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Survival of offspring who experience early parental death: Early life conditions and later-life mortality

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ABSTRACT

We examine the influences of a set of early life conditions (ELCs) on all-cause and cause-specific mortality among elderly individuals, with special attention to one of the most dramatic early events in a child's, adolescent's, or even young adult's life, the death of a parent. The foremost question is, once controlling for prevailing (and potentially confounding) conditions early in life (family history of longevity, paternal characteristics (SES, age at time of birth, sibship size, and religious affiliation)), is a parental death associated with enduring mortality risks after age 65? The years following parental death may initiate new circumstances through which the adverse effects of paternal death operate. Here we consider the offspring's marital status (whether married; whether and when widowed), adult socioeconomic status, fertility, and later life health status. Adult health status is based on the Charlson Co-Morbidity Index, a construct that summarizes nearly all serious illnesses afflicting older individuals that relies on Medicare data. The data are based on linkages between the Utah Population Database and Medicare claims that hold medical diagnoses data. We show that offspring whose parents died when they were children, but especially when they were adolescents/young adults, have modest but significant mortality risks after age 65. What are striking are the weak mediating influences of later-life comorbidities, marital status, fertility and adult socioeconomic status since controls for these do little to alter the overall association. No beneficial effects of the surviving parent's remarriage were detected. Overall, we show the persistence of the effects of early life loss on later-life mortality and indicate the difficulties in addressing challenges at young ages.

Introduction

The seeds of senescence may be sown early in life. How individuals experience aging is attributable to both genetic and environmental forces. In this paper, we consider specifically the influence of deprivations and privileges in early life and the manner in which they alter the mortality risks experienced decades later after age 65. This analysis relies on the Utah Population Database, a premier longitudinal, familial health database that is linked to Medicare diagnostic data.

The research question we address in this analysis asks whether conditions present early in individuals' lives are associated with their mortality risk as elders, focusing specifically on a dramatic change encountered by some dependent children and young adults: the death of a parent. Our strategy is to consider the exogenous circumstances present at the time of these parental deaths and adjust for them while estimating the effects of parental deaths on offspring mortality after age 65. We subsequently assess whether the parental-death/mortality risk is mediated by downstream events of the offspring.

This analysis is distinctive in three key respects. It studies a sizable fraction of the elderly population within a defined population. The design also allows us to control for unique familial and

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biodemographic factors. We are also able to link to medical (Medicare) records thereby allowing us to assess how serious co-morbid conditions mediate the effects of parental death and offspring survival.

Significance

We address a fundamental problem about aging: identifying early life conditions that explain the variability in health status many decades later. While the broad question has been the focus of a number of studies (Galobardes, Lynch, & Smith, 2008; Kuh, 2007; Kuh & Ben-Shlomo, 2004), consensus regarding which early life conditions contribute to these health and longevity differentials remains elusive. Many argue that exposures in the early years are profoundly important and shape mortality shifts among adults (Finch & Crimmins, 2004; Hawkes, Smith, & Blevins, 2012). We also consider the role of familial-specific factors as a key early life factor affecting adult health outcomes. The empirical literature that addresses the health effects of early life conditions has not generally analyzed the role of family history or genetics of health and disease (e.g., family history of suicide or heart disease). Some have acknowledged that these influences exist (Blackwell, Hayward, & Crimmins, 2001; Elo & Preston, 1992) with only a few analyses assessing its importance (Gluckman, Hanson, Cooper, & Thornburg, 2008; Smith, Mineau, Garibotti, & Kerber, 2009).

Several mechanisms have been proposed that link early life conditions (ELCs) to later-life health. These include direct effects where children acquire susceptibilities that generate excess adult mortality risks (Bengtsson & Lindstrom, 2003; Blackwell et al., 2001; Elo & Preston, 1992). Barker (Barker, 1990, 1994) has long argued that poor pre-natal nutrition alters fetal development and programs adult-onset disease risk. Alternatively, acquired immunities from childhood illnesses (Hayward & Gorman, 2004) and hormesis (the beneficial effects of moderate stress) suggest that some early adversity may be beneficial (Mattson, 2008). Preston, Hill, and Drevenstedt (1998b) noted that those with early deprivation are likely to endure many of the same adversities throughout life because conditions encountered when young (e.g., low SES) persist into adulthood, a mechanism that is counter to the idea that early susceptibilities *per se* lead to subsequent poor health (Kuh & Ben-Shlomo, 2004; Mirowsky & Ross, 2008; O'Rand & Hamil-Luker, 2005).

Identifying links between early exposures and later health also raises questions about mortality selection. Robust individuals exposed to harsher environments earlier in life may have better survival at older ages (Corti et al., 1999; Hawkes, Smith, & Robson, 2009; Nam, Weatherby, & Ockay, 1978; Strehler & Mildvan, 1960). This suggests that adversity at young ages may be associated with better health at older ages. Alternatively, survivors to advanced ages may be likely to have endured adversity that led to scarring, a feature that enhances their mortality risks (Myrskyla, 2010; Preston, Hill, & Drevenstedt, 1998a).

What may be regarded as one of the most traumatic ELCs to a child, adolescent, or young adult is the death of a parent. Indeed, parental death may indicate environmental conditions leading to a parent's death that also adversely affect the adult offspring's risk of premature death. A number of investigations have examined how early parental death has increased the risk of adverse health outcomes later in life (van Domburgh, Vermeiren, Blokland, & Doreleijers, 2009; Jacobs & Bovasso, 2009; Mireault & Bond, 1992; Persson, 1981; Roy, 1983; Saler & Skolnick, 1992; Umberson & Chen, 1994). Younger children in these bereaved families are likely to experience the same loss of social and economic support as those encountered by the surviving parent. Certainly childhood and adolescence are phases where psychological and physical change can be tumultuous ordinarily – a loss of a parent at these ages could therefore yield dramatic lasting effects. In studies of Alzheimer's Disease (AD), AD risk past age 65 increased if an individual lost their parents to death early in life (Norton, Ostbye, Smith, Munger, & Tschanz, 2009; Norton et al., 2011). In a study of mortality for subjects born in a much earlier era with higher rates of parental mortality (between 1850 and 1900) that relied on sibling pairs, no support for the presence of excess mortality associated with early parental death was detected (Smith et al., 2009).

The transition from adolescence to adulthood, and the role that parents play during that critical stage, have been studied extensively (Reinherz, Giaconia, Hauf, Wasserman, & Silverman, 1999; Shanahan, 2000; Wickrama, Conger, Wallace, & Elder, 2003). The loss of parents may serve to initiate or exacerbate undesirable outcomes for their young adult offspring as a result of their inability to provide financial and social support at a key juncture in the life course of their offspring, especially as they relate to their children's economic independence or family formation.

Our attention is drawn to ELCs that are present in childhood, adolescence and young adulthood that can be measured on an entire population of seniors alive when we are able to examine morbidity via medical records. As we have argued previously (Smith et al., 2009), a family history of longevity may be one of the best early life measures that predicts adult survival – indeed it may be ***the*** earliest measure as it represents a biodemographic marker for familial health and longevity that exerts an influence from the very beginning. In this previous work we suggested via the use of genealogies that a measure of familial longevity, called Familial Excess Longevity or FEL (the construction of FEL is described below) (Kerber, O'Brien, Smith, & Cawthon, 2001), may be thought of as an observable proxy for frailty. If a family history of longevity is salient, then we should expect to see differences in mortality risks across levels of FEL.

In addition to a family history of longevity, we examine three other key conditions that may confound the mortality effects of parental loss. First, associations have been shown between parental age at birth and offspring health outcomes including longevity (Gavrilov & Gavrilova, 1997; Priest, Mackowiak, & Promislow, 2002). Others argue that longevity is affected by the number of mutations accumulated in germ line (ova and sperm) cells that arise when parents reproduce at advanced ages (Gavrilov & Gavrilova, 1997; Smith et al., 2009). Parental age may affect offspring longevity because children born to older parents have higher educational/occupational attainment and greater access to socioeconomic resources (Mare & Tzeng, 1989). However, older parents share fewer years of life with their children than other parents (Myrskyla & Fenelon, 2012). The adverse effects of early (teenage) parenthood in terms of socioeconomic outcomes, childbearing and mental health characteristics have also been demonstrated (Fergusson & Woodward, 1999; Liu, Zhi, & Li, 2011; Moore & Waite, 1981).

The quality of lives of children, adolescents and young adults may also be affected by the family's socioeconomic status (SES), sibship size, and in the case of Utah, their religious affiliation. We measure all three in this analysis to control for confounding conditions existing prior to parental death. Family-of-origin SES has been shown to affect the fortunes of offspring in adulthood (Smith, Mineau, & Bean, 2002; Smith et al., 2009). We have also demonstrated the importance of parental religious affiliation because members of the Church of Jesus Christ of Latter-day Saints (LDS or Mormons) have lower mortality given their lack of smoking and alcohol consumption and elevated levels of social integration

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