

# Two centuries of marriage and mortality in the United States: Evidence from family histories

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## Abstract

This paper estimates the relationship between marital status and mortality using a large sample of family histories from the United States. The data under analysis contain birth, death and marriage dates on a sample of family-linked individuals. Using both linear models of life expectancy and parametric and semi-parametric hazard models, the analysis finds strong and consistent benefits of marriage that are highest in early adulthood, but diminish steadily over the life course. Generally the effects of being single and widowed are similar, and protective effects of marriage exist for both men and women, though they are stronger for men. Sibling-level random effects are incorporated into both the linear models and the parametric hazard model. In general, the random effects have little impact on the coefficient estimates.

[Preliminary and incomplete—Please do not quote or cite; comments welcome]

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## 1. Introduction

Researchers across the disciplines that study the social determinants of health have found a remarkably consistent empirical regularity: married people are healthier than their unmarried counterparts by every health measure commonly studied. They report fewer chronic illnesses; they have better mental health; they are less disabled by a variety of measures; and they live longer. These regularities are as strong and consistent as those found relating health to other social factors such as education and income. Moreover, the health advantages associated with married exist for women as well as men.<sup>2</sup>

Those who work in this area must concede, however, that little consensus exists on why marriage seems to have a “protective” effect. The methodological challenges in demonstrating a causal link between marriage and health have proved daunting, and documenting the specific family processes that might be responsible for the association is harder still. Serious and legitimate doubts remain concerning whether marriage promotes health or is simply correlated with health because of a third factor that may be positively associated with both the probability of being married and the probability of good health.

The association between marriage and mortality has been extensively studied in recent decades, and comparisons of mortality differentials across different population subgroups and countries have shown to be remarkably consistent. But most analyses are plagued with the same problem that besets many modern demographic studies: the lack of life-course, longitudinal data. Longitudinal, historical data, such as the data employed in this study, provide a potentially fruitful source of information in both testing for and explaining a link between marriage and mortality. Furthermore, if the association between marriage and mortality is found to be robust in historical populations, scholars will be able to refine the explanations for the protective effect of marriage, particularly as they relate to socioeconomic status, health knowledge, and access to medical care.

## 2. Conceptual Background

### *2.1 Families and health: Empirical findings and explanations*

Family effects are known to be important for a variety of economic and demographic phenomena. Family background, containing both genetic and environmental elements, may be an important determinant of fertility, intelligence, education, earning and occupation. However, the number of studies that measure the effect of family background on demographic or economic variables is limited because few data sets are suitable for such analysis.

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<sup>2</sup> It is often inferred from the sex-based gap in the married-unmarried differential that the larger differential for men implies that marriage is structured to serve men more than women. However, at least as plausible is that the differential for men is higher primarily because single men engage in so many risky and unhealthy practices. Waite and Gallagher (2000) summarize the considerable evidence on the “civilizing” (and health-promoting!) effect of marriage on male behavior.

Family background can affect health and mortality through a variety of channels. Most frequently studied has been the influence of genetics. A long history of research has investigated the familial transmission of life expectancy that remains an extremely active area of research today.<sup>3</sup> But as Vaupel and others have noted, genetics can account for only about 25% of the variation in lifespan.<sup>4</sup> Recent research suggests that an additional 25% of the variation may be due to non-genetic characteristics such as educational achievement, socio-economic status, disability and debilitaion, or malnutrition in utero or in childhood (Yashin and Iachine, 1997; Vaupel et al., 1998).<sup>5</sup>

By far the most frequently employed family variable in studies of health and mortality has been marital status, though it is still less studied than race or socioeconomic status.<sup>6</sup> This is particularly true for demographic analyses of mortality.<sup>7</sup> In an early study, Ortmeier (1974) concludes that married persons have lower mortality for almost every major cause of death. Hu and Goldman (1990) perform extensive international comparisons to show that mortality is higher for married persons in every time period and every country included in the study. They also find that mortality risk is highest for divorced persons and that the excess mortality of unmarried persons, as of the date of their study, had been increasing over recent decades. Although the protective effect of marriage is found for both sexes, most studies have found much stronger effects for men.<sup>8</sup>

A variety of health measures other than mortality are also associated with marital status, including self-reported health status, acute or chronic morbidity and disability. Probably the most extensive recent analysis is in Pienta, Hayward and Jenkins (2000), who use the Health and Retirement Study to show that the married dominate the unmarried across all common health measures, and across both genders, all races, and all unmarried categories. Their results confirm the previous findings of numerous other studies concerning marital status and health (including disability).<sup>9</sup>

Little consensus exists concerning causal explanations for the association between health and marital status. The most prevalent explanations focus on characteristics of marriage that affect health. In addition to surveying the empirical findings on marital status and health, seminal papers by Cobb (1976) and Cassell (1976) introduced the concept that marriage provides key “social support,” which in various forms has dominated the

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<sup>3</sup> See Desjardins and Charbonneau, 1990; Bocquet-Appel and Jakobi, 1990; 1991; Brand et al., 1992; Mayer, 1991; Robine and Allard, 1997; Tallis and Leppard, 1997.

<sup>4</sup> Vaupel 1989; McGue, Vaupel, Holm, and Harvald, 1993; Herskind et al., 1996.

<sup>5</sup> Socioeconomic, demographic and ecological influences on health and mortality are still extremely active areas of epidemiological research. See, for example, Feldman 1989; Pappas et al. 1993; Preston and Elo 1995; Sorlie, Backlund and Keller 1995; Rogers, Hummer and Nam 2000.

<sup>6</sup> This literature is reviewed in Ross, Mirowsky and Goldsteen (1990) and more recently in Waite and Gallagher (2000) and Rogers, Hummer and Nam (2000).

<sup>7</sup> See Gove, 1973; Hu & Goldman, 1990; Burman & Margolin, 1992; Trovato, 1992; Rogers et al., 2000.

<sup>8</sup> Zick & Smith, 1991; Goldman, Korenman, & Weinstein, 1995; Lillard & Waite, 1995.

<sup>9</sup> Verbrugge, 1979; Verbrugge, Gates & Ike, 1991; Stewart, Greefield, Hays, Wells, Rogers, Berry et al., 1987; Macintyre, 1992; Wyke & Ford, 1992; Goldman et al., 1997; Murphy, Glaser & Grundy, 1997; Waldron, Weiss & Hughes, 1997.

sociological literature related to health and epidemiology.<sup>10</sup> A marriage provides a person with partners, family members and extended social networks that can assist the individual in maintaining good health.

A spouse can, in addition to providing emotional support, help his or her partner monitor health, encourage healthy behaviors and assist in obtaining medical care. In an analysis of cancer survival, Goodwin, Hunt, Key, and Samet (1987) find that unmarried persons not only have higher mortality (controlling for stage and type of treatment), but that they had later diagnoses and a lower likelihood of treatment, while Gordon and Rosenthal (1995) found that post-hospitalization health outcomes were better for married than unmarried, and Morgan (1980) shows a significantly higher rate of rate of hospitalization by the unmarried. Additionally, the unmarried are more likely to die from “social pathologies” (accidents, suicides and homicides) (Rogers, 1995) and from diseases that are strongly influenced by a person’s behavior (Umberson, 1987). Men, in particular, seem to moderate their behaviors upon marriage (while returning to the risky behaviors after marriage).<sup>11</sup> Married men drink less alcohol, are “more likely to smoke, to drink and drive, to drive too fast, to get into fights, and to take other risks that increase the chances of accidents and injuries” (Waite & Ghallager, 2000). Some evidence also suggests that sharing a residence with someone else does not confer the same advantages on people as does living with a spouse (Kobrin and Hendershot, 1977; Lillard and Waite, 1995).

For those who exit marriage, health is thought to deteriorate because of the loss of social support discussed above. Furthermore, marital dissolution can have strong direct effects on health. The literature on bereavement after the death of a spouse finds a sharp increase in mortality risk, particularly for men, immediately following the death of a spouse, (Bowling, 1987; Kaprio, Koskenvuo, & Rita, 1987), though some have found the risk to diminish after a short period of time (Martikainen & Valkonen, 1996). Maritkainen and Valkonen (1998) also find that the bereavement effects are similar across education and income groups. Although not much is known about the physical processes the raise mortality risk, Kiecolt-Gleiser et al. (1987) find that women whose marriages had recently ended had poorer immune system functioning than married women.

Much has been made of the differences between men and women in the association between health and marital status. As noted above, several studies have found a greater impact of marital dissolution on men, though women who are widowed, divorced or separated also face a higher risks of mortality, morbidity and disability. Lillard and Waite (1995) conclude that men gain from marriage by a change to a more “settled” life-style, whereas women gain predominantly through access to increased financial

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<sup>10</sup> See House, Umberson, and Landis (1988a, 1988b) and Litwick and Messeri (1989) for reviews of the theory and evidence relating social support to health.

<sup>11</sup> Stolzenberg (in press) finds that men’s health deteriorates if they have wives who work more than 40 hours per week, but finds no reciprocal effect of a husband’s employment on the wife’s health. This suggests that wives play an important role in maintaining the health of their husbands, since a demanding work schedule likely reduces the time and energy that wives can devote to spousal health maintenance.

resources. They find that upon marriage, men experience an immediate reduction in the hazard of mortality and that the hazard rate returns to its pre-marriage level following marital dissolution. Women, on the other hand, experience a steady decline in the hazard with each year of marriage.

Some have questioned a direct causal role for marriage and argue, instead, that selection into and out of marriage is responsible for the association between health and marital status. There is some evidence that selection is important, particularly in marriage formation, where those with better health habits (Fu & Goldman, 1996) and higher health status (Waldron, Hughes & Brooks, 1996) are more likely to marry, though the selection hypothesis usually finds considerably less support than the protection hypothesis (Korbin & Hendershot, 1977; Morgan, 1980). Others have found no support for the selection hypothesis (Zick & Smith, 1991).

Recent studies have searched for selection effects through both an analysis of unobservable variables and through direct controls for health at time of marriage. Behrman, Birdsall, and Deolankar (1995) use data on twins to estimate the effect of unobservable, individual human capital endowments on labor market success and success in the marriage market. These endowments are also strongly linked to obesity, which suggests a possibility of marital selection on the basis of health. Similarly, Lillard and Panis (1996) find positive selection on the basis of unmeasured factors that are correlated with both health and marriage, but they also find evidence of adverse selection, which results from an incentive to marry by those in poor health.

The association between marriage and mortality found in modern populations appears to exist in a variety of historical populations as well, though there have been relatively few studies on this topic. Hacker (1997) studied Yale College graduates from 1701 to 1805 and found that married graduates report a 10-year advantage in life expectancy at age 25 over unmarried graduates. In fact, the married-unmarried differential far exceeded urban/rural differences or differences based on occupation. He finds a sharp increase in the relative mortality risk from near unity at 20-24 years, rising to about 2 by age 25-29, leveling off until age 59, and then falling back to unity at older ages. His hazard model analysis finds a 78% higher hazard for single graduates than for married, and an 18% higher hazard for widowed graduates.

Probably the central problem with the historical and modern studies is that issue of selection into marriage, since healthy people may be more likely to marry. In a recent important paper, Murray (2000), attempts to control for selection into marriage by using body mass index as a measure of health status in his study of Amerhest College graduates born between 1832 and 1879. He finds that healthier men were more likely to be married, but that the marriage has a protective effect on health even after controlling for health.

### *2.3 Marital status over the life course*

The causal mechanisms linking marital status and mortality are still very unclear even though the literature showing the empirical regularity is extensive. Although all studies control for age, the question of how the relationship between marriage and mortality varies across the life-cycle can have received little attention. The discussion below draws upon the theoretical strands in the literature to discuss informally how age might be used to discriminate between theories.

Most discussions of marriage and health assume that marriage induces behavioral change, particularly on the part of men. In the modern era, discussion usually involves behaviors with known links to health, such as smoking, diet and exercise. In the historical era, however, there was little widespread or accurate knowledge about the production of health, but there still may have been important behavioral modifications. We would anticipate, for instance, that the prevalence of risky behaviors and heavy alcohol consumption would have decreased with marriage as would have the number of sexual partners.<sup>12</sup> It seems reasonable to assume, therefore, that behavioral modifications in terms of risky behaviors would be most salient at younger ages and strongest for men, since young men have, throughout history, been most likely to engage in risk behaviors. Women, on the other hand, might actually face higher health risks following marriage because of the risk of maternal mortality that came with marriage.

Much of the focus on behavioral change in the modern era assumes that the benefits of marriage are going to accumulate over time. Lillard and Waite (1995), for instance, place a heavy emphasis on the duration of the marriage, particularly for women. We can think of this argument in the context of the health capital model of Grossman (1972), in which health is a latent capital stock that can be augmented by investment and which depreciates over time. According to the capital stock approach, marriage increases the ability of individuals to raise their capital stock.

If the capital stock approach is correct we should expect to see a widening of the gap between the married and the never married over time. Furthermore, widowhood should have no immediate impact on health, but the difference between the married and the widowed should grow gradually over time. The capital stock implies, therefore, not only differences between married and unmarried that vary over time but important differences between the unmarried categories, particularly in later life.

Sociologists' notions of social support play an important role in the discussion of marriage and mortality. However, other than the direct role of emotional support provided by the spouse, it is not clear what role other individuals play in providing emotional support and promoting health. What is clear is that women have a much larger social network outside of marriage than men typically do. Thus, spousal loss should have a more negative and immediate effect on men than on women, as much of the previous literature shows.

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<sup>12</sup> Cigarette consumption did not become widespread until the invention of the mass-produced cigarettes in the end of the 19<sup>th</sup> century.

What is unclear, however, is how the consequences of bereavement will vary with age and with the duration of marriage. Our conjecture is that loss early in life is more unanticipated and, therefore, will be more traumatic. If bereavement is the primary source of marital status effects, then the effects of widowhood should be highest at earlier ages and the widowed should have higher mortality than single persons at young ages

The final source of marital status effects are due to the selection. There are two important sources of selection which have different implications for the analysis. The first type of selection is that robust persons are more likely to select into marriage (and less likely to select out). Although it is not clear that more robust people are going to marry first (their value on the marriage market gives them the incentive to invest in a longer search for high quality mates), a likely marriage market equilibrium (yet to be formally demonstrated) is that single people, as a group, are becoming less robust over time compared to the married. If spousal death is random, then the gap in mortality between singles and married people should increase over time, but the married and the widowed should not differ.

Of course, people do not marry random people, they marry people who are like them in many ways. Because of homogamy, longer-live persons are likely to have long-lived spouses (with the same holding true for short-lived persons). Smith and Zick (1994) have shown the association between spousal life spans, and Wilson (2002) has demonstrated that marriage market effects lead to a strong inter-spousal correlation in health status by a variety of health measures. Homogamy implies that widowed persons will be shorter lived not because of any consequences of marriage, per se, but because spousal lifespan is an indicator of mortality risk. This type of selection, therefore, implies a persistent gap between the married and the widowed in terms of mortality risk that should remain constant over time. It is not clear, however, which type of selection effect is strongest, so it is not known whether the single are worse off than the widowed, though the effects of widowhood, relative to marriage, should remain relatively constant, while the effects of being single should decline up to the point where most of those who will ever marry have already married.

Though the ideas set out here are highly informal, they are more nuanced than is the typical discussion in many studies that discuss “protection v. selection.” There are different types of protection and different types of selection, with the effects of marital status varying across the types. Although the explanations given above are not at all mutually exclusive (most everyone would argue, for instance, that marriage market selection plays some role), an analysis of marriage and health over the life course may allow some discrimination between the predictions of the different theories.

### 3. Data

#### *3.1 The family history data collection*

Data used for this study come from a collection of 34 published genealogies from the United States. While not strictly a random sample, the observations in the sample have

been shown to be roughly representative of the historical U.S. white population prior to about 1900. Currently there are over 108,000 individuals in the family history sample, with birth dates ranging from 1594 to the late 20<sup>th</sup> century. All available vital dates have been collected, and the records have been linked to all the available U.S. census manuscripts. The greatest advantage of this data source over other types of records is the family linkages that allow researchers to conduct family and intergenerational analyses. As discussed below, mortality analysis requires that most of the observations in the sample not be used because they are censored by the publication of the family history book. Additionally, missing vital dates further restricts the sample. The final sample used for analysis here, which also deletes the sample individuals who marry into the sample (because the lack of information typically present for those cases) consists of 7,257 females and 9,301 males.

Family histories have been used as a source of data for intergenerational demographic analysis for a number of years (Fogel 1986; Wahl 1986; Pope 1986 and 1992) and have been an extraordinarily useful source of data. Family histories appear to be a useful source for the study of adult mortality prior to extensive development of a death registration system shortly after 1900, and life tables from genealogies are quite comparable to life table from other sources. The most intriguing finding developed from the genealogies to date is the marked decline in life expectation for both men and women in the antebellum period. Three studies based on three different sample of family history data (Kunze, 1979; Fogel, 1986; Pope, 1992) have all found this decline. Since the life tables based on family histories include very few foreign born individuals, they yield slightly higher life expectancies.

### *3.2 Comparability with other historical estimates*

The sample characteristics of the family history data is a subject of ongoing study. Of particular concern is the representativeness of the sample and the comparability to other data sources that have been used for mortality research. Pope (1992) notes that the U.S. family histories underrepresent blacks and immigrants. Infant and child deaths, since they are underrecorded in the source documents, are also underrepresented in the family histories. Death dates are also less common than birth dates because dispersal of families and women's name changes make death years difficult to find. In general, however, family histories seem to be an appropriate source for studying adult mortality. Although potential biases exist, reconstructed families seem to be representative of the larger population (Norton, 1980). In research with a smaller version of the U.S. family history data used here, Pope (1992) shows that life table estimates from the family histories are remarkably consistent with other existing historical estimates

### *3.3 Variable Definitions*

Other than length of life, marital status is the most important variable under analysis. There are four marital status variables incorporated below: 1) married; 2) single; 3)



widowed; 4) unknown (married at some point, but spousal death date and/or marriage date is unknown). In the linear models, individuals without known marriage dates are treated as married following age 30. In the hazard models, they are treated as married throughout (which assumes that the marriage occurred in the early 20s, an assumption that is likely to make the estimated effects of marriage at early ages less reliable).

As noted above, state-level fixed effects are included for state of birth. Also, when non-bloodline individuals are included in the analysis, bloodline is included as a dummy variable in the regressions. Categories for year of birth are included as dummy variables. Finally, birth order, family size, and age of mother at the birth of the individual were tested, but they were found to have no effect and are excluded from the analysis discussed below.

## 4. Methods

### *4.1 General methodological issues in dealing with genealogical data*

Sample selection issues with the family history data are extremely important in making valid inferences from family history data. Gender differences are generally very small, but the large number of missing dates leaves open the possibility of significant sample selection issues. Previous analysis suggests that infants who die are less likely to be recorded in the history, and people who marry into the family have more missing dates than persons in the bloodline. Because of the possibility that those marrying into the family with sufficient information to be used in the analysis may differ systematically from those without such information, the “non-bloodline” cases are excluded here. This also reduces the need to account for left-censoring of cases who are not at risk to die prior to being married.

The analysis of marriage effects is problematic for at least four reasons. The first is missing data. The key methodological issue is whether the data is randomly or whether it is systematic in some way and, if it is, is it missing in a way that is correlated with explanatory variables in the model, such as marital status. Although the issue is not firmly resolved, no convincing reason has been yet posited concerning how the relationship between marital status and mortality should affect the probability that the genealogist collecting the data would be able to find a particular death date.

The second important issue in studying mortality is the data have no independent controls for health, meaning that selection into marriage on the basis of health status may be a problem, as discussed above. Of course, the data also lack information on a host of other unobservable variables that may impact mortality risk. The approach taken here to deal with this problem is to exploit the family structure of the data by estimating models with sibling-level random effects. Estimating the life expectancy of siblings jointly will provide better controls for unobserved health status to the extent that the impact of genetics and early life variables persist into adulthood. Also, fixed effects for state of birth are included in all regressions.

A third issue is the endogeneity of marital status. In hazard models, the endogeneity of the time-varying covariates (in this case the marital status transitions) can bias the estimates of the parameters. For instance, some people who would have married, die before they have the opportunity to get married. This means that married people will tend to live longer because they have lived long enough to be married. Thus the method may overstate the benefits of marriage because of the early mortality in the sample.

The fourth issue is that of censoring. A large percentage of the individuals in the sample were still living at the time of the creation of the family history. It is necessary, therefore, to limit the analysis to those cases who were born 90 years or more before the last recorded date in the genealogy. This insures that all observations were at risk to die over the entire period of the analysis (other than the slight bias caused by those living more than 90 years). Treating people as alive but censored at the time of the collection of the data is not sufficiently conservative, given the large number of missing death dates in the sample. This sample restriction precludes analysis of mortality for cohorts born in the 20<sup>th</sup> century. Figure 1 shows the distribution of birth years for all individuals and for those in the analysis sample.

#### *4.2 Empirical mortality models with random effects*

A variety of methods exist for estimating the impact of covariates on mortality. The analysis that follows explores essentially three kinds of models. First, simple linear models of life expectancy are estimated at three points in the sample individuals' lifetimes: age 30, 45 and 60. Simple OLS models are used to estimate the effect of marital status and other covariates on life expectancy.

In these models and those to follow, I attempt to account for health by using the data on the individuals siblings. Because of the family linkages in the data, I am able to identify family groupings of siblings and then jointly estimate their life expectancy using a random effects model. In brief, life expectancy for person  $j$  in family group  $i$  is defined as:

$$(1) \quad E_{ij} = BX + u_i + e_{ij}$$

where  $X$  is a vector of covariates, including year effects and state of birth effects,  $e_{ij}$  is the individual-specific error term, and  $u_i$  is the family-specific intercept, which is assumed to be randomly distributed. Generalized least squares provides consistent estimates of the coefficients.

Obviously the simple linear model leaves much to be desired, including allowance for time-varying marital status, a way to deal with censoring of the data and duration dependence. Survival analysis, therefore, is likely a much more appropriate tool in estimating mortality. I estimate here both semi-parametric Cox models and parametric survival models.

The Cox model (1972) has become in recent years a mainstay of survival analysis. This is likely because of its ease of implementation and interpretation. If we let  $h(t|x)$  be the hazard rate of mortality for a person with characteristics  $X$  at time  $t$ .<sup>13</sup> In the semi-parametric model,  $h(t|x)$  is assumed to depend on covariates in the following fashion:

$$(2) \quad h(t|x) = e^{XB} h_0(t),$$

where  $h_0(t)$  is the “baseline” hazard function. The attractive feature of this model is that the baseline hazard is treated simply as nuisance parameter and need not be estimated. The partial likelihood function, in fact, does not depend on survival, but is simply a straightforward function of the order of survival times in the sample. Maximum likelihood estimates of  $B$  can be used to calculate relative mortality risks (or hazard ratios), which are calculated as  $e^b$ , where  $b$  is an individual regression coefficient.

For the sake of robustness, it is also useful to estimate parametric survival models, which are estimated by assuming a particular function for the baseline hazard function and the performing maximum likelihood estimation. Different specifications of the functional form allow for very different shapes of the mortality hazard. Figures 2-4 represent the age-specific annual death rates for the sample as a whole. Since the annual death rate at a particular age approximates the hazard rate, these figures suggest a model for the hazard function that increases sharply and exponentially in later life. Exploration with a variety of models suggests that a Gompertz hazard specification is appropriate, and empirical testing of alternative forms confirms the appropriateness of this specification. The hazard function is parameterized in much the same way as (2), but the baseline function is assumed to follow the Gompertz specification.

An advantage of the parametric approach to hazard models is that it allows treatment of group-effects, which in the case at hand are sibling-level effects on mortality. The standard parametric approach for dealing with unobserved heterogeneity has been the frailty model (Vaupel, Manton, and Stallard, 1979). In the frailty model, the individual hazard is multiplied by a constant,  $a_i$ , that is assumed to proportionally scale his level of mortality risk. Thus, a new parameter is added as follows:

$$(3) \quad h(t|x) = a_i e^{XB} h_0(t).$$

It is necessary to assume a distribution for the frailty term, and I follow the common convention of assuming that  $a_i$  follows a generalized gamma distribution. In the models that follow, (3) will be estimated assuming that  $a_i$  varies randomly at the individual level and that it varies by family, where all the siblings in the family group share the same frailty parameter.<sup>14</sup>

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<sup>13</sup> The hazard rate is the probability of dying at time  $t$  conditional on surviving to time  $t$ .

<sup>14</sup> See Gutierrez (2002) for a description of the likelihood function and estimation method for the shared frailty model.

## 5. Results

The first results are from the linear models. Model estimates are found in Tables 2 and 3. Table 2 gives estimates using the entire data set, while Table 3 estimates the model separately for men and women. Estimates of both OLS and a linear model with sibling-level random effects. Marital status is measured at baseline. Fixed effects for state of birth are also employed for this and all subsequent analysis.

The results in Tables 2 and 3 show, that relative to the married group, unmarried individuals (both single and married) have about a four year advantage in life expectancy. This declines to about 2 years at age 45, and largely disappears at age 60. The high mortality of the “status unknown” category is troubling and does not have an obvious interpretation. We would expect that group to have a higher life expectation, since it includes people who have been married, but the spousal death date is unknown.

The sex-specific results indicate that the marital status effects exist for both men and women, but differences exist across the sexes. At age 30, men face a greater degree of risk from being single than do women, but widowed women experience higher mortality than widowed men (though the number of widowed persons at age 30 is very small). By age 45, the differences between single and widowed level out, and men have slightly lower life expectancy than women for both singles and widowed. By age 60, it appears that the gains from being married have virtually disappeared for both sexes, down to less than half a year (which is statistically insignificant).

The next method employed is hazard model analysis using time-varying covariates for marital status. As noted above, both semi-parametric Cox models and parametric specifications are employed. Individuals with a marriage date, but no death date are right censored at the date of marriage. Individuals are followed from age 15 to death.

Table 4 shows hazard ratio estimates that are broadly consistent across models, though the parametric methods yield higher hazard ratios. In each model there is large and statistically significant effect on estimated hazard of death for the single and widowed groups relative to the excluded married category (again the unknown group is unaccountably high). Unmarried individuals have about a 40% higher hazard of mortality under the Cox model and about 65-70% higher under the parametric specifications. The variance of the sibling-level effect is statistically significant, but it is small and has very little consequence for the parameter estimates.

In Table 5 the hazard models are re-estimated separately for males and females. Under both specifications, protective effect is higher for males than females, though it is strongly significant for both sexes. Neither males or females show a significant difference between being single and being widowed.

Table 6 further subdivides the analysis by estimating models for men and women for different age groups. In each case the cases still alive are right-censored at the end of the age period. Time-varying covariates are maintained throughout. These results show that the assumption of proportionality across the life-cycle is clearly violated. Hazard ratios for men are still higher than for women, but it is clear that the protective effects of marriage are declining sharply across time, virtually disappearing (as we saw earlier) in those over age 60. In the early years, the hazard associated with being unmarried is particularly high, especially for single men (the ratio for widowed is insignificant, given that there are very few widows in this age range).

Finally, Table 7 shows the effects of marriage across three periods of time: pre-1800, 1800-1850 and 1850-1900. The effects of marital status are relatively stable, but they do show some trend over time that differs by gender. In brief, for men, marriage is becoming a less important predictor of mortality. For women it is becoming more important. The trends in both cases, however, are relatively modest.

## 6. Discussion

Four basic findings are found in the analysis above. First, marital status is strongly related to mortality risk prior to the 20<sup>th</sup> century in the United States. Second, the effects are relatively stable across the history of the United States at least until 1900, though they increase for women and decrease for men. Third the relationship between marriage and mortality is high for both women and men, but is consistently higher for men. And fourth, the marital status effects are high for several years, but drop sharply for both men and women over aged 60.

What do the results above imply about the reason that marriage is related with mortality? The most pronounced finding is that marriage becomes successively less important over the life course. This speaks strikingly against behavioral explanations based on an improving health capital stock, since an increasing capital stock implies that the single-married difference should be increasing over time. If marriage changes health behavior, it is through behaviors that have payoffs relatively early in life, such as an avoidance of high risk behaviors such as fighting or excessive drinking. The very high effects of being a single male under age 30 gives this view some further validity. It is challenged, however, by the fact that single women also had a higher mortality rate, which is even more problematic since single women, by and large, didn't face the risk of maternal mortality (the advantage of avoiding maternal mortality, however, may be seen by the fact that widowed women in before age 30 actually had a lower mortality than married women).

The hypothesis that men will face a bigger effect from bereavement than women does find some support here. First, the effects of spousal loss are higher for men than for women across the life-cycle. As noted above, early spousal loss is actually advantageous for women (though we would want to control for the presence of children, which hasn't yet been attempted) and in the post-60 years, widowed men do have a highly statistically

significant mortality disadvantage, though it is smaller in magnitude than the estimates for both men and women earlier in life. However, bereavement cannot account for the gap between single and married people that is found for both men and women (though, perhaps, loneliness can).

The evidence also casts some doubt on the importance of selection (though selection on unobservables can never be totally dismissed based on an analysis of observable variables). The argument was made earlier that selection into marriage implies that the single-married gap should be growing over the life course. Actually, it is falling for both men and women. The sharper fall for men is probably due to behavioral modification among men as they age. The fact that the widowed effect dies over time is also evidence against selection based on assortative mating in the marriage market, since sharing the same risk factors over the course of the marriage should make the assortative mating effects even stronger.

These and other puzzles need to be considered as scholars attempt to understand the association between marriage and health in modern populations. Interpretations of modern data emphasize the role that the spouse (particularly the wife) plays in maintaining the health of her partner. But in the historical period, medical care was, at best, benign, and “knowledge” that existed about promoting health was as likely as not to be incorrect. However, in a period where meeting the basic necessities of life in terms of net nutritional intake was challenging and the realities of life were harsh in so many ways, it may be that having a spouse around to share the burdens was at least as important as it is today.

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TABLE 1: Summary of vital data

	<b>Females</b>	<b>Males</b>
Total Observations	7,257	9,301
% Ever Married	81.7%	83.5%
Distribution by Birth Year		
Birth year<1700	138	204
Year of Birth: 1700-1724	187	234
Year of Birth: 1725-1749	198	340
Year of Birth: 1750-1774	311	564
Year of Birth: 1775-1799	678	1035
Year of Birth: 1800-1824	1244	1566
Year of Birth: 1825-1849	1708	2073
Year of Birth: 1850-1874	1881	2243
Year of Birth: 1875-1899	912	1042
Remaining Life Expectancy at Age 15 ( $e_{15}$ ):		
Total Sample	46.26	47.26
Birth year<1700	44.99	44.08
Year of Birth: 1700-1724	36.90	46.06
Year of Birth: 1725-1749	43.71	47.62
Year of Birth: 1750-1774	49.21	50.85
Year of Birth: 1775-1799	46.86	46.93
Year of Birth: 1800-1824	44.73	46.63
Year of Birth: 1825-1849	44.21	45.27
Year of Birth: 1850-1874	48.03	48.33
Year of Birth: 1875-1899	49.43	49.17

Notes: Data include those with a known lifespan who live until at least age 15 and are not censored by the end of the family history book, which means that they were born at least 90 years prior to the last recorded date in the book; these are the cases used for analysis in the subsequent tables.

TABLE 2: Linear Models

	Life expectancy at Age 30 ( $e_{30}$ )				Life expectancy at Age 45 ( $e_{45}$ )				Life expectancy at Age 60 ( $e_{60}$ )			
	OLS		RE		OLS		RE		OLS		RE	
	Coeff.	Std. Error	Coeff.	Std. Error	Coeff.	Std. Error	Coeff.	Std. Error	Coeff.	Std. Error	Coeff.	Std. Error
Sex:Male	-0.81	0.31 **	-0.79	0.31 **	-1.54	0.26 ***	-1.56	0.25 ***	-1.50	0.21 ***	-1.45	0.21 ***
Single/Never Married	-3.57	0.39 ***	-3.70	0.39 ***	-1.98	0.39 ***	-2.06	0.38 ***	-0.27	0.33	-0.27	0.32
Widowed	-4.56	1.64 **	-4.68	1.61 **	-2.40	0.66 ***	-2.35	0.63 ***	-0.56	0.35	-0.49	0.34
Status Unknown	-4.57	0.36 ***	-4.55	0.36 ***	-1.92	0.28 ***	-1.93	0.29 ***	-0.73	0.23 ***	-0.63	0.24 **
Year of Birth: 1700-1724	-2.01	1.47	-1.35	1.55	0.13	1.27	0.63	1.29	-0.27	1.05	0.05	1.08
Year of Birth: 1725-1749	2.82	1.42 *	3.07	1.49 *	3.83	1.23 **	3.91	1.22 ***	2.91	1.05 *	2.99	1.00 **
Year of Birth: 1750-1774	4.86	1.24 ***	5.03	1.36 ***	3.83	1.08 ***	4.07	1.11 ***	1.77	0.93	1.88	0.90 *
Year of Birth: 1775-1799	1.68	1.16	1.92	1.29	1.42	1.02	1.67	1.05	-0.15	0.88	-0.06	0.86
Year of Birth: 1800-1824	1.37	1.13	1.36	1.25	2.00	0.99 *	2.03	1.02 *	-0.16	0.86	-0.10	0.84
Year of Birth: 1825-1849	1.91	1.13	1.95	1.25	2.29	0.98 **	2.38	1.02 *	-0.25	0.85	-0.17	0.84
Year of Birth: 1850-1874	3.49	1.13 **	3.52	1.24 **	3.08	0.98 **	3.21	1.01 ***	0.29	0.85	0.42	0.83
Year of Birth: 1875-1899	5.17	1.17 ***	4.99	1.29 ***	3.72	1.02 ***	3.80	1.05 ***	0.81	0.88	0.84	0.86
Constant	33.2	3.1 ***	33.52	3.13 ***	24.96	2.67 ***	25.46	2.70 ***	17.93	2.07 ***	17.92	2.18 ***
N	12,066		12,066		10,032		10,032		8,186		8,186	
R <sup>2</sup>	0.034		0.034		0.022		0.021		0.021		0.021	

Notes: Excluded categories are Married and Birth Year<1700. All models contain fixed effects for state of birth. Marital status variables are fixed at their baseline values. Standard errors are robust (heteroskedasticity-consistent). P-values for the test of B=0 are represented by: \*: p<.05; \*\*:p<.01; \*\*\*: p<.001

TABLE 3: Linear models--By sex

	Life expectancy at Age 30 ( $e_{30}$ )		Life expectancy at Age 45 ( $e_{45}$ )		Life expectancy at Age 60 ( $e_{60}$ )	
	OLS		OLS		OLS	
	Coeff.	Std. Error	Coeff.	Std. Error	Coeff.	Std. Error
<b>FEMALES</b>						
Single/Never Married	-2.88	0.64 ***	-1.74	0.58 ***	-0.45	0.49
Widowed	-6.76	2.59 **	-1.65	0.93	-0.43	0.51
Status Unknown	-3.60	0.60 ***	-0.82	0.47	0.03	0.39
Year of Birth: 1700-1724	-3.73	2.58	0.24	2.35	0.43	1.93
Year of Birth: 1725-1749	6.12	2.55 *	6.42	2.21 ***	4.81	1.92 ***
Year of Birth: 1750-1774	6.35	2.09 **	5.24	1.82 ***	1.08	1.62
Year of Birth: 1775-1799	4.03	1.87 *	3.60	1.66 *	-0.03	1.50
Year of Birth: 1800-1824	3.60	1.80 *	3.79	1.60 *	0.00	1.46
Year of Birth: 1825-1849	3.92	1.79 *	4.44	1.60 *	0.15	1.45
Year of Birth: 1850-1874	6.14	1.78 **	5.85	1.59 ***	1.07	1.46
Year of Birth: 1875-1899	7.92	1.85 ***	6.89	1.64 ***	1.74	1.48
Constant	27.62	5.24 ***	19.67	4.59	14.75	3.65
N	4,819		4,056		3,239	
R <sup>2</sup>	0.038		0.031		0.026	
<b>MALES</b>						
Single/Never Married	-4.08	0.49 ***	-2.19	0.54 ***	0.03	0.46
Widowed	-2.65	2.09	-2.87	0.95 ***	-0.46	0.49
Status Unknown	-5.25	0.46 ***	-2.61	0.36 ***	-1.18	0.30 ***
Year of Birth: 1700-1724	-1.60	1.78	-0.41	1.51	-0.61	1.27
Year of Birth: 1725-1749	0.91	1.73	2.29	1.48	2.11	1.25
Year of Birth: 1750-1774	3.64	1.55 *	2.77	1.34 *	2.00	1.13
Year of Birth: 1775-1799	0.05	1.48	-0.08	1.27	-0.33	1.09
Year of Birth: 1800-1824	-0.26	1.46	0.78	1.24	-0.30	1.06
Year of Birth: 1825-1849	0.36	1.45	0.76	1.24	-0.60	1.06
Year of Birth: 1850-1874	1.50	1.45	1.12	1.24	-0.31	1.06
Year of Birth: 1875-1899	3.17	1.50	1.53	1.29	0.16	1.10
Constant	35.45	3.70	26.19	3.15	17.65	2.44
N	7,247		6,246		3,239	
R <sup>2</sup>	0.038		0.023		0.0212	

Notes: Excluded categories are Married and Birth Year<1700. All models contain fixed effects for state of birth. Marital status variables are fixed at their baseline values. Standard errors are robust (heteroskedasticity-consistent). P-values for the test of B=0 are represented by: \*: p<.05; \*\*:p<.01; \*\*\*: p<.001

TABLE 4: Time-varying hazard models--Age 15 to death

	Cox Model			Gompertz		Gompertz w/ Shared (sibling- level) Frailty		
	Haz.Ratio	Std. Error		Haz.Ratio	Std. Error	Haz.Ratio	Std. Error	
Sex:Male	1.120	0.021	***	1.123	0.020	1.128	0.021	***
Single/Never Married	1.428	0.039	***	1.683	0.044	1.754	0.048	***
Widowed	1.436	0.040	***	1.661	0.042	1.678	0.045	***
Status Unknown	1.403	0.032	***	1.485	0.031	1.508	0.036	***
Year of Birth: 1700-1724	1.157	0.105		1.147	0.099	1.125	0.099	
Year of Birth: 1725-1749	0.822	0.075	*	0.844	0.072	0.835	0.070	*
Year of Birth: 1750-1774	0.780	0.062	**	0.782	0.058	0.765	0.059	***
Year of Birth: 1775-1799	0.971	0.073		0.942	0.067	0.929	0.067	
Year of Birth: 1800-1824	0.975	0.072		0.943	0.065	0.936	0.065	
Year of Birth: 1825-1849	1.013	0.075		0.971	0.067	0.956	0.066	
Year of Birth: 1850-1874	0.874	0.064		0.846	0.058	0.830	0.058	**
Year of Birth: 1875-1899	0.827	0.062	*	0.801	0.056	0.781	0.057	**
Variance of frailty parameter						0.043	0.008	***
N	16558			16558		16558		

Notes: Excluded categories are Married and Birth Year<1700. All models contain fixed effects for state of birth. Marital status is treated as a time-varying covariate. The frailty parameter,  $a$ , is a multiplicative constant of the baseline hazard function and is assumed to follow a generalized gamma distribution. Standard errors are robust (heteroskedasticity-consistent). P-values for the test of Haz. Ratio=1 are represented by: \*: p<.05; \*\*:p<.01; \*\*\*: p<.001

TABLE 5: Time-varying hazard models--by sex

Cox Model	Females			Males		
	Haz. Ratio	Std. Error		Haz. Ratio	Std. Error	
Single/Never Married	1.391	0.060	***	1.523	0.052	***
Widowed	1.424	0.067	***	1.415	0.041	***
Status Unknown	1.432	0.057	***	1.380	0.036	***
Year of Birth: 1700-1724	1.357	0.211	*	1.069	0.092	
Year of Birth: 1725-1749	0.730	0.118		0.872	0.070	
Year of Birth: 1750-1774	0.769	0.102	*	0.825	0.061	*
Year of Birth: 1775-1799	0.900	0.108		1.006	0.070	
Year of Birth: 1800-1824	0.928	0.109		1.040	0.070	
Year of Birth: 1825-1849	0.951	0.111		1.097	0.073	
Year of Birth: 1850-1874	0.789	0.092	*	0.970	0.065	
Year of Birth: 1875-1899	0.758	0.090	*	0.933	0.066	
N	7257			9301		

  

Gompertz Frailty Model	Females			Males		
	Haz. Ratio	Std. Error		Haz. Ratio	Std. Error	
Single/Never Married	1.669	0.069	***	1.759	0.063	***
Widowed	1.689	0.073	***	1.687	0.055	***
Status Unknown	1.563	0.060	***	1.457	0.039	***
Year of Birth: 1700-1724	1.366	0.204	*	1.083	0.118	
Year of Birth: 1725-1749	0.781	0.113		0.892	0.096	
Year of Birth: 1750-1774	0.762	0.091	*	0.794	0.078	*
Year of Birth: 1775-1799	0.856	0.093	*	1.002	0.095	
Year of Birth: 1800-1824	0.883	0.093		0.977	0.092	
Year of Birth: 1825-1849	0.889	0.093		1.029	0.096	
Year of Birth: 1850-1874	0.757	0.079	**	0.915	0.085	
Year of Birth: 1875-1899	0.720	0.077	**	0.865	0.082	
N	7257			9301		

Notes: Excluded categories are Married and Birth Year<1700. All models contain fixed effects for state of birth. Marital status is treated as a time-varying covariate. The frailty parameter,  $a$ , is a multiplicative constant of the baseline hazard function and is assumed to follow a generalized gamma distribution. Standard errors are robust (heteroskedasticity-consistent). P-values for the test of Haz. Ratio=1 are represented by: \*: p<.05; \*\*:p<.01; \*\*\*: p<.001

TABLE 6: Piecewise Cox models--By age and sex

	FEMALES			MALES		
<u>AGE=15-30</u>	<u>Haz. Ratio</u>	<u>Std. Error</u>		<u>Haz. Ratio</u>	<u>Std. Error</u>	
Single/Never Married	2.123	0.245	***	3.392	0.409	***
Widowed	0.754	0.542		1.832	0.783	
Status Unknown	2.279	0.283	***	2.028	0.296	***
<u>AGE=30-45</u>	<u>Haz. Ratio</u>	<u>Std. Error</u>		<u>Haz. Ratio</u>	<u>Std. Error</u>	
Single/Never Married	1.592	0.170	***	2.482	0.211	***
Widowed	2.222	0.425	***	2.585	0.494	***
Status Unknown	2.114	0.195	***	2.140	0.165	***
<u>AGE=45-60</u>	<u>Haz. Ratio</u>	<u>Std. Error</u>		<u>Haz. Ratio</u>	<u>Std. Error</u>	
Single/Never Married	1.487	0.157	***	1.685	0.146	***
Widowed	1.624	0.196	***	1.991	0.222	***
Status Unknown	1.382	0.124	***	1.547	0.101	***
<u>AGE=60+</u>	<u>Haz. Ratio</u>	<u>Std. Error</u>		<u>Haz. Ratio</u>	<u>Std. Error</u>	
Single/Never Married	1.058	0.072		1.041	0.058	
Widowed	1.103	0.066		1.256	0.048	***
Status Unknown	1.083	0.063		1.191	0.044	***

Notes: Reference category is "Married." Variables not shown are birth year categories and state of birth. Marital status is treated as a time-varying covariate, but all living observations are censored at the end of the age period. Standard errors are robust (heteroskedasticity-consistent). P-values for the test of Haz. Ratio=1 are represented by: \*: p<.05; \*\*:p<.01; \*\*\*: p<.001

TABLE 7: Piece-wise Cox models--By sex and birth year

	MALES			FEMALES		
<u>Birth Year=1594-1799</u>	<u>Haz. Ratio</u>	<u>Std. Error</u>		<u>Haz. Ratio</u>	<u>Std. Error</u>	
Single/Never Married	1.676	0.133	***	1.260	0.122	*
Widowed	1.580	0.101	***	1.287	0.126	*
Status Unknown	1.347	0.086	***	1.371	0.121	***
<u>Birth Year=1800-1849</u>	<u>Haz. Ratio</u>	<u>Std. Error</u>		<u>Haz. Ratio</u>	<u>Std. Error</u>	
Single/Never Married	1.511	0.084	***	1.381	0.092	***
Widowed	1.472	0.081	***	1.436	0.106	***
Status Unknown	1.446	0.068	***	1.387	0.087	***
<u>Birth Year=1850-1899</u>	<u>Haz. Ratio</u>	<u>Std. Error</u>		<u>Haz. Ratio</u>	<u>Std. Error</u>	
Single/Never Married	1.491	0.100	***	1.532	0.116	***
Widowed	1.431	0.088	***	1.544	0.121	***
Status Unknown	1.417	0.067	***	1.607	0.108	***

Notes: Reference category is "Married." Marital status is treated as a time-varying covariate, but all living observations are censored at the end of the age period. Standard errors are robust (heteroskedasticity-consistent). P-values for the test of Haz. Ratio=1 are represented by: \*: p<.05; \*\*:p<.01; \*\*\*: p<.001