Chapter One
Introduction

Since at least William Farr’s nineteenth century demonstration that the married experience lower mortality across the age spectrum, there has been substantial empirical and theoretical attention to understanding why marriage might affect mortality. (Farr 1858) This mortality advantage has been shown in numerous data sets, around the world. (Hu and Goldman 1990) For many years, these studies were hampered by the unavailability of longitudinal data. (Goldman 1993) In the interval, a striking number of intellectually rigorous and creative explanations have been developed to explain what it might be that marriage provides. Recently improved data and improved analytic techniques allow us to begin to empirically adjudicate among the many possibilities.

In the pages that follow, I will review the more prominent explanations as to why marriage might improve mortality. I will attempt to show that these explanatory frameworks often have clear implications for the pattern that a person’s hazard of death should follow after the death of a spouse. Further, the frameworks generally have implications for how those patterns should vary as a function of the degree and nature of impairment associated with the terminal period and some have implications for how this should vary by gender. In the following chapter, I will examine these patterns in a data set of sufficient size, detail, and follow-up to allow precise
estimation, and, hopefully, some distinctions to be drawn. Thereafter, I will ask: do these differences in mortality have correlates in differences in health services utilization?

Before engaging this project, however, it is worth asking why this topic warrants attention. There is, of course, the argument from pure intellectual curiosity. But this work has further implications along at least three distinct axes. The first is practical. A very large fraction of Americans marry. That means one member of those couples will eventually experience the health consequences of losing a spouse. The literature clearly suggests that those health consequences are quite substantial. (Waite 1995) If we can understand the process by which spousal loss causes people to fall ill or die, we may be able to design interventions to reduce the number of premature deaths – in other words, this work has basic public health implications. A second reason to study the process by which marriage may improve health is more theoretical. While the marriage relationship is in some respects unique, it is also clearly a relationship. We may then be able to use it as a model system in which to try to understand the ways in which other kinds of relationships—or, more generally, social structures and community—impact our health. These other structures have been directly examined by other scholars, and this work is relevant to broader social concerns about the changing nature of American society and community. Finally, we may be interested in understanding marriage itself. To this view, the health consequences of marriage are a way of gaining insight into how marriage works –
what spouses do for each other, and why. The extraordinary interest that the study of marital dynamics holds for the public at large should require little demonstration.

1.1 **Orientation**

How does marriage help? There are two important axes along which explanations for the protective effects of marriage can be described. In Table 1.1, I show the two axes and the location of major theories of the protective effect of marriage and social support on each. The vertical axis is that of mechanism of operation. The horizontal axis looks at the time frame over which marriage acts. (See Table 1.1.)

The first axis has been a focus of intense debate within the literature, some of which will be reviewed shortly. This is the question of mode of operation: does marriage offer protection via physiological, emotional, or instrumental means? Most succinctly: does marriage change your body, your mind, or your world? That is, do bodies just work better when in relationships? Or, are people happier when married, and the happiness makes them healthier? Or, finally, is it that spouses (or other people) do things for the person? The answers need not be mutually exclusive. What we are particularly interested in here is asking: which of the many ways in which marriage changes you have an impact on your mortality?

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1 This typology is very similar to that in House, James S., Karl R. Landis, and Debra Umberson. 1988. "Social Relationships and Health." *Science* 241:540-545.
The second axis is implicit in other discussions. An empirically convenient way is to consider this axis as a question of time. Does marriage act primarily in the current period, or does it have lingering effects? Succinctly: are the effects of marriage now or for forever? In my examinations here, I find it useful to label these “current period” models and “investment” models.

Each of these explanatory frameworks have potentially dozens of implications for research using different data sets, so it is worth taking a moment and introducing the data set in which I will conduct my tests. The Care after the Onset of Serious Illness (COSI) data set is a very large data set of almost 200,000 married elderly couples. One member of each couple was newly diagnosed in 1993 with one of the following serious illnesses: myocardial infarction (“heart attack”), congestive heart failure, hip fracture, stroke, colon cancer, lung cancer, urinary tract cancer, or one of 7 other serious cancers. We call the member of the cohort who was diagnosed the “proband”. Both members of the couple are followed forward in time until their death or until June 30, 1999 (a minimum of 5.5 years of follow-up); we have extremely good health status information on both members. Most of the theories discussed below are really about events at time of transition from marriage to widowhood. We observe large numbers of transitions, as both the sick and well members of couples die during our follow-up, and we can look at the mortality implications this has for the survivor. In particular, we are interested in two issues: (1) the pattern of increased risk for death after the loss of a spouse within a given
disease category (does it stay high, or does it taper off?); and, (2) how those patterns of increased risk vary across diseases.

In particular, we will use two different frameworks, illustrated as the timelines in Figure 1.1. For many of our analyses, we will be interested in the comparison between Couple A and Couple B. That is, we will be interested in the mortality of COSI probands as a function of whether their spouse has died. This lets us look at variation across diseases in how sensitive they make probands to the loss of a spouse. There are two types of “Couple B” included in our analyses: those in which neither member dies before the end of follow-up and those in which the proband dies before the spouse – in either case, from the proband’s perspective, he or she never had to survive the loss of the spouse. (Only one type is illustrated in the figure for simplicity.)

A second set of analyses is possible using this data set. For these, we will be interested in the mortality of spouses as a function of whether the sick proband has died; this is illustrated as Couple C compared to Couple D. The nomenclature gets somewhat awkward here, but we will persist in referring to the sick member of the couple who qualified for enrollment in COSI as the “proband”, and the other member of the couple as the “well spouse” or just as the “spouse”. In this second set of analyses, we are interested in the impact of the loss (or not) of a proband on his or her spouse. These analyses are not quite identical to looking at the impact of spousal loss in the general population – after all, all the probands are sick enough to enter COSI and so are substantially sicker than the population at large. So this second set of
comparisons, between Couple C and Couple D, allows us to look at the impact of the loss of a sick proband on the mortality of a spouse. As in the case of Couple B, there are two types of Couple D: those in which neither member dies before the end of follow-up, and those in which the spouse dies before the proband – in either case, the spouse never experiences bereavement.

There is also important heterogeneity among the diseases which we will exploit. For our purposes, two dimensions of that heterogeneity are important enough to require introduction in advance: (1) degree of impairment of physical functioning, and, (2) responsiveness to day-to-day care.

In the first case, consider recent data from a meta-analysis of the impact of diagnoses on functioning as evaluated using the RAND SF-36, a well-validated survey instrument that evaluates levels of functioning on a number of scales that range from 0 to 100, with 100 being optimal functioning. (Sprangers et al. 2000) They found that cancer patients had the highest physical functioning (67.5), individuals with cardiovascular diseases lower levels (59.3), and stroke and patients with musculoskeletal disease yet lower (51.1 and 49.6, respectively). Yet on the social functioning scale, all of these diseases were tightly clustered (68.2 – 76.1, with stroke lowest). These results further accord with our general clinical perception of disease. It is worth noting that the cardiovascular disease is an highly heterogeneous subgroup. It includes those who have had a myocardial infarction, and who have on the whole quite high levels of function (Ickovics, Viscoli and Horwitz 1997; Vaccarino et al. 1997), as well as those with congestive heart failure, who by
definition have at least moderate impairment. Thus it seems that in general, M.I. patients have the least physical impairment, cancer patients an intermediate level (that may be almost as low), and C.H.F., stroke and hip fracture patients have the highest levels of physical impairment. I must note in passing that other orderings of these disease, such as by pain-free days, or days with no impairment caused by the disease, are possible; on these schemes, cancer appears noticeably worse than the other conditions. (Centers for Disease Control and Prevention 2000)

The second issue is the degree to which these diseases are responsive to day-to-day care. Based primarily on clinical impression, four levels appear. Congestive heart failure (C.H.F.) is exquisitely responsive to day-today variation in compliance with medications and dietary restrictions. Patients suffering from hip fracture and stroke may be quite impaired in their mobility, and require substantial help with their activities of daily living, but negligence on these issues will not induce the rapid clinical deterioration that is the hallmark of life with C.H.F. Cancer patients may be at increased susceptibility to a variety of opportunistic infections, and their pain needs may be highly benefited by attentive nursing, but they are yet less dependent from a mortality perspective. Finally, patients who have survived a myocardial infarction may – aside from an increased number of medications oriented to secondary prevention – lead a life otherwise quite like their life before diagnosis.
1.2 Caveats

There are two further introductory comments necessary. The first is on the role of gender in these models. Some models are explicitly gendered, such as the social support literature. Others, such as Umberson’s social control theory (see below), have found gendered empirical support – but the intrinsic logic is not gendered. Finally, there are certain classes of economic models to which no reference to gender is necessarily made. In general, I present the models in an ungendered way, unless such gendering is integral to the logic of the model. However, for a variety of reasons, I will estimate separate models by gender: thus, it will be possible that one class of explanations may find empirical support in the case of men, and a different one in the case of women.

The second caveat relates to the life course implications of this work. As Williams (Williams 2001) has suggested, the implications of a being widowed may be quite different for a 40 year-old man and for a 80 year-old man – that such transitions are normative in the latter case, but not the former. The current project will look only within the elderly, for a number of reasons. Not least is that, given current life expectancies at birth, a substantial majority of all deaths, and much disease burden, occurs among the elderly. This is the time in life when most people experience the interaction between social institutions and biology in terms of decreasing health. This, then, is the time during life where health effects may be most important, and so it might be most useful to understand them here rather than in some other population.
1.3  **Current Period Models**

In the pages that follow, a large number of theories yield testable predictions. In Table 1.2, we will accumulate all of the predictions made in this chapter for the reader’s convenience. Each will be given a identifying number and a short moniker.

1.3.1  **Physiologic Benefits**

Berkman (1995) has reviewed a series of direct physiologic effects of the loss of social support on the neuroendocrine and immunologic systems. The general pattern of her findings is that there is depressed functioning in these important systems in the face of the loss of social support. Similar studies and conclusions were reviewed in the *Handbook of Bereavement.* (Stroebe, Stroebe and Hansson 1993)

More recently, Cohen *et al.* (1997) have found that prospectively measured social support is directly associated with immunologic functioning in an excellent example of this line research. They took a group of volunteers, assessed their social support and baseline health, then experimentally exposed the volunteers to a randomly selected virus while the volunteers were in quarantine. Great care was taken to quantify the degree of disease caused by the experimental exposure and insure that the results were not confounded by previous exposure to the virus or other pathogens. It was consistently found that those with less social support outside of quarantine were more likely to have more severe infections in quarantine from the particular virus to which they were exposed.
These theories have only been elaborated over a very short-term basis. Consistent with a general homeostatic view of system derangements as becoming cumulatively worse, the physiologic theories would imply an increased susceptibility to death that should be additive or multiplicative with the overall burden of disease. Thus, the effects of the loss of a spouse should be least bad for those with relatively silent disease (such as survivors of myocardial infarction), worse for those with diseases that cause ongoing functional deficits (such as those with congestive heart failure, or who survived a stroke or hip fracture), and marriage loss should be worst for those who have heavy systemic burdens of disease, such as those with malignancy. These predictions are tabulated in Table 1.2 as hypothesis A. Either a gradual tapering off of the increased hazard of death as the organism habituates to the new environment or a prolonged course (if the organism does not habituate) would seem plausible – additional work needs to be done to look at the long-term time courses.

1.3.2 Emotional Benefits

An important tradition in social support is related to the psychological study of stress. (Pearlin 1989; Thoits 1995) In the stress literature, it is often argued that support is not a generally health-producing phenomenon; rather support serves to “buffer” the negative effects of stress. Some formulations of this have been cogently critiqued and reviewed elsewhere. (Cohen and Syme 1985; Thoits 1982) In general, there seems to be some consensus that social support both improves individuals’
psychological well-being and provides a buffer against negative shocks of diverse types. The logic runs that social support in some way improves mental health (Mastekaasa 1994) (an interesting finding in its own right) and that improved mental health improves mortality. (Berkman 1995) It is argued that these effects are independent of the effects of social support on immune functioning, diet, and other instrumental intervening variables. That is, there may be real health benefits to emotional closeness, reduced loneliness, and having a confidant.

Perhaps more relevant here is the argument as to whether it is the instrumental support per se that provides the benefits of social support. An alternative hypothesis is that what matters are the emotional gains of the perceptions of the availability of support. That is, what matters is whether or not individuals have people they think would help, regardless of whether or not those people actually did help. At least one interesting study has attempted to directly compare the results of emotional and instrumental support, and finds that it is the perceived rather than the actual support that matters. (Seeman et al. 1995) (But see also (Blazer 1982).) Given the lack of emotional or perceptual measures in much of the instrumental work, this gives reason to pause. Unfortunately, it is not clear whether these results can be reliably distinguished from measurement complexities; that is, how do we know that our “objective perceptions” of the amount of social support available to the individual is

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2 This earlier study argues that the perceived support and objective support measures have largely independent actions.
more accurate than the respondents’ “perceptions” in order to compare objective and subjective measures?

These theories are quite difficult to operationalize in the sort of data to which we will turn in the next chapter. However, consider that patients suffering from Alzheimer’s Disease have a loss of the ability to recognize, remember, and in general feel close to those around them. As such, if the primary mode by which marital status improves health is by providing emotional support, then patients suffering from Alzheimer’s should be less responsive to emotional care from others than similarly sick patients without Alzheimer’s. This implies that patients with Alzheimer’s Disease—whatever their primary diagnosis—should suffer less of an increased hazard of death upon loss of a spouse than those without this dementia. This is hypothesis B1 in Table 1.2. Furthermore, it might also be argued that those with functional decrements are likely to be facing more stress on a day-to-day basis; hence, the loss of emotional buffering should be worse and more enduring for those with disease such as C.H.F., stroke, and hip fracture that impose a high functional decrement than those patients suffering from diseases such as myocardial infarction which may be associated with little functional decrement. This is hypothesis B2 in Table 1.2.
1.3.3 Instrumental Benefits

1.3.3.1 Debra Umberson: Social Control and the Imposition of Normativity

Next, let’s consider Debra Umberson’s influential work on “Social Control as a Dimension of Social Integration”. (Umberson 1987; Umberson 1992) Umberson argues that spouses exercise normative force over behavior. This would account for the cross-sectional finding that married men smoke less, drink less, and are less likely to be involved in automobile accidents, engage in crime or commit suicide. (Gove 1973) Given the oft-cited importance of such preventive health measures to later survival (Berkman and Breslow 1983; Force 1996), the argument runs, the reduction in risks early in life affords great reductions in mortality later in life. This argument possesses great theoretical and intuitive appeal as it ties health behaviors and mortality outcomes to the long traditions of studying social integration (Durkheim 1951) and deviance (Becker 1991 (1963); Sampson and Laub 1993). Steven Nock (Nock 1998) has demonstrated that marriage is associated with more socially normative behaviors in many domains, from church attendance and philanthropic donations to spending time at work rather than bars. In the particular case of health behaviors, however, Umberson (Umberson 1992) herself was unable to detect significant reductions in risk-behaviors during the transition into marriage, suggesting at the minimum a need for additional empirical work; she did show an increase in risk-behaviors upon transition out of marriage.

This line of reasoning argues that immediately upon loss of a spouse, probands begin to engage in nonnormative behavior. This implies an immediate
increase in their hazard of death as a result of the immediate increased risks they take on (e.g. from drinking and driving more). The relative hazard of death should continue to increase thereafter, as the accumulated bad habits have time to begin to take a health toll (e.g. the reduced liver function from the alcohol). (This latter component is, strictly speaking, an investment model, but is included here for expository convenience.) Moreover, for diseases that are particularly responsive to day-to-day monitoring, the rate of increase of the hazard of death should be greater. The clearest examples of this are the cases of diabetes mellitus and C.H.F., where daily diet- and medication-compliance are of the utmost importance and strongly cumulative. Thus, hypothesis C of Table 1.2 predicts that the loss of a spouse should cause a greater hazard of death in the case of C.H.F. than in the cases of M.I. or cancer and that the relative hazard of the widowed should rise with time since the loss of the spouse.

These theories draw heavily on a sociologic tradition that gives substantial weight to social norms. For those who prefer rational choice interpretations, it is worth noting that Becker and Kilburn (Becker and Kilburn 1992) provide a model with almost identical implications based on rational gambles. The similarity results if one assumes that individuals act as if norms contain information on the health implications of behavior, and norms tend to steer people towards healthier behavior.
1.3.3.2 Instrumental Social Support: Knowledge, Skills, Labor

There has been a large collection of small findings suggesting marriage and other social networks provide a diverse set of tangible forms of help. Litwak and colleagues (Litwak et al. 1989) have reviewed the particular findings and have proposed an appealing interpretation. They argue that the social networks under study represent “informal groups.” Drawing on organizational theory, they continue, suggests that informal groups provide help that needs to be flexible, readily available and is nontechnical; in contrast, the medical system will provide the formal, highly technical services that also ostensibly improve people’s health. This informal help can take a variety of forms, from information about who a good doctor is to cooking healthy meals. They explicitly theorize that this help may come after the onset of disease, not merely in leading to the prevention of certain types of disease.

In the original Litwak piece, diseases are arrayed on a continuum of amenability to informal help, and it is generally found that those diseases viewed as most amenable to informal help display the largest marital mortality benefits—operationalized as greater differences in the relative mortality rates based on death certificate data. Based on the scores they provide for different diseases, they state that the marital mortality differences should be largest and most enduring for stroke (after initial hospitalization) and hip fracture, smaller for colon cancer and lung cancer, yet smaller for the cluster of M.I., C.H.F., urinary tract cancer, and quite small for diseases such as CNS cancer and pancreatic cancer. This is hypothesis D1 of Table 1.2. However, examining these rankings, it appears that Litwak et al.’s expert
panel’s ranking were driven primarily by the amenability of the disease to prevention by instrumental, informal social support. (That is, the ranking is quite similar to that implied by an Umberson-like focus on prevention.)

An alternative test might distinguish between informal social support that provides day-to-day care, and informal social support that provides access to information. (This distinction occurs in their discussion, but is not one they highlight themselves.) If social support acts primarily through short-term day-to-day care, the loss of a spouse should have a greater and more enduring effect for those conditions with functional-decrements and nursing care requirements than for otherwise. Practically, this suggests that C.H.F., stroke, and hip fracture patients should show larger and more enduring increases in their hazard of death following the loss of a spouse than should those with M.I. This is hypothesis D2 of Table 1.2. If social support acts by providing information about how to get good services, then diseases for which there has been more technological innovation should show greater differentials after the loss of a spouse than those without. Thus, probands with myocardial infarction—for which markedly effective new treatments have been developed—should be more responsive to the loss of a spouse than diseases such as lung cancer, for which health care offers little help. (Important differentials as a function of educational status have been noted in M.I. survival. See review by Lauderdale 2001.) This is hypothesis D3 of Table 1.2. Likewise, if social networks serve to provide access to better information about health, we would expect to find the married going to better physicians and high-quality hospitals – both of which have
been shown to have mortality impacts. (Ayanian et al. 1997; McClellan, McNeil and Newhouse 1994)

The social support literature is also able to make explicit gendered predictions. The loss of a spouse should be worse for men than for women because women are “kin-keepers” and so are not nearly as cut-off from social networks with spousal loss as are men. If this mechanism—spouse as social network connection—is substantively important, it implies that the male loss of spouses suffering from Alzheimer’s should not be so bad as male loss of physically-impaired spouses since demented spouses already stopped providing kin-keeping, unlike physically-impaired spouses. This is hypothesis D4 of Table 1.2.

1.3.3.3 Spousal Social Capital

An interesting model of the benefits of marriage can be developed by analogy to the work of Daniel on the productivity of husbands. (Daniel 1993) Daniel found that not only does marriage increases men’s wages, but so does cohabitation (although to a lesser degree). Men’s wages rise over the first few years, then find a steady state. Termination of the marriage results in a sharp decline in wages. Anticipated terminations (such as divorce) are preceded by a gradual decline in men’s wages. Men with working wives receive substantially smaller benefits. Other work has shown that an increase in the probability of divorce reduces the wage premium. (Gray and Vanderhart 2000) Daniel’s interpretation of this is that the men are able to earn a rate of return on a so-called “augmentation capital” possessed by the wives.
That is, the wives have something (possibly something physical, maybe the wives’ time and energy) that they can give to their husbands that increases their husbands’ marginal productivity – that is, the wives can somehow make the husbands’ effective human capital go up. Daniels argues that wives invest in such augmentation capital instead of in their own human capital until the usual point at which they can earn more by alternative investments of their time.

It is reasonable to assume that such payoffs may occur not only in labor market but also with health. That is, wives may possess a stock of capital (of some unspecified form) that increases the health of their husbands. The loss of the wife leads to the loss of this extra health capital, and lower health for the husband. This pattern is very consistent with some empirical results on the marital mortality benefit, such as those of Lillard and Waite (Lillard and Waite 1995). Similar explanatory frameworks have been suggested in other contexts. (Waite and Joyner 2001) In Daniel’s formulation, the capital inheres in the wife, and can be distributed as she sees fit. There is little reason then to expect a beneficial effect of marriage on wife’s productivity. However, the gendering of the story is not essential, particularly as we move from the domain of capital productive of labor market outcomes to capital productive of health outcomes. That is, while men may not increase women’s labor market productivity, men may still be able to increase women’s health productivity. Whether men in fact do so is an empirical question.

What is more, it is equally possible to think of this health “augmentation capital” as inhering not in the spouse, but in the relationship between the spouses –
that is, as a form of social capital. This has certain implications that will not be testable in our data set, but help make the distinction more clear. Consider the case in which the augmentation capital inheres in the spouse. If a given woman could be followed through several relationships, and if her partner’s “health” could be observed, than for each relationship, this implies that the health advantage to the new partner would be both quick in onset and of similar magnitude.\(^3\) In contrast, if the health benefits inhere in the relationship, then the new health capital would need to be recreated with each new relationship, leading to a slower onset of the increase in health, and greater variability across relationships. Intriguingly, this second interpretation is more consistent with the Lillard and Waite findings about the slow time course of the decreasing hazard of death that women experience with the onset of relationships.

In operationalizing this explanatory framework, the most insight can be gained by looking at the hazards of death of the spouses of our probands, following the loss of the probands. That is, referring again to Figure 1.1, we are in this case interested in the comparison between Couple C and Couple D. If the augmentation capital is primarily the result of the physical actions of the spouse, then the increase in the hazard of death should be greater for those who lost a proband due to conditions with little functional decrement (\textit{e.g.} MI, cancer) than those with serious functional

\(^3\) This is a minor elaboration of an analytic strategy used to understand the relationship between couples’ wages in Waite, Linda J., and Lee A. Lillard. 2001. "The Decision to Marry and the Work and Earning Careers of Spouses." at Population Association of America meetings in Washington, D.C.
decrement (e.g. CHF, hip fracture). This is because the spouses of probands with physical decrements have already lost their partners’ physical usefulness – so the marginal loss on the actual death of a spouse should be much smaller. This is hypothesis E1 of Table 1.2. By a similar argument, if the augmentation capital a proband provides is primarily mental, then the loss of nondemented spouses should result in a greater increase in the hazard of death than the loss of spouses suffering from Alzheimer’s Dementia; this is hypothesis E2 of Table 1.2.

It is worth noting that if caregiver burden is, in fact, quite consequential for mortality, that implies a similar pattern. The absence of important differences in the mortality of a spouse as a function of the way they lose their proband would argue strongly against caregiver burden being particularly important from a mortality perspective. That is, given the heterogeneity in our diseases in their impact on all kinds of functioning, we would expect that there should be differences in the burden that a proband exerts on his or her spouse. (For example, it has been argued that caregiver burden is only a problem for the spouses of those with at least some degree of disability. (Schulz and Beach 1999)) Note, further, that looking at time from diagnosis to death provides more analytically insightful differentiation between the hypotheses of spousal social support and caregiver burden as explanations for mortality patterns. The longer a person is ill, the greater the caregiver burden associated with that person. This is schematized in Figure 1.2. This progressive burden implies longer durations of illness should be associated with smaller increases in the hazards of death when the proband finally dies. In contrast, for an
augmentation capital to be important, the spouse must provide something that it is prohibitively costly to acquire elsewhere – or there never would be differences in the first place. In that case, duration of illness is not associated with any substantial changes in the difference in the hazard of death associated with losing a proband to various causes. (That is, losing a proband after, say, 6 months of C.H.F.-induced loss of their physical augmentation capital would be just as bad as losing a proband after 3 years of loss of their physical augmentation capital. But losing a proband after only 6 months of caregiving would be worse than losing a proband after 3 years of caregiving – after three years of caregiving, the baseline mortality hazard of the caregiving married has risen to a point where it approaches the hazard of death of a noncaregiving widow, so the transition is much smaller. This is hypothesis H in the “Alternative Hypotheses” section of Table 1.2.)

1.4 Investment Models

The preceding set of models have all been focused on what it is a spouse provides by his or her direct presence. Only Umberson’s work explicitly contemplates long-term effects that might endure; she sees the improved health behaviors associated with the onset of relationships as having long-term preventive consequences. In the introduction to their early collection on the health benefits of social support, Cohen and Syme (1985) have asked why it is that the emotional benefits of social support are always presumed to flow directly through the person’s presence. Could not stress “buffering” be passed on by the inculcation of more
adaptive habits of response to stress? I have not discovered empirical work that carries forward this insight. Nor have I found work that seeks to discover long-term physiologic direct benefits of relationships, although this line of research is still relatively new. Thus, the work on investment theories of the protective effect of marriage has focused on the instrumental forms of investment.

1.4.1.1 Pure Investment

The canonical economic approach to the study of health is itself a general investment model. In Michael Grossman’s argument, health can be modeled as an unmeasurable stock of human capital that yields a flow of illness-free days. (Grossman 1972) Death is viewed as occurring once the stock falls below some level. Investments are made in the stock in a variety of forms from healthy living to frequent medical care. There has been relatively little empirical work specifying the particular forms of investment as a subject of research per se; applications of the model to the choice of environmental quality (Cropper 1981), and to career choice (Cropper 1977; Thaler and Rosen 1975) have been made. (A substantial review is

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4 In Grossman’s work, for simplicity’s sake, he views this as a known deterministic level. Extensions have been suggested that view the stock of health as influencing the hazard of death at any point. Cropper, Maureen L. 1977. "Health, Investment in Health, and Occupation Choice." *Journal of Political Economy* 85:1273-1294. While these models substantially reduce the empirical tractability of an already complicated model, it is not clear that it matters whether one views death as occurring deterministically with known (but unobservable to outsiders) stock, deterministically with imperfectly known stock, or probabilistically with known stocks—or at least, it is not clear that it matters for the uses to which the model will be put here.
(Tolley, Kenkel and Fabian 1994). Lillard and Panis (1996) and Zick and Smith (1991) provide some application to marriage, but focus on Grossman’s notion of a marriage as just one of a diverse set of inputs to health production.\textsuperscript{5} The logic is straightforward. Marriage presumably acts by lowering the costs of investment in health: either because spouses facilitate the production of health capital, directly invest themselves, or decrease the depreciation rate. Given lower costs of investment, more health capital is built, and the married are healthier. The existing literature has not yet examined within this general framework what form these investment activities might take in daily life; although the other applications have generally examined instrumental approaches, there is no reason for excluding physiologic or emotional forms of investment in the marital production of health capital.

If health capital is physically embodied in individuals \textit{and} spouses are helpful only by changing the rate of investment, then the loss of a spouse should be associated with a gradual increase in the hazard of death, as the capital stocks of the widowed depreciate faster (and are not replenished) than the stocks of the married. This relative hazard should continuously rise after the loss of a spouse. Differences across diseases in the rates of relative increase after spousal loss would presumably be attributed to differences in the degree to which the disease itself impaired the (surviving) proband’s ability to reinvest. This admittedly simplistic formulation is presented as hypothesis F1 in Table 1.2.

\textsuperscript{5} There is not so clear a focus on the enduring nature of the capital stock in their modeling.
This theory also suggests the possibility of a distinctly gendered pattern. Given the importance of income to men’s contribution to the marriage, women have substantial incentives to keep men healthy and in the labor market. If women possess an intrinsic biological survival advantage or if women’s home production efficiency is less responsive to health, men have much less incentive to invest in the health of their wives.6

A simple generalization of the Grossman logic allows the loss of a spouse to be a capital shock that decreases the capital stock, causing a sudden increase in the hazard of death at the point of loss of a spouse. This shock could come in many forms – most obviously, as the emotional trauma of losing a loved one. Thereafter, in the simplest cases, the health capital would continue to depreciate at an increased rate among the widowed as a result of the loss of the lost spouses’ inputs as in the previous case. As noted in Table 1.2 as hypothesis F2, this would imply a step increase in the hazard of death at the point of loss of the spouse, and continued increased hazards thereafter.

A more nuanced version would allow probands who so chose to reinvest in some health capital at an increased rate after the loss of their spouse—that is, to take corrective action for this stock decrement. In that case, assuming no differences

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6 This is not to assume that all women make their only contribution in the home, but women assuredly account for a disproportionate share of the home production. See South, Scott J., and Glenna Sopitze. 1994. "Housework in Marital and Nonmarital Households." *American Sociological Review* 59:327-347. Women—at least among the elderly—do seem to have a survival advantage; I have no information on the second conditional.
between diseases in their impact on health capital formation efficiency, you would expect those with the least bad diseases, and hence the longest time to reap the returns from capital investment, to invest most heavily. (This assumes, of course, that the various diseases do not vary in their impact on the relative marginal utilities of non-health consumption.) Hence low mortality diseases should have a more rapid decay of the difference in the hazard of death between the married and the unmarried than should high mortality diseases, as those with low mortality diseases invest more rapidly; this is specified as hypothesis F3 in Table 1.2

1.4.1.2 Instrumental Social Support: Income and Assets

Lillard and Waite (1995) note that “the mortality risks of currently married women and widowed women appear to be quite similar. … Recall that these effects of marital status hold constant income, which suggests that, although widowed women often suffer a fall in income with their husband’s death, if their income position had remained the same, their risk of dying would not go up. This is not the case for never married, divorced or separated women.” (p. 1149) Quite plausibly, they “speculate that widowed women with the same level of household income as divorced women are actually better off financially since they more often have access to [undivided] assets that remain from a marriage, especially a house.” (p. 1154) In studies that have the information to examine household finances, household income is generally controlled. Yet, given the substantially greater earnings of men and the likely positive benefits of income for health, this may underestimate the benefits of
marriage to women. Of course, to adequately take this into account would demand a model of what the women earn in the absence of marriage, a difficult task with a long history in labor economics. (Goldin 1990) The SUPPORT study asked families to assess the impact of losing a member to serious diseases; in crosstabulations, they found that congestive heart failure was associated with much higher rates of savings depletion than colon cancer or lung cancer. (Zhong 2001) This implies that for female spouses, the loss of a proband to C.H.F. should be associated with longer bereavement than loss of a proband to cancer, if continuity of assets is an important reason for the better mortality of women. (Our estimates will only control for income measures at the time of diagnosis, not thereafter unlike the PSID estimates.) This is hypothesis G in Table 1.2.

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1.5 Alternatives: Selection Models

A major concern that plagues all work on the possible mortality implications of marriage goes under the label of “selection.” All of the preceding models suggest that there is a real effect of marriage – in the language of experimental design, that marriage is a treatment which has a significant treatment effect. However, given that there is clearly non-random assignment to marriage, to any particular spouse, and to the loss of a spouse, differences in outcomes might be attributable to unobserved differences between the two treatment groups that are independent of the treatment, per se. There are three versions of this concern, of progressively increasing subtlety, that bear discussion; they are not mutually exclusive.

The simplest concern about selection can be phrased as follows: “Maybe only the healthy get married”. This concern has been rehashed excruciatingly often in the literature. (See, for a review, several elegant pieces by Noreen Goldman such as Goldman 1993; Goldman 1994; and Goldman, Korenman and Weinstein 1995.) Two comments are relevant here. First, among elderly Americans in 1990, less than 5% had never been married – in general, most people in most populations get married or form other long-term relationships. (U.S. Bureau of the Census 1996) Second, our dataset will look exclusively at transitions out of marriage in order to examine these possibilities.

This focus on transitions out of marriage leads to a second selection possibility. Rather than a simple healthy/not-healthy binary notion of health that leads to marriage or not, there might be assortative mating along health axes. (Lillard
and Panis 1996) If there is so-called positive assortative mating, than healthy individuals will tend to marry healthy individuals. In this case, the loss of a spouse would be associated with an increased hazard of death not because of some effect of the spouse, but because the kind of person whose spouse dies is one who him- or herself is sicker. In the case of negative assortative mating, sick individuals seek out healthy spouses in order to benefit from their services. In that case, the loss of a spouse should be associated with *improved* mortality, as individuals who lose a spouse are marked as the “healthy” half of the couple, and the healthy half of the population has improved mortality (by definition) relative to the population as a whole. There are two ways we can examine these possibilities. If we had perfect health controls, than neither selection effect could bias our results. We have very good health controls in the data set – and can compare the apparent effects of marriage with and without controls. Secondly, selection on health would have to occur at the time of courtship and marriage – typically in this cohort’s 20’s or 30’s. There must, assuredly, be increasing error in the measurement of health implied by selection as time since the matching occurs. For example, if positive assortative mating is important, the effects of the loss of a spouse should *decrease* as the age of the couple increases; moreover, this effect should be precisely gender symmetric. We can test this relatively easily by interacting age with the effects of marriage – the magnitude of the change in hazard associated with the loss of a spouse should vary as a function of the age at which that loss occurs; this is listed as hypothesis S1 in Table 1.2.
The most subtle of the selection theories argues for the critical importance of the fact that married couples live together. While this may be obvious, it means that they share a common environment and are likely to have common health habits: exercise (or not) together, smoke (or not) together, eat ice cream (or fruit) together. This also suggests that couples experience joint health shocks, negative or positive. (These could be “fast”, as when the couple is in a car accident, or “slow”, as the result of sharing an prolonged exposure to radon or bad diets.) In this case, the loss of a spouse is associated with increased risk not because of some initial correlation in their health, but because they have both damaged their health together.

There are three approaches to this problem. The first is to see if couples are more likely to get sick at the same time – that is: are members of COSI more likely than chance alone to have a spouse also in COSI. This is hypothesis is hypothesis S2 in Table 1.2. The second test is to see if there are differences in the impact of the loss of a spouse as a function of how the spouse died; relatively exogenous causes of death, such as being hit by a bus or having a cardiac arrest, should be associated with less of an increased hazard of death if the joint health shocks are driving the apparent marital effect. This is hypothesis S3 in Table 1.2. And, finally, we can look for heterogeneity across diseases. Some diseases, such as lung cancer, congestive heart failure, and head and neck cancer, are closely linked to health behaviors that have been known to be negative for a long time. Others, such as colon cancer and hip fracture, are associated with behaviors, but behaviors whose consequences have only recently become clear – after our cohort would have engaged in them. Last, there are
almost “exogenous” diseases, such as pancreatic cancer, lymphoma, and leukemia, which have very little known behavioral components. This selection model would suggest that the hazard of death associated with the loss of a spouse should be greatest for the earlier classes of diseases. This is hypothesis S3 in Table 1.2. In closing, it is worth noting that the caregiver fatigue models that were discussed earlier are similar in spirit to the joint-health-shocks models, with the minor elaboration that the illness of the sick partner is, in fact, the health shock the “well” partner experiences.

1.6 Summary

Clearly, an enormous amount of thought has been devoted to understanding how it could be that marriage alters mortality. This chapter has attempted to review some of the more prominent explanatory frameworks. Most likely, all have some degree of truth – but their relative importance needs to be determined.
Figure 1.1: Comparisons to be Used

Couple A

vs.

Couple B

Proband

Spouse

Proband

Spouse

Diagnosis

Death of Spouse

? 

Diagnosis

No Death of Spouse

? 

Couple C

vs.

Couple D

Proband

Spouse

Proband

Spouse

Diagnosis

Death of Proband

? 

Diagnosis

No Death of Proband

?
Figure 1.2: Time Courses of Spousal Social Capital vs. Caregiver Burden

Spouse's Hazard of Death

onset

Social Capital

Caregiver Burden

Time Since Loss of Function
Table 1.1: How Might Marriage Work?

<table>
<thead>
<tr>
<th></th>
<th>Current Period</th>
<th>“Now”</th>
<th>Investment</th>
<th>“Forever”</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Direct – Physiological</strong></td>
<td>• Neuroendocrine effects</td>
<td>• Immunological effects</td>
<td>• Unmeasured</td>
<td></td>
</tr>
<tr>
<td></td>
<td><strong>“Change your body”</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Emotional</strong></td>
<td>• Buffering of stress</td>
<td></td>
<td>• Learning of buffering skills</td>
<td>• Stronger mental health base</td>
</tr>
<tr>
<td><strong>“Change your mind”</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Instrumental</strong></td>
<td>• Umberson social control</td>
<td>• Instrumental social support</td>
<td>• Umberson social control</td>
<td>• Grossman health capital</td>
</tr>
<tr>
<td><strong>“Change your world”</strong></td>
<td>• Inter-Spousal social capital</td>
<td></td>
<td>• Lillard &amp; Waite income benefits</td>
<td></td>
</tr>
</tbody>
</table>
Table 1.2: Summary of Hypotheses

<table>
<thead>
<tr>
<th>Theory</th>
<th>Focus</th>
<th>Disease Ranking</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Current Period Models</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A Direct Physiology</td>
<td>Proband</td>
<td>Cancer &gt; CHF, stroke, hip &gt; MI</td>
</tr>
<tr>
<td>B1 Emotional – Stress Buffering</td>
<td>Proband</td>
<td>CHF, stroke, hip &gt; MI</td>
</tr>
<tr>
<td>Social Control (Umberson)</td>
<td>Proband</td>
<td>CHF &gt; MI, Cancer</td>
</tr>
<tr>
<td>Social Support – Informal Groups (Litwak)</td>
<td>Proband</td>
<td>Stroke, hip &gt; colon, lung &gt; MI, CHF &gt; CNS, pancreatic cancer</td>
</tr>
<tr>
<td>Social Support – Day to Day Care</td>
<td>Proband</td>
<td>CHF, hip, stroke &gt; MI</td>
</tr>
<tr>
<td>Social Support – Information Provision</td>
<td>Proband</td>
<td>MI &gt; others</td>
</tr>
<tr>
<td>D4 Kin-Keeping</td>
<td>Proband</td>
<td>Non-Alz. Spouse &gt; Alz. Spouse</td>
</tr>
<tr>
<td>E1 Social Capital – Physical</td>
<td>Spouse</td>
<td>MI, Cancer &gt; CHF, hip, stroke in Proband</td>
</tr>
<tr>
<td>E2 Social Capital – Intellectual</td>
<td>Spouse</td>
<td>Non-Alz &gt; Alz. in Proband</td>
</tr>
</tbody>
</table>

Time Course: tapering as adaptation occurs

Time Course: unrelated to time proband sick
<table>
<thead>
<tr>
<th>Theory</th>
<th>Focus</th>
<th>Disease Ranking</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Investment Models</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F1 Pure Investment</td>
<td>Proband</td>
<td>Time Course: gradual increase of relative hazard</td>
</tr>
<tr>
<td>F2 Investment + Shocks</td>
<td>Proband</td>
<td>Time Course: up, and increasing thereafter</td>
</tr>
<tr>
<td>F3 Investment + Shocks + Reinvestment</td>
<td>Proband</td>
<td>Time Course: Bereavement: Bad Ca, Lung Ca &gt; CHF, Stroke &gt; MI, Colon Ca</td>
</tr>
<tr>
<td>G Lillard &amp; Waite Assets</td>
<td>Spouse</td>
<td>For women: CHF &gt; Colon Ca, Lung Ca in probands</td>
</tr>
<tr>
<td><strong>Alternative Hypotheses</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>H Caregiving Fatigue Selection:</td>
<td>Spouse</td>
<td>within CHF, hip, stroke, the Hazard Ratio of loss decreases with time since diagnosis.</td>
</tr>
<tr>
<td>S1 Positive Assortative Mating</td>
<td>Either</td>
<td>Impact of marital status loss decreases with age</td>
</tr>
<tr>
<td>S2 Selection: Joint Health Shocks</td>
<td>Both</td>
<td>Increased likelihood of both being in COSI</td>
</tr>
<tr>
<td>S3 Selection: Joint Health Shocks</td>
<td>Either</td>
<td>Impact of loss less if spouse lost to cardiac arrest MI, CHF, lung &gt; Colon, hip &gt; CNS Ca.</td>
</tr>
<tr>
<td>S4 Selection: Joint Poor Health Behavior</td>
<td>Proband</td>
<td>Lymphoma/Leukemia</td>
</tr>
</tbody>
</table>