final reported models when they are significant.

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controlled—including socio-demographic, health-behavior-related covariates and psychological distress. To assess potential age differences in the relationship between marital quality and cardiovascular risk, Model 2 adds interaction terms of age by marital quality.

We also address the possibility of temporal early cardiovascular risk and later marital quality. 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 and covariates.

Correction for Sample Selection Bias

Our analyses are restricted to married respondents in both waves so the samples are selective of those with relatively good marital quality; marriages of poor quality are more likely to have ended. Moreover, sample attrition between waves, due to mortality, poor health, refusal or inability to locate the respondent is not random. Therefore, we apply the approach, developed by Heckman (Heckman 1979) to adjust the sample selection biases that are due to selection though marriage and mortality. See Umberson et al. (2006) and Liu (2012) for similar applications. 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 of 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.

RESULTS

Tables 4–7 present results from models of hypertension, rapid heart rate, high CRP, and general CVD events. Table 4 presents results for all outcomes for women as predicted by negative marital quality. Table 5 presents the same outcomes of women for positive marital quality. 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 are derived by exponentiation.

Model 1 of Table 4 shows no significant effect of negative marital quality at Wave 1 on 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 at Wave 2 for women (Model 1 of Table 4). Specifically, for women, with every one unit increase of negative marital quality between Waves 1 and 2, the relative risk of controlled and uncontrolled hypertension at Wave 2 increases by 1.68 (i.e., exp(0.52)) and 1.57 times, respectively, and the odds of having high CRP at Wave 2 also increase by 1.68 times. Moreover, the relationship between Wave 1

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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. For women ages 57–65, negative marital quality at Wave 1 is not significantly associated with any of the cardiovascular risk outcomes at Wave 2 and the expected associations do not appear for women ages 65–74. Indeed, among women ages 65–74, the odds of undiagnosed hypertension (b = −0.05–1.25) and rapid heart rate (b = 0.20–1.97) are both *lower* for women with higher levels of negative marital quality, which is contrary to our expectations. However, among women at the oldest age group we see a strong and consistent relationship between negative marital quality and cardiovascular risks, in contrast to the lack of expected effects at younger ages (Model 2). Specifically, among women ages 75–85, 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. We also estimated all models separately for the three age groups (results not shown). These results show statistically significant effects of negative marital quality on hypertension, rapid heart rate and CVD events among women ages 75–85 ($p < 0.05$).

Table 5 shows similar models for positive marital quality among women. Results from Model 1 show no significant relationship of positive marital quality at Wave 1 to any of the cardiovascular risk outcomes at Wave 2. However, this relationship varies by age, in particular for hypertension and CVD events. Results from Model 2 show that positive marital quality at Wave 1 is not significantly associated with the risks of hypertension or CVD events at Wave 2 among women ages 57–64 or 65–75. However, among women ages 75–85, positive marital quality at Wave 1 is negatively associated with hypertension and CVD events by Wave 2. Specifically, among women ages 75–85, 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%, respectively; and the odds of reporting any CVD events at Wave 2 decrease by 52.29%. Moreover, women ages 75–85 who experience an increase in positive marital quality between Waves 1 and 2 tend to face lower odds of CVD events at Wave 2 (b = 1.35–2.10), although this relationship does not appear among women ages 57–64 or 65–74 (Model 2). Models estimated separately for women ages 75–85 show significant effects of positive marital quality on hypertension and CVD events ($p < 0.05$).

Tables 6 and 7 present results for men. Table 6 shows effects of negative marital quality on cardiovascular outcomes. Results from Model 1 show that men whose marriages increase in negative quality (i.e., characterized by increases in criticism and demands) face lower relative risks of controlled hypertension at Wave 2 ($b = −0.57$, $p < 0.05$) than others. However, coefficients are smaller among older men (Model 2). Indeed, models estimated for each age group separately show that change in negative marital quality is not significantly ($p > 0.05$) associated with hypertension at Wave 2 among men ages 65–74 or 75–85. Men with higher levels of negative martial quality at Wave 1 also 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).

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Negative marital quality is not significantly related to the odds of either general CVD events or rapid heart rate for men in any of the age groups.

Table 7 shows results for positive marital quality among men. These results suggest that positive marital quality is not related to any cardiovascular risk outcome 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 ages 75–85 than among men ages 57–64 (Model 2). However, models estimated separately by age group show no significant effect for this measure among men ages 75–85.

Cardiovascular risks may also affect later marital quality, as poor health may put a strain on the marriage. 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 our preliminary results (not shown but available upon request) show virtually no significant interaction of age with cardiovascular risks, we only present the main effects models in Table 8. Results from Table 8 suggest that neither heart rate nor CVD events at Wave 1 are 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, with patterns that vary by gender. Men who had controlled hypertension at Wave 1 have reduced 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 reduced 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 more negative marital quality ($b = 0.28$, $p < 0.01$) and less positive marital quality ($b = -0.23$, $p < 0.05$) at Wave 2 than do women with low or normal CRP at Wave 1. Thus, we see a mixed picture of effects of cardiovascular risks at Wave 1 on changes in marital quality by Wave 2.

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 (Umberson et al. 2006). This study highlights the importance of marital quality for cardiovascular risk—the central mechanism in the pathogenesis of many acute and chronic critical conditions among older adults in the U.S. We move beyond previous laboratory-based clinical studies by providing the first nationally representative population-based evidence of this overall relationship. We also contribute to our understanding of the importance of age and gender in moderating this relationship. Below, we outline our major findings and implications from this study in relation to each research hypothesis.

A life course approach emphasizes the importance of 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 hypothesized that those with high levels of negative marital quality would subsequently experience greater cardiovascular risk than those with low levels

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of negative marital quality; and those with high levels of positive marital quality would subsequently experience lower cardiovascular risk than those with low levels of positive marital quality, but this difference would be relatively modest (Hypothesis 1). Our results support this hypothesis but highlight important age and gender variations in this relationship. We will discuss these shortly. Moreover, although we find associations of both negative and positive marital quality with cardiovascular risk, especially among women and at older ages, our results show that negative marital quality is associated with more types of cardiovascular risk than is positive marital quality. This is consistent with clinical studies (and also our Hypothesis 1), which have found more significant effects of marital strain than of marital support on cardiovascular health (Robles and Kiecolt-Glaser 2003). Family and health researchers argue that both the support and stress processes related to marriage produce health outcomes (Umberson et al. 2006). Our results suggest that the stress process operating within marriage may be more important than the supportive aspect of marriage in shaping individuals' cardiovascular health.

Gender and Age Variations in the Link between Marital Quality and Cardiovascular Risk

Both age and gender have been identified as strong predictors of cardiovascular risk (Izzo and Black 2003). Family scholars also emphasize that marital quality varies by both age and gender (Umberson et al. 2005). The gendered 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 health differently for men and women over the life course. We hypothesized that the association between marital quality and cardiovascular risk would become stronger at older ages (Hypothesis 2) and it would be more pronounced for women than for men (Hypothesis 3). Consistent with these hypotheses, we find that the expected association between marital quality and cardiovascular risk is primarily present among women and at older ages; it is less so among men or at younger ages. This finding is in line with clinical literature that finds that marital strain tends to evoke greater and more persistent physiological and cardiovascular changes in women than in men. A long-held sociological tenet is that the state of being married promotes the health of men more than of women (Bernard 1972). However, a handful of clinical studies suggest that marital quality tends to be more important for women's health than for men's (Kiecolt-Glaser and Newton 2001). 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 (Umberson et al. 1996). Depression associated with marital strain may impose a variety of health threats by altering cardiovascular function of women (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 strong even after depression is controlled. Physiological adjustment to social relationships appears different for men and women (Kiecolt-Glaser and Newton 2001). 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.

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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 have greater impacts on individuals' health as they get older. This is especially the case for women. Social networks and contacts with friends tend to decrease at later ages (Cornwell, Laumann and Schumm 2008). Therefore, the marital relationship becomes increasingly important 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 CVD. Younger adults, especially women, may be stressed by negative marital quality, but the effects on their hearts may take years to emerge. Moreover, aging is a process often accompanied by a decline of the functioning of the immune system and an increase in functional limitations (Lakatta and Levy 2003). Thus, older individuals, especially those at very old ages, may be more vulnerable to a negative relationship than they were earlier in their lives. Criticism, demands and failure to get along may stimulate greater cardiovascular response at very old ages as a result of changes in physical fitness, body fat percentage, and the presence of chronic conditions. Our results point to the need for research on specific social and physiological mechanisms that underlie the relationship between marital quality and cardiovascular risk at very old ages.

Some general patterns across all ages appear in the link between marital quality and changes in cardiovascular risk, although sometimes for only one gender. We find that for women at all old ages, an increase in negative marital quality leads to higher risks of hypertension. Since hypertension is an early stage in the progression of cardiovascular disease—increasing the risks of more serious disease, it may appear at younger ages than the later events such as heart disease or stroke. Additionally, we find that negative martial quality tends to increase the risk of high CRP, also an early stage in the CVD process, for both men and women at all old ages. This finding differs from that of a recent study, which found minimal association between negative marital quality and CRP levels for both men and women (Donoho et al. 2013). These divergent results may be due to different measures of CRP, as well as to their failure to examine differences by age. Our results are indeed more consistent with previous clinical studies that suggest a stronger effect of negative marital quality than positive marital quality on health (Ewart et al. 1991; Robles and Kiecolt-Glaser 2003).

Reverse Causation

Although we develop a causal framework from which we draw hypotheses on how marital quality affects cardiovascular risk, it is also likely that cardiovascular risk shapes later marital quality (Hypothesis 4). We find mixed evidence on this hypothesis, depending on gender and specific cardiovascular risk outcomes. Inconsistent with this hypothesis, we find that marital quality affects later heart rate and CVD events, especially for women at older ages, but that rapid heart rate and CVD events have little impact on later changes in marital quality. The results on hypertension and CRP vary by gender. For men, we find results contrary to our expectations: men who had controlled hypertension at Wave 1 experience

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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 the meanings of “controlled hypertension”. On the one hand, 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 life style changes to reduce 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 reverse causation between marital quality and cardiovascular risk. We find that controlled hypertension and high CRP are both significant predictors of subsequent declines in marital quality for women; at the same time, marital quality also affects the subsequent risk of hypertension (especially among older women) and high CRP for women. These findings for women are consistent with the long-standing observation that husbands are less likely than wives to provide support and care 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 causal relationships, we were limited by sample size, especially for separate gender and age groups. To understand the role of selection and causal processes in the links between marital quality and cardiovascular risk, future studies should employ longitudinal data with larger sample size and more waves of follow-up. The NSHAP is currently collecting the third wave of data, which will provide opportunities to further untangle causality. 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. However, we emphasize that although our conclusions are more relevant to the population of older adults who are not in very poor health, less likely to die, and more likely to stay married, this study is based on a random sample from that segment of the population. Indeed, those who died between waves and those who were not married in both waves probably also faced relatively high cardiovascular risk, making our estimates conservative. Third, this study focuses on later life course stages. Aging is a life-long process which starts at much earlier ages. Future studies should examine a broader range of ages to fully understand the life course patterns in the relationship between marital quality and cardiovascular risk. It is likely that people at very young ages—who are more likely to lack coping skills to deal with their marital conflicts—as well as at very old ages are more likely to experience cardiovascular responses to marital quality than middle-aged adults, suggesting a non-linear age pattern. Future studies should test this possibility using other datasets with a wider range of ages. Finally, various social, biological, psychological, and behavioral mechanisms underlie the link between marital quality and cardiovascular risk.

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Future studies should seek to identify the precise mechanisms and processes through which marital quality and cardiovascular risk affect each other, and to address how those mechanisms and processes vary across gender and age.

CONCLUSION

A large body of evidence points to a strong linkage between social relationships and health (Waite, Iveniuk and Laumann 2014). Much of this work considers different types of social relationships and highlights the marital relationship 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 builds on clinical evidence on the importance of marital quality for 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 defines social context, moderating the role that marital quality plays in cardiovascular risk and disease. We find strong evidence that women's cardiovascular health is more sensitive to marital quality than is men's. Given 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 applying an interdisciplinary approach to advance our understanding of the interactions of social and biological processes, as they affect cardiovascular health.

Acknowledgments

In all models, we control for age, 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. We are grateful for comments and suggestions from the anonymous reviewers as well as participants at the conference of Integrated Research in Health and Aging: Early Results from NSHAP Wave 2.

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Biographies

Hui Liu is an associate professor of sociology at Michigan State University. Her research examines how marriage and family processes are related to population health and well-being using innovative quantitative methods. She currently receives a Mentored Research Scientist Development Award (K01) from the National Institute on Aging to study the biological links between marriage and health using interdisciplinary approaches. Her other current

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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.

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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 W1	38.8	Normal W2	30.98
Controlled hypertension W1	20.17	Controlled hypertension W2	25.13
Undiagnosed hypertension W1	15.62	Undiagnosed hypertension W2	20.85
Uncontrolled hypertension W1	25.41	Uncontrolled hypertension W2	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	
57–64	53.6
65–74	32.93
75–85	13.47
Race-ethnicity	
Non-Hispanic white	86.94
Non-Hispanic black	5.57
Hispanic	5.33
Others	2.16
Education	
No diploma	10.13
High school graduate	26.13

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Covariates	
Some college	38.17
College graduate	25.57
Relative family income	
Below average	15.85
Average	39.79
Above average	32.24
Missing	12.12
Psychological distress W1	4.51(4.32)
Smoke W1	
No	89.5
Yes	10.5
Drink W1	
No	38.47
Yes	61.53
BMI W1	
Normal or underweight	27.33
Overweight	41.85
Obesity	28.01
Morbidly obese	2.81
Physical activity W1	
< 3 times per week	36.15
>= 3 times per week	63.85
Antihypertensive medication use W1	
No	48.05
Yes	51.95
Probability of death at W2	0.06(0.06)
Probability of remaining married in both waves	0.44(0.18)

W1: Wave 1. W2: Wave 2.

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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 W1	31.49	Normal W2	25.25
Controlled hypertension W1	20.41	Controlled hypertension W2	26.1
Undiagnosed hypertension W1	17.88	Undiagnosed hypertension W2	19.23
Uncontrolled hypertension W1	30.22	Uncontrolled hypertension W2	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	
57–64	54.54
65–74	30.43
75–85	15.03
Race-ethnicity	
Non-Hispanic white	83.41
Non-Hispanic black	6.12
Hispanic	8
Others	2.47
Education	
No diploma	13.2
High school graduate	21.8

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Covariates	
Some college	29.21
College graduate	35.79
Relative family income	
Below average	17.16
Average	33.04
Above average	34.07
Missing	15.73
Psychological distress W1	4.07(4.23)
Smoke W1	
No	84.53
Yes	15.47
Drink W1	
No	31.22
Yes	68.78
BMI W1	
Normal or underweight	18.4
Overweight	41.79
Obesity	36.04
Morbidly obese	3.77
Physical activity W1	
< 3 times per week	30.14
>= 3 times per week	69.86
Antihypertensive medication use W1	
No	46.44
Yes	53.56
Probability of death at W2	0.11(0.08)
Probability of remaining married in both waves	0.63(0.18)

W1: Wave 1. W2: Wave 2.

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

*** p<0.001,

** p<0.01,

* p<0.05.

a Wald tests: difference between men and women is significant at p<0.05.

b Wald tests: difference between men and women is significant at p<0.1.

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.

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

*** p<0.001,

** p<0.01,

* p<0.05.

^{a}Wald tests: difference between men and women is significant at p<0.05.

^{b}Wald tests: difference between men and women is significant at p<0.1.

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.

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

*** p<0.001,

** p<0.01,

* p<0.05.

^{a} Wald tests: difference between men and women is significant at p<0.05.

^{b} Wald tests: difference between men and women is significant at p<0.1.

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.

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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 vs. normal	Undiagnosed vs. normal	Uncontrolled vs. normal	Controlled vs. normal	Undiagnosed vs. normal	Uncontrolled vs. normal						
Age W1 (ref=57–64)												
65–74	0.42 (0.45)	0.78 (0.41)	0.17 (0.37)	0.38 (0.44)	0.78 (0.40)	0.14 (0.37)	−0.14[b] (0.35)	−0.13[b] (0.34)	−0.50 (0.27)	−0.43 (0.27)	0.84* (0.32)	0.81* (0.32)
75–85	−0.78 (0.59)	0.26[b] (0.69)	−1.09* (0.52)	−0.80 (0.58)	0.21[b] (0.70)	−1.08 (0.55)	0.41 (0.49)	0.32 (0.51)	0.03 (0.43)	0.25 (0.45)	1.19 (0.61)	1.18 (0.61)
PMQ W1	−0.02 (0.16)	0.00 (0.19)	0.10 (0.18)	−0.13 (0.22)	0.00 (0.26)	0.02 (0.25)	−0.09 (0.17)	0.02 (0.22)	−0.17 (0.14)	0.10 (0.19)	0.22 (0.17)	0.14 (0.24)
PMQ W1 X age 65–74				0.38 (0.37)	−0.13 (0.37)	0.32 (0.38)		−0.07 (0.33)		−0.60[a] (0.36)		0.18 (0.34)
PMQ W1 X age 75–85				−0.12 (0.62)	0.41 (0.63)	−0.19[b] (0.73)		−0.61 (0.55)		−0.55 (0.51)		0.13 (0.52)
Change of PMQ W2-W1									0.11 (0.14)	0.39 (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 (1.09)	2.85 (1.47)	1.80 (0.95)	−0.55 (1.10)	2.80 (1.44)	1.83 (0.96)	−2.15 (1.43)	−2.08 (1.40)	−1.26 (1.03)	−1.28 (1.01)	−2.31 (1.51)	−2.28 (1.50)

*** p<0.001,

** p<0.01,

* p<0.05.

[a] Wald tests: difference between men and women is significant at p<0.05.

[b] Wald tests: difference between men and women is significant at p<0.1.

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.

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Table 8

Regression Coefficients for Cardiovascular Risks Predicting Marital Quality from Ordinary Least Squares Regression Models

	NMQ W2		PMQ W2	
	Men	**Women**	**Men**	**Women**
Hypertension W1 (ref=normal)				
Controlled hypertension W1	−0.22* (0.10)	0.15 *a* (0.09)	0.04 (0.08)	−0.18* *b* (0.08)
Undiagnosed hypertension W1	−0.00 (0.10)	0.02 (0.09)	0.00 (0.09)	0.00 (0.10)
Uncontrolled hypertension W1	−0.13 (0.09)	0.07 (0.09)	0.05 (0.09)	0.06 (0.08)
Rapid heart rate W1	−0.13 (0.07)	0.02 (0.08)	0.02 (0.06)	−0.09 (0.06)
CRP W1 (ref: CRP<=3)				
CRP>3	0.22 (0.13)	0.28** (0.09)	−0.16 (0.10)	−0.23* (0.09)
Not applicable	−0.01 (0.07)	−0.03 (0.06)	−0.04 (0.05)	0.12 (0.08)
CVD W1	0.02 (0.07)	0.07 (0.09)	−0.07 (0.05)	−0.13 (0.11)
NMQ/PMQ W1	0.54*** (0.04)	0.71*** *a* (0.04)	0.59*** (0.04)	0.71*** *a* (0.04)
Constant	−0.12 (0.33)	−0.57** (0.20)	−0.05 (0.25)	0.24 (0.22)
N	613	388	613	388
R-squared	0.37	0.53	0.45	0.58

*** p<0.001,

** p<0.01,

* p<0.05.

a Wald tests: difference between men and women is significant at p<0.05.

b Wald tests: difference between men and women is significant at p<0.1.

Standard errors in parentheses.

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