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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 4533 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.

Suicide was the 10th leading age-adjusted cause of death in the United States for 2011 (Hoyert and Xu, 2012). While proximal stressors are known to affect suicide risk (Denney et al., 2009), early-life stressor have also been implicated (Agerbo et al., 2002). A potential early-life stressor implicated in suicide risk (and overall mortality) is early-life parental death (PD) (Niederkrotenthaler et al., 2012; Smith et al., 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 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 and 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).

1. 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 1406 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

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correlated with increased suicide risk, though they did not examine the effects of RWP *per se*. Linking data from multiple Swedish registers between 1969 and 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 and Ben-Shlomo, 2004; Pearlin et al., 1997), considers the "linked lives" of family members (George, 2003), and views development and health as lifelong processes (Elder and 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 et al., 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 et al., 2007; Rostila et al., 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 et al., 2006). As childhood is a period of great physiological plasticity (Heim et al., 2008), regulatory responses may be conditioned far into adulthood (Luecken and 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 and Roubinov, 2012; McEwen and 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 et al., 2011; Heim et al., 2008; Rostila and 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 and 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 et al., 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 et al., 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 and 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 and 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 and Roubinov, 2012). For example, the foregone income due to PD may also require the widowed parent to devote more time to paid employment (Biblarz and 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 and Wilson, 2005). But, since RWP is beneficial for other health outcomes (Andersson et al., 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 and Stellar, 1993; Rostila et al., 2013; Rostila and 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 et al., 2010). Furthermore, CVD is the leading cause of death in the United States (Hoyert and 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