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Marriage Selection and Mortality Patterns: Inferences and Fallacies*

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Researchers have long wondered whether marital-status differences in mortality arise largely from selection mechanisms or from causal processes typically known as *marriage protection*. Unfortunately, many investigators have relied on aggregate patterns of mortality differentials—such as age schedules of excess mortality in the single population or the relationship between the level of excess mortality and the relative size of the single population—to make inferences about the relative importance of selection and causal processes. In this paper, a simple mathematical simulation model is used to demonstrate that many inferences derived from observed patterns are simply not justified. This finding highlights the importance of prospective data for assessing the relative importance of selection and causal factors in accounting for the excess mortality of the unmarried.

Few demographic issues have been studied as frequently, yet remain as poorly understood, as the relationship between marital status and mortality. Hundreds of studies in a large number of industrialized countries have demonstrated that married men and women have greater longevity and experience better health than do single, divorced, and widowed persons. At the same time, considerably less is known about the extent to which these differences result from *marriage selection* or from causal mechanisms sometimes referred to as *marriage protection*. In other words, do married people fare better than their unmarried counterparts because mentally and physically healthier persons are more likely to marry in the first place (*marriage selection*), or because of the presumed social, psychological, economic, and environmental benefits associated with having a spouse (*marriage protection*)?

The potential importance of selection as a competing explanation with marriage protection has been recognized since the mid-1800s. In a study of the influence of marriage on mortality in France, William Farr (1858) notes:

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> Cretins do not marry; idiots do not marry; idle vagrants herd together, but rarely marry. Criminals by birth and education do not marry to any great extent. . . . The children of families which have been afflicted with lunacy are not probably sought in marriage to so great an extent as others; and several hereditary diseases present practically some bar to matrimony. The beautiful, the good, and the healthy are mutually attractive; and their unions are promoted by the parents in France (Farr 1858, p. 509).

Many other investigators since Farr's time have referred explicitly to selection as a possible contributor to the better health profiles and greater longevity of the married population as compared with the single population (see, for example, Carter and Glick 1970; Durkheim 1951; Sheps 1961; Shurtleff 1956). Spouses may be selected for better health not only through the direct exclusion of mentally and physically ill persons from marriage, but also through a wide range of selection criteria including income, physical appearance, risk-taking behaviors, health-related habits such as smoking and excessive drinking, and emotional stability. Although the importance of these health-related factors in relation to other attributes of potential spouses has not been evaluated, evidence from a number of studies suggests that health-related characteristics indeed are used as selection criteria for marriage.

A recent study, for example, concluded that the mate selection process was probably a major factor in generating exceptionally high death rates among single Japanese males and females during the past several decades. In particular, the importance of excluding potential spouses with hereditary or psychological diseases in the family line and the use of relatives, neighbors, friends, go- betweens, and private detectives in the arranged marriage process appear to be largely responsible for the 15-year disadvantage in life expectancy experienced by Japanese singles (compared to married Japanese) in the middle of this century (Goldman and Hu 1992). Other studies in Britain, Japan, and Bangladesh found that persons with serious genetic diseases or severe physical handicaps were more likely to be single than were their healthy counterparts (Higashi, Ishihara, and Wada 1979; Imaizumi 1989; Kiernan 1988; Rahman 1991). A study in the United States demonstrated that marriage selection may operate on health-related behaviors, and not simply on health status: differences in smoking behavior by marital status were found to result from personal characteristics or experiences that increased the likelihood of smoking adoption and of marriage and divorce, rather than from the effects of marriage or parenting per se (Waldron and Lye 1989).

Although a considerable body of research has explored the roles of physical attractiveness and personality in mate selection (Murstein 1972, 1980), little is known about the extent to which persons are denied marriage (or choose to remain single) on the basis of these factors. Nevertheless it is plausible that the single population disproportionately includes the physically unattractive, who may be more likely than others to suffer from health problems including obesity. It is also possible that behavioral traits which hinder the formation of stable relationships include a propensity to engage in risky behaviors and hence lead to a higher incidence of accidents, mental problems, and disease among the single.

Although the marriage selection mechanisms described above typically favor healthier persons, this need not always be the case. Indeed, Farr (1858) recognized that the unmarried classes "contain some of the highest members of their race, ascending from the idiot up to Newton." Analyses in the United States demonstrated that the most highly educated persons (who, on average, have the highest life expectancy) are *least* likely to marry (Carter and Glick 1970). Similarly, increases in the propensity of highly educated Japanese to remain unmarried are believed to be partly responsible for recent improvements in life expectancy among single Japanese (Goldman and Hu 1992).

Bernard (1982) notes that whether persons choose not to marry or are selected involuntarily out of marriage,

> [w]e are faced with the irrepressible, inevitable, and—most researchers concede—insoluble chicken-and-egg, cause-and-effect question. Do the married . . . look so much better than the never-married because marriage is good for them or because the less good prospects were selected out of the married population in the first place? . . . Short of a controlled experiment . . . we have to pick and choose our way around and through the data (pp. 18–19).

Some observers would argue that even if a controlled experiment were feasible, it could never address the issue properly because the potential benefits of marriage stem in part from psychological and social advantages of sharing life with a loved one, not with a randomly chosen mate!

In the absence of experiments, what data have scholars used to identify or quantify marriage selection mechanisms as they relate to health or longevity? Until the most recent decade, the great majority of investigations have been essentially cross-sectional—that is, they have been based either on a single-round survey or on aggregate death registration or health utilization data for a short period. Since the late 1970s, researchers have relied increasingly on prospective survey data to explore the relationship between marital status (and social support, more generally) and health status (House, Landis, and Umberson 1988). Without doubt, these longitudinal community surveys in the United States and Europe (Berkman and Breslow 1983; Blazer 1982; House, Robbins, and Metzner 1982; Schoenbach et al. 1986; Welin et al. 1985; Zuckerman, Kasl, and Ostfeld 1984) have been the most promising studies to date for establishing the effects of marital status, and of related social and economic factors, on health. Specifically, by introducing adjustments for baseline health status and related risk factors, these prospective investigations have demonstrated that the health benefits of marriage and social support persist in the presence of controls for selection effects. One drawback of the prospective studies, however, is that they have not attempted to evaluate directly the impact of marital selection effects or to determine the relative importance of selection and causal factors on differential mortality by marital status.

A major issue motivating the present study concerns the types of data and analyses that would permit researchers to assess the impact of marital selection on the excess mortality of singles. (Note that we restrict the focus of our analysis to the single population because several potentially different types of selection processes determine whether a person becomes divorced or widowed, as well as whether a formerly married person remarries.) One could reasonably argue that the ideal data set for this purpose would be a prospective survey which follows a young unmarried sample through the adult life span, collecting repeated measures of marital status, health status, health-related risk factors and behaviors, and socioeconomic status. Such a survey would be based on a sample large enough to produce sufficient numbers of deaths at young and middle ages and to distinguish among the single, divorced, and widowed groups.[1]

A related issue is whether prospective data offer the only possibilities for this type of research or whether legitimate conclusions about the relative effects of selection versus protection can be drawn from carefully designed analyses of cross-sectional data. As described in detail in the next section, many researchers in the past made inferences about the importance of marriage selection on the basis of particular patterns of mortality by marital status, estimated from cross- sectional data. In each of these instances, conclusions were based on the degree of similarity between an observed pattern and a pattern hypothesized to result from selection.

Much of the remainder of this paper is concerned with evaluating some of these methods, used repeatedly since Durkheim's classic study of suicide (Durkheim 1897, 1951). In particular we focus on two types of approaches, and examine how several scholars have

applied each to draw conclusions about the importance of marriage selection in producing the excess mortality of the single population. The first approach is based on age patterns of mortality differentials; the second, on the direction and strength of the relationship between the magnitude of the mortality differential and the relative size of the single population. If these types of analyses are theoretically sound, they could advance our understanding of the mechanisms underlying marital status differences in mortality on the basis of readily available data. If they are flawed, we will be forced to reevaluate many of the conclusions already derived from such cross-sectional patterns.

NATURE OF THE ARGUMENTS

Age Patterns of Mortality Differentials

The great majority of analysts focusing on marital status differentials in mortality have relied on a measure of relative risk known as the relative mortality ratio (RMR)—the ratio of the death rate of a specified unmarried group (e.g., singles) to the death rate of the married group. In most populations, age schedules of relative mortality among singles are characterized by rising values from the twenties to the mid-thirties or the forties, followed by declining ratios through the oldest age groups (see, for example, Hu and Goldman 1990). This observed age pattern of the RMR has led to several (sometimes subtle) inferences in support of marriage selection. For example, Livi-Bacci (1985, p. 104) argues that "if marriage operates its selection of the healthier lives," we should expect increasing levels of excess mortality of singles between ages 25 and 40—a growth which "should level off and stop altogether when nuptiality falls to very low levels . . . after 40 years of age or so." In addition, he notes that "the decline of single excess mortality after a certain age is to be expected." Such hypothesized age schedules of excess mortality in the single population fit the observed Italian patterns, and Livi-Bacci ultimately favors selection theories over hypotheses related to the protective role of marriage.

Other analysts have made similar arguments relating typical age patterns of excess single mortality to selection mechanisms.[2] For example, Zalokar (1960, p. 57) notes that because the marriage rate falls off rapidly by ages in the forties, "the influence of medical selection, then, can be expected to decline to a relatively small proportion of all factors in the death rates by marital status of women over age 50." Sheps (1961, p. 552) notes that the decrease in single women's relative mortality with increasing age "supports the notion that selective factors may play an important role in producing the observed excess mortality in the nonmarried groups."

Other researchers faced with unexpected age patterns of RMRs have concluded that the observed age patterns of the differentials are not consistent with selection hypotheses and that by default, causal mechanisms are responsible for the observed mortality patterns. For example, Goldman, Lord, and Hu (1992) surmise that the relatively constant ratios observed through the middle and older age groups of Japanese women could *not* arise predominantly from marriage selection processes. Dùrkheim (1951) uses a related argument to conclude that selection could not be the driving force behind the higher suicide rates of singles:

> If it [the differential suicide rate] were a result of matrimonial selection, it should grow from the start of this selection, or the age when young men and women begin to marry. At this point, a first difference should be noted which should increase with the progress of selection . . . In short, the maximum should be reached when the good grain is completely separated from the tares, when the whole population admissible to marriage has actually been admitted. . . . This maximum should occur between 30 and 40 years of age; few marriages are made later (p. 182).

Durkheim goes on to note that the observed *coefficients of preservation* (the suicide rate of the single divided by the suicide rate of the married population, a measure equivalent to the relative mortality ratio restricted to deaths from suicide) are not consistent with selection because the maximum ratio is achieved too early (e.g., in the 25-to-30 age group for France in 1889–1891).

Relationship between the RMR and the Relative Size of the Single Population

An alternative strategy that has been used to draw conclusions about selection relies on the relationship between the relative mortality ratio and the relative size of the single population. The underlying hypothesis, implicit in the work of several researchers (Fuchs 1974; Hu and Goldman 1990; Kisker and Goldman 1987; Livi-Bacci 1985), is the notion that populations in which the great majority of persons marry should be characterized by greater selectivity effects among those who remain single than populations in which substantial proportions never marry. Livi-Bacci states the premise as follows:

> Let us suppose, for a moment, that the selective efficiency of marriage acts with the same strength in different populations. By efficiency, we indicate the "screening" action made with respect to health, etc. of the partner. It follows that the lower is the proportion remaining single (the proportion successfully unscreened) the higher must be the frequency—among the unmarried—of the less healthy and the impaired, and the higher should be the excess mortality of the singles (1985, p. 104).

Other researchers do not present such a formal argument, but the underlying logic is similar. For example, a smaller proportion of the U.S. population was married in the 1980s than in the 1970s; according to Keyfitz (1988, p. 110), this fact implies that "selective effects on longevity have more room to operate."

Researchers typically have tested such hypotheses by estimating the correlation between the proportion single at various ages (usually ages near the end of the marriage span) and the relative mortality ratio at corresponding ages (Fuchs 1974; Kisker and Goldman 1987; Livi-Bacci 1985). In most cases, the resulting correlation coefficients are negative. For example, on the basis of data for 26 countries, Livi-Bacci reports a coefficient of -0.603 between the proportion of females single (at ages 40–44) and the relative mortality ratio (at ages 35–44). Kisker and Goldman obtain even stronger correlations when comparisons are restricted to cohorts within a given country (notably Japan and France).

The first inference which researchers have drawn from these analyses is that selection is important in accounting for the observed mortality differentials. Further deductions occasionally are drawn from the size of the correlation coefficient. For example, Livi-Bacci offers one possible reason why the resulting coefficients are more negative for women than for men:

> Among females, . . . most of the excess mortality can be attributed to the permanent selective role of marriage—the negative relation between the proportion single and excess mortality conforming to the expectations. Among males, the "protective" role of marriage has a higher efficiency, explains a larger share of single excess mortality, obscuring the relationship between this latter and the proportion single (1985, p. 105).

For the remainder of this paper, we use a mathematical simulation model to demonstrate the weaknesses of the arguments presented above. Some statements are proved false; others are shown to be inconclusive. In particular we demonstrate that marriage

selection mechanisms, operating in the absence of any marriage protection factors, can produce unanticipated patterns in the relative mortality ratio with respect to both age and the relative size of the single group. As a consequence, we show the incorrectness of many inferences about the importance of selection or causal mechanisms that have been derived from observed patterns of the relative mortality ratio.

A SIMULATION MODEL

We consider a simple mathematical model in an attempt to explore the nature of the mortality differentials resulting from marriage selection on the basis of health characteristics. Although we recognize that observed mortality differentials by marital status are almost certainly the result of both causal and selection factors, we consider hypothetical populations in which causal effects are entirely absent so that we can distinguish the potential effects of selection mechanisms from those of marriage protection. In essence we construct a hypothetical population (cohort) in which marriage offers no health benefits over the single state, but in which healthy persons are more likely to marry than are unhealthy individuals. Thus, married persons will appear healthier than their single counterparts (i.e., the relative mortality ratios will exceed unity) completely as a result of the marriage selection process.

This marriage process has three essential components: first, members of the population differ with regard to underlying frailty (measured by risks of dying); second, characteristics related to frailty are observable to the potential spouse;[3] and third, the likelihood of marriage is related inversely to measured frailty. Were all members of the population to have either identical mortality or identical marriage schedules, the resulting relative mortality ratios would necessarily be unity at all ages. We consider a population composed of only two types of individuals: "healthy" persons, who have a risk of dying of $\lambda_1\mu(x)$, and "frail" persons, who have a risk of dying of $\lambda_2\mu(x)$, where $\lambda_1 < \lambda_2$.[4] Beginning at some defined age, healthy and frail individuals marry with risks $\theta_1\nu(x)$ and $\theta_2\nu(x)$ respectively, where $\theta_1 > \theta_2$. Thus $\mu(x)$ and $\nu(x)$ denote the baseline mortality and marriage schedules of the cohort, λ_2/λ_1 (which exceeds unity) denotes the relative mortality risk of the frail, and θ_2/θ_1 (which is less than unity) denotes the relative marriage risk of the frail.[5] (Although the mathematics would become more complicated, this simple model could be expanded to allow for a continuous distribution of frailty across the population.) The final parameter c refers to the proportion of individuals in the healthy group at the first possible age at marriage.

The description of the parameters is summarized below, and the marriage selection process is shown schematically in Figure 1. This model can be viewed as an extension of the simple "heterogeneity model" that Vaupel and others have used to distinguish between the mortality curve of a cohort and that of each of its members (Vaupel, Manton, and Stallard 1979; Vaupel and Yashin 1985).

Parameters:

$\lambda_1\mu(x)$	mortality risk of healthy
$\lambda_2\mu(x)$	mortality risk of frail
$\theta_1\nu(x)$	marriage risk of healthy
$\theta_2\nu(x)$	marriage risk of frail
c	proportion healthy at first age of marriage

Because our interest focuses on the single population (in comparison with everyone else), we consider the ever-married population as one group rather than distinguishing

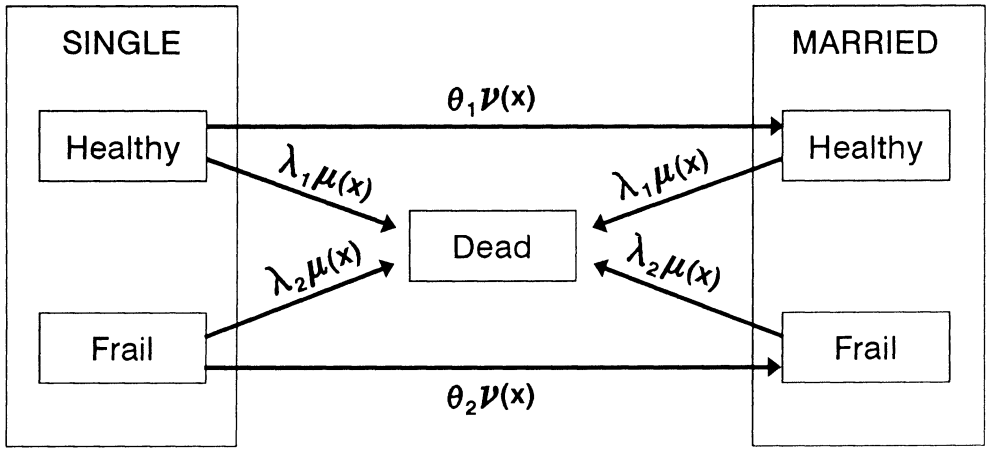


Figure 1. A Simple Marriage Selection Model

among the currently married, divorced, and widowed subgroups. Thus, for the remainder of the analysis, we redefine the relative mortality ratio as the age-specific mortality risk of the single population divided by the corresponding mortality risk of the ever-married population.[6] This typology also enables us to keep the mathematics tractable because we can restrict the model to one sex by ignoring divorce and widowhood. Note that in this one-sex model, we do not make any explicit assumptions about the nature of assortative mating. For example, we do not disaggregate the marriage risk of the frail in terms of the two component risks, namely the risks that a frail individual marries a frail versus a healthy spouse. Rather we invoke a common assumption underlying one-sex models—that the "supply" of spouses of the opposite sex is large enough to support the average marriage rates specified in the model.

The parameters and baseline schedules defined above specify completely the mortality risks of the single and the ever-married population and hence define the RMR schedule at any age (greater than the first age at marriage). As described in detail in the appendix, the mortality risks of the single and of the ever-married population each can be expressed as a weighted average of the risks for the healthy and the frail subgroups; the weights refer to the proportions of single (and ever-married) persons who are healthy and frail respectively. In the remainder of this paper, for convenience, we use the term *married* interchangeably with *ever-married* and the term *rate* to denote a hazard or risk function.

Before making any actual calculation of the RMR, we must consider a plausible set of baseline schedules and parameter values. We represent the underlying mortality schedule by a Gompertz curve and the underlying marriage schedule by a model nuptiality schedule (Coale and McNeil 1972); the baseline curves, derived from 1980 data for Japanese females, are shown graphically in Figure 2. Because we have no empirical information with which to identify the remaining parameters (for example, the initial proportion frail in the population or the risks of dying and marrying among the frail relative to the healthy), we explore a range of values that are consistent with observed data. That is, although the parameters pertaining to the frail and the healthy subgroups never could be identified from actual data, the set of parameters taken together defines the overall mortality and marriage rates of the population. As described in the appendix, we consider only values of the parameters that lead to plausible estimates of life expectancy and celibacy in the overall population. The resulting RMRs, presented in the examples below, range between 1 and 4. A recent comparative analysis of relative mortality ratios determined that the RMRs

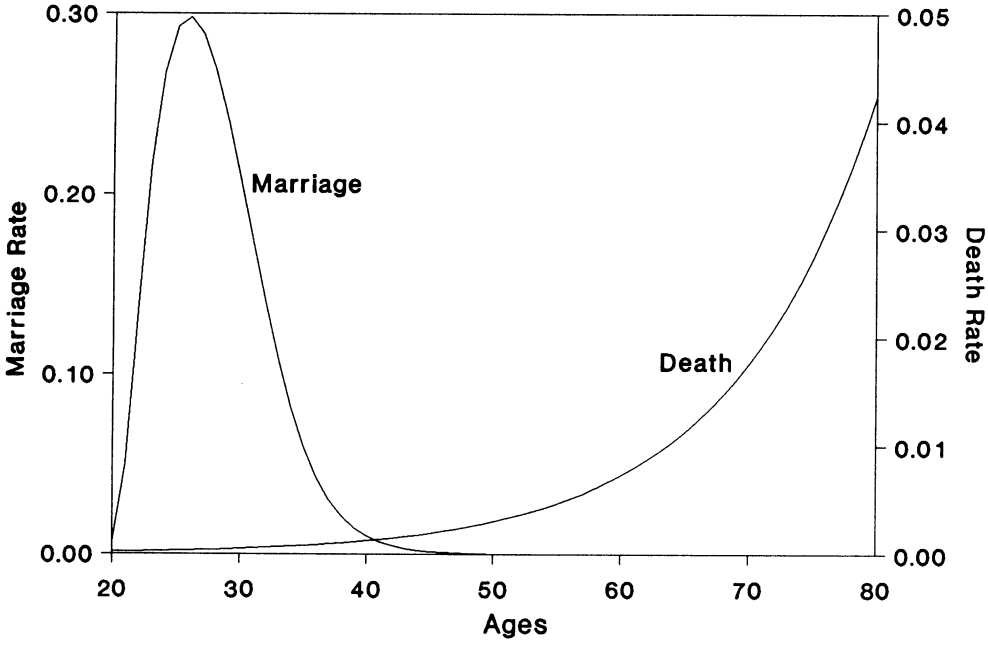


Figure 2. Baseline Marriage and Death Rates

averaged to about 1.5 for females and 2.0 for males across 16 industrialized countries and were as high as 5 for middel-aged Japanese singles in the mid-1900s (Hu and Goldman 1990).

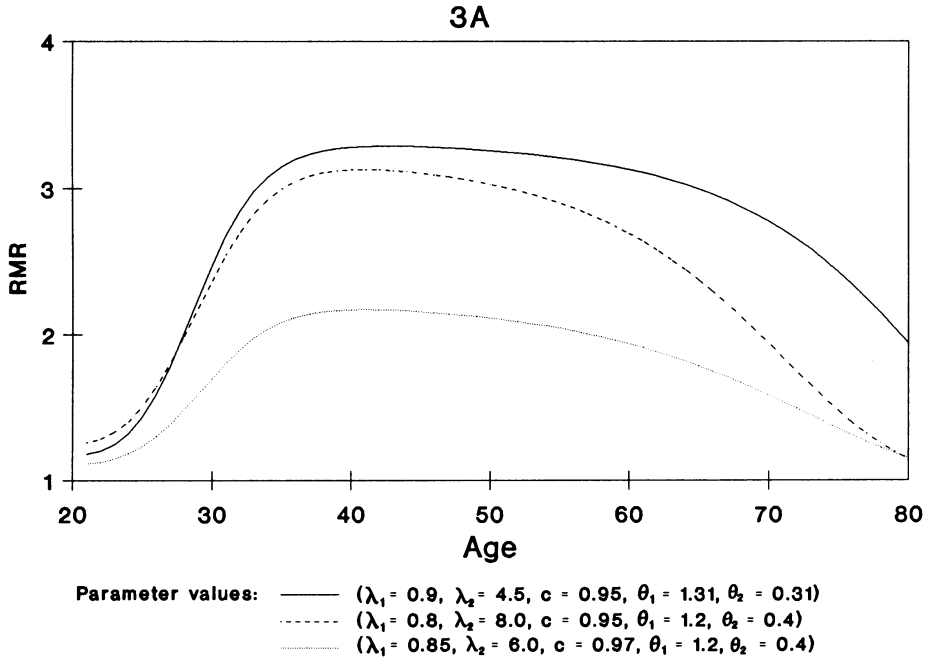
RESULTS

Age Patterns of the RMR

As described earlier, a pervasive hypothesis in previous research has been that marriage selection mechanisms lead to relative mortality ratios which increase through much of the marriage span but begin to decline once marriages no longer take place—that is, around age 40. The underlying notion is that once marriages cease, the fraction of frail persons in both the single and the ever-married populations diminishes progressively. The frail population presumably declines more rapidly among the singles (because the frail form a greater part of the single than of the married group) and the relative mortality ratio of singles decreases accordingly.

The RMR schedules shown in Figure 3A support this hypothesis. The RMR schedules are derived from the baseline marriage curve shown in Figure 2, in which the marriage rate peaks at age 25 and declines continuously to very low values by age 40. As hypothesized, the resulting RMR schedules shown in Figure 3A increase progressively from age 20, reach maximum values near age 40, and decline steadily thereafter toward unity.

Through the choice of different parameters, however, unexpected age patterns also may result from the same type of marriage selection process. In general the relative mortality ratios still increase from about age 20 to 40.[7] As shown in Figure 3B, however, the RMR schedule can remain relatively flat through the middle and older ages or can rise well beyond the end of the marriage span. Analyses presented in Goldman et al. (1992)



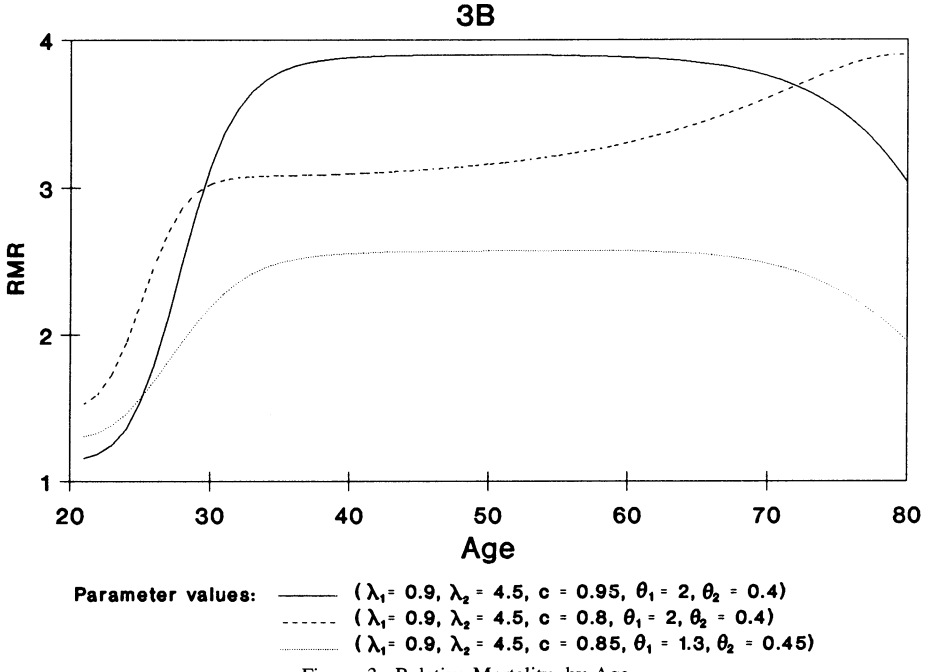


Figure 3. Relative Mortality, by Age

show that the relationships between the parameters of the selection model and the age pattern of the RMR are extremely complicated and often counterintuitive. In some instances, a change only in the value of c (the proportion of the population that belongs to the healthy subgroup at the initial age at marriage) drastically alters the shape of the resulting RMR schedule.[8]

How do these unanticipated RMR patterns arise? As hypothesized, once marriages no longer take place, both the married and the single populations "improve" their composition as they lose their frailest members (through death) more quickly than their healthy members. Consequently the death rates of the married and of the single eventually begin to approach one another, and the relative death rate (i.e., RMR) *ultimately* converges to unity. Mathematical analysis, however, reveals that the age at which the convergence begins depends on the *relative rates of change* of the two death rates and is not predictable: convergence need not begin at the end of the marriage span or even within the human life span.

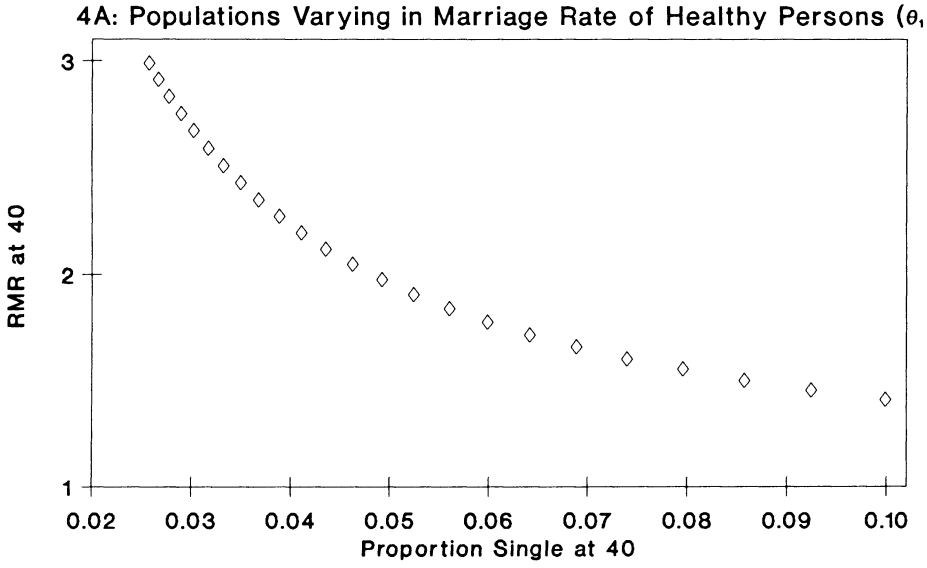
In summary, the schedules shown in Figure 3B are *inconsistent* with many of the inferences previously made by researchers. Although the excess mortality of the singles derives entirely from marriage selection mechanisms in our examples, the RMRs fail to decline at the point when single individuals (virtually) stop marrying. Many researchers would have refuted the importance of marriage selection and would have embraced various marriage protection theories on the basis of the type of schedules shown in 3B.

Relationship between the RMR and the Size of the Single Group

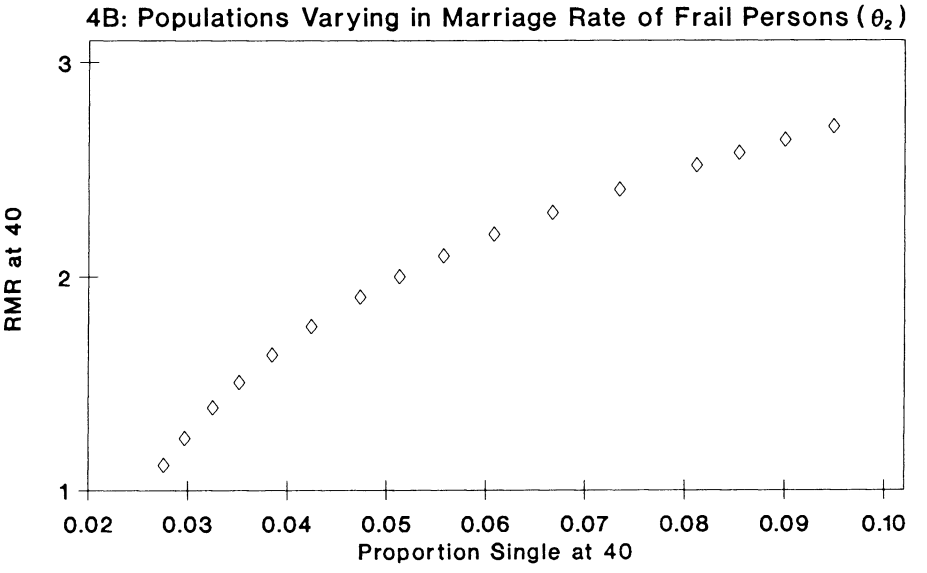
The second type of approach used by researchers to make inferences about marital selection derives from the apparent negative relationship between the excess mortality of the singles and their relative size: populations in which relatively few individuals remain single are typically those in which single persons experience high excess mortality. We use the simulation model described here to demonstrate that marriage selection on the basis of health characteristics need not result in such a negative relationship. We begin by considering two highly simplified scenarios in which only the marriage risks of the healthy (θ_1) or of the frail (θ_2) vary across populations. We demonstrate that the relative size of the single population will have the hypothesized negative relationship with the RMR when populations differ with regard to the marriage rates of the healthy. By contrast, when populations differ with respect to the frail subgroup's marriage rates, the relative size of the single population will be related *positively* to its relative mortality.

These findings are presented in Figures 4A and 4B and can be understood in the following way. Changes in θ_1 and changes in θ_2 have the same directional impact on the proportion remaining single, but they have opposite effects on the relative mortality of singles. Specifically, an increase in the marriage risk of either the healthy or the frail subgroup, in the absence of a change in any other parameter, necessarily lowers the proportion of the overall population that remains single at any specified age. An *increase* in the marriage rate of the healthy (θ_1), in the absence of any other change, leads to a quicker depletion of healthy persons from the single state, and consequently to a higher death rate among singles and a lower death rate among the married; hence an *increase* in θ_1 necessarily leads to an increase in the RMR. The same effect would be achieved by a *decrease* in the marriage rate of the frail (θ_2). Stated differently, an *increase* in the marriage rate of the frail (θ_2) leads to a quicker depletion of frail persons from the single state, and consequently to a decrease in the death rate of singles and an increase in the death rate of ever-married persons—that is, to a decrease in the RMR.

Figure 4A depicts a series of simulated populations (which could represent countries or



Parameter values: $\lambda_1 = 0.9$, $\lambda_2 = 4.5$, $c = 0.9$, θ_1 varies between 1.06 and 1.98, $\theta_2 = 0.5$. Parameters of baseline marriage schedule are $\mu = 25$, $\sigma = 3$, $C = 0.9$.



Parameter values: $\lambda_1 = 0.9$, $\lambda_2 = 4.5$, $c = 0.8$, $\theta_1 = 1.6$, θ_2 varies between 0.42 and 1.47. Parameters of baseline marriage schedule are $\mu = 25$, $\sigma = 3$, $C = 0.9$.

Figure 4. Relative Mortality Ratio versus Proportion Single at Age 40

cohorts within a country). These populations differ only with regard to θ_1, the marriage rate of the healthy; proportions single and relative mortality are measured as of age 40. As expected, populations with lower proportions single (larger θ_1) have higher relative mortality ratios; thus the hypothesized negative relationship between the relative size of the single population and the magnitude of excess mortality is confirmed. Figure 4B depicts simulated populations differing only with regard to the value of θ_2. In this case the resulting relationship between the RMR and the relative size of the single population is *positive*.

Thus the relationship between the magnitude of the excess mortality among singles and the relative size of this group depends critically on how the marriage rates for the healthy and the frail subgroups vary across populations. If differences across populations result predominantly from varying degrees of discrimination against frail persons, the relationship is likely to be positive. This counterintuitive finding results from the fact that in this scenario, populations with low fractions remaining single are precisely those which achieve near-universal marriage because of the relatively easy entry of the frail into marriage; hence these populations are characterized by relatively little excess mortality among the singles. By contrast, if differences across populations result primarily from varying degrees of voluntary celibacy (for example, among well-educated professional women), the relationship is likely to be negative: small single populations contain primarily frail individuals who are unable to marry, whereas large single populations are dominated by healthy individuals. Although many analysts may have had the latter scenario in mind—believing, for example, that variations in marriage rates across countries are determined by the extent to which (healthy) women and men elect to remain single—such a premise has never been stated explicitly.

In fact, Livi-Bacci (1985, p. 104) is the only researcher to present any condition associated with the hypothesized negative relationship. We repeat part of his argument presented earlier: "Let us suppose . . . that the selective efficiency of marriage acts with the same strength in different populations. It follows that the lower is the proportion remaining single . . . the higher should be the excess mortality of the singles." An important component of this argument is that populations are characterized by selection processes *of the same strength*. In the simple simulation model presented in this paper, the *strength* of marriage selection is measured by the relative marriage risk of the frail (θ_2/θ_1): a relative risk of unity indicates the absence of marriage selection (on the basis of health status), whereas successively smaller values indicate stronger selection processes in which the frail are progressively less likely than their healthy counterparts to marry. It should be clear that the two scenarios shown in Figure 4 violate Livi-Bacci's assumption because variation in the marriage rate of *either* the healthy *or* the frail subgroup necessarily alters the relative marriage risk or the strength of the selection process.

What, according to our model, is the effect of changing marriage rates—in a selection process of fixed strength—on the ensuing relationship between the relative size of the single group and the RMR? We answer this question by simulating different populations so that the marriage rates of both the healthy and the frail groups vary across populations but the relative marriage rate remains unaltered. The results are more complicated than in the previous examples, and require that we distinguish carefully between the absolute death rate and the relative mortality ratio of the single population.

As a result of increases in the marriage rates of the healthy and the frail, lower proportions remain single at any specified age. Mathematical results (not shown here) reveal that increases in the marriage rates θ_1 and θ_2 (in the absence of any other change) lead to higher absolute death rates among the single. Hence a consistent negative relationship exists between the relative size and the *absolute* death rate of the single population. Mathematical arguments, however (also not shown here), demonstrate that contrary to intuition, higher marriage rates also result in higher death rates among the ever-married.[9] The net effect of

these increases in the absolute death rates of both the single and the ever-married populations on the relative mortality ratio is unpredictable.

Indeed, the results presented in Figure 5 show that the relationship between the percentage single and the relative mortality ratio can be negative, positive, or even nonmonotonic. The particular nature of the association depends critically on the values of the parameters in the model. In the examples shown in Figure 5, we generated the different panels by varying either the relative marriage risk or the initial proportion of the population that was healthy. Within a given panel, however, the only parameters that vary are the absolute marriage risks; they vary in such a way as to keep the relative marriage risk (i.e., strength of marriage selection) constant across populations. Clearly, the results are not consistent with Livi-Bacci's claim.

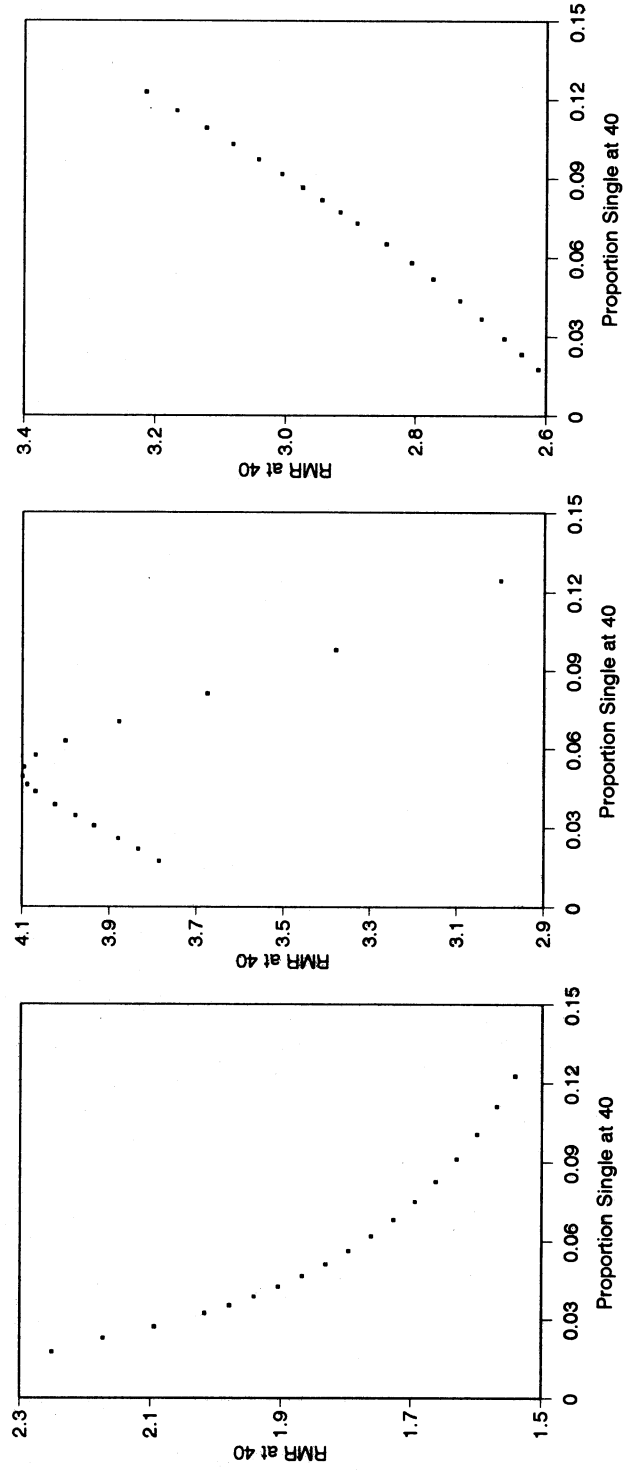
The findings in Figure 5 highlight the complicated nature of the relative mortality ratio and ultimately illustrate the difficulty of drawing inferences from resulting patterns of the RMR. Because changes in the RMR depend on the relative rates of change of its numerator and denominator, the behavior of the RMR can be unpredictable even when the behaviors of the death rates of the single and the ever-married populations are understood.

These results suggest that correlations between the proportion single and the death rate of the singles may be more informative than those between the proportion single and the RMR. Yet it is crucial to recognize that such a conclusion depends entirely on two assumptions: 1) that variation across populations arises solely from differences in the marriage rates θ_1 and θ_2, and 2) that the marriage rates vary in such a way as to keep the relative marriage risk of the frail constant across populations. Clearly, such assumptions are unlikely to be satisfied in typical data sets. For example, variations in the overall level of mortality across populations probably would obfuscate the negative relationship between the absolute death rate of singles and the proportion single described above.[10] More generally, variations in several aspects of the mate selection process—absolute risks of marriage and mortality, the initial distribution of frailty, and the marriage and mortality differentials between the frail and the healthy—would add considerable scatter to the relationships depicted in Figures 4 and 5.

In summary, the results in Figures 4 and 5 reveal that a consistent negative relationship between the proportion single and the magnitude of the RMR occurs in only one of three highly simplified scenarios—namely, when variation across populations results entirely from differing marriage rates among the healthy (e.g., from different rates of voluntary singlehood; see Figure 4A). The graphs also demonstrate that it is meaningless to use the resulting correlation coefficient between the proportion single and the RMR to make inferences about the strength of the marriage selection process. When the strength of this process is constant across populations, the relationship need not even be monotonic, let alone linear. When the relationship is consistently negative (Figure 4A), the strength of the selection process must vary from one population to another.

ALTERNATIVE ARGUMENTS BASED ON MARRIAGE PROTECTION THEORIES

The simulation exercises described here refute some of the arguments presented in earlier research by demonstrating that marriage selection acting alone can produce patterns which are often inconsistent with our intuition. Moreover, even if marriage selection necessarily resulted in the hypothesized patterns—namely, declining RMRs beyond marriageable age and a negative relationship between the RMR and percent single—the occurrence of the predicted patterns in itself would not justify causal inferences about the



Parameter values: For all panels, $\lambda_1 = 0.9$, $\lambda_2 = 4.5$ and parameters of baseline marriage schedule equal $\mu = 25$, $\sigma = 3$, and C = 0.9. For Panel A, $(\theta_2/\theta_1) = 0.5$ and c = 0.9. For Panel B, $(\theta_2/\theta_1) = 0.1$ and c = 0.9. For Panel C, $(\theta_2/\theta_1) = 0.1$ and c = 0.75.

Figure 5. Relative Mortality Ratio versus Proportion Single at Age 40, under Constant Strength of Marriage Selection

importance of marriage selection in producing the observed mortality differentials. As described below, the *same* patterns of mortality differentials that some researchers have used to advocate selection hypotheses have been employed by others to support marriage protection theories.

Some sociologists claim that the decline of relative mortality ratios with increasing age is consistent with theories of social integration. For example, Durkheim (1951, p. 209) asserts that "suicide varies inversely with the degree of integration of the social groups of which the individual forms a part." In defense of this premise, Durkheim suggests that the family environment (primarily the presence of children) is responsible for the observed age pattern of RMRs for suicide.[11] Several scholars have expanded Durkheim's reasoning to encompass many or all causes of death. They maintain that the protective aspects of marriage, including the presence of children in the home, are likely to be greatest at ages below the forties, thereby producing a general decline in relative mortality ratios through middle and older ages (see, for example, Gove 1973).[12]

Sociologists also have argued that status integration theory can be used to predict the relationship between the RMR and the percentage of persons in each marital state. The theory, which originates with Durkheim's study, postulates that the suicide rate of a population varies inversely with the level of status integration in the population. This relationship derives from the hypothesis that persons in infrequently occupied status configurations (i.e., low status integration) are less able to maintain stable social relationships and to conform to societal expectations, and thus are more likely to experience role conflict (Gibbs 1982; Gibbs and Martin 1958; Stafford and Gibbs 1985). On the basis of a similar but more general theory, Dodge and Martin (1970) postulate that the degree of status integration varies inversely with the level of socially induced stress and hence with the rate of morbidity and mortality from chronic diseases. Gibbs and colleagues measure status integration by the percentage of persons occupying a particular combination of states, typically including marital status, age, and gender (as well as, for example, occupation and religion). Hence, without any reference to the role of marriage selection, status integration theory predicts that smaller proportions remaining single should be associated with higher suicide death rates and higher rates of stress, illness, and general mortality in the single population.

CONCLUSIONS

Although most causal relationships among social and demographic factors are potentially obfuscated by selection mechanisms, the causal relationship between marital status and mortality seems to be linked inextricably with selection. In part this occurs because some of the criteria for selection into marriage (e.g., health status) are related *directly* to the outcome of interest (mortality) and because other criteria (e.g., factors related to personality) are largely unobservable to the analyst but are clearly associated with health-related behaviors (e.g., drinking and risk taking).

In spite of the difficulties, many researchers continue their struggle to identify the relative roles of marriage selection and causal mechanisms in producing observed differences in health and longevity. Although the use of prospective community studies in recent years has confirmed the existence of protection mechanisms and has enhanced our understanding of some of the social and economic pathways underlying such causal processes, relatively little of this research has focused on the importance of selection factors in relation to causal factors.

As described in this paper, a number of researchers have attempted to distinguish between selection and protection mechanisms on the basis of particular patterns of

differential mortality by marital status, typically derived from cross-sectional data. The mathematical simulations presented here demonstrate that inferences drawn from such observations are generally not justifiable. Although the hypothesized patterns, such as declining relative mortality beyond the upper age at marriage, could result entirely from marriage selection, so could many contrasting patterns. Hence it is indefensible either to use hypothesized patterns to support arguments in favor of selection or to use deviant patterns to refute selection arguments in favor of marriage protection theories. Were this not sufficient cause for concern, a review of relevant sociological studies reveals that some advocates of marriage protection posit virtually the same patterns of mortality differentials by marital status as do some proponents of selection theories. It seems highly unlikely that the analyst ever could use the types of aggregate patterns described here to distinguish between selection and causal explanations. More generally, this result casts doubt on the usefulness of cross-sectional data for evaluating the relative roles of causal and selection processes in producing the excess mortality of the unmarried population; it suggests that longitudinal data may offer the only promising approach.

NOTES

[1] Unfortunately, most of the prospective studies that have been used to assess the relationship between social support and health are restricted to persons beyond typical marriageable age and are based on samples too small to allow separate consideration of the different unmarried groups.

[2] One drawback associated with almost all of these inferences is that they are derived from a hypothesis about cohorts, but the inferences typically are based on period data (presumably because cohort data on marital status differentials in mortality are scarce). It is evident that a wide range of observed (age) patterns could be obtained from period data simply by positing changes over cohorts in the nature of the marriage selection process. Thus the researchers assume implicitly that the marriage selection process is stationary over time. In this paper we do so as well—that is, we treat inferences drawn from earlier research as if they were based on cohort data.

[3] Such characteristics include not only diseases and disabilities but also health-related behaviors such as diet, smoking, drinking, and other forms of risk-taking behavior.

[4] By assuming that the risk of mortality is a function of age alone (and not of marital status), we also are assuming implicitly that the mortality risks of spouses are independent of one another.

[5] Note that by setting $\lambda_1 = \theta_1 = 1$, we could have defined the model in terms of two baseline curves and only three parameters. We have chosen to use five parameters, however, in order to avoid modifying the baseline curves for successive calculations.

[6] Some analysts prefer the measure defined with respect to ever-married persons. For example, Zalokar (1960) argues that the conventional measure presumes a beneficial effect of marriage, whereas the measure defined with respect to the ever-married population is more appropriate for examining the potential influence of selection processes. The age patterns resulting from the two measures of the RMR are generally similar to one another (Goldman et al. 1992).

[7] Examples not presented here demonstrate that the RMRs can begin to decline somewhat before the end of the marriage span (i.e., age 40).

[8] The only intuitive relationship is the following one between the RMR and the marriage risks. Specifically, an increase in the relative marriage risk of the frail (θ_2/θ_1), caused either by an increase in θ_2 or by a decrease in θ_1, signifies a decrease in the strength of the marriage selection process; the relative mortality ratio necessarily decreases. By contrast, the relationship between the RMR and the mortality risks λ_1 and λ_2 is extremely complex.

[9] Although it may seem implausible that increases in the marriage rates θ_1 and θ_2 can simultaneously increase the death rates of the single and the ever- married groups, we must remember that changes in the marriage rates also lead to changes in marital-status composition—namely, the percentages that remain single and that marry.

[10] Hu and Goldman (1990) examined the relationship between the proportion in a given marital state (married, single, widowed, and divorced) and the death rate of that marital state, after

introducing controls for time period and age. Log-linear regression models were fitted to reported numbers of deaths and of persons for males and females in 16 countries. In 28 out of 32 cases, the resulting coefficient between the relative size of the single population and its mortality rate was negative.

[11] Durkheim (1951) does not state explicitly how changes in the family environment over the life cycle result in the observed age pattern of RMRs (for suicide), but he suggests that the rise in the ratios from the twenties to the maximum and the subsequent decline "evolve[s] in accordance with the prolongation of family life" (Durkheim 1951, p. 184).

[12] Several studies have examined directly the effect of parenting status on mortality (Kobrin and Hendershot 1977), health status (Anson 1989), and negative health behaviors (Umberson 1987).

APPENDIX. CALCULATION OF THE RMR

In the marriage selection model described above, the relative mortality ratio (RMR) at any age is specified completely by five parameters (λ_1, λ_2, θ_1, θ_2, and c) and by the baseline mortality and marriage schedules [$\mu(x)$ and $\nu(x)$]. For convenience, we present again the description of the parameters:

Parameters:

$\lambda_1\mu(x)$	mortality risk of healthy
$\lambda_2\mu(x)$	mortality risk of frail
$\theta_1\nu(x)$	marriage risk of healthy
$\theta_2\nu(x)$	marriage risk of frail
c	proportion healthy at first age of marriage

The RMR is not defined for ages below the first age at marriage (a_0). The RMR at any age $x > a_0$ can be expressed as the ratio of two death rates, each of which is simply a weighted average of the rates for the healthy and the frail subgroups:

$$RMR(x) = \frac{S(x)}{M(x)} = \frac{\text{death rate of single at x}}{\text{death rate of ever-married at x}} = \frac{W^S(x)\lambda_1\mu(x) + [1 - W^S(x)]\lambda_2\mu(x)}{W^M(x)\lambda_1\mu(x) + [1 - W^M(x)]\lambda_2\mu(x)} \tag{A1}$$

where $W^S(x)$ is the proportion healthy among singles age x and $W^M(x)$ is the proportion healthy among ever-married persons age x. Because healthy persons age x exit the single state, by marriage or by death, at a rate of $[\lambda_1\mu(x) + \theta_1\nu(x)]$ and frail persons at a rate of $[\lambda_2\mu(x) + \theta_2\nu(x)]$, we can express the proportion healthy among singles age x, $W^S(x)$ as

$$W_S(x) = \frac{c \exp\{-\int_{a_0}^{x} [\lambda_1\mu(y) + \theta_1\nu(y)]\, dy\}}{c \exp\{-\int_{a_0}^{x} [\lambda_1\mu(y) + \theta_1\nu(y)]\, dy\} + (1-c) \exp\{-\int_{a_0}^{x} [\lambda_2\mu(y) + \theta_2\nu(y)]\, dy\}}. \tag{A2}$$

The expression for the proportion healthy among the ever-married age x, $W^M(x)$, is more

complicated. Recognizing that the number of healthy ever-married persons is the difference between the total healthy population and the healthy single population (forming the numerator of Equation (A3), below) and that a corresponding term pertains to the number of frail ever-married persons, we can express $W^M(x)$ as follows:

$$W_M(x) = \frac{[c\ \{\exp[-\int_{a_0}^{x} \lambda_1\mu(y)dy] - \exp\{-\int_{a_0}^{x} [\lambda_1\mu(y) + \theta_1\nu(y)]\ dy\}\}}{[c\ \{\exp[-\int_{a_0}^{x} \lambda_1\mu(y)dy] - \exp\{-\int_{a_0}^{x} [\lambda_1\mu(y) + \theta_1\nu(y)]\ dy\}\} + (1-c)\ \{\exp[-\int_{a_0}^{x} \lambda_2\mu(y)dy] - \exp\{-\int_{a_0}^{x} [\lambda_2\mu(y) + \theta_2\nu(y)]dy\}\}]} \quad \text{(A3)}$$

Note that once the parameters and baseline schedules are specified, Equations (A1), (A2), and (A3) taken together yield the RMR schedule at any age $x > a_0$. In performing the actual calculations, we (1) represent the baseline survival curve (i.e., $\exp[-\int_{a_0}^{x} \mu(y)dy]$) by the closed-form expression for the Gompertz distribution[a] and 2) estimate the baseline survival curve for first marriage (i.e., $\exp[-\int_{a_0}^{x} \nu(y)dy]$) as $1 - \int_{a_0}^{x} g(y)dy$, where g(y) is represented by the Coale-McNeil closed-form expression for the frequency of first marriage[b] and its integral is evaluated numerically (by the trapezoidal rule). The first age at marriage, a_0, is set equal to 20 throughout the calculations.

The baseline mortality and marriage schedules were derived from 1980 data for Japanese females. The mortality rates imply a life expectancy at age 20 (for the total population) of about 60 years. The model nuptiality schedule is based on a mean age at marriage of 25 years, a standard deviation of 3 years, and a proportion eventually marrying of either 0.90 or 0.95. In all of the examples, the estimated life expectancy for the total population between ages 20 and 80 equals approximately 55 years. The proportion married by age 40 varies between about 0.87 and 0.98.

APPENDIX NOTES

[a] The hazard rate of the Gompertz distribution can be expressed as $\mu(x) = Be^{ax}$ and the corresponding survival curve as $S(x) = \exp[\frac{B}{a}(1-e^{ax})]$. In the examples presented here, B equals 0.000037 and a equals 0.088; these values imply a life expectancy at age 20 of almost 60 years for the baseline population. The corresponding life expectancies of the healthy and the frail subgroups can be obtained from the corresponding survival functions, namely $S(X)^{\lambda 1}$ and $S(X)^{\lambda 2}$ respectively.

[b] The frequency curve of first marriages, g(y), typically is expressed as

$$g(y) = \frac{0.1946\ C}{k} \exp\{-0.174\ (z - 6.06) - \exp[-0.2881\ (z - 6.06)]\},$$

where $z = (y - a_0)/k$, a_0 denotes the age at which marriages first take place, k denotes the pace of marriage (relative to a Swedish standard), and C denotes the proportion that eventually marry (Coale

and McNeil 1972). On the basis of the transformations $\mu = a_0 + 11.36k$ and $\sigma = 6.58k$, g(y) can be reexpressed in terms of the mean age at first marriage (μ), the standard deviation of the age at first marriage (σ), and the proportion eventually marrying (C) (Rodríguez and Trussell 1980). (Note that because some members of the population fail to marry, g(y) is not a probability density function and $\nu(x)$ is technically not a risk function, according to statistical definition.)

REFERENCES

Anson, Ofra. 1989. "Marital Status and Women's Health Revisited: The Importance of a Proximate Adult." *Journal of Marriage and the Family* 51:185–94.

Berkman, Lisa F. and Lester Breslow. 1983. *Health and Ways of Living*. New York: Oxford University Press.

Bernard, Jessie. 1982. *The Future of Marriage*. New Haven: Yale University Press.

Blazer, Dan G. 1982. "Social Support and Mortality in an Elderly Community Population." *American Journal of Epidemiology* 115:684–94.

Carter, Hugh and Paul C. Glick. 1970. *Marriage and Divorce: A Social and Economic Study*. Cambridge, MA: Harvard University Press.

Coale, Ansley J. and Donald R. McNeil. 1972. "The Distribution by Age of the Frequency of First Marriage in a Female Cohort." *Journal of the American Statistical Association* 67(340):743–49.

Dodge, David L. and Walter T. Martin. 1970. *Social Stress and Chronic Illness*. Notre Dame, IN: University of Notre Dame Press.

Durkheim, Emile. 1897. *Le Suicide: Etude de Sociologie*. Paris: F. Alcan.

_____. 1951. *Suicide*. Glencoe, IL: Free Press.

Farr, William. 1858. "The Influence of Marriage on the Mortality of the French People." Pp. 504–13 in *Transactions of the National Association for the Promotion of Social Science*, edited by G.W. Hastings. London: John W. Parker.

Fuchs, Victor R. 1974. "Some Economic Aspects of Mortality in Developed Countries." Chapter 11 in *The Economics of Health and Medical Care*, edited by Mark Perlman. New York: Wiley.

Gibbs, Jack P. 1982. "Testing the Theory of Status Integration and Suicide Rates." *American Sociological Review* 47:227–37.

Gibbs, Jack P. and Walter T. Martin. 1958. "A Theory of Status Integration and Its Relationship to Suicide." *American Sociological Review* 23:266–77.

Goldman, Noreen and Yuanreng Hu. 1992. "Excess Mortality among the Unmarried: A Case Study of Japan." Forthcoming in *Social Science and Medicine*.

Goldman, Noreen, Graham Lord, and Yuanreng Hu. 1992. "Marriage Selection and Age Patterns of Mortality: A Mathematical Investigation." Paper presented at Annual Meetings of the Population Association of America, CO. Forthcoming in *Mathematical Population Studies*.

Gove, Walter R. 1973. "Sex, Marital Status, and Mortality." *American Journal of Sociology* 79:45–66.

Higashi, T., O. Ishihara, and T. Wada. 1979. "Medicosocial Aspect of People with Epilepsy in Japan: A Survey from Standpoint of Epilepsy Center." *Folia Psychiatrica et Neurologica Japonica* 33(3):399–405.

House, James S., Karl R. Landis, and Debra Umberson. 1988. "Social Relationships and Health." *Science* 241:540–45.

House, James S., Cynthia Robbins, and Helen L. Metzner. 1982. "The Association of Social Relationships and Activities with Mortality: Prospective Evidence from the Tecumsah Community Health Study." *American Journal of Epidemiology* 116:123–40.

Hu, Yuanreng and Noreen Goldman. 1990. "Mortality Differentials by Marital Status: An International Comparison." *Demography* 27(2): 233–50.

Imaizumi, Yoko. 1989. "Mortality Rate of Huntington Disease in Japan: Secular Trends, Marital Status, and Geographic Variations." *Japan Journal of Human Genetics* 34:169–78.

Keyfitz, Nathan. 1988. "A Markov Chain for Calculating the Durability of Marriage." *Mathematical Population Studies* 1:101–22.

Kiernan, Kathleen E. 1988. "Who Remains Celibate?" *Journal of Biosocial Science* 20:253–63.

Kisker, Ellen E. and Noreen Goldman. 1987. "Perils of Single Life and Benefits of Marriage." *Social Biology* 34:135–52.

Kobrin, Frances E. and Gerry E. Hendershot. 1977. "Do Family Ties Reduce Mortality? Evidence from the United States, 1966–1968." *Journal of Marriage and the Family* 39:737–45.

Livi-Bacci, Massimo. 1985. "Selectivity of Marriage and Mortality: Notes for Future Research." Pp. 99–108 in *Population and Biology*, edited by Nathan Keyfitz. Liège, Belgium: Ordina Editions.

Murstein, Bernard I. 1972. "Physical Attractiveness and Marital Choice." *Journal of Personality and Social Psychology* 22(1):8–12.

———. 1980. "Mate Selection in the 1970s." *Journal of Marriage and the Family* 42:777–92.

Rahman, M. Omar. 1991. "Marriage and Young Adult Mortality in Rural Bangladesh: The Impact of Selection." Unpublished manuscript, RAND, Santa Monica.

Rodríguez, Germán and James Trussell. 1980. *Maximum Likelihood Estimation of the Parameters of Coale's Model Nuptiality Schedule from Survey Data*. Voorburg, Netherlands: International Statistical Institute.

Schoenbach, Victor J., Berton H. Kaplan, Lisa Fredman, and David G. Kleinbaum. 1986. "Social Ties and Mortality in Evans County, Georgia." *American Journal of Epidemiology* 123:577–91.

Sheps, Mindel C. 1961. "Marriage and Mortality." *American Journal of Public Health* 51:547–55.

Shurtleff, Donald A. 1956. "Mortality among the Married." *Journal of the American Geriatrics Society* 4:654.

Stafford, Mark C. and Jack P. Gibbs. 1985. "A Major Problem with the Theory of Status Integration and Suicide." *Social Forces* 63:643–60.

Umberson, Debra. 1987. "Family Status and Health Behaviors: Social Control as a Dimension of Social Integration." *Journal of Health and Social Behavior* 28:306–19.

Vaupel, James W., Kenneth G. Manton, and E. Stallard. 1979. "The Impact of Heterogeneity in Individual Frailty on the Dynamics of Mortality." *Demography* 16:439–54.

Vaupel, James W. and Anatoli I. Yashin. 1985. "Heterogeneity's Ruses: Some Surprising Effects of Selection on Population Dynamics." *The American Statistician* 39:176–85.

Waldron, Ingrid and Diane Lye. 1989. "Family Roles and Smoking." *American Journal of Preventive Medicine* 5(3):136–41.

Welin, L., G. Tibblin, K. Svardsudd, B. Tibblin, S. Ander-Peciva, B. Larsson, and L. Wilhelmsen. 1985. "Prospective Study of Social Influences on Mortality." *Lancet* April 20, 1985:915–918.

Zalokar, Julia B. 1960. "Marital Status and Major Causes of Death in Women." *Journal of Chronic Diseases* 11:50–60.

Zuckerman, Diana M., Stanislav V. Kasl, and Adrian M. Ostfeld. 1984. "Psychosocial Predictors of Mortality among the Elderly Poor: The Role of Religion, Well-Being, and Social Contacts." *American Journal of Epidemiology* 119:410–23.