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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.

Keywords

Early parental death; Mortality; Life-course; Cumulative disadvantage

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 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 (Myrskylä, 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 (Myrskylä & Fenelon, 2012). The adverse effects of early (teenage) parenthood in terms of socioeconomic outcomes, child-bearing 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 (Mineau, Smith, & Bean, 2004). Sibship size has also been identified as a childhood condition affecting later-life health. Some suggest that members of larger sibships and latter born siblings will have lower educational achievement that leads to unhealthy lifestyle choices (Downey, 1995; Hart & Smith, 2003; Modin, 2002). The resource dilution model (Downey, 1995; Guo & VanWey, 1999; Modin, 2002) argues that parents have finite levels of resources, and more offspring means greater resource dilution. Alternatively, siblings may improve survival, due to the social support they provide in adulthood (Garibotti, Smith, Kerber, & Boucher, 2006). Sibship size and birth order may therefore have both protective and risk effects.

Several important events or circumstances are considered that may mediate the pathway connecting parental death experienced early in life and subsequent elderly mortality risk. Mediators considered here are the offspring's adult marital events, parity and health status. The effect of marital status on mortality has been demonstrated consistently though some

argue whether it is selection or causation (Elo & Preston, 1996; Goldman, 1993; Smith & Waitzman, 1997). While beneficial health outcomes are associated with being married (and adverse outcomes with marital dissolution), there are also distinct mortality effects of fertility *per se* (Gagnon et al., 2009; Smith et al., 2002). But the presence of parents (or to-be grandparents) has been found to be associated with longevity but also fertility (i.e., the grandmother hypothesis (Hawkes & Smith, 2009)). Finally, cumulative disadvantages, which may have been triggered by early parental death, have been shown to increase adult morbidity risks, indicating the need to consider adult health status as an important potential mediator (Ferraro & Kelley-Moore, 2003; Turrell, Lynch, Leite, Raghunathan, & Kaplan, 2007).

Data and methods

This study utilizes data drawn from the Utah Population Database (UPDB). The UPDB is one of the world's richest sources of linked population-based information for demographic, genetic, and epidemiological studies. UPDB has supported biodemographic studies in large part because of its size, pedigree complexity, and linkages to numerous data sources. The majority of life-span epidemiological studies examine health influences of ELCs with relatively modest sample sizes. The full UPDB now contains data on nearly 7 million individuals due to longstanding efforts to update records as they become available including all statewide death certificates (1904-present) and Medicare claims. For this study, we have identified members of birth cohorts from the first half of the 20th century, individuals for whom early and midlife conditions are measured and who are linked to their adult medical records generated decades later. It is these complex data links that provide unparalleled data quality and depth that focus on families and health outcomes that span entire life spans of individuals and their relatives. This study has been approved by the University of Utah's Resource for Genetic and Epidemiologic Research and its Institutional Review Board.

Given the large sample sizes and the quickly changing morbidity risks by age and gender, we conducted all survival analyses by gender and estimated age-stratified Cox regressions (each 5 year age group being allowed to have their own baseline hazard). The first age category begins at age 66 to eliminate the problems of prorating the partial year coverage of individuals who became age eligible for Medicare part-way into a year when they turned age 65. Ages are assessed in 1992, the first year in which we have Medicare data. The restriction to age 65 for all analyses was imposed to allow for the assessment of the mediation effects of health status as measured by Medicare data.

As noted later, we also conducted age-specific models for those who were less than 75 and those 75+ in 1992. Individuals were followed for a maximum of 19 years (to 2011). The total sample size is $N = 92,618$ (N (females) = 50,687 and N (males) = 41,931). All cases in the study have complete data on all variables with one exception, as noted below, with respect to a simple imputation for missing paternal SES.

Key measures

We begin by describing the two key health measures: the Charlson Comorbidity Index and all-cause/cause-specific mortality. The health status of individuals is measured by the

Charlson comorbidity index (Charlson, Pompei, Ales, & MacKenzie, 1987). The Charlson index was adapted for use with ICD-9 codes by Deyo, Cherkin, and Ciol (1992) and Romano, Roos, and Jollis (1993). Both modifications were intended for use with the Medicare Part A records (Klabunde, Potosky, Legler, & Warren, 2000). Klabunde, Warren, and Legler (2002) introduced information from physician claims data that significantly enhanced the index's predictive value for the risk of mortality. In the present study, we have adopted this variant of the Charlson Comorbidity index based on the Surveillance, Epidemiology and End Results (SEER) Program (SEER) Medicare comorbidity SAS macros.

A second SEER-Medicare macro calculated the comorbidity index with respect to cancer. Given that cancer originally was the index disease, it was not included as a co-morbid condition in this SEER-Medicare program. The Deyo version of the Charlson index uses ICD-9-CM codes. Two diseases not included in the Deyo version are cancer and metastatic carcinoma because this macro assumes that co-morbidities are relative to cancer. Accordingly, we have added cancer as a co-morbid disease. We identified specific episodes of the following 17 morbidities occurring at baseline in 1992 that form the basis of the Charlson Comorbidity Index: myocardial infarction, congestive heart failure, peripheral vascular disease, cerebrovascular disease, dementia, chronic pulmonary disease, rheumatologic disease, peptic ulcer disease, mild liver disease, diabetes (mild to moderate), diabetes with chronic complications, hemiplegia/paraplegia, renal disease, any malignancy, moderate or severe liver disease, metastatic solid tumor, and AIDS.

Our goal was to avoid characterizing someone as being disease-free when in fact their health events were simply not well represented in the Medicare data. CMS provides a monthly HMO indicator variable that describes when a beneficiary was enrolled in a managed care plan. For Utah, managed care enrollment, where the individual would not have complete data represented in the Medicare claims file, peaked at 15.8% of all Medicare enrollees. Another complication is Medicare Part C (Medicare Advantage Plan). Part C is the combination of Part A and Part B, but is different in that it is provided through private insurance companies approved by Medicare. As expected, few claims existed in the file for individuals during the time they were enrolled in part C. For this analysis, we excluded persons that had more than 50% coverage from an HMO or were enrolled in Part C in 1992.

Mortality information was based on Utah death certificates linked to UPDB. Deaths could occur in any year spanning 1992–2011. In each instance, cause of death was available in ICD-9 or ICD-10 codes. These causes were aggregated into larger categories for the selected cause-specific analyses and they represent the leading causes of death found among this sample of seniors. We have added external causes as a special case given that they represent more immediate external causes and to serve as a contrast to the other explicitly disease-oriented categories:

Circulatory :	ICD9 : 390—459; ICD10 : I00-I99
Cancer, Neoplasms :	ICD9 : 140—239; ICD10 : C00-C99; D00-D48
Endocrine, nutritional & metabolic diseases :	ICD9 : 240—279; ICD10 : E00—E90
Respiratory	ICD9 : 460—519; ICD10 : J00-J99

Nervous System and Sense Organs :

ICD9 : 320—389; ICD10 : G00-G99; H00-H95

External :

ICD9 : 800—999; ICD10 : S; T; VW; X; Y 00-99

Measures of age at parental death, parental remarriage, sibship size, marital status, maternal and paternal LDS status (parental data when subject was a child), parental birth age, familial excess longevity (FEL) and parental SES were derived from the UPDB. For the parental ages at the offspring's birth, the parental ages at death and familial excess longevity, we acknowledge the possibility of problematic collinearity. The pairwise correlations of these variables were all quite low ($r(\text{parental age at birth, parental death age}) = 0.07$ for fathers and 0.01 for mothers; $r(\text{parental death age, FEL}) = 0.17$ for fathers and 0.17 for mothers; and $r(\text{parental age at birth, FEL}) = 0.04$ for fathers and 0.05 for mothers).

For fathers who died in Utah and for whom we obtained a Utah death certificate, we captured their usual industry and occupation from the death certificate. These data have been converted to a socioeconomic index developed by Nam and Powers (Nam & Powers, 1983). Higher scores are associated with higher SES. Approximately 25 percent of fathers in the sample did not link to a Utah death certificate. These individuals had missing SES data, were identified by a dummy variable and were assigned the group mean for the Nam-Powers socioeconomic index.

Family history of longevity was measured using FEL, a statistic developed using deep genealogical data of multigenerational pedigrees drawn from the UPDB (Kerber et al., 2001), and applied to other life-span studies using UPDB (Garibotti et al., 2006). At its foundation, the FEL is based on the assumption that family history of longevity follows Mendelian patterns of inheritance. Higher levels of FEL correspond to a stronger history of family longevity. To construct FEL, we calculated individual-level excess longevity measured by the difference between an individual's attained age and the age that that individual was expected to live based on a lognormal accelerated failure time model using two basic covariates: gender and birth year. We included only persons who reached age 65 so that the measure was less affected by deaths from external causes. This measure was calculated on all blood relatives living to age 65 in UPDB. FEL is simply the weighted average of all excess longevity of all such relatives. The weights are the kinship coefficient, which is the probability that an individual shares a particular allele with another individual identical by descent from a common ancestor.

Two additional likely mediating variables were also considered for a large subsample of individuals who eventually became parents. First, fertility was measured as the total number of children born based on all source records in the Utah Population Database. The second measure was adult socioeconomic status again based on Nam-Powers scores constructed from occupation information appearing on Utah birth certificates. The maximum SES score was used for those having more than one child. For men, the SES score reflects their own occupation, while for women it reflects their spouse's unless no husband was noted.

Statistical methods

Sex-specific Cox proportional hazard models were estimated for all analyses. Tests of the proportionality assumption for the effects of early parental death indicated the assumption

was upheld in these models. Adjustments for clustering by common parents were made for all Cox models. Given the powerful effects of age on mortality for older-aged samples, we included aggressive controls by stratifying the Cox models by age.

Two sets of models were estimated under two different sample inclusion conditions. For the first set of conditions, two models were examined that considered the effects of early parental death and remarriage for existing (or nearly so) conditions into which an individual was born; this model was then expanded by adding potentially mediating marital and health measures that capture potentially mediating life circumstances in adulthood. In the second set of conditions, we again examined the same early life conditions into which an individual was born, but on the subsample comprising individuals who eventually became parents. For this subsample, we considered the mediating effects of fertility as well as socioeconomic status. For this latter set of models, the potentially mediating effects of adult conditions were again considered, but now included parity and adult SES in addition to marital and health status measures in adulthood.

The sequence of models can be summarized as follows:

$$h(t|b)=h_o(t) \exp(X_1 B_1) \quad (1)$$

for early life conditions only. Here, X represents early measures in the individual's life including FEL, father's Nam-Power SES measures, mother's and father's ages at birth, mother's and father's religious affiliation, birth order, number of siblings, and then the central variables of the child's age when the mother and father died and whether they remarried. For the full sample, this model is expanded to:

$$h(t|b)=h_o(t) \exp(X_1 B_1 + X_2 B_2) \quad (2)$$

where X_2 represents adult variables measuring whether the individual ever married, age at widowhood, and the Charlson Co-Morbidity index.

These two equations are re-estimated with the subsample of individuals who became parents. What is different is an expansion of Equation (2) which now becomes:

$$h(t|b)=h_o(t) \exp(X_1 B_1 + X_2 B_2 + X_3 B_3) \quad (3)$$

where X_3 represents adult variables measuring parity and adult SES. All models were estimated using PROC Phreg in SAS v9.3.

Results

Descriptive characteristics of the sample are shown in Table 1. Several aspects of the sample are noteworthy. First, all subjects were 65 or older in 1992, so all were born in 1927 at the latest with mean birth year of 1916. Accordingly, their parents were born in the decades just before or slightly after 1900. This is important to keep in mind since the specific early life circumstances of the subjects coincided with key historical events, including the 1918 Influenza Pandemic, World War I and a period that lacked any formal programs approximating death or survivor benefits. We note that we found no evidence of specific

mortality effects associated with being born during the WWI years or during the 1918 flu pandemic.

The pattern of early parental death reflects the rising rates of adult mortality with age – about 2% of parental deaths occurred when the offspring was under age five, and the rate rose to 8% and 14% when aged 18–29 for maternal and paternal deaths, respectively. Surviving fathers had a 50% higher rate of remarriage than surviving mothers (7.7–7.9% versus 5.2–5.4%).

Female and male Cox proportional hazard models were estimated for the full sample with results shown in Tables 2 and 3, respectively. Females faced a small but elevated risk of all-cause mortality after age 65 if they experienced an early parental death. As Table 2 shows, women whose mothers died when they were ages 5–17 had the highest risks, when compared to those whose parents died when the individual was age 30 or older. When adult mediators were excluded the hazard rate ratio was 1.13 ($p < .0001$), a risk that changed minimally to 1.12 ($p < .0001$) with the addition of these mediators. For paternal deaths, the pattern was similar in that the largest significant adverse effects were detected for those who lost their fathers when they were ages 5–17, though smaller adverse effects were also found for ages 0–4 and 18–29. Again, the adult mediators made little difference in these risk patterns. Small beneficial but insignificant effects of parental remarriage were found whether the surviving parent was the mother or father. For female offspring, the fact of paternal remarriage did not contribute toward a reduction in the excess mortality risk associated with early parental death.

For men, the associations between mortality and early parental death mirror those found for females but with somewhat attenuated effects. A large significant effect was found for men whose mothers died when they were young adults (18–29) (HRR = 1.11, $p < .0001$), an association that declined slightly to 1.10 ($p < .0001$) with adjustments for adult characteristics. For paternal deaths, they too were only significant when they occurred in early adulthood though the effects were smaller (HRR = 1.05, $p < .01$ without the adult mediators and HRR = 1.04, $p < .01$ after their inclusion). These mediation results, though modest in magnitude, suggest that for men the mechanisms that connect early trauma to adult survival are partially though not substantially operating through their effects on marital and health status in later adulthood.

For both gender-specific analyses, we tested whether early parental death, when examined in combination with other early or subsequent life stressors, yielded any significant interaction effects on all-cause mortality. No significant interactions were detected (results not shown). No significant interaction effects were found for the specific interaction between an early maternal and an early paternal death. Overall, the fact or timing of early parental death did not significantly interact with other early life characteristics or subsequent mediating health and marital variables.

Before describing elaborations of the gender-specific main effects models, we briefly describe how other early life conditions altered the life chances of our sample of men and women. In many cases, these influences are similar across genders. In previous work (Smith

et al., 2009), we emphasized the unique role that familial excess longevity (FEL) played in serving as a pseudo-frailty measure when estimating survival models. In the current analysis, we again demonstrate the significant influence that FEL represents. FEL had the largest explanatory value of any covariate (note its Chi-square value in Tables 2 and 3, second only to the Charlson comorbidity index). At the same time, however, the actual effect size of being in the bottom quartile of the FEL distribution conferred a survival penalty that was similar to the penalty encountered by early parental death. With this comparison, we observe an inherited risk on par with an acquired risk. Note that the addition of controls for adult marital status and comorbidities did not attenuate the effects of FEL.

Marital status and health status comprised the two adulthood mediators we considered in Tables 2 and 3. For men and women, never marrying had a significant mortality effect in relation to those who ever married. Widowhood before age 1992 and incident widowhood after 1992 were associated with excess mortality for both men and women, with the strongest effects arising for recently widowed men. Of all the characteristics examined, the comorbidity index was the single largest predictor of mortality. An increase in the 1992 comorbidity index of one point (generally, representing one distinct chronic medical condition) represented a 34% and 26% increase in the mortality hazard rate for women and men, respectively.

Gender-specific models were re-estimated by two broad age categories (under 75 and 75+; the single-year-age Cox stratification was maintained within these two larger age groupings) in an attempt to assess general interaction effects between the younger old and the older old. Overall, the pattern of effects was similar to those from the all-age analyses, except that effect sizes were attenuated in the 75 + models (see Fig. 1).

A central developmental element of childhood and adolescence is the transition to adulthood as represented by parenthood and occupational attainment. Here we summarize the effects of early parental death and whether their influence on adult mortality is mediated by parity and adult SES, all based on the subsample of individuals who became parents (Tables 4 and 5). We report models that now include the potential mediating influence of parity and adult SES. The sample sizes decline to 29,256 for females and 26,367 for males where all sample members have complete data on all variables. The patterns observed for the full sample are largely replicated with this additional analysis suggesting that parity and adult SES are not strong mediators.

In these models, we show a small but significant protective effect of increasing parity for men but not for women. This result differs from previous work which showed an excess mortality risk with rising parity, although for a natural fertility cohort in a much earlier historical period (Smith et al., 2002). This difference in findings is likely due to the fact that those with more siblings but at lower overall levels of fertility (as is the case in the present study) enjoy more net social support; increasing numbers of siblings when family size is generally large may represent a burden to parents and create more resource competition among siblings. Finally, for both men and women, adult SES now becomes one of the strongest factors in affecting later-life mortality, even though it is not a strong mediator of the effects of early parental death.

Finally, we consider cause-specific analyses as a way to gain insight on possible mechanisms linking early adversity and privilege to later-life mortality risk. Supplemental Tables S1 and S2 report the results of the full models for the subsample of parents and include parity, marital status/widowhood measures, adult SES, and the Charlson Comorbidity Index. For both genders, the association between early parental death and mortality was largely confined to death due to cardiovascular disease. For women, the excess risks were seen for maternal and paternal deaths after age 5. For men, excess deaths from cardiovascular disease were largely associated with the death of mothers.

The effects of early parental deaths on other cause-specific mortality risks were noteworthy in three other instances. For men, mother's death in childhood elevated the risk of diabetes mortality and to a degree for women. Also, men and women whose fathers died when young are at an increased risk of death due to respiratory diseases. This is an instance where exposures that led to an early paternal death may have directly affected the mortality risks of the offspring even decades later. Included in respiratory causes of death is chronic obstructive pulmonary disease (COPD).

Discussion

Students of human aging and the life course have long called for the characterization of life course trajectories to better describe and understand aging from birth to death and within key age ranges (Hsu, 2009; Marin, Chen, & Miller, 2008). Each life comprises a trajectory beginning with a family history of health and longevity, which then unfolds with distinctive family (of origin) characteristics and exogenous shocks (e.g., early parental death) followed by transitions due to childbearing, marriage, widowhood, and health. Our focus has been to consider these effects on mortality and to assess the extent to which these early insults have persistent influences on a person's life chances.

The hypothesized adverse late-life health effects of early parental death were largely supported. For all-cause mortality assessed with the full model and the full sample for all subject ages combined, women were adversely affected by early parental death at nearly all offspring ages; for men, the more robust effects were detected for parental loss when experienced as a young adult. For both genders, the strength of these associations were greater under age 75 than over age 75.

Early parental death represents a number of potential pernicious influences including shared genetic risk that affects both the parent's and the adult child's health, loss of parental support (both emotional and financial) needed for optimal development, and the possibility that the larger environmental circumstances that led to the parents' death in the first place (including a shared event, such as a family accident) were likely encountered by the child. This may be the case for respiratory diseases where both parent (who died young) and child were plausibly exposed to the same environmental exposures. For a range of outcomes, adverse influences of early parental death have been reported (Jacobs & Bovasso, 2009; Roy, 1983; Smith et al., 2009). The effects of early parental death may have been attenuated because of mortality selection, whereby the most adversely affected children with early parental death may have died prior to age 65, leaving a healthier subset of individuals that

are less susceptible to the effects of parental loss. Also, when children are young, social interventions on the part of family and friends may attenuate the stress that would otherwise have been endured by the young child whose parent had died. This possible mechanism was not directly addressed in this study given the lack of data needed to perform a strong test of this idea.

Adolescents and young adults are vulnerable to the influences of parental death given that those are stages in life when adult development and independence are paramount. The loss of a mother generates large effects, a testament to the enduring effects of a mother's death on older offspring since mothers are most likely to provide emotional support and assistance in childrearing as the transition to adulthood occurs. Since this finding controls for whether surviving fathers remarry, the loss of historically strong maternal involvement (that is not replenished via a step-mother) when the launch into adulthood is under way appears to have lasting health effects in terms of mortality risks.

The beneficial effects of remarriage were initially observed (Table 2) but generally this was not a robust association. We had argued that parents who remarry, and whose economic fortunes are thereby likely to have improved, could provide some benefits later in the lives of their adult children. This benefit may reflect positive selection, since parents who remarry may be better off to begin with, though in the end we did not find support for the beneficial effects of remarriage. We did not yet consider whether step-parents brought their own children into the resulting blended family.

The case for the presence of early life effects on later-life mortality risks is strongest with respect to FEL. As in our previous work, FEL was found to be a powerful predictor of mortality risks in the latter decades of life (Garibotti et al., 2006; Kerber et al., 2001; Kerber, O'Brien, Smith, & Mineau, 2008; Smith et al., 2009). Our findings here demonstrate that higher values of FEL serve to protect individuals from all-cause and most cause-specific forms of mortality, after controlling for comorbidities at baseline. This association is fairly general since individuals in the lowest quartile of FEL have significantly higher mortality for almost all causes of death considered. It is worth noting that, conversely, with the exception of external causes, those in the top 25% of FEL enjoy the best survival. The other exception is cancer where we show members of the higher levels of FEL have an excess mortality risk for cancer (the association though is insignificant). This is consistent with the argument that the same mechanisms promoting longevity also promote cancer (i.e., telomerase allows cells to continue to divide, something that is good for healthy normal cells but bad for malignant cells) (Caruso, Lio, Cavallone, & Franceschi, 2004; Kenyon, 2010). We have suggested previously that an individual's family history of longevity may be the earliest of life's conditions affecting mortality risk, given that it is present from the beginning (Smith et al., 2009).

One of the surprising findings of this analysis is the relatively weak influence that comorbidities had on mediating the association between early parental death and subsequent mortality. Our interpretation of this result is that the putative stresses and challenges associated with early life trauma do not necessarily manifest themselves in terms of disease, at least not as measured using the Charlson scale at baseline. One intriguing line of inquiry

regarding this result is the possible manner in which early stress not only “gets under the skin” but “into the cell.” In previous work using the UPDB, we have shown how telomeres, the tips of chromosomes, experience a gradual loss of telomeric DNA as somatic cells divide and are now implicated in aging and apoptosis (Cawthon, Smith, O’Brien, Sivatchenko, & Kerber, 2003). This is relevant because a small but growing number of studies have shown how stress in childhood or younger adulthood contribute to telomere shortening and thus enhance aging in exposed individuals (Epel et al., 2004; Heidinger et al., 2012). Accelerated aging from telomere shortening may result in specific disease endpoints (cardiovascular disease for example) (Cawthon et al., 2003) but may also hasten the demise of an individual for reasons unassociated with a specific pathology.

The role of other hypothesized early and midlife characteristics in affecting later life mortality found support, but not universally. In general, individuals born to more socioeconomically disadvantaged fathers or younger mothers faced higher mortality risks, consistent with a vast literature linking SES with mortality as well as some growing evidence regarding adult offspring health penalties arising from having a teen mother (Jaffee, Caspi, Moffitt, Belsky, & Silva, 2001). For this sample, no evidence was detected regarding the adverse effects of being born to older parents. This is inconsistent with studies of more historic samples that have shown that older parents represent a significant mortality risk factor (Gavrilov & Gavrilova, 1997; Smith et al., 2009), a difference that may be attributable to greater variance in death ages and weaker sources of medical intervention in times past. Across models, there is evidence indicating a mortality risk associated with being a latter-born child, though the effect size is quite small (Modin, 2002; Smith et al., 2009). This has been attributed to preferential access to parental resources from first or lower order births. Results also support longstanding associations related to the benefits of religious affiliation (Mineau et al., 2004) and the adverse influences of widowhood (Alter, Dribe, & Van Poppel, 2007; Smith & Zick, 1996).

Our efforts, while novel in several aspects, were faced with some challenges that are noteworthy. Foremost among these is the fact that we relied strictly on the Charlson Comorbidity Index at baseline drawn from all Medicare claims as our measure of health status. In its defense, it is a composite of the major diseases that afflict seniors with many desirable features, such as its wide population coverage that is far less subject to response rate problems typically found in surveys. Nonetheless, our measure is based on Medicare claims data from which the disease counts were generated. Clearly other measures could be used, though obtaining annual measures from other sources spanning a decade or more for a large population may prove difficult. We also acknowledge that the Medicare claims are based on prevalent rather than incident cases. This raises questions about health status prior to 1992. Also, we were unable to examine the effects of divorce, because divorces were not as observable in the data as were marriages.

In order to construct the data file with the measures of interest, we needed to limit the sample in a number of ways, mostly requiring that we had the key family variables of interest. These restrictions likely limited the sample to families with stronger connections to Utah with more complete pedigree data. One aspect of this restriction is that the sample has stronger ties to the LDS Church. Accordingly, the effect of early parental death may have

been attenuated because of the social support potentially available to a recently bereaved child.

The parental loss aspects of the paper addressed loss due to death, not due to divorce. The UPDB, for all its advantages, does less well in capturing divorces beyond the past 20 years or so. Nonetheless, divorces that occurred when subjects were children would have been relatively rare given the likely historical period in which these would have occurred. For parental deaths, we have not yet considered the nature of the parental death to better ascertain the circumstances of the death (e.g., suicide, homicide, protracted illness).

The path from childhood to the seventh decade of life is a long one with many possible permutations. Indeed, residual biases in our findings may be present given that the permutations represented by some unmeasured confounders could not be exhaustively included. Our focus here has been to assess key early life circumstances while investigating selected midlife factors that might mediate the links spanning the beginning to the end. We have demonstrated the power of using linked records at the population level, as recommended by many (Jutte, Roos, & Brownell, 2011; Moceri et al., 2001), for the purposes of assessing the late-onset health effects of early life conditions. This design of using linked records may be the optimal approach for studying lives over such periods of time since it relies on observations and measurement that are derived objectively.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at <http://dx.doi.org/10.1016/j.socscimed.2013.11.054>

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Fig. 1.

Mortality Hazard Rate Ratios for Early Parental Death, By Gender of Offspring, Age and Gender of Parent Death Stratified by Age of Offspring. All Cox models adjust for family history of longevity, childhood SES, parental age at time of child's birth, religious participation of parent, birth order, number of siblings, remarriage of the surviving parent, individual's own marital status, adult SES, and comorbidity score in 1992.

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 1

Gender-specific descriptive statistics for all early and later-life variables.

Variable	Females		Males	
	Mean	Std Dev	Mean	Std Dev
Duration Years (Measured from 1992)	9.64	5.62	9.15	5.67
Birth Year	1915.31	7.30	1916.85	6.66
Age in 1992	76.73	7.30	75.20	6.66
Top 25 %tile of FEL	0.250	0.433	0.251	0.434
Bottom 25 %tile of FEL	0.249	0.433	0.248	0.432
Father's Nam-Power Score (/10)	4.831	1.592	4.837	1.583
Father a Farmer (=1)	0.312	0.463	0.309	0.462
Whether Father's Nam-Power Score Imputed	0.279	0.449	0.289	0.453
Mother <20 when born	0.053	0.224	0.055	0.228
Mother >=40 when born	0.082	0.275	0.081	0.273
Father <20 when born	0.020	0.139	0.021	0.143
Father >=50 when born	0.032	0.176	0.033	0.177
Mother Active LDS	0.874	0.332	0.870	0.337
Father Active LDS	0.838	0.368	0.842	0.364
Birth Order	4.087	2.776	4.050	2.726
Number of Siblings	5.938	3.482	5.767	3.388
Mother died when S 0–4	0.019	0.136	0.017	0.130
Mother died when S 5–17	0.060	0.238	0.058	0.235
Mother died when S 18–29	0.079	0.270	0.074	0.262
Father died when S 0–4	0.019	0.138	0.019	0.137
Father died when S 5–17	0.080	0.271	0.079	0.269
Father died when S 18–29	0.145	0.352	0.142	0.349
Surviving Father Remarries by S age 30	0.079	0.270	0.077	0.266
Surviving Mother Remarries by S age 30	0.052	0.222	0.054	0.225
S Not Married (=1)	0.049	0.215	0.040	0.197
Number of S Children (Parity>=1) (for parous individuals)	3.656	2.092	3.798	2.110
S Max. Nam Powers Score (for parous individuals)	58.705	20.907	60.684	20.985
S was a Farmer (=1) (for parous individuals)	0.164	0.370	0.143	0.350
S Widowed <30 (=1)	0.012	0.107	0.004	0.064
S Widowed Between 30 and 44 (=1)	0.029	0.169	0.011	0.102
S Widowed Between Age 45 and Year 1992 (=1)	0.347	0.476	0.117	0.321
S Charlson Comorbidity Index in 1992	0.608	1.118	0.757	1.307

Table 2

Female mortality hazard rate ratios (HRR's) for early parental death based on Cox proportional hazards regressions. Models adjust for early conditions only and adding later life conditions, $N = 50,687$.

Parameter	HRR	P	χ^2	HRR	P	χ^2
Top 25 %tile of FEL	0.857	<0.0001	176.4582	0.866	<0.0001	138.3221
Bottom 25 %tile of FEL	1.187	<0.0001	210.6807	1.167	<0.0001	157.5539
Father's Nam-Power Score (/10)	0.990	0.0018	9.7503	0.992	0.0158	5.8210
Father a Farmer (=1)	0.944	<0.0001	22.9945	0.950	<0.0001	17.1318
Whether Father's Nam-Power Score Imputed	0.972	0.0181	5.5854	0.982	0.1429	2.1467
Mother <20 when born	1.078	0.0030	8.7878	1.075	0.0065	7.4049
Mother >=40 when born	0.985	0.4499	0.5709	0.990	0.6258	0.2378
Father <20 when born	1.083	0.1547	2.0248	1.049	0.4224	0.6437
Father >=50 when born	0.995	0.8432	0.0391	0.995	0.8655	0.0287
Mother Active LDS	0.964	0.0251	5.0138	0.962	0.0202	5.3960
Father Active LDS	0.968	0.0237	5.1178	0.974	0.0743	3.1863
Birth Order	1.005	0.0471	3.9431	1.006	0.0237	5.1184
Number of Siblings	1.000	0.8496	0.0360	0.999	0.4548	0.5586
Mother died when S 0-4	1.080	0.0444	4.0417	1.059	0.1346	2.2389
Mother died when S 5-17	1.131	<0.0001	25.7954	1.121	<0.0001	21.5527
Mother died when S 18-29	1.057	0.0053	7.7630	1.043	0.0390	4.2591
Father died when S 0-4	1.082	0.0278	4.8390	1.082	0.0339	4.5012
Father died when S 5-17	1.099	<0.0001	23.9955	1.087	<0.0001	16.8898
Father died when S 18-29	1.055	0.0002	14.1920	1.053	0.0004	12.6473
Surviving Father Remarries by S age 30	0.949	0.0311	4.6470	0.957	0.0777	3.1130
Surviving Mother Remarries by S age 30	0.963	0.1152	2.4820	0.956	0.0864	2.9400
Not Married (=1)				1.319	<0.0001	86.5038
Widowed <30 (=1)				1.074	0.0940	2.8052
Widowed Between 30 and 44 (=1)				1.095	0.0007	11.4065
Widowed Between Age 45 and Year 1992 (=1)				1.032	0.0077	7.0994
Widowed after 1992 (Time-varying) (=1)				1.040	0.0067	7.3460
Charlson Comorbidity Index in 1992				1.335	<0.0001	1990.7229

Parameter	HRR	P	χ^2	HRR	P	χ^2
Model χ^2 , df, p-value	$\chi^2 = 732.1$, df = 21, $p < .0001$ $\chi^2 = 4696.9$, df = 27, $p < .0001$					

‘S’ = subject.

Table 3

Male mortality hazard rate ratios (HRR's) for early parental death based on Cox proportional hazards regressions. Models adjust for early conditions only and adding later life conditions, $N = 41,931$.

Parameter	HRR	P	χ^2	HRR	P	χ^2
Top 25 %tile of FEL	0.840	<0.0001	192.6898	0.850	<0.0001	154.5767
Bottom 25 %tile of FEL	1.160	<0.0001	131.2480	1.141	<0.0001	97.7693
Father's Nam-Power Score (/10)	0.990	0.0029	8.8391	0.992	0.0204	5.3808
Father a Farmer (=1)	0.970	0.0223	5.2233	0.973	0.0428	4.1044
Whether Father's Nam-Power Score Imputed	0.989	0.4112	0.6753	0.987	0.3351	0.9292
Mother <20 when born	1.064	0.0180	5.5960	1.048	0.0824	3.0163
Mother >=40 when born	0.974	0.2151	1.5367	0.986	0.5084	0.4374
Father <20 when born	1.030	0.5858	0.2970	1.021	0.6961	0.1526
Father >=50 when born	1.020	0.5099	0.4343	0.992	0.8019	0.0629
Mother Active LDS	0.947	0.0014	10.2354	0.940	0.0004	12.7662
Father Active LDS	0.977	0.1305	2.2863	0.977	0.1355	2.2278
Birth Order	1.010	<0.0001	15.3889	1.011	<0.0001	15.6470
Number of Siblings	0.998	0.3927	0.7305	1.000	0.8574	0.0323
Mother died when S 0-4	1.041	0.3349	0.9297	1.004	0.9360	0.0064
Mother died when S 5-17	1.078	0.0044	8.1024	1.045	0.1009	2.6910
Mother died when S 18-29	1.113	<0.0001	23.2136	1.096	<0.0001	15.5530
Father died when S 0-4	1.034	0.3862	0.7509	1.047	0.2291	1.4464
Father died when S 5-17	1.036	0.0924	2.8320	1.039	0.0836	2.9936
Father died when S 18-29	1.051	0.0012	10.4900	1.043	0.0089	6.8344
Surviving Father Remarries by S age 30	0.969	0.2440	1.3572	0.983	0.5386	0.3782
Surviving Mother Remarries by S age 30	1.044	0.0971	2.7521	1.043	0.1203	2.4138
Not Married (=1)				1.376	<0.0001	120.3273
Widowed <30 (=1)				1.104	0.1597	1.9768
Widowed Between 30 and 44 (=1)				1.080	0.0986	2.7288
Widowed Between Age 45 and Year 1992 (=1)				1.062	0.0002	14.3604
Widowed after 1992 (Time-varying) (=1)				1.099	<0.0001	37.6062
Charlson Comorbidity Index in 1992				1.261	<0.0001	1893.0117

Parameter	HRR	P	χ^2	HRR	P	χ^2
Model χ^2 , df, p-value	$\chi^2 = 623.3$, df = 21, $p < .0001$		$\chi^2 = 3525.5$, df = 27, $p < .0001$			

‘S’ = subject.

Table 4

Female mortality hazard rate ratios (HRR's) for early parental death based on Cox proportional hazards regressions. Models adjust for early conditions only and adding later life conditions that includes parity and adult SES. Restricted to Parous individuals, $N = 29,256$.

Parameter	HRR	P	χ^2	HRR	P	χ^2
Top 25 %tile of FEL	0.839	<0.0001	124.3380	0.856	<0.0001	86.8369
Bottom 25 %tile of FEL	1.186	<0.0001	118.8321	1.163	<0.0001	84.8704
Father's Nam-Power Score (/10)	0.986	0.0006	11.8952	0.992	0.0650	3.4055
Father a Farmer (=1)	0.936	<0.0001	18.3696	0.935	<0.0001	16.6664
Whether Father's Nam-Power Score Imputed	0.975	0.1651	1.9272	0.975	0.1940	1.6870
Mother <20 when born	1.105	0.0025	9.1481	1.099	0.0065	7.3933
Mother >=40 when born	0.964	0.1758	1.8328	0.985	0.5895	0.2911
Father <20 when born	1.076	0.3223	0.9797	1.050	0.5713	0.3205
Father >=50 when born	0.989	0.7764	0.0807	0.979	0.6149	0.2530
Mother Active LDS	0.980	0.4070	0.6875	0.984	0.5394	0.3767
Father Active LDS	0.964	0.0744	3.1839	0.970	0.1577	1.9960
Birth Order	1.006	0.0728	3.2181	1.005	0.1591	1.9824
Number of Siblings	1.002	0.4802	0.4983	0.998	0.4861	0.4852
Mother died when S 0-4	1.120	0.0310	4.6526	1.066	0.2409	1.3755
Mother died when S 5-17	1.144	<0.0001	15.4492	1.123	0.0010	10.8917
Mother died when S 18-29	1.073	0.0107	6.5170	1.050	0.0949	2.7896
Father died when S 0-4	1.091	0.0659	3.3829	1.110	0.0320	4.5975
Father died when S 5-17	1.131	<0.0001	22.8021	1.107	0.0004	12.4331
Father died when S 18-29	1.052	0.0094	6.7499	1.048	0.0204	5.3802
Surviving Father Remarries by S age 30	0.953	0.1544	2.0286	0.978	0.5351	0.3848
Surviving Mother Remarries by S age 30	0.968	0.3007	1.0710	0.965	0.3162	1.0048
Not Married (=1)				1.311	0.6640	0.1886
Number of Children				0.995	0.1037	2.6471
S Max. Nam Powers Score				0.998	<0.0001	47.0182
S was a Farmer (=1)				0.950	0.0071	7.2443
Widowed <30 (=1)				1.141	0.0243	5.0756
Widowed Between 30 and 44 (=1)				1.161	<0.0001	17.1973

Parameter	HRR	P	χ^2	HRR	P	χ^2
Widowed Between Age 45 and Year 1992 (=1)				1.084	<0.0001	23.1702
Widowed after 1992 (Time-varying) (=1)				1.083	<0.0001	18.8514
Charlson Comorbidity Index in 1992				1.373	<0.0001	1105.4605
Model χ^2 , df, p-value	$\chi^2 = 472.5$, df = 21, $p < .0001$ $\chi^2 = 3039.3$, df = 30, $p < .0001$					

‘S’ = subject.

Table 5

Female mortality hazard rate ratios (HRR's) for early parental death based on Cox proportional hazards regressions. Models adjust for early conditions only and adding later life conditions that includes parity and adult SES. Restricted to Parous individuals, $N = 26,367$.

Parameter	HRR	P	χ^2	HRR	P	χ^2
Top 25 %tile of FEL	0.829	<0.0001	133.7452	0.844	<0.0001	101.5245
Bottom 25 %tile of FEL	1.152	<0.0001	76.5325	1.121	<0.0001	45.7707
Father's Nam-Power Score (/10)	0.988	0.0026	9.0890	0.995	0.2873	1.1323
Father a Farmer (=1)	0.968	0.0348	4.4552	0.964	0.0285	4.7989
Whether Father's Nam-Power Score Imputed	1.020	0.3073	1.0424	1.025	0.2256	1.4681
Mother <20 when born	1.064	0.0601	3.5334	1.042	0.2303	1.4388
Mother >=40 when born	0.952	0.0633	3.4495	0.960	0.1308	2.2826
Father <20 when born	1.042	0.5483	0.3604	1.045	0.5100	0.4342
Father >=50 when born	0.977	0.5542	0.3498	0.945	0.1776	1.8179
Mother Active LDS	0.936	0.0070	7.2625	0.919	0.0006	11.6572
Father Active LDS	0.990	0.6280	0.2347	0.986	0.5132	0.4275
Birth Order	1.011	0.0009	10.9926	1.014	0.0001	14.7386
Number of Siblings	0.997	0.2249	1.4729	0.996	0.1076	2.5893
Mother died when S 0-4	1.080	0.1668	1.9117	1.060	0.3056	1.0496
Mother died when S 5-17	1.080	0.0281	4.8198	1.038	0.3117	1.0236
Mother died when S 18-29	1.120	<0.0001	15.2523	1.107	0.0008	11.3525
Father died when S 0-4	1.000	0.9921	0.0001	1.027	0.5889	0.2921
Father died when S 5-17	1.024	0.3918	0.7333	1.040	0.1619	1.9559
Father died when S 18-29	1.059	0.0036	8.4668	1.065	0.0018	9.7142
Surviving Father Remarries by S age 30	0.954	0.1744	1.8450	0.957	0.2336	1.4187
Surviving Mother Remarries by S age 30	1.042	0.2118	1.5587	1.022	0.5126	0.4288
Not Married (=1)				2.928	0.0235	5.1282
Number of Children				0.990	0.0013	10.3433
S Max. Nam Powers Score				0.996	<0.0001	93.7160
S was a Farmer (=1)				0.946	0.0072	7.2227
Widowed <30 (=1)				1.159	0.0929	2.8227
Widowed Between 30 and 44 (=1)				1.139	0.0147	5.9488

Parameter	HRR	P	χ^2	HRR	P	χ^2
Widowed Between Age 45 and Year 1992 (=1)				1.066	0.0017	9.8776
Widowed after 1992 (Time-varying) (=1)				1.120	<0.0001	38.2222
Charlson Comorbidity Index in 1992				1.278	<0.0001	1219.5019
Model χ^2 , df, p-value	$\chi^2 = 403.1$, df = 21, $p < .0001$ $\chi^2 = 2357.1$, df = 30, $p < .0001$					

‘S’ = subject.