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I've Got You Under My Skin: Marital Biography and Biological Risk

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Abstract

Social relationships shape adult health in profound ways. This study informs our understanding of this association by investigating how the transitions, timing, and exposures to marriage are associated with types of biological risk presumed to serve as pathways to disease and disability. Drawing on the 2005 – 2006 National Social Health and Aging Project ($N = 1,062$), the authors evaluated how marital biography was associated with cardiovascular, metabolic, and chronic inflammation risk. The results showed that the effects of marital biography were highly sensitive to gender, the dimension of marital biography, and type of biological risk. For example, marital exposure was protective of cardiovascular risk for women, but not men, whereas an earlier age at first marriage had a pernicious effect on chronic inflammation among men, but not women. Health behaviors did not explain these associations. The implications of these findings are discussed as they pertain to under-the-skin risk processes and chronic morbidity.

Keywords

aging; health; life course; marriage

Although the life course perspective is a useful paradigm for understanding how health differentials among older adults arise (Halfon & Hochstein, 2002), the importance of social relationships over the life course, especially those pertaining to marriage, in shaping these differentials are often overlooked. We argue that marriage is fundamentally important for understanding life course origins of health because of the consequences of marital status transitions and timing and the importance of marital exposure. Several recent studies illustrate the importance of considering marital biography regarding heart disease, chronic conditions, functional limitations, self-reported health, and all-cause mortality (Dupre, Beck, & Meadows, 2009; Hughes & Waite, 2009; Zhang & Hayward, 2006). Although these studies represent a significant step forward in documenting the importance of marital biography for health outcomes, data limitations have precluded their assessment of possible “under-the-skin” processes that are thought to mediate these relationships. Biological risk measures capture under-the-skin processes involved in the development of several major diseases prevalent among older adults, in particular cardiovascular disease and type 2 diabetes.

This study builds on previous research in that we examined how three types of biological risk—cardiovascular, metabolic, and inflammation—are related to marital biography. Understanding these relationships is important for at least three reasons. First, although biological risk is frequently invoked to explain the marriage – health association, no one has explicitly tested how marital biography is related to biological risk. Evidence that such a relationship exists would strengthen claims that marriage has a direct effect on morbidity and mortality. Second, self-reported health measures in the marriage and health literature may reflect reporting biases. By using biological markers, we traversed this problem. Although work on marital biography and mortality also overcomes this issue, these studies tend to examine all-cause mortality and thus do not capture specific biological pathways. Third, our analyses provide clues as to how marital biography influences the etiology of prevalent health conditions such as cardiovascular disease and type 2 diabetes. For instance, certain systems may be more affected than others by marital biography, or different dimensions of marital biography may differ in their impact on biological risk. Finally, our analyses contribute to an ongoing discussion regarding the differential effects of marriage on health for men and women.

CONCEPTUAL FRAMEWORK

Marital biography refers to key components of the marital life course, including transitions into and out of marriage, age at first marriage, and marital exposure (Hughes & Waite, 2009). Specifically, we addressed five questions concerning the association between these features of marital biography and biological risk. First, to what extent do biological risk profiles among continuously married persons differ in comparison to those whose marriage dissolved and are now remarried or previously married? Second, do multiple dissolutions have more of a pernicious effect on biological risk? Third, how is age at first marriage related to biological risk? Fourth, does the time spent married since first marriage impact biological risk? Finally, to what extent do health behaviors explain these relationships?

Marriage and Biological Risk

Married persons are better off financially, live longer, and experience better mental and physical health than unmarried people (Carr & Springer, 2010; Waite, 1995). Whether marriage has a causal protective effect over health remains an open line of inquiry. Other explanations for these associations include spuriousness and selection. Previous work has shown, however, that accounting for potential confounders that are related to both marital biography and health, such as age, race, education, health behaviors, and religious involvement, does not explain these associations (Waite & Lehrer, 2003). Moreover, because of the increasing prevalence of longitudinal data, researchers have been able to begin addressing the issue of selection. Positive selection into marriage and negative selection out of marriage by persons who are healthier and more socio-economically advantaged does partially explain many of the associations between marriage and health (Fu & Goldman, 1996; Lillard & Panis, 1996). However, a significant body of research provides evidence supporting the idea that marriage exerts a protective effect on health and well-being through the accumulation of economic, behavioral, and psychological resources (Brockmann & Klein, 2004; Carr & Springer, 2010; Waite & Gallagher, 2000).

Burgeoning research also indicates that social relationships may protect or harm cardiovascular, metabolic, neuroendocrine, and immune system functioning (Ryff & Singer, 2001), and the association documented between social relationships and physiological functioning in general likely extends to marriage (Kiecolt-Glaser & Newton, 2001). Evidence from nonrepresentative samples suggests that divorce can alter biological processes (e.g., atherosclerosis), although population-based studies documenting the influence of marital biography on biological processes are sparse (Robles & Kiecolt-Glaser, 2003). In this study, we considered three dimensions of biological risk that capture long-term aspects of health risk: (a) cardiovascular risk, (b) metabolic risk, and (c) chronic inflammation. It is important to recognize that these biological risk markers do not necessarily represent independent processes.

Marital biography potentially influences cardiovascular function in several ways. The *reactivity hypothesis*, for example, predicts that in the presence of repeated stressors the body undergoes a physiological response that affects cardiovascular functioning (Smith & Ruiz, 2002). The sympathetic – adrenal – medullary and hypothalamic – pituitary – adrenal (HPA) axes likely trigger this process via the flow of catecholamines and glucocorticoids (the body's stress hormones) in the presence of a perceived threat. Activation of the stress response results in, among other things, substantially increased heart rate, accelerated breathing, and constriction and dilation of various blood vessels. The ordeal of going through a marital dissolution, as well as dealing with the stressful aftermath of marital dissolution, may repeatedly trigger this response. Also, marriages that ended in divorce may have ended after years of marital conflict. Experimental studies have found that marital conflict increases epinephrine, norepinephrine, and cortisol levels (Malarkey, Kiecolt-Glaser, Pearl, & Glaser, 1994) as well as blood pressure (Ewart, Taylor, Kraemer, & Agras, 1991), although these studies are not representative of the general U.S. population. Conversely, marriage may buffer the harmful effects of stress associated with daily life, thereby reducing cardiovascular reactivity via social support. Using an experimental design, Holt-Lunstad, Birmingham, and Light (2008) showed that, among healthy married couples, warm physical contact was associated with decreased alpha-amylase—an indicator of sympathetic nervous system activity—and increased oxytocin, a hormone thought to reduce stress responses. This protective support may accumulate over time and become noticeable in midand late life vis-à-vis biological risk.

Perhaps just the perception of available support from one's spouse lowers cardiovascular reactivity. In one experimental study, the researchers presented participants with a public speaking challenge in which one group was offered support, if needed, and the other was not. No support was actually available, but the group that thought support was available recorded lower systolic and diastolic blood pressure (Kamarck, Manuck, & Jennings, 1990). Also, married persons tend to live healthier lifestyles that include less smoking and drinking, two risk factors consistently associated with cardiovascular disease.

Marital biography is associated with numerous factors that may affect the body's metabolic system, such as social support, negative emotions, a sense of control, and financial strain. For instance, the financial strain evoked from marital dissolution may lead to a regular activation of the body's HPA axis, which releases glucocorticoids, such as cortisol. Chronic

exposure to cortisol has many effects on the metabolic system, such as the production of excess glycogen and the suppression of insulin and hence increased blood sugar (McEwen & Lasley, 2002). Moreover, evidence is growing that changes in the HPA axis due to prolonged stress can lead to the dysregulation of appetite and visceral fat distribution (Adam & Epel, 2007). For these reasons, repeated activation may raise the risk for obesity and type 2 diabetes. Marital biography may also affect the metabolic system through lifestyle factors such as physical activity. At least some evidence suggests that the transition into marriage is associated with weight gain and a reduction in physical activity, which may offset some of marriage's protective influence (Wade & Pevalin, 2004).

Chronic inflammation may contribute to the development of many health conditions, such as cardiovascular disease, type 2 diabetes, and certain types of cancers. Marital biography may have a strong impact on chronic inflammation through increased social support (or perceived support), exposure to stressors, emotions, mental health, and health behaviors. Many of these factors have been tied with chronic inflammation in prior research. For instance, proinflammatory cytokine (e.g., interleukin-6) production can be triggered by negative emotions and stressful experiences (Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002). Moreover, persistent stressors have been shown to prompt chronic inflammation even after the cessation of stress. For example, Kiecolt-Glaser and colleagues (2003) found that the caretaking of a spouse resulted in higher levels of interleukin-6—a precursor to the production of C-reactive protein (CRP)—over time among both current and former caretakers. Marriage may also influence the inflammation through the social support received over the life course. At least one nationally representative study provides evidence for this relationship between marriage and chronic inflammation: Sbarra (2009), using data from older adults, found that being married reduced the risk of elevated CRP levels for men, but not for women.

The physiological processes alluded to above, in particular HPA axis dysregulation, that are argued to link marriage and biological risk are long-term interactive processes. Each dimension of marital biography considered in our study likely has long-term effects on people's lives. For example, although marital dissolutions are often thought of as acute stressors, they actually represent a constellation of both acute and chronic stressors that can occur long before a dissolution (e.g., conflict leading to divorce) and continue long after the dissolution (e.g., financial strain). Moreover, the social and physiological results of different aspects of marital biography likely work quite interactively to extend beyond the domain of marriage. For instance, repeated HPA axis activation is thought to both decrease the efficacy of the negative feedback loop responsible for shutting off the stress response—via decreased glucocorticoid receptors in the brain—and increase sensitivity to HPA activation of the stress process, via heightened amygdala functioning (McEwen & Lasley, 2002). In other words, individuals who experience repeated HPA activation are more likely to become, and remain, physiologically stressed. Such tendencies may have a harmful effect on other areas of life (e.g., work, nonmarital social relationships). Detriments in these social domains may lead to deterioration of psychosocial resources, such as a sense of control, that in turn produce even more HPA axis activation and thereby reinforce the processes that influence cardiovascular, metabolic, and inflammation risk mentioned in the preceding paragraphs.

Marital Biography and Biological Risk

Marital dissolution is associated with a variety of social stressors that lead to deleterious health behaviors and outcomes (Wang & Amato, 2000), but less is known regarding whether marital loss continues to exert a noxious effect even after remarriage. A small literature on the topic suggests that this is the case. For example, Nystedt (2006), using 12 years of longitudinal data from Sweden, reported that remarried individuals were more likely to smoke and less likely to quit smoking than those who remained continuously married. Other studies have also documented more chronic conditions, functional limitations, and depressive symptoms, as well as a higher prevalence of cardiovascular disease, among the remarried compared to the continuously married (Hughes & Waite, 2009; Zhang & Hayward, 2006). Transitioning into marriage can alter health through several pathways, such as improved health behaviors and mental health or increases in health care consumption. Marital dissolution may foster deleterious health behaviors, such as smoking and drinking, which remain after remarriage. On the basis of these arguments, we formulated our first and second hypotheses:

Hypothesis 1: Previously married individuals will have higher levels of biological risk than those who are currently married.

Hypothesis 2: Remarried individuals will have higher levels of biological risk than the continuously married.

The dissolution of a marriage, either through divorce or widowhood, is a major life event that affects health and well-being. The literature on life events and health provides considerable evidence that negative life events are associated with poor health outcomes. For example, among participants exposed to a cold virus, those who reported more negative life events were more likely to develop a cold than others (Cohen, Tyrrell, & Smith, 1993). In terms of marital events, multiple dissolutions were positively associated with functional limitations and future mortality (Dupre, Beck, & Meadows, 2009; Hughes & Waite, 2009). There are numerous ways marital dissolutions can influence health. For example, one or more marital dissolutions may foster a lower sense of control over one's life. Individuals with a lower sense of control tend to be more inclined to engage in unhealthy lifestyles and are especially susceptible to the effects of stressors (Mirowsky & Ross, 2003). Indeed, each marital disruption may lead to a variety of acute and chronic stressors, such as financial strain, emotional distress, and conflict in personal relationships. On the basis of these arguments, we put forth our third hypothesis:

Hypothesis 3: Individuals with more than one marital disruption will have higher biological risk than those with one or zero disruptions, net of current marital status.

Early marriage has deleterious consequences that reverberate over the life course and proliferate with time. Marrying at younger ages is associated with lower levels of education, income, and wealth; more marital conflict and instability; and worse mental health (Amato & Rogers, 1997; Dahl, 2010; Wilmoth & Koso, 2002). To the extent that the effects of age at first marriage extend out over the life course, they may also influence biological risk. For instance, to the extent that a college degree is associated with less marital conflict or financial strain (two factors thought to prompt biological stress responses), age at marriage

may have a subsequent effect on biological risk at older ages. The consequences of early marriage also extend to health behaviors and mental health. Marriage at younger ages often precedes an early age at first birth; age at first birth, in turn, is associated with pernicious health-related outcomes such as higher alcohol use and depressive symptoms later in adulthood (Mirowsky & Ross, 2002; Wolfe, 2009). On the basis of these arguments, we formulated our fourth hypothesis:

Hypothesis 4: Individuals with a lower age at first marriage will have higher levels of biological risk than those who married at later ages.

The amount of time one spends married since his or her first marriage may influence health beyond both marital status and the number of dissolutions. The cumulative time spent married potentially represents the accrued interest from increased social support, decreased financial strain, a more orderly lifestyle, and health care consumption. The accumulated time married, for example, has been shown to be protective of chronic conditions, functional limitations, and mortality net of marital status and number of dissolutions (Dupre et al., 2009; Hughes & Waite, 2009; Pienta, Hayward, & Jenkins, 2000). Moreover, research in other national contexts using linked census and death records also suggests that marital exposure is protective over mortality (Blomgren, Martikainen, Grundy, & Koskinen, 2012; Grundy & Tomassini, 2010). On the basis of these arguments, we put forth our fifth hypothesis:

Hypothesis 5: Individuals with more years of marital exposure age will have lower biological risk than those with less exposure.

Health behaviors represent one key pathway by which marital biography can influence biological risk. In particular, smoking and heavy drinking are thought to lead to increased biological risk, including hypertension and elevated glycosylated hemoglobin, body mass, and CRP levels (Eckel, Grundy, & Zimmet, 2005; Puddey & Beilin, 2006; Volpato et al., 2004). Conceptually, each dimension of marital biography that we consider potentially has a positive or negative influence on various health behaviors that directly affect biological risk. In order to test the extent to which health behaviors serve as a pathway from marriage to health, it seems important to account for nonbeneficial health behaviors associated with marriage, such as physical activity. On the basis of these arguments, we put forth our final hypothesis:

Hypothesis 6: The effects of marital biography on biological risk will operate in part through health behaviors.

The literature is split as to whether men or women garner greater benefit from marriage (e.g., Waite, 1995; K. Williams & Umberson, 2004; Zhang & Hayward, 2006). For instance, K. Williams and Umberson showed that marital dissolution undermined the self-reported health of men, but not of women, whereas Zhang and Hayward (2006) reported that marital dissolution increased the incidence of cardiovascular disease for middle-aged women, but not men. Others, however, have found no gender differences for a host of health outcomes (Hughes & Waite, 2009; Pienta, Hayward, & Jenkins, 2000). Trying to understand these differences is complicated by distinctive study designs and multiple health outcomes. More research is required to understand these complex patterns. Our study contributes to this

ongoing discussion by examining gender differences in the effect of marital biography on biological risk.

METHOD

Survey

The analyses are based on data from the first wave of the 2005 – 2006 National Social Life, Health, and Aging Project (NSHAP). Respondents were selected at random from a nationally representative multistage area probability sample of U.S. households, and the data were gathered between July 2005 and March 2006. NSHAP is nationally representative of the U.S. civilian noninstitutionalized population age 57 through 85 ($N = 3,005$). Detailed information about the NSHAP sample design is available elsewhere (O’Muircheartaigh, Eckman, & Smith, 2009). This study is uniquely qualified to test the relationships between marital biography and biological risk because information was gathered both for individuals’ marital histories and biomarkers of biological risk. In addition to in-home interviews and take-home questionnaires, anthropometric measurements and blood, salivary, and vaginal mucosal specimens were taken in the home. The survey had an unweighted response rate of 75%, and among those in the survey 83% were randomly asked to provide blood spots, of whom 85% agreed.

In an effort to minimize the effects of mortality selection and match the age of our sample to recent relevant studies, we restricted our analyses to respondents age 75 or younger. The number of respondents with blood spots (and other biomarkers) who were 75 years or under was 1,241. This analysis excluded the never-married and those currently living with a partner. The number of never-married and cohabiting men and women in our sample is small and would not provide enough statistical power to make any meaningful claims about these demographics. Moreover, prior research on marriage and mortality in the United States suggests that never-married older persons are a very select group; this is particularly true for older never-married men (Liu, 2009; Zhang & Hayward, 2006). As Zhang and Hayward pointed out, it is plausible that the never-married men who manage to survive to older ages are an especially select group because never-married men at older ages have a much higher risk of death than do ever-married men. Individuals who had a CRP value greater than 10 mg/L were also dropped from the analysis because values above 10 typically indicate acute rather than chronic inflammation. After using listwise deletion of missing values to deal with item nonresponse, complete data were available for 528 women and 534 men for a total sample size of 1,062. The NSHAP data use a complex survey design to account for oversamples of Blacks, Hispanics, and men. Accordingly, the analyses used in this study are weighted and adjust for clustering.

Biological Indicators

Following prior research (Seeman et al., 2008), we classified individuals as “high risk” (1 = *high risk*, 0 = *not high risk*) for each indicator using the risk criteria shown in Table 1. These dummy variables were summed to give count variables for cardiovascular and metabolic risk. Chronic inflammation was based solely on having high levels of CRP. Our study therefore contained three dependent variables: (a) cardiovascular risk (range: 0 – 3), (b)

metabolic risk (range: 0 – 2), and (c) chronic inflammation risk (range: 0 – 1). Because of the small number of cases that had three cardiovascular risk factors, we collapsed these individuals into the two-risk-factor category, giving a new range (0 – 2). Measures were constructed only for respondents with valid data for all biological indicators.

Marital Biography

We identified marital status by creating five mutually exclusive dummy variables. Respondents who had never experienced a marital dissolution are referred to as *continuously married*. Among currently married respondents, we distinguished those who had experienced one marital dissolution (*remarried 1 dissolution*) from those who had experienced two or more dissolutions (*remarried 2+ dissolutions*). Among those who were not married we distinguish those who had witnessed one dissolution (*previously married 1 dissolution*) from those who had witnessed two or more dissolutions (*previously married 2+ dissolutions*). The cumulative length of time a respondent has been married since his or her first marriage was calculated for all respondents to give a measure of marital exposure that is referred to as *decades married*. Last, we determined from a person's reported age and marital history his or her *age at first marriage*.

Health Behaviors

We measured smoking status with two items that asked "Do you smoke cigarettes now?" and "Have you ever smoked cigarettes regularly?" On the basis of responses to these items we constructed three dichotomous smoking indicators: (a) *current smoker*, (b) *former smoker*, and (c) *nonsmoker*. Alcohol consumption was based on two items that asked "Do you drink any alcoholic beverages?" and "In the last three months, on how many days have you had four or more drinks?" On the basis of responses to these items we constructed three dichotomous indicators of alcohol consumption: (a) *heavy drinker*, (b) *light drinker*, and (c) *nondrinker*. In our analyses, respondents who reported having four or more drinks in one sitting during the last 3 months are considered heavy drinkers, and respondents who drank but did not ingest four or more drinks in one sitting are considered light drinkers. Finally, we included measures of physical activity based on an item that asked "How often do you participate in physical activity such as walking, dancing, gardening, physical exercise or sports?" These include *low* (<4 times/month), *moderate* (1 – 2 times/week), and *high activity* (≥ 3 times/week).

Controls

We included several controls for age (years), race/ethnicity, highest education degree, and religious attendance. Race/ethnicity consisted of four dummy variables indicating whether the respondent was *White*, *Black*, *Hispanic*, or *other*. The highest education degree was broken down into five dummy variables indicating whether the respondent had *less than a high school degree*, *a high school degree*, *associate's degree*, *bachelor's degree*, or *a master's degree or higher*. Respondents were also asked how often they had attended religious services in the last 12 months, with responses ranging from 0 (*never*) to 6 (*several times a week*).

Analytical Strategy

We began by examining the distribution of biological risk and health behavior measures and how they vary by marital status. Next, we examined the bivariate correlations for all variables in the study. We then investigated how marital biography is associated with biological risk through the use of multivariate modeling. We estimated three gender-specific nested models. The first set of models establishes the total association between each dimension of marital biography and biological risk. The second adds basic demographic controls to the previous model. The third adds health behaviors to the previous model in order to test the extent to which the relationship between marital biography and biological risk was explained by health behaviors. We estimated models examining cardiovascular and metabolic risk using Poisson regression, and we used logistic regression to estimate models examining inflammatory risk. Odds ratios are presented for all models. The odds ratio can be interpreted as a unit increase in the independent variable produces a percentage change in the expected count or odds. For example, analyses employing Poisson regression can be interpreted such that, for all variables, coefficients above 1 produce a $(\beta - 1) \times 100$ percentage increase in the expected count, whereas coefficients below 1 produce a $(1 - \beta) \times 100$ percentage decrease in the expected count.

We performed several ancillary analyses using different model specifications as well as different modeling techniques. All analyses were replicated using various other covariates, including measures of fertility history and additional measures of socioeconomic status. NSHAP contains items asking about the number of live children, live births (women only), and household income and wealth. All of these measures were excluded from the final models because they had a nontrivial number of missing cases. Even with this loss of statistical power, all marital biography effects paralleled those reported here in regard to statistical significance and relative magnitude. When examining cardiovascular and metabolic risk, we replicated our analyses using negative binomial and ordinal regression techniques. These models produced results consistent with all of the substantive conclusions reported in the text. Note that the models do not include current age as a covariate because current age, age at first marriage, and marital exposure are linearly dependent among the continuously married. In preliminary analyses, we explored several alternative specifications of the models that controlled current age, but doing this did not alter our substantive conclusions. We also explored the possibility that the effects of the health behavior variables were canceling each other out. However, when we included each of the health behavior measures separately in Model 3, the results did not differ meaningfully from the ones we present.

RESULTS

How is marital status related to biological risk and health behaviors? The distributions of these factors, reported as the percentage of individuals in each category for each marital status, are reported in Tables 2 and 3. For example, Table 2 shows that 57.2% of continuously married women had zero cardiovascular risks. Among this sample, approximately 79% of respondents had at least one biological risk factor (results not shown). Table 2 provides some evidence to suggest that remarried women with two disruptions and

previously married women had higher levels of biological risk than their continuously married counterparts. Specifically, the percentage of remarried women with multiple disruptions who had no cardiovascular risks (34.6%, $p < .10$) was lower than the continuously married women (57.2%). Moreover, a significantly higher percentage of women in this group had two risks (42.3%) relative to their continuously married counterparts (15.8%). The percentage of previously married women with one or more disruptions who had zero metabolic risks was lower than the continuously married women (27.2% and 20.3% vs. 46.2%), and the multiple-disruptions group had a higher percentage of individuals with two risks (35.6% vs. 13.0%). Chronic inflammation was distributed similarly among women in each marital status group. The percentage of remarried women with one disruption who never smoked was lower than continuously married women (33.3% vs. 57.2%). Similarly, when compared with their continuously married counterparts, a higher percentage of previously married women who had experienced multiple disruptions currently smoked (30.5% vs. 11.5%, $p < .10$). Previously married women with one disruption were less likely to drink than those who were continuously married. The distribution of physical activity did not vary by marital status. Overall, Table 2 suggests that some modest differences exist in the distribution of biological risk and health behaviors across marital status among women.

Among the male sample, approximately 82% of respondents had at least one biological risk factor (results not shown). As shown in Table 3, the distribution of biological risk variables generally did not vary by marital status among men. The results suggest some possible exceptions to this general pattern, however. Although marginally significant, the results in Table 3 suggest that, compared to continuously married men, a higher percentage of previously married men with two disruptions had zero metabolic risks. Similarly, there is limited evidence to suggest that chronic inflammation was more prevalent among previously married men with one marital disruption in comparison to continuously married men (34.4% vs. 22.3%, $p < .10$). There is some evidence to suggest that previously married men with one disruption were more likely to smoke and that men with two disruptions were more likely to fall into the heavy drinking category than continuously married men. Continuously married and remarried men had similar distributions for all variables. Overall, Table 3 suggests that continuously married men had little advantage in regard to biological risk than had others, but they had more optimal health behaviors than previously married men.

The bivariate relationships among all variables used in this study are displayed in Table 4, with the correlations for women falling below the diagonal and those for men above the diagonal. This table displays modest associations between various dimensions of marital biography and biological risk, marital biography and health behaviors, and health behaviors and biological risk for both women and men. The magnitudes of these associations vary by the dimension of marital biography or biological risk and by gender. The largest bivariate correlation coefficient existed between age at first marriage and marital exposure among men ($\rho = -.50$); thus, multi-collinearity is unlikely to present a problem.

Table 5 reveals how marital biography was related to each of the three measures of biological risk among women. The baseline model for cardiovascular risk shows that decades married was negatively associated with cardiovascular risk such that a unit increase

was approximately associated with a 13% decrease in the expected risk count. The standardized odds ratio (not shown) reveals that a standard deviation change of 1.32 decades (or 13.2 years) was associated with an 18% decrease in the expected count. Being remarried with multiple dissolutions was associated with a 40% increase in the expected risk count compared to continuously married respondents ($p < .10$). The magnitude of this coefficient was not statistically larger than that for remarried individuals with only one disruption. When the demographic controls were added to Model 2, the coefficient for remarried respondents with multiple dissolutions dropped below marginal significance, but the association between decades married and biological risk did not change. The inclusion of health behaviors seen in Model 3 did not change the effect of decades married. The model fit improved from the previous model based on a likelihood ratio test. Overall, evidence for Hypothesis 5 was found regarding cardiovascular risk.

Model 4 reveals that previously married women who experienced multiple disruptions were disadvantaged in regard to metabolic risk such that being in this category was associated with a 41% increase in the expected risk count compared to the continuously married. A comparison of the coefficients in Model 4 suggested that there are no statistically significant differences in the magnitude of the associations between continuously married and remarried women who experienced one marital dissolution. Moreover, the demographic controls introduced in Model 5 did not meaningfully influence the association between being previously married with multiple disruptions and risk. Introducing health behaviors in Model 6 did not change the association between multiple marital disruptions and biological risk, but the effect of decades married on biological risk became marginally significant ($p < .10$) such that a decade of marriage was associated with a 7% decrease in expected risk. Health behaviors were important as the model fit improved from Model 5. Overall, the results for women uncovered some evidence for Hypothesis 1. Models 7 through 9 show that all dimensions of marital biography were unrelated to chronic inflammation. Of note is that no evidence was found for any of the study hypotheses regarding chronic inflammation among women.

Table 6 reveals how marital biography was related to each of the three measures of biological risk among men. Although marginally significant, the baseline model suggests that age at first marriage was negatively associated with cardiovascular risk such that each additional year increase was associated with a 2.2% decrease in the expected biological risk ($p < .10$). For instance, according to this finding, a man first married at 23 will have an 11% lower expected risk count than a man first married at 18—*ceteris paribus*. The effect of age at first marriage was not influenced by the inclusion of demographic and health behavior variables seen in Models 2 and 3. The other marital biography components in Model 3 were unrelated to cardiovascular risk. Models 4 through 6 show the same rather modest finding regarding age at first marriage. One unexpected finding is that the remarried with one marital disruption were found to have lower levels of metabolic risk than the continuously married by approximately 21%.

Health behaviors did not account for these relationships. For men, the relationship between marital biography and chronic inflammation was similar to cardiovascular and metabolic risk. Age at first marriage was statistically different from zero and was associated with a 6%

drop in the odds of chronic inflammation per year of delayed age at first marriage. This implies that a 5-year delay in age at first marriage was associated with a more than 30% decrease in the estimated odds of chronic inflammation. There is also evidence that individuals with multiple disruptions experienced lower levels of chronic inflammation, although these effects were only marginally significant. Overall, these results provide support that men who married at younger ages were more likely to witness chronic inflammation than those who married at later ages (Hypothesis 4). We found no evidence for any of the other hypotheses among men.

DISCUSSION

This research addressed the relationship between marital biography and under-the-skin biological risk. Overall, the evidence connecting marital biography to biological risk points to the specificity of the results, because key associations differ depending on different dimensions of marital biography and biological risk as well as gender. For women, evidence for Hypothesis 1 was found regarding metabolic risk and evidence for Hypothesis 5 was found vis-à-vis cardiovascular risk. Evidence was found for Hypothesis 4 among men in regard to inflammation risk and, to a lesser extent, cardiovascular and metabolic risk.

These findings suggest that marital biography gets under the skin through different mechanisms and into different bodily systems over different time scales. For example, it appears that marital exposure may be especially important for cardiovascular risk. The *cumulative exposure model* suggests that the body remembers the psychosocial insults experienced over a lifetime (Davey, Ben-Shlomo, & Lynch, 2002). The increased emotional, perceived, and actual social support available from marriage may accumulate over time to buffer the insults the body receives from the daily hassles of life. This finding is consistent with the hypothesis that life marital histories, in particular exposure, are related to health dimensions that develop slowly, such as cardiovascular disease (Zhang & Hayward, 2006).

In contrast to the impact of exposure for women's cardiovascular risk, nonmarried women who had experienced multiple disruptions were disadvantaged vis-à-vis metabolic risk. This pattern may indicate that marital transitions influence the metabolic system over relatively short periods of time compared to cardiovascular risk; that is, the time scales for the development of these types of biological risk may differ. Indeed, marital transitions may prompt fairly rapid changes in diet and physical activity (Umberson, Liu, & Powers, 2009). Transitions into and out of marriage may influence metabolic risk on the time scale of years, or perhaps months, not decades as with marital exposure and cardiovascular risk. Although the idea of different time scales of risk development seems plausible, it should be tempered by the fact that previously married individuals with one disruption did not have higher levels of risk than the continuously married.

For men, an earlier age at first marriage was associated with higher biological risk. This finding was consistent with a small body of work that detected the same pattern for various health outcomes, including chronic conditions, functional limitations, psychiatric disorders, and mortality (Dupre et al., 2009; Forthofer, Kessler, Story, & Gotlib, 1996; Hughes & Waite, 2009). We have argued that age at marriage influences biological risk in older

adulthood because the consequences tied to an early or late first marriage proliferate across the life course (e.g., acquisition of wealth). This relationship, however, may reflect the causal effect of other factors correlated with age at marriage (e.g., childhood environment), although at least one study, which used an instrumental-variables approach, provided compelling evidence that this is not the case. Specifically, Dahl (2010) reported that marriage at younger ages is likely at least partly causal in shaping future education and poverty status and hence, possibly, future biological risk.

Overall, the results suggest that marriage is more protective for women than men. Although the weight of the evidence in the field seems to suggest that marriage is more protective for men, at least three prominent studies that investigated marital biography did not find marriage to be more protective for men (Dupre et al., 2009; Hughes & Waite, 2009; Zhang & Hayward, 2006). Our results, in fact, parallel those of Zhang and Hayward in the sense that marital biography mattered for women's cardiovascular risk but not for men's risk. Zhang and Hayward suggested that men and women may be at very different points in the disease process and that women may be more vulnerable in terms of biological risk at this point in their life. For example, women's menopause may make them more vulnerable in their 50s and 60s compared to men, and this might shape gender differences in cardiovascular risk. Men are also further along in the disease process than women, given their different hormonal exposures (Crimmins, Hayward, Ueda, Saito, & Kim, 2008). One may therefore see different patterns of relations in different age groups. Moreover, to the extent the gender differences in biological vulnerability are playing an important role, it behooves social scientists to more carefully understand the intersection of social structured life course biographies and sex differences in biomedical processes.

There are several factors to consider that may explain the lack of a more demarcated relationship between marital biography and biological risk given that the relationship between marital biography and health and mortality tends to be more pronounced (Waite, 1995). First, most population-level surveys using biomarkers, like ours, were collected at one point in time. The effect of marital biography on changes in biological risk may share a very different relationship than levels in biological risk. Medical research shows that changes in biological risk are particularly important for the development of clinical disease (Briel et al., 2009; LaRosa, He, & Vupputuri, 1999). Second, these snapshot associations do not reveal how marital biography affects the onset and progression of biological dysregulation. Marital biography may influence the onset of risk differently than the progression of dysregulation that may, in turn, have different effects on health. Similarly, on the basis of National Health and Nutrition Examination Survey data—the gold standard of population-level biomarker data—Crimmins, Kim, and Vasunilashorn (2010) showed that the extent to which biomarkers predict morbidity and mortality differs by gender, age, and socioeconomic status. Therefore, the influence of biological risk on morbidity and mortality may also differ by marital biography. Our focus on biological risk levels is the first step in a long-term agenda to understand how marital status transitions influence morbidity and mortality via biological risk trajectories.

Third, we emphasize that selection processes are of the utmost importance. In order to truly understand the pathways connecting marriage to biological risk, it is imperative that

researchers untangle how selection and assortative mating processes influence the marriage and health relationship. For instance, because women live longer than men, which shapes marriage markets in men's favor, might the healthiest women be the most likely to remarry? Conversely, older unmarried men with health problems may be equally or more likely to marry than their healthy counterparts. Might remarriage among women possibly entail copious caretaking responsibilities that actually erode health? In short, researchers know relatively little about these processes. Much work remains to uncover not only how marital biography influences health but also the role health plays in marriage markets among older adults. Although research is sparse in this area, at least one study found that healthy women were more likely to date than their less healthy counterparts; among men, however, there was no difference between the healthy and unhealthy (Bulcroft & Bulcroft, 1991). Although speculative, this may suggest that healthier women are more likely to remarry, but both healthy and unhealthy men are equally likely to marry, although we found that remarried men had lower metabolic risk. The next frontier in marriage and health research must account for the roles of assortative mating, marital markets, and mortality selection by using multiwave longitudinal data with large sample sizes. In order to unveil the processes connecting marriage, biological risk, and health, these topics must be rigorously addressed.

Finally, there are a number of measurement issues that are particularly important for this study and likely population-level biomarker research more broadly. First, the biomarkers collected were neither the only nor the optimal markers to reflect physiological functioning but were the best available using an in-home collection procedure (S. R. Williams, Pham-Kanter, & Leitsch, 2009). Second, there are a number of ways in which population-level biomarker collection can lead to inaccurate measurement. Validation studies, however, have shown a close association between biomarkers collected using both population-level and venipuncture procedures (see McDade, Williams, & Snodgrass, 2007, for an overview). Third, available biomarker data provided an unequal assessment of different physiological systems, ranging from 1 (inflammation) to 3 (cardiovascular). Our results, for example, suggest that, for women, the cardiovascular and metabolic systems were affected by marital biography, but inflammation was not. This may represent the underlying relationship or represent the fact that inflammation was measured with a sole indicator (CRP). Fourth, because of the study's cross-sectional design, we could make only between-person comparisons. The "normal" range for biological indicators may vary both within and between persons, so additional biomarkers at different times would be useful. Finally, our scoring system of biological risk using clinically defined cutpoints was relatively crude as information on gradients was lost, although others have shown the usefulness in employing this type of measurement (Seeman et al., 2008). This latter point is of particular importance because theories of biological dysregulation suggest a gradual process (McEwen & Lasley, 2002). Testing the functional forms marital biography shares with biological risk and formulating their conceptual underpinnings should be a high priority.

This study has other limitations that deserve mention. First, the average age of this sample was over 65 years. Men tend to have higher mortality at these ages than women; hence, selection through mortality may have muted the relationship between marital biography and biological risk for this reason. Second, gender-reporting biases may have confounded our results. For instance, women may have been more accurate in reporting marital histories.

Third, selection factors may have influenced our results. For example, our sample excluded institutionalized older adults.

This study represents the first nationally representative investigation of marital biography and biological risk. These findings provide some evidence for the commonly invoked argument that biological risk serves as a pathway from marriage to health. The important point here is that biological risk in old age was related to transitions into and out of marriage decades earlier, which highlights the importance of considering temporally distal factors in explaining why people differ in biological risk. This study represents an initial step toward understanding how marital biography influences trajectories of biological risk and the pathways by which marriage gets under the skin to shape health.

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Table 1**High-Risk Criteria for Biological Risk**

Variable	High-Risk Cutpoint
Cardiovascular risk	
Systolic blood pressure (mmHg)	140 (Chobanian et al., 2003)
Diastolic blood pressure (mmHg)	90 (Chobanian et al., 2003)
Resting heart rate (beats/minute)	90 (Seccareccia et al., 2001)
Metabolic risk	
Waist circumference (inches)	35 Women, 40 men (National Institutes of Health, 1998)
Glycosylated hemoglobin (%)	6.4 (Golden et al., 2003)
Inflammation	
C-reactive protein (mg/L)	3.1 (Ridker, 2003)

Table 2
Distributions of Biological Risk and Health Behavior Variables Among Females ($N = 528$)

Variable	Continuously Married ($n = 208$) ^a	Remarried 1 Disruption ($n = 66$)	Remarried 2+ Disruptions ($n = 26$)	Previously Married 1 Disruption ($n = 169$)	Previously Married 2+ Disruptions ($n = 59$)
Cardiovascular risk					
0	57.2	56.1	34.6 [†]	53.3	37.3 [*]
1	26.9	19.7	23.1	25.4	33.9
2	15.8	24.2	42.3 [*]	21.3	28.8
Metabolic risk					
0	46.2	47	46.2	27.2 [*]	20.3 [*]
1	46.9	40.9	50	52.7	44.1
2	13	12.1	3.9	20.1	35.6 [*]
Inflammation risk					
0	72.6	78.8	76.9	70.4	62.7
1	27.4	21.2	23.1	29.6	37.3
Smoking					
Nonsmoker	57.2	33.3 [*]	26.9	56.2	40.7
Former smoker	31.3	51.5 ^b	46.2 ^b	32	28.8
Current smoker	11.5	15.2	26.9	11.8	30.5 [†]
Drinking					
Nondrinker	43.8	42.4	30.8	61.5 [*]	52.5
Light drinker	50	40.9	53.9	33.1 [*]	39
Heavy drinker	6.3	16.7	15.4	4.7	8.5
Physical activity					
Low	20.2	24.2	11.5	21.9	20.3
Medium	19.7	10.6	19.2	16.6	20.3
High	60.1	65.2	69.2	64.5	59.3

Note: Values are the percentage of respondents in each category.

^a Each marital group is compared with the continuously married.

d Indicates a percentage difference between the remarried with one disruption and the remarried with two disruptions ($p < .05$).

$\dagger p < .10$.

$* p < .05$ (two-tailed) based on a proportion difference test.

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Table 3
Distributions of Biological Risk and Health Behavior Variables Among Males ($N = 534$)

Variable	Continuously Married ($n = 292$) ^a	Remarried 1 Disruption ($n = 107$)	Remarried 2+ Disruptions ($n = 38$)	Previously Married 1 Disruption ($n = 64$)	Previously Married 2+ Disruptions ($n = 33$)
Cardiovascular risk					
0	48.6	49.5	57.9	48.4	33.3
1	32.9	29	21.1	31.3	33.3
2	18.5	21.5	21.1	20.3	33.3
Metabolic risk					
0	35.6	40.2	31.6	39.1	51.5 [†]
1	44.9	44.9	60.5	45.3	30.3
2	19.5	15	7.9	15.6	18.2
Inflammation risk					
0	77.7	82.2	86.8	65.6	84.9 ^b
1	22.3	17.8	13.2	34.4 [†]	15.2
Smoking					
Nonsmoker	32.2	35.5	23.7	29.7	30.3
Former smoker	54.1	49.5	57.9	35.9 [*]	45.5
Current smoker	13.7	15	18.4	34.4 [*]	24.2
Drinking					
Nondrinker	37.7	30.8	29	32.8	21.2
Light drinker	42.8	42.1	44.7	40.6	30.3
Heavy drinker	19.5	27.1	26.3	26.6	48.5 ^{*b}
Physical activity					
Low	11.6	10.3	15.8	21.9	9.1
Medium	18.5	19.6	5.3	9.4	18.2
High	69.9	70.1	79	68.8	72.7

Note: Values are the percentage of respondents in each category.

^a Each marital group is compared with the continuously married.

Indicates a percentage difference between the previously married with one disruption and the previously married with two disruptions ($d = .05$).

$d = .10$
* Indicates a percentage difference based on a proportion difference test.

Table 4

Correlation Matrix of All Study Variables Among Women (Below Diagonal, $n = 528$) and Men (Above Diagonal, $n = 534$)

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23
1. Cardiovascular	—	0.11	0.17	0	-0.03	0	0.09	-0.08	-0.06	-0.07	0.06	0.16	-0.09	0.02	0.05	0.1	0	-0.05	0.03	0.03	-0.04	-0.09	-0.06
2. Metabolic	0.15	—	0.19	-0.03	-0.01	-0.02	-0.05	-0.05	0.04	-0.05	-0.06	-0.07	-0.05	0.12	0.16	0.06	0.04	0.02	0.06	0.02	-0.07	-0.10	0
3. C-reactive protein	0.09	0.24	—	-0.05	-0.06	0.11	-0.04	-0.03	-0.04	0.01	0.11	-0.03	-0.05	0.02	0.11	0.16	0.04	0.04	-0.03	-0.03	-0.11	-0.04	-0.01
4. Remarried 1	0	-0.08	-0.06	—	-0.14	-0.18	-0.13	-0.05	-0.03	-0.01	-0.03	0.03	0	-0.04	0.04	-0.01	0.04	0.05	0.03	-0.03	-0.02	0.01	-0.01
5. Remarried 2+	0.11	-0.07	-0.03	-0.09	—	-0.10	-0.07	-0.12	-0.05	0.04	0.01	0.01	0.02	0.03	-0.08	-0.03	-0.04	-0.02	-0.04	0.15	0.05	-0.07	-0.10
6. Prev. married 1 disrupt.	-0.01	0.13	0.02	-0.26	-0.16	—	-0.09	0.07	-0.38	-0.11	0.17	0.02	-0.01	0.1	-0.07	0.15	-0.02	0	0	-0.06	0.01	-0.03	-0.11
7. Prev. married 2+ disrupt.	0.1	0.18	0.07	-0.13	-0.08	-0.24	—	-0.07	-0.18	-0.03	0.05	0.15	-0.06	-0.03	0.01	0.03	-0.07	-0.02	0.01	-0.01	0.01	-0.01	-0.14
8. Age at first marriage	-0.02	-0.01	-0.02	-0.07	-0.10	0.06	-0.13	—	-0.50	-0.03	-0.05	-0.03	0	-0.03	0.07	0.1	0.05	0.05	-0.08	-0.05	0.01	0.06	-0.01
9. Marital exposure	-0.15	-0.20	-0.06	0.02	0	-0.37	-0.27	-0.27	—	0.13	-0.16	-0.11	-0.01	-0.03	-0.04	-0.16	-0.03	-0.01	0.09	-0.02	-0.07	0.03	0.16
10. Former smoker	0	-0.01	-0.01	0.14	0.06	-0.04	-0.04	-0.03	-0.01	—	-0.47	0.05	-0.02	-0.02	0.05	-0.08	-0.03	-0.04	0.03	-0.04	-0.09	0.05	-0.02
11. Smoker	0.06	-0.05	0.08	0	0.08	-0.06	0.15	0.01	-0.10	-0.30	—	0.09	-0.12	0.09	-0.05	0.14	-0.07	0.02	0	0.04	0.05	-0.15	-0.18
12. Heavy drinker	0.1	-0.11	-0.04	0.13	0.06	-0.08	0.01	0.04	-0.11	0.15	0.08	—	-0.48	0.07	0.01	-0.04	0.07	0.09	-0.01	-0.09	0.03	-0.02	-0.18
13. Light drinker	0.02	-0.17	-0.09	-0.01	0.05	-0.12	-0.03	-0.01	0.1	0.11	-0.05	-0.25	—	-0.07	-0.08	-0.03	-0.11	-0.07	-0.04	0.09	0.1	0.06	-0.01
14. Light activity	0.09	0.12	0.12	0.03	-0.05	0.02	0	0.08	-0.05	-0.07	0.14	-0.01	-0.11	—	-0.17	0.1	0.01	0	0.04	0.02	-0.06	-0.05	-0.05
15. Moderate activity	0.09	0.03	0.01	-0.06	0.02	-0.06	0.03	-0.05	0	0.05	0.04	0	0.01	-0.23	—	0.01	0.05	-0.02	0.01	-0.03	-0.06	0	0.08
16. Black	0.03	0.22	0.13	-0.06	-0.04	0.16	0.06	0.04	-0.19	0.01	0.04	-0.08	-0.13	0.06	0.04	—	-0.12	-0.08	0.03	-0.03	-0.06	-0.11	0.09
17. Hispanic	-0.09	0.15	0.03	-0.01	-0.08	0.09	-0.03	0.07	-0.08	-0.11	-0.05	-0.08	-0.12	0.01	-0.10	-0.15	—	0.55	-0.13	-0.03	-0.16	-0.12	0.08
18. Other race	-0.05	0.11	0.03	-0.03	-0.06	0.04	-0.02	0.07	-0.06	-0.07	0.02	-0.05	-0.11	0	-0.07	-0.11	0.65	—	-0.09	-0.03	-0.10	-0.09	0.05
19. High school	0.03	0.01	-0.04	-0.07	0.03	-0.07	0.07	0	0.06	-0.05	0.12	0.05	0	0.11	0.06	-0.06	-0.12	-0.10	—	-0.30	-0.31	-0.27	-0.01
20. Associates	-0.03	-0.09	0.04	0	0.05	0.02	-0.08	0.01	0	0.04	-0.02	-0.06	0.08	-0.02	0.01	0.02	-0.11	-0.08	-0.42	—	-0.21	-0.18	-0.01
21. College	-0.02	-0.04	-0.08	0.03	-0.02	-0.02	-0.03	0.09	-0.06	0.03	-0.15	0.04	0.07	-0.10	-0.04	-0.07	-0.03	-0.01	-0.28	-0.18	—	-0.18	0.02
22. Advanced degree	-0.04	-0.02	0.03	0.11	-0.07	-0.01	0.01	0.03	-0.06	0.06	-0.08	0.1	0.07	-0.09	-0.03	0.05	-0.03	-0.04	-0.23	-0.15	-0.10	—	0
23. Religious attendance	-0.12	0.08	0.03	0.03	-0.18	0.06	-0.01	-0.01	0.1	-0.11	-0.16	-0.21	-0.06	-0.12	-0.02	0.22	0.08	0.03	-0.07	-0.01	0.04	0.07	—

Note: Coefficients in boldface type indicate statistical significance at $p < .05$ (two-tailed), disrupt. = disruption(s).

Table 5
Odds Ratios for Regression Models of Marital Biography on Biological Risk Among Women ($N = 528$)

Variable	Cardiovascular Risk			Metabolic Risk			Inflammation Risk ^a		
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8	Model 9
Remarried 1 disruption	0.89	0.93	0.93	0.89	0.92	0.90	0.75	0.78	0.69
Remarried 2+ disruptions	1.40†	1.32	1.40†	0.77	0.81	0.88	0.66	0.62	0.65
Previously married 1 disruption	1.0†	1.06	1.12	1.22	1.18	1.14	0.64	0.60	0.56
Previously married 2+ disruptions	1.12	1.06	1.13	1.41*	1.39*	1.44*	0.88	0.87	0.80
Age at first marriage	0.99	1.00	1.00	0.99	0.99	0.99	0.97	0.98	0.97
Decades married	0.87*	0.86**	0.87*	0.93	0.93	0.93†	0.89	0.90	0.89
Black		1.00	1.01		1.44**	1.32*		2.10*	1.70†
Hispanic		0.58†	0.58†		1.37*	1.34*		0.89	0.88
Other race		1.12	1.19		1.02	0.99		1.56	1.47
High school degree		1.01	0.94		1.05	1.07		0.86	0.89
Associate's degree		0.76	0.74		0.90	0.95		1.28	1.52
Bachelor's degree		0.80	0.78		0.96	1.09		0.37†	0.55
Master's-plus degree		0.68	0.64†		0.73	0.90		0.69	1.16
Religious attendance		0.96	0.98		1.02	1.02		1.00	1.01
Former smoker			0.87			1.18			1.21
Current smoker			0.84			0.89			1.57
Heavy drinker			1.58**			0.60**			0.48
Light drinker			1.20			0.74*			0.61†
Light physical activity			1.47**			1.41**			2.35**
Moderate physical activity			1.37*			1.19			1.12
Constant	0.23	0.43	0.18	0.04	-0.06	-0.05	0.32	0.20	0.17
Log likelihood ^b	-568.40	-562.60	-556.70†	-568.10	-558.20*	-551.70*	-311.20	-304.80	-298.40*

Note: Each marital group is compared to the continuously married.

^a Logistic regression models were used for inflammation risk. Those with C-reactive protein values of 3.1 -10 mg/L were compared to those 0-3.09 mg/L.

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Likelihood ratio tests were performed on nested models and show if a model differed from the previous model.

$\dagger p < .10.$
 $* p < .05.$
 $** p < .01$ (two-tailed).

Table 6
Odds Ratios for Regression Models of Marital Biography on Biological Risk Among Men ($N = 554$)

Variable	Cardiovascular Risk			Metabolic Risk			Inflammation Risk ^a		
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8	Model 9
Remarried 1 disruption	1.04	1.02	0.98	0.80 [†]	0.79*	0.79*	0.88	0.81	0.79
Remarried 2+ disruptions	0.83	0.75	0.76	0.80	0.78	0.81	0.31 [†]	0.30 [†]	0.31
Previously married 1 disruption	0.89	0.83	0.79	0.82	0.80	0.81	1.30	1.01	0.99
Previously married 2+ disruptions	1.11	1.03	0.92	0.71	0.70	0.71	0.33 [†]	0.28	0.26
Age at first marriage	0.98 [†]	0.98 [†]	0.98 [†]	0.99 [†]	0.99	0.99 [†]	0.94*	0.94*	0.94*
Decades married	0.90	0.91	0.93	0.96	0.96	0.95	0.77	0.76	0.78
Black		1.05	1.08		1.22	1.12		1.83	1.71
Hispanic		0.92	0.90		1.00	0.90		0.61	0.65
Other race		0.66 [†]	0.61 [†]		1.16	1.22		1.39	1.37
High school degree		0.88	0.92		1.05	1.01		0.52*	0.56 [†]
Associate's degree		0.89	0.97		1.02	1.01		0.43*	0.44*
Bachelor's degree		0.73	0.72		0.93	0.94		0.41 [†]	0.41 [†]
Master's-plus degree		0.57*	0.60*		0.72 [†]	0.71 [†]		0.40***	0.50 [†]
Religious attendance		0.96 [†]	0.97		1.00	0.97		0.96	0.98
Former smoker			0.82 [†]			0.83*			1.36
Current smoker			0.90			0.69*			2.87**
Heavy drinker			1.57***			0.83			0.82
Light drinker			1.00			0.82***			0.91
Light physical activity			0.98			1.33*			1.04
Moderate physical activity			0.99			1.45***			1.60
Constant	1.77	2.23 [†]	1.84	1.44	1.42	2.03	3.79	9.12	4.72
Log likelihood ^b	-580.60	-574.70	-568.60 [†]	-584.10	-582.70	-568.70***	-273.20	-258.60***	-253.10 [†]

Note: Each marital group is compared to the continuously married.

^a Logistic regression models were used for inflammation risk. Those with CRP values of 3.1–10 mg/L were compared to those 0–3.09 mg/L.

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Likelihood ratio tests were performed on nested models and show if a model differed from the previous model.

χ^2

* $p < .10$.

** $p < .05$.

*** $p < .01$.

*** $p < .001$ (two-tailed).