WIVES AND EX-WIVES: A NEW TEST FOR HOMOGAMY BIAS IN THE WIDOWHOOD EFFECT*

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Increased mortality following the death of a spouse (the "widowhood effect") may be due to (1) causation, (2) bias from spousal similarity (homogamy), or (3) bias from shared environmental exposures. This article proposes new tests for bias in the widowhood effect by examining husbands, wives, and ex-wives in a longitudinal sample of over 1 million elderly Americans. If the death of an ex-wife has no causal effect on the mortality of her husband, then an observed association between the mortality of an ex-wife and her husband may indicate bias, while the absence of an effect of an ex-wife's death on her husband's mortality would discount the possibility of homogamy bias (and also of one type of shared-exposure bias). Results from three empirical tests provide strong evidence for an effect of a current wife's death on her husband's mortality yet no statistically significant evidence for an effect of an ex-wife's death on her husband's mortality. These results strengthen the causal interpretation of the widowhood effect by suggesting that the widowhood effect is not due to homogamy bias to any substantial degree.

The "widowhood effect" describes the increased probability of the recently bereaved to die. Demographers have documented this effect for more than 150 years (Farr 1858) in numerous countries (Hu and Goldman 1990). Recent longitudinal studies put the long-term excess risk of death associated with widowhood compared with marriage at around 20%, net of controls (Elwert and Christakis 2006; Martikainen and Valkonen 1996a; Schaefer, Quesenberry, and Wi 1995).

Although the existence of the widowhood effect is descriptively settled for most populations (Stroebe, Schut, and Stroebe 2007), its explanation remains contested. Following the classification of Kraus and Lilienfeld (1959), modern research considers three competing explanations for why the death of one spouse may be associated with increased mortality for the remaining spouse: causality, homogamy, and shared exposure. First, the widowhood effect may represent a causal effect, capturing the stress of losing a loved one; the loss of psychological, social, and economic resources; and the burden of adjusting to widowhood. Second, the widowhood effect may be a noncausal artifact of spousal similarity—homogamy—by which the mortality of both members of the married couple is spuriously correlated because "like marries like." Third, the widowhood effect may be a noncausal artifact of shared exposure to environmental risk factors that affect husband and wife equally. Among these three possibilities, recent theoretical and methodological advances have fostered confidence in the causal interpretation of the widowhood effect. Nevertheless, the contribution of homogamy bias and shared-exposure bias to previous estimates of the widowhood effect remains unclear.

Here, we present new identification strategies to purge estimates of the widowhood effect of both homogamy bias and one dimension of shared-exposure bias to better

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isolate the causal effect of widowhood on mortality. Our strategies draw upon unique longitudinal samples of marital dyads and marital triads comprising married men, their current wives, and their ex-wives. In short, we argue that the death of an ex-wife should have no causal effect on the mortality of her ex-husband, such that the presence of an association between the mortality of ex-wives and ex-husbands may indicate the presence of, and the absence of such an association should indicate the absence of, certain dimensions of bias in the widowhood effect. Extracting the observed association between the death of an ex-wife and the mortality of her husband from a simultaneous estimate of the effect of the current wife's death on her husband's mortality should reduce bias in the latter and, therefore, strengthen the causal interpretation of the widowhood effect. Expanding on this logic, this article specifies three distinct yet related empirical tests capitalizing on the availability of ex-wives in two newly assembled longitudinal data sets of American husbands, wives, and ex-wives.

THREE ACCOUNTS OF THE WIDOWHOOD EFFECT

Causation

Over the past 15 years, advances in theory, data, and methods have greatly strengthened the causal interpretation of the widowhood effect (Elwert and Christakis 2006). Investigators have identified several mechanisms to support a causal interpretation. First among these is the difficult transition to widowhood itself. Following the death of a spouse, the survivor must shoulder the burden of grief, adjust to new social roles and daily routines, and develop functional substitutes for the contributions of the deceased spouse, all of which may increase a spouse's mortality. Supporting this focus on the transition to widowhood, several recent, large studies found that excess mortality remains high for several years but is especially high during the first few months following the death of a spouse (Elwert and Christakis 2006; Johnson et al. 2000; Martikainen and Valkonen 1996b). Other possible causal mechanisms engage the long-term difference between the salubrious attributes of marriage and the detrimental qualities of widowhood. To account for the long-term survival advantage of married individuals relative to widowed individuals, sociologists emphasize social integration in marriage, which provides spouses with a ready source for emotional support and direct care in case of illness (Lillard and Waite 1995; Litwak and Messeri 1989). According to Umberson (1987, 1992), spouses—particularly wives—promote healthy behaviors and discourage unhealthy behaviors. Economic approaches advance similar arguments, phrased in terms of marital economies of scale and household division of labor (Becker 1981). Upon the death of a spouse, many health benefits of marriage decrease or disappear. For example, men traditionally lose their primary caregiver (Umberson, Wortman, and Kessler 1992), and women suffer reduced economic resources (Lillard and Waite 1995). Widows and widowers report less healthy lifestyles than married individuals (Umberson 1987, 1992) and receive lower-quality medical care (Iwashyna and Christakis 2003).

The robustness of the widowhood effect across large longitudinal studies with extensive controls further contributes to confidence in a causal interpretation. Martikainen and Valkonen (1996a) studied a Finish census cohort with six years of follow-up and found widowhood effects among men and women after controlling for age, period, income, home ownership, household size, and region of residence. Elwert and Christakis (2006), who analyzed longitudinal data from more than 400,000 elderly married couples in the United States, found widowhood effects among white (but not black) men and women, even after controlling for detailed measures of baseline health for both spouses, among other things. With similar data, Elwert and Christakis (2008) reported cause-specific widowhood effects for almost all major causes of death. Schaefer et al. (1995) studied California state health records and found robust widowhood effects, even after controlling for age, education, marriage order, smoking, alcohol consumption, body mass

index (BMI), and psychological symptoms, among other factors. Critically, all of the foregoing studies agreed that the inclusion of control variables—beyond the age of both spouses—makes surprisingly little difference for the estimated size of the widowhood effect. Finally, Lillard and Panis (1996) analyzed male mortality in the Panel Study of Income Dynamics using a simultaneous equations strategy to control for one component of unobserved heterogeneity, in addition to observed confounding variables, and found the widowhood effect robust to both.

That said, most researchers acknowledge that evidence for the causal interpretation of the widowhood effect is incomplete (Elwert and Christakis 2006; Lillard and Panis 1996; Martikainen and Valkonen 1996a; Schaefer et al. 1995). Because randomized experiments are unavailable (researchers cannot randomize spouses to die), evidence necessarily came from conventional observational studies that, by design, are vulnerable to omitted variable bias (Rosenbaum 2002). And simultaneous equation models that control for certain components of omitted variable bias can do so only at the cost of strong assumptions about exclusion restrictions or functional form. The credibility of the causal interpretation of the widowhood effect thus rests on researchers' ability to explore complementary identification strategies in hopes of collectively overcoming the limiting assumptions of each approach (Manski 1995; Morgan 2004).

Homogamy Bias

Homogamy (spousal similarity) and the subsidiary phenomenon of positive assortative mating (the marriage of likes) offer an alternative, noncausal account for the widowhood effect (Kraus and Lilienfeld 1959; Martikainen and Valkonen 1996b; Schaefer et al. 1995). If husband and wife resemble each other with respect to personal traits associated with their mortality, and if these traits are insufficiently controlled for in the empirical analysis, then the mortality of both spouses may be associated observationally even if the death of one has no causal effect on the mortality of the other.

Research has documented homogamy along a wide range of social, psychological, and biological dimensions (Kalmijn 1998). Sociologists and social psychologists have found strong homogamy for age (Dean and Gurak 1978), race (Qian and Lichter 2004), religious affiliation (Sherkat 2004), educational attainment (Schwartz and Mare 2005), socioeconomic status (Jacobs and Furstenberg 1986), and class background (Kalmijn 1991). Homogamy has been documented for psychosocial afflictions, such as alcoholism and phobic disorders (Boye-Beaman, Leonard, and Senchak 1991; Yamaguchi and Kandel 1993) as well as for biological variables, such as height and weight (Schafer and Keith 1990).

Many known dimensions of homogamy are directly or indirectly related to mortality: most importantly, age. Education (Lauderdale 2001), height, and weight are well-established predictors of mortality across all age groups (Calle et al. 1999; Stevens et al. 1998), as are parental class background (Osler et al. 2005) and various psychological disorders (Cohen and Benjamin 2006).

Whereas most studies on the widowhood effect control for age, few studies control for education, place of residence, BMI, psychological conditions, or detailed measures of baseline health—and none control for all of them, let alone for all unmeasurable characteristics that might place spouses at increased risk of death. To the extent that spousal similarity is inadequately controlled for in previous empirical work, the causal interpretation of the widowhood effect remains open to challenges from homogamy bias.

Shared-Exposure Bias

Shared exposure to environmental conditions offers another noncausal account for the widowhood effect (Elwert and Christakis 2008; Kraus and Lilienfeld 1959; Martikainen and Valkonen 1996b; Schaefer et al. 1995). If a husband and wife are jointly exposed to detrimental external conditions that are insufficiently controlled in the empirical analysis,

such factors may induce an association between the mortality of the husband and wife, even though the death of one spouse does not cause an increase in mortality for the other.[1]

Spousal coresidence gives rise to several potential sources of bias from shared environmental exposure. For example, neighborhood composition and poverty levels around the place of residence correlate strongly with mortality (Geronimus et al. 2001) but are rarely observed in studies of the widowhood effect. Similarly, exposure to residential toxins—or air pollution, more generally—are known to raise the risk of death (Field 2001), yet they are not included in studies of the widowhood effect. Other sources of shared exposure may originate more specifically from shared spousal behaviors, such as diet and smoking. Although some studies of the widowhood effect include controls for residential environment (Subramanian, Elwert, and Christakis 2008), smoking behavior (Schaefer et al. 1995), or accidents (Martikainen and Valkonen 1996b), none control for all potential sources of shared exposure. To the extent that past work omits salient features of the shared marital environment, the causal interpretation of the widowhood effect remains open to challenges from exposure bias.

ANALYTIC STRATEGY: USING EX-WIVES TO TEST FOR BIAS IN THE WIDOWHOOD EFFECT

Expected Patterns of Association

It is difficult to distinguish causation from the confounding biases of homogamy and shared environmental exposure using observational data on coresident married *couples* alone because all three explanations predict the same positive association between a husband's and a wife's mortality. It may, however, be possible to distinguish causation from homogamy bias and shared-exposure bias using data on married couples and *ex-spouses* because, in this case, the pattern of predicted associations varies across explanations.

Table 1 summarizes the expected associations between the mortality of husbands (H), wives (W), and ex-wives (E) under the three theoretical scenarios. Each row gives the predictions for one pairwise association between the mortality of H, W, and E across scenarios. Plus signs indicate an expected positive association; blanks indicate no expected association. We discuss the table from the perspective of husbands' mortality because the following empirical analysis focuses on husbands' mortality as the primary outcome.

The first column shows the pattern expected if the apparent effect of the wife's death on her husband's mortality is exclusively due to causation. The wife's death would then be associated with an increase in husband's mortality, net of controls, by the causal mechanisms reviewed earlier in this article. Because the causal interpretation of the widowhood effect rests on mechanisms engendered by spousal affiliation and coresidence, one would not expect an association between an ex-wife's death and her husband's mortality.[2] Similarly, we would not expect to observe an association between the mortality of the current wife and an ex-wife.

The second column shows the associations expected because of homogamy between husbands and wives. If like marries like, then the current wife's death should be positively associated with her husband's mortality even if the death of one does not cause the death of the other. For example, to the extent that men of low education with congenital heart

1. Bias from shared exposure differs from homogamy bias. Shared exposure bias arises from joint exposure to a confounding variable that is simultaneously associated with mortality in both the husband and the wife. Homogamy bias, by contrast, arises from a positive correlation between the individual characteristics of the husband and the wife that are similarly but separately associated with mortality in the husband and wife, respectively.

2. A husband's death, however, may exert a causal effect on an ex-wife's mortality if her financial situation deteriorates upon his death (e.g., because of reduced financial transfers). To maintain the exclusion restriction that an ex-spouse's death has no causal effect on the survivor's mortality, we restrict the analysis to widowhood effects experienced by men.

Table 1. Expected Associations Under Competing Scenarios

			Shared Environmental Exposure		
	Causation	Homogamy	Past	Present	Permanent
Current Wife and Husband	+	+		+	+
Ex-wife and Husband		+	+		+
Ex-wive and Current Wife		+			+

Notes: Summary of expected associations between the mortality of the husband, current wife, and ex-wife under three scenarios, as discussed in the text. A plus sign (+) denotes a positive expected association. A blank denotes no association expected.

disease marry women who are similarly disadvantaged, we would expect their deaths to be correlated because both would likely die sooner than otherwise similar highly educated, healthy men married to similarly advantaged women. By the same reasoning, we would expect to find an association between husbands and their current wives as well as between husbands and their ex-wives (and even between current wives and ex-wives). The presence of an association between ex-wives' and husbands' mortality, net of controls, may therefore indicate homogamy bias in the apparent effect of current wives' death on husbands' mortality. Conversely, the absence of an association between the mortality of husbands and their ex-wives, net of controls, would discount the possibility of homogamy bias and, consequently, raise the credibility of a causal interpretation for the widowhood effect.

The last three columns show the patterns of associations expected in the presence of insufficiently controlled characteristics of the shared spousal environment. We differentiate the shared spousal environment into three types of exposures—past, present, and permanent—because each has different implications for bias in the widowhood effect.

"Past shared" exposures are shared between husbands and ex-wives, but not between husbands and current wives. For example, an ex-wife's smoking habits may have exposed the husband to the deleterious effects of secondhand smoke, thus inducing a positive association between a husband's and ex-wife's mortality. By definition, past shared exposures cannot induce correlations between a husband's and his current wife's mortality, nor between a current wife's and an ex-wife's mortality. Therefore, the existence of past shared environmental exposures alone does not induce bias in the estimate for the causal effect of a current wife's death on a husband's mortality, and an association between an ex-wife's and a husband's mortality does not necessarily indicate a bias in the estimated effect of a current wife's death on a husband's mortality.

"Present shared" exposures are shared between husbands and current wives, but not between husbands and ex-wives. For example, the mortality of husbands and their current wives may be positively correlated because the current wife smokes at home. Insufficiently controlled characteristics of the present shared environment would, therefore, induce bias in the widowhood effect. Note that because husbands do not share this present environment with their ex-wives, the absence of an association between an ex-wife's and her husband's mortality cannot rule out the presence of present shared exposure bias in the widowhood effect. This is a limitation for the usefulness of ex-wives to control for bias in the widowhood effect; including ex-wives in the analysis improves the identification of the widowhood effect, but it does not address all possible dimensions of unobserved heterogeneity. We correct this limitation by including observed controls for present shared environment in our empirical analysis.

Finally, "permanent shared" exposures are those features of the marital environment that are anchored in the husband such that all three individuals would have been exposed. For example, if the husband is a lifelong smoker, his smoking may induce a positive correlation between his own mortality and that of both his current and his ex-wife. The

existence of an association between ex-wife's and husband's mortality may, therefore, indicate the presence of bias from permanent shared exposure. Conversely, the absence of an association between an ex-wife's and her husband's mortality, net of observed controls, would indicate the absence of bias from permanent shared exposure in the widowhood effect.

In any real-life setting, all three scenarios (causation, homogamy bias, and shared-exposure bias) may occur simultaneously. However, to the extent that the data provide evidence for some associations over others, it becomes possible empirically to narrow the field of potential explanations for the widowhood effect. Specifically, evidence against an apparent effect of an ex-wife's death on a husband's mortality would help discount the possibility of bias both from homogamy and from permanent shared environmental exposure. Although estimates of the widowhood effect could still suffer bias from present shared environmental exposure, or from similarities that the husband shares with his current wife but not with his ex-wife, the exclusion of two noncausal explanations (homogamy and permanent shared environment) would critically strengthen the credibility of a causal interpretation of the widowhood effect.

Three Tests Using Marital Dyads and Triads

We offer three empirical tests for bias in the widowhood effect by drawing on two different samples of husbands, wives, and ex-wives. The first test is based on a sample of two kinds of *marital dyads*: pairs of husbands and their current wives (HW) and pairs of men and their ex-wives (HE), in which each man is linked to only one woman. The second and third tests are based on a sample of *marital triads*, in which each man is linked to *both* his current wife and one (not remarried) ex-wife (HWE).

To understand the specific assumptions and comparative strengths and weaknesses of each test, it is helpful to partition the unobserved health-relevant dimensions of homogamy, u, into two components, u_W and u_E. Let u_W denote the component that a husband shares with his current wife, and let u_E denote the component that a husband shares with his ex-wife. Define u_C as the intersection between u_W and u_E—that is, the unobserved health-relevant characteristics that the husband shares with both his current wife and with his ex-wife. Note that the dyads test deals only with u_W and u_E because each man is linked to only one woman. In the triads tests, however, each man is linked to two women, such that u_C provides additional traction.

Dyads test. The dyads test estimates the effect of current wives' death and the effect of ex-wives' death on husbands' mortality and compares these two effects. The difference between the two effects exactly purges the effect of the current wife's death on the husband's mortality (the estimated widowhood effect) of homogamy bias, if two assumptions hold: (D1) The death of an ex-wife has no causal effect on the mortality of her ex-husband; and (D2) u_W contributes to the association between the mortality of husbands and their current wives as u_E contributes to the association between the mortality of husbands and their ex-wives.

Assumption D1 is well justified: because the theoretical explanations for the causal effect of the current wife's death on husband's mortality rest on spousal coresidence and/or affiliation, the death of an ex-wife should not cause an increase in an ex-husband's mortality. Significantly, a violation of D1 in the sense that the death of an ex-wife may causally increase the mortality of her ex-husband would actually strengthen the dyads test because differencing the estimated effects of current wives' and ex-wives' deaths on husband mortality would then subtract more than necessary from the estimated widowhood effect. (Bias from past shared exposures in the estimated effect of an ex-wife's death on an ex-husband's mortality in HE dyads would similarly render the dyads test more conservative because past shared exposures do not contribute to the association between the current wife's and the husband's mortality.)

Regarding assumption D2, research suggests that both ongoing marriages (HW dyads) and dissolved marriages (HE dyads) are strongly homogamous with respect to age, education, occupational status, and religious affiliation (Dean and Gurak 1978; Jacobs and Furstenberg 1986; Whyte 1990), although first marriages tend to be somewhat more homogamous. The most consequential difference in homogamy between dissolved and ongoing marriages is the greater age difference between husband and wife in later marriages, since age is a critical predictor of mortality. Our empirical analysis compensates for this departure from assumption D2 by explicitly controlling for the ages of all spouses, among other factors. In sum, previous findings of strong homogamy among HW dyads and HE dyads, as well as our ability to control directly for some possible departures from assumption D2, suggest that the dyads test may eliminate much—although possibly not all—homogamy bias from the widowhood effect.

Two triads tests. Like the dyads test, both triads tests estimate the effects of current wives' and ex-wives' deaths on husband mortality. Additionally, they take into account that every man is linked to both his current and ex-wife. The triads tests, therefore, are more powerful than the dyads test because they can draw on u_C, which allows us to purge the widowhood effect of homogamy bias in two complementary ways.

The first triads test assumes that controlling for an ex-wife's vital status proxies for health-relevant spousal similarities between a husband and his current wife, such that the estimated effect of the current wife's death on husband's mortality, net of the ex-wife's vital status and other controls, by itself gives the widowhood effect purged of homogamy bias. Specifically, this test assumes the following: (F1) A husband's unobserved similarities with his ex-wife encompass his unobserved similarities with his current wife, $u_W \square u_E$ (i.e., $u_W = u_C$); and (F2) An ex-wife's vital status captures the relationship between u_C and husband's mortality, net of controls.

Research strongly supports assumption F1—that is, the claim that remarried individuals resemble their current spouses as much as they resembled their ex-spouses, at a minimum with respect to educational attainment, religious affiliation, occupation (Dean and Gurak 1978; Jacobs and Furstenberg 1986), and even age (Whyte 1990).[3] It is difficult to assess the validity of assumption F2, except to note that the ex-wife's vital status may have weak signal strength for the effect of unobserved dimensions of homogamy on mortality. In contrast to the dyads test (assumption D1), this first triads test does not require that the ex-wife's death have no causal effect on husband's mortality or that the association between an ex-wife's death and husband mortality be free of bias from past shared exposure. In this sense, the first triads test may be more robust than the dyads test.

The second triads test mimics the dyads test by computing the difference between the apparent effects of the current wife's and the ex-wife's death on husband's mortality in order to purge the widowhood effect of homogamy bias. Specifically, this test assumes the following: (S1) The death of an ex-wife has no causal effect on the mortality of her ex-husband; and (S2) u_W contributes to the association between the mortality of the husband and his current wife, as u_E contributes to the association between the mortality of the same man and his ex-wife.

The defense of S1 and S2 is similar to the defense of the analogous assumptions, D1 and D2, in the dyads case earlier in this article. However, S2 appears more credible in the triads case than D2 is in the dyads case because research more strongly supports the notion that remarried individuals resemble their current spouses as much as they resemble their former spouses (as discussed previously regarding assumption F1).

3. Assumption F1 compares the degree of homogamy across successive marriages of the same focal individual, whereas assumption D2 compares the degree of homogamy in disjoint samples of ongoing and dissolved marriages. Thus, it is not surprising that F1 receives stronger empirical support from the empirical literature. To the best of our knowledge, all previous research on marital homogamy in higher-order marriages has been conducted from the perspective of remarried women rather than remarried men.

Similar reasoning supports the use of ex-wives to test for bias from permanent shared exposure (which predicts the same pattern of associations as does homogamy). However, using ex-wives to control for bias from permanent shared exposure additionally depends on the degree to which permanent shared exposures, which (in the case of ex-wives) occurred in the past, continue to affect the mortality of ex-wives after divorce. In support of this assumption, recent work documented the long reach of childhood exposures on old-age morbidity and mortality (Hayward and Gorman 2004). Finally, note that the dyads test and the second triads test will be conservative if past shared exposure bias inflates the estimated effect of ex-wives' death on husbands' mortality. Because these tests subtract the estimated effect of an ex-wife's death from the estimated effect of the current wife's death, an upward bias in the effect of an ex-wife's death on her husband's mortality would lead to a downward bias in the difference between these two effects, thus underestimating the causal effect of the current wife's death on her husband's mortality. Because the burden of proof is on the existence of a causal effect, conservative bias in the dyads and second triads test appears unproblematic.

Obviously, controlling for observed dimensions of homogamy and shared exposure (or variables on the pathway connecting these omitted variables to husbands' mortality) reduces the need to rely on any of the foregoing assumptions. Without omitted variables, there is no bias from homogamy or shared environmental exposure. Central in this respect is our ability to control for age and health of all individuals, since they are among the best predictors of mortality. Thus, we need only be concerned about the components of bias from homogamy and shared environmental exposure that operate independently of observed controls.

Although arranging these three tests in a strict order of strength or credibility is not logically possible, we believe that the assumptions behind the second triads test are the weakest, thus rendering this test the strongest and most credible. Nonetheless, we are satisfied that the first triads test and the dyads test are also based on reasonable assumptions, and thus merit an empirical investigation. Since these three tests trade off complementary strengths, together they provide an important opportunity to gain insights into the role of homogamy bias and permanent shared-exposure bias in the widowhood effect.

DATA

Dyad and Triad Detection

We extract large longitudinal samples of husbands, wives, and ex-wives from Medicare databases of the U.S. Centers for Medicare & Medicaid Services. Medicare records are well suited for our purpose because they capture an estimated 97% of Americans aged 65 and older (Kestenbaum 2000), are longitudinal, and contain rich covariate information to control for confounding. Most importantly, Medicare records permit the detection of married couples as well as ex-spouses (Iwashyna et al. 1998).

In the first step of data development, all Medicare beneficiaries between ages 65 and 99 on January 1, 1993, were subjected to a spousal-detection algorithm adapted from Iwashyna et al. (1998). The algorithm uses information encoded in beneficiaries' unique individual-level identifiers, consisting of a Health Insurance Claim number (HIC) and a Beneficiary Identification Code (BIC). Individuals who derive their Medicare coverage through their personal entitlement history are assigned an HIC that equals their Social Security number and a BIC identifying them as "primary claimant." Individuals who derive coverage as dependents of a primary claimant receive an HIC that equals the Social Security number of the primary claimant, and a BIC specifying their relationship to the primary claimant (e.g., "current wife," "divorced wife"). These identifiers are closely monitored and therefore highly accurate because the disbursement of funds depends on them. Searching the Medicare Denominator File for individuals with identical HICs and appropriate BICs

thus enables the unambiguous identification of current and former spouses (Iwashyna et al. 1998). In this research, we restrict our attention to male primary claimants and their dependent current and former wives.

We divide the pool of all identified (ex-)spouses into three mutually exclusive groups. The first group, HW dyads, contains conventional married couples consisting of husbands (H) and their current wives (W). HW dyads were married at baseline (January 1, 1993) and are not known to have been married previously. The second group, HE dyads, contains marital dyads consisting of previously married men (H) and their ex-wives (E). HE dyads are legally divorced couples and comprise husbands who are not known to have remarried and their ex-wives who are known not to have remarried. The third group—HWE triads—contains marital triads consisting of a husband, his current wife, and one ex-wife. Husbands and wives in HWE triads are known to be married at baseline, and the ex-wives are known not to have remarried since divorce.

Previous validations against the 1990 census document that the pool of HW dyads is representative of all elderly married couples in the United States with respect to the age, race, poverty status, and region of residence of both spouses, as well as the age difference between spouses (Elwert and Christakis 2006; Iwashyna et al. 2002).[4] Nevertheless, the algorithm selects dyads and triads nonrandomly with respect to the relative earnings of spouses and marriage duration. Women identified as dependent wives, whether current or former, will generally have had lower lifetime earnings than their primary claimant husbands. Although men of this generation commonly outearned their wives, the algorithm thus selects on gender role traditionalism. Current wives must generally have been married to the primary claimant for at least two years, and ex-wives must generally have been married to the primary claimant for at least 10 years and cannot have remarried since their divorce. These marriage duration requirements and the remarriage elimination rule limit our ability to detect ex-wives in the data. At the same time, the marriage duration requirements improve the usefulness of ex-wives as controls for shared-exposure bias because they guarantee that husbands and ex-wives long shared the same marital environment.

After applying all relevant sample restrictions (detailed later), we retain a 20% simple random sample of all identified HW dyads ($N = 444{,}685$, representing about 8% of all married couples in the corresponding population), all identified HE dyads ($N = 54{,}465$), and all identified HWE triads ($N = 2{,}138$). Overall, this analysis thus draws on information from 1,004,714 elderly Americans.

Variables and Sample Restrictions

We derive the key variables of this study from three different Medicare databases. Records from these databases were linked on the basis of unique individual-level identifiers. The record linkage rate was 100%.

The Medicare Vital Status File, drawn in late 2002, provides exact dates of death for all deceased sample members. From this information, we derive the outcome (husband's time to death since January 1, 1993) and the two key independent variables of interest (death of current wife, and death of ex-wife). Husbands who were still alive by the end of follow-up were censored on January 1, 2002.

We extract detailed health measures to control for differences in baseline morbidity from the Medicare Provider Analysis and Review (MedPAR) File, which contains diagnostic information for all hospitalizations among Medicare beneficiaries during the latter half of 1992. Baseline health is an important predictor of widowhood and death (Christakis and Allison 2006), and our detailed, physician-ascertained controls for confounding by health status exceed the (usually self-reported) health information in previous studies.

4. These are all variables shared by Medicare and the 5% census PUMS. We are not aware of means to gauge the representativeness of the detected HE dyads and HWE triads.

We summarize the chronic disease burden at baseline by computing Charlson comorbidity scores (Charlson et al. 1987) from hospitalization records separately for all husbands, wives, and ex-wives; we trichotomize this measure into low, moderate, and severe (Charlson scores of 0, 1, and 2 or higher, respectively). We further include counts of the number of days (coded in weeks) that each individual spent in the hospital in the latter half of 1992. Because most beneficiaries enter the Medicare program at age 65, we restrict the analysis to marital dyads and marital triads in which all individuals were aged 65.5 or older at baseline in order to guarantee six months of health look-back for the entire sample.

We derive race information for all individuals from the race and ethnicity codes in the Medicare Vital Status File. Because large-sample research found no detectable widowhood effect among blacks (Elwert and Christakis 2006), we restrict the analysis to marital dyads and marital triads in which all individuals are white. Note that the age and race restrictions increase the homogamy in this sample, thus providing for more exacting tests.

The Medicare Denominator File provides additional control variables from Social Security records. The ages of all individuals are entered as continuous main effects, age squared, and age relation between husbands and (ex-)wives. We derive a poverty indicator for each individual based on dual eligibility for Medicare and Medicaid services in 1993 (Clark and Hulbert 1998). Mailing addresses give each individual's place of residence to the ZIP code level in 1993. Because the causal mechanisms behind both the widowhood effect and bias from shared exposure rest on marital coresidence, we exclude HW dyads in which husbands and their current wives do not reside in the same ZIP code. To minimize contamination from direct social contact between husbands and their ex-wives, we also exclude all HE dyads in which husbands and their ex-wives reside in the same ZIP code. We limit the analysis to husbands residing in the 50 U.S. states and the District of Columbia, and include indicators for census division of residence as control variables in the analysis to capture regional variation in mortality (Subramanian et al. 2008).

STATISTICAL METHODS

We use continuous-time, semiparametric (Cox) hazard models to analyze the effect of a wife's and an ex-wife's death on a husband's mortality. We present two main analyses (giving three tests): one for the combined sample of marital dyads (HE and HW), and another for HWE marital triads. These two analyses differ in setup and interpretation, and they are independent because their respective samples are disjunct.

The first model (Eq. (1)) combines HW dyads and HE dyads into one analysis to test whether the effect of current wife's death on husbands' mortality is different from the effect of an ex-wife's death, net of controls. In this analysis, each husband either has a current wife or an (unmarried) ex-wife at baseline, but not both.

$$h(t) = h_0(t)\ \exp[S(t)\beta_1 + F^*S(t)\beta_2 + F\beta_3 + \mathbf{X}_H\beta_4 + \mathbf{X}_S\beta_5]. \quad (1)$$

The model partitions husband's hazard of death at time t, $h(t)$, into the product of a baseline hazard that varies freely with time, $h_0(t)$, and a function of the vector of explanatory variables, such that changes in the explanatory variables induce proportional shifts in the baseline hazard. Husband's hazard of death is a function of a time-varying indicator for spouse's death, $S(t)$. The time-invariant indicator variable F is coded to 1 if the spouse is an ex-wife (the reference category is current wife). Thus, $\beta_1 \neq 0$ tests for the presence of the traditional widowhood effect attributable to the death of a current wife. The coefficient on the interaction between F and $S(t)$, β_2, estimates the difference in effects between a current wife's death and an ex-wife's death on husband's hazard of death; and $\beta_2 < 0$ tests whether the effect of current wife's death is greater than the effect of ex-wife's death. This test is critical because it indicates whether the effect of the current wife's death contains components beyond homogamy bias and permanent shared-exposure bias, under

maintained assumptions. The sum of $\beta_1 + \beta_2$ gives the estimated main effect of the death of an ex-wife on the husband's hazard of death. The coefficient on F, β_3, estimates the difference in the hazard of death between currently married men and divorced men whose ex-wife is still alive. We include two vectors of control variables: one for the characteristics of the husband, $\mathbf{X}_H$, and one for the characteristics of the spouse, whether current or former, $\mathbf{X}_S$.[5]

Our second model analyzes husband's mortality in marital triads and provides the first and second triads tests for homogamy bias. Moving from the analysis of marital dyads to the analysis of marital triads has consequences for the parameterization and interpretation of the model, shown in Eq. (2).

$$h(t) = h_0(t)\ \exp[W(t)\beta_1 + E(t)\beta_2 + \mathbf{X}_H\beta_3 + \mathbf{X}_W\beta_4 + \mathbf{X}_E\beta_5]. \quad (2)$$

The model includes a time-varying indicator for current wife's death, $W(t)$; a separate time-varying indicator for ex-wife's death, $E(t)$; and the same set of time-invariant baseline control variables entered separately for husbands, wives, and ex-wives: $\mathbf{X}_H$, $\mathbf{X}_W$, and $\mathbf{X}_E$. The coefficients β_1 and β_2 estimate the main effects of current wife's death and ex-wife's death on husband's mortality. To the extent that an ex-wife's vital status proxies for unobserved dimensions of homogamy between a husband and his wife and ex-wife (assumptions F1 and F2), controlling for the ex-wife's death purges the conventional widowhood effect estimate, β_1, of homogamy bias (and permanent shared-exposure bias). The test of $\beta_1 > 0$, while controlling for $E(t)$, thus provides the first triads test for the presence of a widowhood effect net of homogamy bias and permanent and shared exposures.

Alternatively, we can turn to the second triads test and test for a positive difference between the effects of current and ex-wife's death on husband's mortality, $\beta_1 - \beta_2 > 0$, which would indicate the existence of a widowhood effect net of homogamy bias under assumptions S1 and S2.

In all models, outcome and time-varying covariates are measured to the day. Time starts at cohort inception on January 1, 1993. Surviving husbands are censored on January 1, 2002, providing up to nine years of mortality follow-up. Because all data come from administrative registries, there is negligible loss to follow-up. Following established conventions, all statistical tests reported are two-sided.

RESULTS

Dyads Test

Descriptive statistics. Table 2 shows summary statistics for all variables in the multivariate analysis of the dyads sample. Overall, about one-half of all 499,150 husbands died during follow-up—49% of the 444,685 HW husbands and 39% of the HE 54,465 husbands died. Among women, 20% of HW wives and 28% of HE ex-wives predeceased their husbands and ex-husbands, respectively. All combinations of dyad type and husband's and (ex-)wife's death appear sufficiently populated to enable efficient multivariate estimation. The mean age at cohort inception is 75.4 years for men and 73.0 years for women, and 21% of current wives and 28% of ex-wives are older than their (ex-)husbands. Most individuals had low Charlson comorbidity scores at baseline, but husbands were somewhat sicker than their (ex-)wives and spent more time in the hospital prior to baseline. HW dyads differ from HE dyads chiefly in that ex-wives are much more likely to be poor than current wives (33% versus 3%), and are older than current wives relative to their (ex-)husbands by 0.6

5. Including interactions between X_S and F in the model to account for differences in the effects of a wife's and an ex-wife's characteristics on a husband's death did not change the central coefficients of interest, β_1 and β_2 (not shown).

Table 2. Descriptive Statistics for the Dyads Sample (husband-wife and husband–ex-wife)

Variable	Means/Fractions		
	All	Husband-Current Wife	Husband–Ex-wife
Death			
Husband dies	0.49	0.49	0.39
Spouse dies first	0.21	0.20	0.28
Age, 1993			
Husband	75.40	75.65	73.38
Spouse	73.04	73.22	71.57
Age Squared, 1993			
Husband	5,720	5,758	5,411
Spouse	5,366	5,393	5,145
Woman Older Than Husband	0.21	0.21	0.28
Poverty			
Husband	0.03	0.03	0.06
Spouse	0.06	0.03	0.33
Charlson Score (second half of 1992)			
Husband			
Low	0.88	0.88	0.93
Moderate	0.05	0.05	0.03
Severe	0.07	0.07	0.04
Spouse			
Low	0.93	0.92	0.94
Moderate	0.03	0.04	0.03
Severe	0.04	0.04	0.03
Weeks Hospitalized (second half of 1992)			
Husband	0.34	0.36	0.20
Spouse	0.27	0.27	0.21
Census Division			
New England	0.04	0.04	0.04
Mid-Atlantic	0.13	0.14	0.09
East-North Central	0.18	0.19	0.14
West-North Central	0.10	0.10	0.06
South Atlantic	0.17	0.17	0.20
East-South Central	0.07	0.06	0.07
West-South Central	0.11	0.11	0.12
Mountain	0.06	0.06	0.08
Pacific	0.14	0.13	0.20
N	499,150	444,685	54,465

Notes: Each husband in the sample is linked to only one woman. All individuals are white, alive, and aged 65.5+ at baseline on January 1, 1993. Follow-up ends January 1, 2002.

Source: Authors' calculations.

Table 3. Main Effects of Current Wife's and Ex-wife's Deaths on Husband's Mortality in a Multivariate Cox Model: Dyads Sample

Effect on Husband's Hazard of Death	Estimate		
	Hazard Ratio	*SE*	*p* Value
Ex-wife Dies	0.978	0.015	.147
Current Wife Dies	1.207**	0.007	.000
Ratio of Ex-wife to Wife	0.810**	0.013	.000

Notes: Key results (estimated main effects) for dyads sample from Cox model including all control variables. N = 499,150 marital dyads (husband-wife and husband–ex-wife). See Appendix Table A1 for complete output.

Source: Authors' calculations.

**$p < .01$ (two-sided)

years. Although the baseline health of current wives and ex-wives appears comparable, ex-husbands suffered a slightly lower chronic health burden and spent less time in the hospital than currently married husbands.[6]

Regression results. Table 3 shows the key results of the covariate-adjusted Cox model for the HE and HW dyads (full results shown in Appendix Table A1). The death of a current wife is found to increase husband's hazard of death by 21% (hazard ratio (HR) = 1.207; CI_{95}[1.19;1.22]; p value <.001). This estimate of the conventional widowhood effect, net of controls, is substantively large, highly statistically significant, and consistent with previously published estimates. By contrast, the death of an ex-wife appears to have no effect on the hazard of death of her former husband, as the point estimate is substantively close to null and not statistically significant (HR = 0.978; CI_{95}[0.95;1.01]; p value =.147). The difference between the effects of a current wife's and an ex-wife's death on a husband's hazard of death (measured as the ratio of these two effects) is substantively large and highly statistically significant (HR = 0.978 / 1.207 = 0.810; CI_{95}[0.79;0.84]; p value < .001). The size, direction, and statistical significance of the difference between the effects of a current wife's death on her husband's hazard of death compared with an ex-wife's death on her husband's hazard of death in this analysis strongly supports the hypothesis that the widowhood effect is not entirely due to homogamy bias or to bias from permanent shared exposure. Indeed, the absence of evidence of any association between the death of an ex-wife and her ex-husband's hazard of death, net of controls, further supports the stronger claim that the estimated widowhood effect may be entirely free of bias from unobserved dimensions of homogamy and permanent shared exposure. This conclusion is further supported by our ancillary finding that controlling for spouses' observed characteristics (beyond age) made little difference for size and statistical significance of the estimated effects of a current wife's and an ex-wife's death on husbands' mortality (not shown).

Triads Tests

Descriptive statistics. Table 4 shows descriptive statistics for the sample of 2,138 marital triads in which each man is linked to both his current wife and his ex-wife. Overall, 57% of husbands die during follow up; 20% of husbands were predeceased by their current wife; 33% were predeceased by their ex-wife; and 8% were predeceased by both women. The mean age at cohort inception was 75.5 years for husbands. Ex-wives and current wives,

6. We reexecuted the analysis on a subset of 85,194 dyads that were matched (using propensity scores) to eliminate observable differences between HW and HE dyads, yielding quantitatively similar and qualitatively identical results (details available upon request).

Table 4. Descriptive Statistics for the Triads Sample (husband-wife–ex-wife)

Variable	Mean/ Fraction
Death	
Husband dies	0.57
Wife dies before husband	0.20
Ex-wife dies before husband	0.33
Both die before husband	0.08
Age, 1993	
Husband	75.49
Current wife	71.39
Ex-wife	73.65
Age Relation	
Wife is older than husband	0.21
Wife is older than ex-wife	0.34
Ex-wife is older than husband	0.27
Poverty 1993	
Husband	0.04
Current wife	0.05
Ex-wife	0.36
Charlson Score (second half of 1992)	
Husband	
Low	0.93
Moderate	0.03
Severe	0.04
Current wife	
Low	0.95
Moderate	0.02
Severe	0.03
Ex-wife	
Low	0.91
Moderate	0.04
Severe	0.05

(continued)

on average, were 1.8 and 4.1 years younger than their (ex-)husbands, respectively. Poverty levels and baseline health of husbands and (ex-)wives in the triad sample appear roughly comparable to those in the dyads sample.[7]

Regression results. Table 5 shows the results of the covariate-adjusted Cox models for the sample of marital triads. The death of a current wife, adjusted only for the ages of the

7. The geographic distribution of triads, however, differs from that of HE and HW dyads, particularly with respect to a surfeit of triads in the West (33% of triads vs. 20% of dyads reside in the Mountain and Pacific divisions of the census).

(Table 4, continued)

Variable	Mean/ Fraction
Weeks Hospitalized (second half of 1992)	
Husband	0.18
Current wife	0.12
Ex-wife	0.27
Region of Residence (husband-wife)	
New England	0.05
Mid-Atlantic	0.07
East-North Central	0.13
West-North Central	0.05
South Atlantic	0.20
East-South Central	0.06
West-South Central	0.11
Mountain	0.09
Pacific	0.24
N	2,138

Notes: Triads consisting of husbands linked to both the current wife and the ex-wife. All are white, alive, and aged 65.5+ at baseline on January 1, 1993. Follow-up ends January 1, 2002.

Source: Authors' calculations.

husband and wife, increases the hazard of death among remarried men by 24% (column 1).[8] Additionally controlling for poverty, health, and place of residence reduces the estimated effect to 21% (column 2). The death of a current wife, net of observed controls and net of an ex-wife's vital status (column 3), increases the hazard of death among remarried men by 20% (HR = 1.197; CI_{95}[1.01;1.42]; *p* value =.039). This widowhood effect is substantively large, statistically significant, and consistent both with previously published results and our own results for HW dyads, reported earlier in this article. Again, we find that the inclusion of additional controls beyond age has little effect on the estimated widowhood effect. By being substantially robust to the introduction of controls for important *observed* dimensions of spousal similarity (beyond age), as well as to the inclusion of an ex-wife's vital status as a control of *unobserved* dimensions of spousal similarity, these results suggest at best a small role for homogamy bias in the estimated widowhood effect. The widowhood effect thus passes the first triads test.

Under assumptions S1 and S2, we can further use the coefficients of the same model (column 3) to execute the second triads test. Rather than using ex-wife's vital status as a control variable to *absorb* confounding by unobserved dimensions of homogamy bias, as we did in the first triads test, we now view its coefficient as *measuring* the degree of noncausal bias in the widowhood effect. Subtracting the coefficient on an ex-wife's death from the coefficient on the current wife's death then gives an estimate for the widowhood effect purged of bias. Column 3 shows that the death of an unremarried ex-wife in the final model is itself associated with a 10% increase in husband's hazard of death. This may suggest the possibility of nontrivial bias in the widowhood effect, although it is

8. Controlling for age substantially changes the estimated widowhood effect. However, since the ages of all involved are highly collinear because of homogamy, no age variable is statistically significant by itself.

Table 5. Cox Model for Current Wife's and Ex-wife's Death on Husband's Mortality: Triads Sample

Variable	Model Controls for: Wife's Death and Age	+ Husband and Wife Controls	+ Ex-wife's Death and Controls
Bereavement			
Current wife dies	1.24* (1.04–1.47)	1.21* (1.02–1.44)	1.20* (1.01–1.42)
Ex-wife dies			1.10 (0.95–1.27)
Age			
Husband	0.98 (0.79–1.21)	0.96 (0.78–1.19)	0.96 (0.75–1.23)
Current wife	1.16 (0.90–1.50)	1.19 (0.91–1.55)	1.26 (0.96–1.64)
Ex-wife			0.96 (0.73–1.25)
Age Squared			
Husband	1.00 (1.00–1.00)	1.00 (1.00–1.00)	1.00 (1.00–1.00)
Current wife	1.00 (1.00–1.00)	1.00 (1.00–1.00)	1.00 (1.00–1.00)
Ex-wife			1.00 (1.00–1.00)
Age Relation			
Wife is older than husband	0.87 (0.71–1.08)	0.90 (0.73–1.11)	0.94 (0.75–1.18)
Wife is older than ex-wife			0.78* (0.64–0.95)
Poor			
Husband		1.37 (0.85–2.22)	1.35 (0.83–2.18)
Current wife		0.96 (0.63–1.47)	0.92 (0.60–1.41)
Ex-wife			1.12 (0.99–1.27)

(continued)

similarly compatible with an inflation only of the estimated effect of an ex-wife's death on husband mortality attributable to past shared exposures.[9] That being said, the estimate is quite imprecise and fails conventional standards of statistical significance (HR = 1.096; CI_{95}[0.95;1.27]; p value =.225). The difference between the effects of a current and an ex-wife's death is substantively large and in the expected direction, but it fails to reach

9. Recall that past shared exposure would inflate the estimated effect of an ex-wife's death on her husband's mortality—but, by definition, it does not bias the widowhood effect of a current wife's death on her husband's mortality. The presence of past-shared exposure would, thus, render the second triads test conservative.

(Table 5, continued)

Variable	Model Controls for: Wife's Death and Age	+ Husband and Wife Controls	+ Ex-wife's Death and Controls
Charlson Score (second half of 1992)			
Husband			
Moderate		1.52* (1.09–2.12)	1.48* (1.06–2.06)
Severe		2.36** (1.73–3.23)	2.32** (1.70–3.17)
Current wife			
Moderate		1.15 (0.78 –1.71)	1.16 (0.78–1.71)
Severe		0.88 (0.56–1.36)	0.85 (0.55–1.33)
Ex-wife			
Moderate			1.2 (0.90–1.60)
Severe			0.93 (0.66–1.31)
Weeks Hospitalized (second half of 1992)			
Husband		1.11* (1.02–1.20)	1.11** (1.03–1.21)
Current wife		1.03 (0.92–1.14)	1.03 (0.92–1.15)
Ex-wife			0.98 (0.94–1.04)
Region of Residence (husband-wife)			
Mid-Atlantic		1.46* (1.07–2.00)	1.44* (1.05–1.97)
East-North Central		1.24 (0.91–1.68)	1.21 (0.89–1.65)
West-North Central		1.12 (0.78 –1.60)	1.09 (0.76 –1.56)
South Atlantic		1.15 (0.86–1.54)	1.14 (0.85–1.53)
East-South Central		1.23 (0.86–1.77)	1.15 (0.79–1.66)
West-South Central		1.15 (0.84–1.58)	1.13 (0.82–1.55)
Mountain		1.00 (0.72–1.39)	0.98 (0.70–1.37)
Pacific		1.06 (0.79–1.41)	1.05 (0.79–1.40)
N	2,138	2,138	2,138

Notes: Figures are hazard ratios. Numbers in parentheses are 95% confidence intervals.

Source: Authors' calculations.

*$p < .05$; **$p < .01$ (two-sided)

statistical significance ($HR_{Difference} = 1.092$; $CI_{95}[0.87;1.37]$; p value =.450). Thus, although point estimates suggest that the death of a current wife has a meaningfully larger effect on husband's hazard of death than does the death of an ex-wife, and although there is no statistically significant evidence for an effect of ex-wife's death on husband's mortality, a wide confidence interval indicates that there is not enough information in the data to distinguish between the two effects at conventional levels of statistical significance.

DISCUSSION

Researchers long have argued that the widowhood effect may be due to causality, homogamy bias, shared-exposure bias, or some mixture of all three. Conventional observational studies must restrict themselves to addressing homogamy bias and shared-exposure bias by controlling for observed dimensions of spousal similarity. This article, by contrast, advances a new strategy to test also for unobserved dimensions of bias in the widowhood effect using unique data on elderly white men and their wives and ex-wives.

Specifically, we proposed three tests: one based on groups of marital dyads, and two based on marital triads. Empirical results broadly are supportive of the absence of homogamy bias and of permanent shared-exposure bias in the widowhood effect. The analysis of marital dyads finds a strong and statistically significant widowhood effect, but no evidence that the death of an ex-wife affects husband's mortality. Furthermore, the difference between the effects of a current and an ex-wife's death is substantively large and highly statistically significant. The data thus pass the dyads test.

The analyses of marital triads similarly find strong evidence for the widowhood effect, even net of controlling for an ex-wife's vital status as a proxy for certain unobserved components of bias. The data thus pass the first triads test. The analysis of marital triads, like the analysis of marital dyads, also shows no statistically significant evidence for an effect of an ex-wife's death on husband mortality. However, although the estimated effect of a current wife's death is substantially larger than the estimated effect of an ex-wife's death on husband mortality, there is not enough information in the triads sample to conclude that the difference between the effects of a current wife's and an ex-wife's death is itself statistically significant. The second triads test, therefore, points in the right direction but remains statistically inconclusive.

Together, these analyses provide new empirical evidence against the presence of bias in the widowhood effect. Or, more precisely, our results support the claim that the widowhood effect is not entirely due to homogamy bias or to bias from permanent shared environment, although it may still be biased by dimensions of homogamy that are idiosyncratic to the marriage between husbands and their current wives or their present shared (as opposed to permanent shared) environment—beyond those observed dimensions explicitly controlled for in the analysis. Our work thus strengthens the causal interpretation of the widowhood effect.

Testing for omitted variable bias is impossible in the absence of identifying assumptions. We know of two other studies that attempted such tests for the widowhood effect, both relying on different assumptions. Lillard and Panis (1996) made assumptions about exclusion restrictions and functional form. Espinosa and Evans (2008) made assumptions about the randomness of spouse's causes of death to exploit exogenous variation in widowhood. Our tests assume that relevant unmeasured dimensions of homogamy are comparable between current and previous marriages. Previous research, although limited, supports this assumption for the dyads test and even more so for the triads tests. Homogamy in dissolved marriages (HE dyads) is somewhat weaker—although still strong—than homogamy in ongoing marriages (HW dyads), and the degree of homogamy of first and later marriages of remarried spouses (HWE triads) is substantially comparable along several dimensions of spousal similarity (Dean and Gurak 1978; Jacobs and Furstenberg 1986; Whyte 1990). Although our work and that of Lillard and Panis (1996) and Espinosa

and Evans (2008) use different data and rely on different identifying assumptions, they all find evidence for an effect of a wife's death on her husband's mortality net of certain components of unobserved heterogeneity. The complementariness of the respective identifying assumptions, together with the similarity in results, further strengthens the causal interpretation of the widowhood effect.

We note several limitations of our study. First, the study is restricted to studying the widowhood effect among elderly white men. Second, although the sample of HW dyads is representative of the population of elderly married couples in the United States with respect to age, poverty status, and region of residence (Elwert and Christakis 2006), it is not representative with respect to the relative earnings of husband and wife. Suitable national comparison data sets are not available to assess the representativeness of divorced couples (HE dyads) and HWE triads in this study. Certainly, the comparatively small number of identified ex-spouses suggests that HE dyads and HWE triads are a more select subgroup of the population than are the HW dyads. However, we have no reason to suspect that the sampled HE dyads and HWE triads should be any more or less homogamous than the corresponding populations beyond the characteristics already mentioned. Departure from representativeness would limit the generalizability of the present results if there is strong heterogeneity in the widowhood effect among different subsets of elderly white men. In this respect, we note that the estimated widowhood effect among HWE triads closely agrees with the estimated widowhood effect among HW dyads, which in turn agrees with published results from other U.S. data (Schaefer et al. 1995). To the extent that our data are nonrandom samples, this agreement would suggest either that there is limited effect heterogeneity in the widowhood effect among elderly white men or that our data are not systematically selective with respect to the widowhood effect, both of which would support the generalizability of our results.

This article exploits the existence of present or past ties between individuals to understand the social transmission of mortality in marriage. More broadly speaking, our work thus explores an interesting connection between network dynamics and social contagion. The appearance and disappearance of social ties in a large-scale network (and in small-scale networks, such as the dyads and triads examined here) is itself an important focus of inquiry; and yet the utility of changing network configurations for estimating causal effects in longitudinal data has hitherto remained underappreciated.

We suspect that our strategy of using the patterning of social ties to advance inference about social contagion may prove useful in realms other than mortality in married couples. Most immediately, the strategy may help to improve our understanding of other dimensions of suspected spousal influence, such as the correlation between spouses' chronic disease (Christakis and Allison 2006). Similar strategies may even help empirically illuminate the role of dyadic and triadic peer influence on teenage behavior (Harding 2005; Sharkey 2006), educational aspirations (Morgan 2005), or the criminal propensities of friends (Laub and Sampson 2003). Finally, it may be possible to generalize the present strategy, given appropriate data, to improve the identification of causal effects in the nascent study of the far-flung transmission of health behaviors and health events along dyadic or hyperdyadic network paths involving kin, friends, and neighbors (Christakis and Fowler 2007). One person's illness, disability, health behaviors, or death may affect similar outcomes in others—and generally only those others—to whom they are connected.

Appendix Table A1. Cox Model for Current Wife's and Ex-wife's Death on Husband's Mortality: Dyads Sample (husband-wife and husband–ex-wife)

Variable	Hazard Ratio
Spouse Dies	1.21** (1.19–1.22)
Spouse Dies and Is Ex-Wife	0.81** (0.79–0.84)
Spouse Is Ex-Wife	1.24** (1.22 –1.26)
Age	
Husband	1.12** (1.10–1.14)
Spouse	1.02** (1.01–1.04)
Age Squared	
Husband	1.00* (1.00 –1.00)
Spouse	1.00** (1.00–1.00)
Spouse Is Older Than Husband	1.06** (1.05–1.07)
Poverty	
Husband	1.48** (1.44 –1.51)
Spouse	1.10** (1.08–1.13)
Charlson Score (second half of 1992)	
Husband	
Moderate	1.55** (1.52–1.57)
Severe	2.35** (2.32–2.38)
Spouse	
Moderate	1.03* (1.01–1.05)
Severe	1.020 (1.00 –1.04)
Weeks Hospitalized (second half of 1992)	
Husband	1.05** (1.05–1.05)
Spouse	1.00 (0.99–1.00)
Region of Residence (husband-wife)	
East-North Central	1.11** (1.09–1.13)
East-South Central	1.18** (1.15–1.21)

(continued)

(Appendix Table A1, continued)

Variable	Hazard Ratio
Region of Residence (husband-wife) (cont.)	
Mid-Atlantic	1.07** (1.05–1.10)
Mountain	1.03* (1.01–1.06)
Pacific	1.00 (0.98–1.02)
South Atlantic	1.08** (1.06–1.10)
West-North Central	1.04** (1.01–1.06)
West-South Central	1.13** (1.11–1.16)
N	499,150

Note: Numbers in parentheses are 95% confidence intervals. The table shows complete model output for key results for dyads shown in Table 3.

Source: Authors' calculations.

*$p < .05$; **$p < .01$ (two-sided)

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