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Life and Death in the Family: Early Parental Death, Parental Remarriage, and Offspring Suicide Risk in Adulthood

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Abstract

Early-life parental death (PD) may increase suicide and other mortality risk in adulthood. The potential implications of subsequent remarriage of the widowed parent (RWP) for suicide have not been well examined. Data came from the Utah Population Database for birth cohorts between 1886 and 1960, yielding a sample of N=663,729 individuals, including 4,533 suicides. Cox models showed PD was associated with increased adult suicide risk before age 50, and with increased risk of cardiovascular disease deaths (CVD) for adults of all ages. For females, RWP attenuated the suicide relationship before age 50 (though not statistically significant), but significantly exacerbated it after age 50. RWP had no significant impact for males. Further, for females, PD's positive association with suicide was stronger than with CVD before age 50. These findings reinforce the importance of biological and social mechanisms in linking early-life stressors to adult mental and physical health.

Keywords

suicide; cardiovascular disease; parental death; remarriage; widowhood; competing risks; sensitive stages; life-course

Suicide was the 10th leading age-adjusted cause of death in the United States for 2011 (Hoyert & Xu, 2012). While proximal stressors are known to affect suicide risk (Denney, Rogers, Krueger, & Wadsworth, 2009), early-life stressor have also been implicated (Agerbo, Nordentoft, & Mortensen, 2002). A potential early-life stressor implicated in suicide risk (and overall mortality) is early-life parental death (PD) (Niederkrötenhaler, Floderus, Alexanderson, Rasmussen, & Mittendorfer-Rutz, 2012; Smith, Mineau, Garibotti, & Kerber, 2009). Given that approximately four percent of children in the United States experience PD before age 18 (Social Security Administration 2000), PD may represent a

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significant suicide risk for the population, and research into possible mechanisms might direct attention to effective interventions.

A life-course framework draws our attention to “linked lives” (George, 2003), where losing the parent deprives the surviving child of a prominent social tie. Remarriage of the widowed parent (RWP) might replace that lost tie thereby attenuating the risk triggered by PD, or further compound the risk through increased conflict. Yet, this potential dynamic has not been well researched. Since such associations across the life-course likely involve chronic stress mechanisms of allostatic load (McEwen & Stellar, 1993), comparisons of long-term suicide risk to a competing cause of death might also help clarify the operations of these mechanisms (Thoits, 1995).

We utilized the Utah Population Database (UPDB) to test the importance of RWP in the PD-suicide relationship, and to compare associations with cardiovascular disease death (CVD), the leading cause of death. Such research should provide health professionals with information from which to base interventions. This study deals with two generations—parent and child. To avoid ambiguity, we refer to the adult child (who might have experienced early parental death, and who may die by suicide) as the “subject”. All other relationships are addressed in relation to the subject (e.g., subject's mother, subject's stepfather).

Background

Large population-based studies examining the relationship of PD on risk of completed suicide are rare. Agerbo et al. (2002) compared all 496 suicides from the population of Denmark aged 10-21 years between 1981 and 1997 with 24,800 sex- and age-matched controls. They showed that experiencing maternal death in early life increases suicide risk. Gravseth et al. (2010) studied the population of Norway born from 1967 to 1976 and followed them until 2004. Of the 610,359 individuals that still lived in Norway at their 19th birthday, they identified 1,406 suicides. They showed a slightly higher risk of adulthood suicide for those experiencing PD by age 19. Also, a history of mother's marital instability was correlated with increased suicide risk, though they did not examine the effects of RWP *per se*. Linking data from multiple Swedish registers between 1969 to 2004, Wilcox et al. (2010) studied over 4 million parent-offspring pairs. After matching by sex and birth-year of parent and offspring, they found the risk of suicide increased among offspring whose parents died by suicide, but not among offspring whose parents died by other causes. Niederkrotenthaler et al. (2012) studied the population of Sweden born between 1973 and 1983 in a matched case-control study. They found PD after age 10 increased suicide risk, but PD previous to age 10 only significantly increased suicide risk when the PD was itself a suicide.

This body of literature is limited both in size and complexity. We build upon it by discussing and testing potential biological and social mechanisms within a life-course framework, which directs our attention to ensuing secondary chronic stressors (George, 2003; Kuh & Ben-Shlomo, 2004; Pearlin, Aneshensel, & Leblanc, 1997), considers the “linked lives” of family members (George, 2003), and views development and health as lifelong processes

(Elder & Johnson 2003). PD can be separated into two key subcomponents, each of which may independently affect adulthood mortality risk. The first is the experience of death, which may directly “scar” (Preston, Hill, & Drevenstedt, 1998, p. 1232) the subject for life. The other is lost social integration and associated secondary stressors.

The stress associated with the death itself may lead to physical and emotional scarring, initiating direct psychobiological changes. Childhood is a sensitive period of development, and attachment theory predicts that death of a parent (particularly the mother) will devastate the child who relies upon the parent to sustain life (Bowlby, 1980). The grief following parental death may be intense, and affect health through acute or chronic mechanisms. If the grief-related shock is intense enough, a survivor may suffer from “broken heart syndrome”, which might increase acute health responses such as myocardial infarction (Cramer, De Boeck, Melman, & Sieswerda, 2007; Rostila, Saarela, & Kawachi, 2013). Familial death may also lead to sudden suicide (Ajdacic-Gross et al., 2008). However, the chronic nature of stress should also be considered. Grief may persist throughout life (Bowlby, 1980), particularly since children may not be as capable as adults in proceeding through healthy grieving processes (Sood, Razdan, Weller, & Weller, 2006). As childhood is a period of great physiological plasticity (Heim, Newport, Mletzko, Miller, & Nemeroff, 2008), regulatory responses may be conditioned far into adulthood (Luecken & Roubinov, 2012). This suggests an increased likelihood of allostatic load, affecting health through such physiological mechanisms as the sympathetic nervous system, the hypothalamo-pituitary-adrenal (HPA) axis, the neuroendocrine system, the immune system, and inflammatory responses (Luecken & Roubinov, 2012; McEwen & Stellar, 1993; Rostila et al., 2013). These physiological mechanisms might increase the risk for prevalent chronic physical illnesses such as cardiovascular disease (CVD), psychiatric disorders such as major depression, borderline personality disorder, post-traumatic stress disorder, and substance abuse; and suicide (Beauchaine, Neuhaus, Zalewski, Crowell, & Potapova, 2011; Heim et al., 2008; Rostila & Saarela, 2011).

Unhealthy behaviors, such as smoking and substance abuse, may further increase the likelihood of poor health outcomes as the subject tries to cope (Martikainen & Valkonen, 1996). These behaviors might be compounded by a diminution in parental supervision following PD, because parental expectations often serve as a deterrent against unhealthy behaviors (Nash, McQueen, & Bray, 2005). This phenomenon highlights the fact that PD not only acts as a substantial psychogenic stressor, but that it also decreases social integration, which is related to suicide (Durkheim, [1897] 1951).

At the individual level, *social integration* may constitute the existence of a relationship of given type (House, Umberson, & Landis, 1988). When PD occurs, subjects lose one or two people in their social (parental) network, altering health through mechanisms involving changes in *social support*, *social regulation*, and *conflict* (House et al., 1988). *Social support* consists of instrumental or emotional assistance, both of which affect health (House et al., 1988). Losing a parent decreases potential socioeconomic resources. Early familial disruption can result in downward social mobility for the surviving family members (Biblarz & Gottainer, 2000), which can increase suicide risk (Breed, 1963). *Lost social regulation* here refers to the aforementioned diminution of informal social control against unhealthy

behaviors such as substance abuse (Nash et al., 2005). *Conflict* may decrease if the relationship with the parent was unhealthy or even abusive (Umberson & Chen, 1994), though some research suggests families with high levels of conflict may actually fare worse following PD (Dowdney, 2000). Bereavement will also require emotional and instrumental adaptation of the surviving spouse, thereby temporarily decreasing their efficiency in the parental role (Luecken & Roubinov, 2012). For example, the foregone income due to PD may also require the widowed parent to devote more time to paid employment (Biblarz & Gottainer, 2000), reducing time available to emotionally support the subject. Therefore, in some ways the surviving child “loses” social resources from both parents.

Examining the moderating role of widowed parent's remarriage can help clarify these chronic mechanisms since RWP can restore lost social integration. A stepparent might not simply “replace” a biological parent, as step-parental relationships are often of poorer quality (Daly & Wilson, 2005). But, since RWP is beneficial for other health outcomes (Andersson, Högberg, & Åkerman, 1996; Norton et al., 2011), it likely also attenuates any increased suicide risk.

Examining multiple outcomes in the same study has been enjoined as another approach for clarifying stress-related mechanisms (Thoits, 1995). The physiological mechanisms discussed thus far influence one's risk not only for suicide, but possibly for other causes of death. Does PD increase the risk for suicide *per se*, or is it simply increasing the risk for bad health overall, of which suicide is but one indicator? While many potential causes of death could be compared to suicide, CVD provides an excellent comparative cause because it is positively associated with familial death and is believed to operate through similar mechanisms of allostatic load (McEwen & Stellar, 1993; Rostila et al., 2013; Rostila & Saarela, 2011). Even psychiatric disorders such as major depression and borderline personality disorder are risk factors for both CVD and suicide (but not so much other common causes of death such as diabetes) (El-Gabalawy, Katz, & Sareen, 2010). Furthermore, CVD is the leading cause of death in the United States (Hoyert & Xu, 2012), and might therefore conceptually be viewed as a general indicator of compromised health.

If PD is more strongly associated with suicide than with CVD, we might infer that some mechanisms are more specific to suicide. We therefore suggest that comparing these competing causes of death may help clarify the most salient chronic mechanisms, and provide guidance on potential interventions. Furthermore, the fact that many mechanisms generate similar mortality patterns suggests some subjects who might have died by suicide will die of a competing cause first, so comparisons might help clarify age-specific mechanisms.

Beyond the key foci of remarriage and competing risks, the relevance of sensitive stages and dose-response relationships should also be considered. Physiological plasticity may peak during specific sensitive substages of development within the childhood period (Niederkrotenthaler et al., 2012), conditioning the magnitude of associations. Attachment theory applies more specifically during the first few years of life when the child is completely dependent upon the parental figure (Bowlby, 1980); therefore, traumas occurring during this period might especially heighten the subject's adverse chronic stress responses.

Sensitive stages also have implications for RWP's impact. Younger stepchildren are more likely to “accept” a stepparent (Adler-Baeder & Higginbotham, 2004). Earlier PD increases the likelihood of RWP at an earlier age, possibly improving the step-relationship's quality.

If PD of one parent is a risk factor for suicide, then PD of both parents might be a stronger risk factor, consistent with a dose-response relationship. The term “orphanhood” may be used to refer to a special type of dual-PD where an individual comes under the care of the state with potential supports such as foster care or legal adoption (Triseliotis, 2002). This is probably more likely when both parents die at the same time or in close succession (very rare), and would further complicate the dynamics of social integration. However, it is worth noting that even in such cases, the individual is without the support of either biological parent. Showing a dose-response relationship can increase confidence that an observed association is not artifactual (Gordis, 2008).

Research Hypotheses

We propose the following six testable hypotheses.

H₁: PD will increase suicide risk for all subjects.

H₂: Dual-PD will increase suicide risk more than PD of one parent.

H₃: Maternal PD will increase suicide risk more than paternal PD.

H₄: Among subjects exposed to PD, those experiencing RWP will experience a smaller increased suicide risk than those without RWP.

H₅: The relationship between PD and suicide risk will vary by the age PD was experienced.

H₆: The excess mortality risk following PD will be greater for suicide than for CVD.

Methods

The UPDB served as the basis for the data. UPDB is a genealogical and medical database containing information on nearly eight million individuals, and includes genealogies of the founders of Utah and their descendants up to the present time. It is a dynamic database that receives annual updates of Utah births, deaths, driver licenses, and health records. The database includes over two million Utah birth certificates and around 800,000 death certificates spanning a century. The Utah Resource for Genetic and Epidemiologic Research (RGE) is a special regulatory body created to administer access to UPDB for research purposes. All research requires IRB and RGE approval (Wylie & Mineau, 2003). The confidentiality of individuals represented in these records is maintained based on agreements between RGE and the data contributors.

First, we constructed mother, father and subject triads. We identified 1,491,140 subjects born between 1886 and 1960, inclusive. Since our cause-of-death records began in 1904, and 18 years of age was our baseline age for measuring suicide risk, no subjects born prior to 1886 were included. We excluded subjects born subsequent to 1960 to improve availability of SES and baptism data (necessary controls). Linking subjects to parents

yielded 1,255,818 triads. In order to satisfactorily test the hypotheses, we refined potential triads to those with complete data for all members. We excluded triads where the subject's sex or last date of Utah residence was unknown, birth year of either parent was unknown, the parents' follow-up data were inadequate, or the father was known to have died prior to the subject's birth. The new refined subsample consisted of 901,579 triads. We also excluded 5,808 triads where the subject was known to be adopted and 10,109 triads where any of their members were polygamous. Since we were examining suicide risk in adulthood, we then excluded 198,927 triads where the subject did not have follow-up data to at least age 18 (note that of these, 35,969 were known to have died, 73 from suicide, with the rest lost to follow-up due to emigration or limited data). Finally, we excluded 23,006 triads where the mother or father did not have a recorded death date and we did not have follow-up information past the subject's 18th year.

The final sample comprised N=663,729 subjects, including 4,533 subjects who died by suicide. This represents 52.85% of the potential triads originally identified. The retained were significantly more likely to be female (53.91% vs. 51.87%), and had a higher mean birth-year (1931.41 vs. 1925.54). And those dying by suicide were significantly more likely to be retained (64.85% vs. 52.79%).

Measures

Health Outcomes—*Suicide* was measured using ICD codes (versions 6-10) and “manner of death” indicators obtained from Utah death certificates, which were available for years 1904-2010. Suicide was indicated by ICD codes E963, E970-E979 (ICD6/7); E950-E959 (ICD8/9); X60-X84, Y87.0, U03 (ICD 10)—or a manner of death indicating suicide. The risk of suicide in our sample peaked for men after age 75, and for women around age 50, consistent with prior research (National Center for Injury Prevention and Control, 2012). *CVD* was included as a competing risk of death with ICD codes 400-469 (ICD6/7); 390-459 (ICD8/9), I00-99 (ICD10) from death certificates.

Key Exposures—*Early Life Death of Parent (PD)* was determined from parent's death year and subject's birth year. If the parent's death year occurred previous to the subject reaching age 18, then that subject experienced PD. This specification was utilized in the *Basic Sequence* (see “Analytic Strategy” below). In the *Age Sequence*, this variable was separated into exposure categories *Age 0-5*, *6-11*, and *12-17*.

Remarriage of widowed parent (RWP) was considered when the widowed parent remarried before the subject reached age 18 and within five years of PD. This five-year requirement measured ongoing social integration throughout post-PD childhood by ensuring the subject had substantial time with the stepparent, and to address the potential confounding of RWP and age at PD. Regardless of age at PD, imposing the five-year requirement allotted fairly equal time periods for RWP. When both parents died in childhood (i.e. dual-PD), RWP was technically possible if the deaths occurred in different years, but was not considered due to small sample sizes.

Control Variables—*Father and mother suicides* were coded 1 when that parent died by suicide previous to the subject's death or censoring, and 0 otherwise, to account for possible

familial suicide risk. Note this variable does not require PD, as the suicide may have occurred during subject's adulthood. *Maternal and paternal age at subject's birth* were continuous variables. *Subject's year of birth* was utilized to stratify Cox models. *Subject's baptism* by age nine measured membership in the Church of Jesus Christ of Latter-day Saints (LDS or Mormon), and was coded as a binary variable. As members of this church played a large role in the founding of the state of Utah, a large proportion of the population has historically belonged to this faith. Normative baptism age is shortly after age eight (Lyon, Gardner, & Gress, 1994), and membership may indicate abstinence from alcohol, tobacco and other substances, and a supportive community structure (Mineau, Smith, & Bean, 2004).

Subject marriage was treated as a time-varying covariate, assuming a value of 1 following the subject's year of first marriage, and 0 otherwise. *Early-life SES* was measured with the Nam-Powers SES Score, which recodes one's usual occupation (a qualitative measure) into a quantitative value on a scale (1-99), with higher values representing higher SES (Nam & Powers, 1983). Father's and mother's usual occupation as recorded on a Utah death certificate were the basis for the subject's Nam Powers score. Consistent with prior PD-suicide research (Niederkrötenhaller et al., 2012), the greater of the two parental scores represented the subject's SES. We coded this score to a six-category variable: including four ranked quartiles (1-25, 26-50, 51-75, 76-99), farming (a score of 40), and missing. Farming was considered separately, because empirical analyses showed a large number of subjects in this group. Basic descriptive statistics for the sample are displayed in Tables 1 and 2.

Analytic Strategy

Analyses were conducted in two phases. In the first phase, we estimated two series of nested Cox hazard models, where the outcome of interest was time to suicide after age 18 and censoring occurred at age the subject was last known alive in Utah. This censoring age was chosen because individuals may have emigrated and died by suicide in other states. Each model was stratified by subject's birth-year to control for cohort and period effects (Kom, Graubard, & Midthune, 1997). Models were estimated separately for males and females, because they differ in suicide risk (National Center for Injury Prevention, 2012) and responses to stress (Heim et al., 2008). Comparisons between female and male subjects are not formally part of this analysis.

Two nested model sequences were utilized. A likelihood ratio test (G^2) tested each hypothesis by comparing the nested (unconstrained) model to its parent (constrained) model via their log-likelihood fit statistics. This nesting strategy enabled us to test the complex configurations of PD and RWP in one integrated statistical sequence.

The nesting configuration is diagrammed in Figure 1, which shows the names of each model and the number of variables (including controls) each contains. The *Basic Sequence* informed whether each new variable set provided information that significantly affected hypotheses H_1 - H_4 , and the *Age Sequence* specifically tested H_5 (i.e., that age of the child at PD affected suicide risk). As each new set of variables was added, an associated variable for the interaction of each variable with age 50+ was also included. This heaviside function addressed the Cox model's proportionality assumption (Kleinbaum & Klein, 2005). Age 50

is deemed an appropriate life-course threshold, because couples often end childbearing and shift priorities towards later life about age 50 (Karp, 1989).

The *Basic Sequence* began with the *Null* model, which included all control variables. The *Parental Death* model added the additional variable of PD of at least one biological parent. In the three remaining models, this PD category was further segmented into mutually exclusive groups of increasing complexity to test hypotheses 2 through 4. The *Dual-PD* model divided PD into the categories of (1) exactly one PD and (2) dual-PD. The *Sex of Parent* model separated PD into categories of (1) maternal PD, (2) paternal PD, and (3) dual-PD. The *Parental Remarriage* model used PD categories of (1) exactly one PD without RWP; (2) exactly one PD with RWP; and (3) dual-PD. Note some models could not be nested within others, and the reader is referred to Figure 1 for nesting configurations.

The *Age Sequence* included the same models from the *Basic Sequence*, except each key variable was further segmented into the three age groups of 0-5, 6-11, and 12-17. For example, the Age Sequence Parental Remarriage model included 25 variables: the seven variables PD age 0-5 without RWP, PD age 6-11 without RWP, PD age 12-17 without RWP, PD age 0-5 with RWP, PD age 6-11 with RWP, PD age 12-17 with RWP, dual PD; seven associated later-life interaction terms; and eleven control variables. Each of these models was nested within its *Basic Sequence* counterpart, and the associated G^2 tested H_5 .

In the second analysis phase, those models that best informed H_1 - H_4 were selected for further examination and testing, including comparisons to CVD mortality as a competing risk and additional Wald tests for equality of coefficients where appropriate. Here, suicide and CVD mortality were jointly modeled using the method of Lunn and McNeil (1995). This method modeled suicide considering all other outcomes as censored, while simultaneously modelling CVD mortality considering all other outcomes as censored. Modelling each separately would yield identical parameter and standard error estimates, but this simultaneous approach enabled direct statistical comparisons between suicide and CVD mortality hazards via Wald tests (Kleinbaum & Klein, 2005; Lunn & McNeil, 1995). Models were estimated in SAS 9.3 using PROC PHREG, and Wald tests for equality of coefficients were performed using SAS's "test" command within that procedure.

Findings

Model Fit

Females—Subtable 3a displays the model fit statistics within the *Basic Sequence* for females. Cells directly testing hypotheses are shaded gray. Testing the *Null* model against the *Parental Death* model yielded a p-value <.001, warranting further investigation of the *Parental Death* model. The other statistically significant comparison was *Dual-PD* vs. *Parental Remarriage*. Here, the crucial element was remarriage (RWP). In fact, the *Parental Remarriage* model significantly improved upon all more parsimonious models. Accordingly, remarriage may be an important moderator for females with respect to how parental death correlates with later suicide risk. Comparing the *Base* to *Age Sequences* (subtable 3b) did not significantly improve model fit. Thus, we found no evidence suggesting sensitive substages were important prior to age 18. Though not explicitly shown, we note that within

the *Age Sequence*, comparing the *Dual-PD* to *Parental Remarriage* model significantly improved model fit. So, even when considering age at PD, remarriage proved important.

Males—In subtable 4a, the only statistically significant key comparison was between the *Null* and *Parental Death* models. Considering age at PD (subtable 4b) did not significantly improve model fit. Therefore, we found no evidence suggesting the sex of the parent, death of both parents, remarriage of widowed parent, or childhood substage were important moderating factors related to male suicide risk.

Cox Model Parameter Estimates

We present and interpret results for the models that improved upon their simpler nested counterparts. For females, this includes the *Parental Death* and *Parental Remarriage* models in the *Basic Sequence*. For males, this includes only the *Parental Death* model in the *Basic Sequence*.

Females—Table 5 shows the hazard ratios (HR's) and significance levels predicting suicide, and cardiovascular death, for each variable in the two Cox models for females. It also displays statistical significance levels from Wald tests for equality between suicide and CVD HR's. Estimates for control variables barely varied across model specifications and were not our main focus. We do not interpret these estimates, but they are presented in Table 6 for the *Basic Parental Death Model*.

In the *Parental Death* model, for females under age 50, PD was associated with a 65% increased risk of suicide. The association with CVD was only 20%, and significantly smaller than the risk for suicide. This lends support for the hypothesis that PD will increase suicide risk more than CVD. This changed after age 50, where the association between PD and suicide disappeared, but the association with CVD remained and was statistically higher than the risk for suicide. In the *Parental Remarriage* model, PD of exactly one parent without remarriage of the widowed parent was associated with a significantly increased suicide risk, but there was no significant increase in suicide risk when remarriage occurred. An additional Wald test for equality between these two parameters yielded an insignificant p-value, so we cannot be certain the hazard ratio for those with RWP was smaller than for those without. Suicide risk for dual-PD females under 50 trebled the risk of those with no PD. An additional test showed the hazard for dual-PD was higher than for single-PD with remarriage, but not without remarriage. So once remarriage is considered, the death of both parents becomes significant, suggesting a possible dose-response to putative lost social integration. After age 50, we found a reverse association. Remarriage was associated with statistically higher suicide risk than no remarriage, suggesting possible late-onset harmful associations between parental remarriage and suicide.

Males—Table 5 also displays the *Parental Death* model for males. Before age 50, parental death was significantly associated with a 24% increased risk of suicide. The association was absent after age 50. There was no difference between suicide and CVD risk under age 50, but CVD risk was statistically greater than suicide risk after 50.

Additional Tests

To deal with possible issues of independence of study subjects since sibships are present in the data, we performed two sensitivity analyses for the parameters specific to risk of suicide in the *Basic Sequence* models presented in Table 5. First, we re-estimated the models with the sandwich estimator (Kleinbaum & Klein, 2005) by computing robust standard errors clustered about the mother. The findings were substantively similar. Next, we re-estimated the models using a subsample formed by randomly selecting one sibling from each mother. Again, the findings were similar.

To test whether the cause of parental death was an important moderator, we further segmented the *Basic Sex of Parent* model into paternal and maternal PD by causes of CVD, respiratory, cancer, other, and unknown. G^2 comparing these models were not statistically significant, suggesting that the cause of the parental death was not an important confounder in our sample. Sample sizes prohibited further segmenting cause of death. Finally, though not an explicit focus of this paper, additional analyses also suggested PD increased suicide risk more for females than males before age 50.

Discussion

Consistent with previous research, we found that early-life parental death was associated with an increased risk of suicide and CVD after age 18 for both sexes. We observed a dose-response relationship, where women with no living biological parents alive at age 18 had a higher risk for suicide than those with one biological parent and a stepparent before age 50. We suspect this association with remarriage may reflect the importance of lost social integration following PD, particularly since social relationships have been shown to be especially important for female health (House et al., 1988). However, this is not a foregone conclusion, since those who remarry are a non-randomly selected group, and RWP may indicate a latent propensity for healthy adaption that also protects against suicide.

For women over age 50, suicide risk was significantly greater in the presence of remarriage than in its absence. It is known women often provide support to aging parents (Fuller-Thomson, Minkler, & Driver, 1997). We suspect those women who experienced RWP may feel pressure to care for the stepparent, possibly providing additional stress since step-relationships are more prone to conflict (Daly & Wilson, 2005). We suggest future research attempt to replicate this finding and test possible explanations.

For males, we found no evidence for a moderating impact of widowed parent's remarriage, and no dose-response relationship with PD. It is possible the male suicide hazard is already so high that increasing it further is difficult. Additionally, since the ensuing suicide risk was not greater than CVD risk before age 50, males exposed to early parental death may be dying of CVD before dying by suicide.

Suicide risk generally attenuated after age 50, possibly due to developed resilience (Rattan, 2008; Wheaton, 1999) or mortality selection (Vaupel & Yashin, 1985) over the life-course. We also found that PD increased CVD risk for both males and females, which is consistent with many of the proposed chronic mechanisms linking PD to both health outcomes. For

females, PD was a stronger risk for suicide than CVD mortality before age 50. Since this is the period when remarriage appears to be protective, social integration may be more specifically related to suicide. We also note that suicide requires an action by the victim, whereas CVD does not. It may be that certain women learn coping skills to avoid suicide, and that capacity increases over time (Wheaton, 1999); but the allostatic load (McEwen & Stellar, 1993) may accumulate for women and be reflected in higher later-life CVD risk.

We suggest our most important findings imply that the widowed parent's remarriage moderates the association of early parental death with later-life suicide risk for females. Future research should consider how stepsiblings, grandparents and other extended kin networks may further moderate these associations. Future research might also attempt to further disentangle how RWP's effects relate to the child's age at exposure to PD, since such stages have previously shown to be important for predicting suicide risk (Niederkrötenhaler et al., 2012). We note that in cases where the parent dies very early, the widowed parent has more time to remarry. This study tried to adjust for this possibility by requiring the parent to remarry within a five-year time period, and we further showed that considering subject's age at PD did not improve upon our basic predictive models. Further, the evidence suggested that even once age was considered, RWP was still an important moderator.

Subjects in our sample were required to live to age 18. Some research suggests mortality may be higher before age 18 following early parental death (Rostila & Saarela, 2011). Thus, deaths to adolescents may have culled the most susceptible from the population, and therefore we suggest our findings are more relevant to chronic than acute mechanisms. We also suspect some of our subjects that did not experience PD experienced early parental divorce, which is another prevalent form of early familial disruption known to increase suicide risk (Agerbo et al., 2002). Due to data limitations, we could not account for divorce in our study. Survival requirements and the omission of divorce data likely promote a conservative statistical bias. We were unable to measure the subjective quality of family relationships, and could not account for possible external institutions, such as foster home programs, that might become part of a child's life following single or dual-PD. Finally, we note that to obtain the large sample size needed for examining suicide, we coalesced a broad range of birth cohorts from 1886 through 1960. Early PD might be considered an off-time event that is more stressful because of its unexpectedness (Rostila & Saarela, 2011), and therefore parental death and remarriage might yield stronger associations in later cohorts when it is less common. While we stratified by birth-year in the analysis, this cohort heterogeneity may have nevertheless statistically biased estimates towards the null. Despite these potential limitations, this study offers a substantial contribution to the literature on early-life stress and suicide. Health professionals should keep these trends in mind when planning potential interventions.

In terms of policy, efforts aimed at preserving or restoring social resources may prove helpful. The most obvious primary policy implication involves preventing premature deaths of parents. Anything shown to protect the health of new parents, including resources such as education, healthcare, prenatal care, childcare and social welfare services (Marmot, Allen, Bell, Bloomer, & Goldblatt) could reduce exposure to the stressor of early parental death. Secondary preventions might involve economic support to the widow or child, including

timely survivor benefits and therapies that help survivors cope. Tertiary preventions would include an individualized treatment approach as the clinician works to prevent suicide amongst at-risk individuals, and manage chronic illnesses associated with the early parental death.

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Highlights

- Early-life parental death (PD) was associated with later suicide risk before age 50
- For females, PD's association was larger for suicide than CVD death prior to age 50
- For females, remarriage of parent attenuated increased suicide risk prior to age 50
- For females, remarriage of parent further increased suicide risk after age 50

Figure 1. Nested Model Sequence Diagram

Table 1
Descriptive Statistics for Health Outcomes, and Parental Death (PD) and Remarriage of
Widowed Parent (RWP) Exposure Categories

	Female		Male	
	Frequency	Percentage	Frequency	Percentage
Total	327,159	100.0%	336,570	100.0%
Health Outcomes				
Suicide	951	.3%	3,582	1.1%
Cardiovascular Death	53,353	16.3%	57,849	17.2%
Censored	272,855	83.4%	275,139	81.7%
PD Category				
No PD	287,164	87.8%	297,026	88.3%
At Least One PD	39,995	12.2%	39,544	11.7%
0-5	10,141	3.1%	9,672	2.9%
6-11	13,060	4.0%	13,000	3.9%
12-17	16,794	5.1%	16,872	5.0%
Exactly One PD	38,040	11.6%	37,733	11.2%
0-5	9,300	2.8%	8,930	2.7%
6-11	12,319	3.8%	12,311	3.7%
12-17	16,421	5.0%	16,492	4.9%
One PD, RWP	30,087	9.2%	29,981	8.9%
0-5	5,987	1.8%	5,854	1.7%
6-11	9,260	2.8%	9,302	2.8%
12-17	14,840	4.5%	14,825	4.4%
One PD, no RWP	7,953	2.4%	7,752	2.3%
0-5	3,313	1.0%	3,076	.9%
6-11	3,059	.9%	3,009	.9%
12-17	1,581	.5%	1,667	.5%
Maternal PD Only	14,515	4.4%	13,942	4.1%
0-5	3,940	1.2%	3,646	1.1%
6-11	4,906	1.5%	4,665	1.4%
12-17	5,669	1.7%	5,631	1.7%
Paternal PD Only	23,525	7.2%	23,791	7.1%
0-5	5,360	1.6%	5,284	1.6%
6-11	7,413	2.3%	7,646	2.3%
12-17	10,752	3.3%	10,861	3.2%
Dual-PD	1,955	.6%	1,811	.5%

Table 2
Descriptive Statistics for Control Variables

	Female		Male	
Categorical Variables	Frequency	Percentage	Frequency	Percentage
Total Count	327,159	100.0%	336,570	100.0%
Categorical Variables				
Maternal Suicide	467	.1%	471	.1%
Paternal Suicide	2,012	.6%	2,127	.6%
Baptized	175,426	53.6%	171,010	50.8%
Married	234,288	71.6%	236,257	70.2%
<i>Nam Powers Score (SES)^a</i>				
76-99	47,946	14.7%	49,867	14.8%
51-75	65,934	20.2%	67,943	20.2%
26-50, excluding 40	43,764	13.4%	45,567	13.5%
1-25	20,891	6.4%	20,910	6.2%
40 (Farming)	66,291	20.3%	68,510	20.4%
Missing	82,333	25.2%	83,773	24.9%
Continuous Variables	Mean	Std. Dev.	Mean	Std. Dev.
Subject's Birth year	1931.0	21.4	1931.8	21.3
Maternal Age	28.3	6.4	28.2	6.4
Paternal Age	32.0	7.6	31.9	7.6
Subject's Years of Follow-up (From Birth Year)	63.5	19.4	61.1	18.1

Notes:

^aThe greater of the subject's mother's or father's Nam Powers score obtained from parent's death certificate. Higher values represent higher SES.

Table 3

Females: Likelihood Ratio Tests Comparing Model Fits

Females N=327,159						
Subtable 3a: Comparisons between Models within Basic Sequence						
Model Name	LL	df	Null	Parental Death	Dual-PD	
			-7797.19	-7789.08	-7787.65	
Parental Death	-7789.08	13	16.22, 2 ***	13	15	
Dual-PD	-7787.65	15	19.08, 4 ***	2.86, 2		
Sex of Parent	-7787.22	17	19.95, 6 **	3.73, 4		
Parental Remarriage	-7783.45	17	27.48, 6 ***	11.26, 4 *	0.87, 2	8.40, 2 *
Subtable 3b: Comparisons of Models between Basic and Age Sequences						
Model Name	Base Models	Age Models	Chi-squared			
	LL	df	LL	df		
Parental Death	-7789.08	13	-7786.96	17	4.25, 4	
Dual-PD	-7787.65	15	-7786.04	19	3.21, 4	
Sex of Parent	-7787.22	17	-7784.43	25	5.58, 8	
Parental Remarriage	-7783.45	17	-7778.70	25	9.50, 8	

Notes: Each subtable consists of model fit log likelihood (LL) statistics and degrees of freedom (df) for nested model specifications.

In subtable a, constrained models are in the columns and unconstrained in rows. The cells are populated with the chi-square statistics from likelihood ratio tests in the format: chi-square value, df followed by statistical significance.

Subtable b compares the constrained *Basic* model to its counterpart unconstrained *Age* model in rows only.

* p<.05

** p<.01

*** p<.001

Table 4

Males: Likelihood Ratio Tests Comparing Model Fits

Males N=336,570						
Subtable 4a: Comparisons between Models within Basic Sequence						
Model Name	LL	df	Null	Parental Death	Dual-PD	
			-29285.02	-29280.58	-29280.04	
		11		13	15	
Parental Death	-29280.58	13	8.87, 2 *			
Dual-Bereaved	-29280.04	15	9.95, 4 *	1.08, 2		
Sex of Parent	-29278.09	17	13.84, 6 *	4.98, 4	3.9, 2	
Parental Remarriage	-29278.43	17	13.18, 6 *	4.32, 4	3.23, 2	
Subtable 4b: Comparisons of Models between Basic and Age Sequences						
Model Name	Base Models LL	df	Age Models LL	df	Chi-squared	
Parental Death	-29280.58	13	-29278.47	17	4.23, 4	
Dual-PD	-29280.04	15	-29277.86	19	4.37, 4	
Sex of Parent	-29278.09	17	-29274.12	25	7.94, 8	
Parental Remarriage	-29278.43	17	-29274.82	25	7.21, 8	

Notes: Each subtable consists of model fit log likelihood (LL) statistics and degrees of freedom (df) for nested model specifications.

In subtable a, constrained models are in the columns and unconstrained in rows. The cells are populated with the chi-square statistics from likelihood ratio tests in the format: chi-square value, df followed by statistical significance.

Subtable b compares the constrained *Basic* model to its counterpart unconstrained *Age* model in rows only.

* p<.05

** p<.01

*** p<.001

Table 5
Cox Model Hazard Ratios and Significance Levels for Suicide and Cardiovascular Death (CVD), and Lumm-McNeil Wald Tests

Females	Parental Death Model		Parental Remarriage Model	
N=327,159	Suicide	=^b	Suicide	= CVD
Under 50				
At Least One PD	1.65***	[*]	1.20***	
Exactly One PD				
One PD, no Remarriage			1.70***	[**] 1.18**
One PD, Remarriage			1.07	1.23*
Dual PD			3.06**	[*] 1.33
50-and-Over^a				
At Least One PD	1.01	[***]	1.12***	
Exactly One PD				
One PD, no Remarriage			0.83	1.13***
One PD, Remarriage			1.77*	[*] 1.05
Dual PD			0.77	1.20***
Males	Parental Death Model		Omitted	
N=336,570	Suicide	=	CVD	
Under 50				
At Least One PD	1.24**		1.34***	
50-and-Over^a				
At Least One PD	1.02	[*]	1.10***	

Notes: Reference group is no PD. Models include all control variables (not shown).

^aFor 50-and-over risk period, HR calculated as [HR for under 50 * HR for over-50 interaction term (not presented)], significance level calculated with Wald test.

^bP-values from Wald tests between suicide and CVD HR's.

* p<.05

100>d

10>d
**

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Table 6
Parameter Estimates for Control Variables in Basic Sequence Parental Death Model

Controls Variable	Females; N=327,159		Males; N=336,570	
	Suicide	= ^b CVD	Suicide	= CVD
Maternal Suicide	2.22	1.20	2.62***	[***] 0.96
Paternal Suicide	1.49	1.04	2.12***	[***] 1.01
Maternal Age	0.99	0.99**	0.99*	[**] 1.01
Paternal Age	1.01	1.01	1.01*	[**] 0.99*
Baptized	0.50***	[***]	0.88***	[***] 0.86***
Married	0.86*	0.88	0.73***	[*] 0.81***
<i>Nam Powers Score (SES)^a</i>				
51-75	0.88	[**] 1.17***	1.03	[*] 1.17***
26-50, excluding 40	0.85	[*] 1.20***	1.14*	1.21***
1-25	0.81	[**] 1.30***	1.11	1.27***
40 (Farming)	0.62***	[***] 1.17***	1.02	[*] 1.16***
Missing	0.76**	[***] 1.07***	0.93	[*] 1.06***

Notes:

^aThe greater of the subject's mother's or father's Nam Powers score obtained from parent's death certificate. Higher values represent higher SES. Reference group is 76-99 (highest SES).

^bP-values from Wald tests between suicide and CVD HR's.

* p<.05

** p<.01

*** p<.001