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Effects of Childhood and Middle-Adulthood Family Conditions on Later-Life Mortality: Evidence from the Utah Population Database, 1850-2002

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Abstract

We examine how key early family circumstances affect mortality risks decades later. Early life conditions are measured by parental mortality, parental fertility (e.g., offspring sibship size, parental age at offspring birth), religious upbringing, and parental socioeconomic status. Prior to these early life conditions are familial and genetic factors that affect life-span. Accordingly, we consider the role of parental and familial longevity on adult mortality risks. We analyze the large Utah Population Database which contains a vast amount of genealogical and other vital/health data that contain full life histories of individuals and hundreds of their relatives. To control for unobserved heterogeneity, we analyze sib-pair data for 12,000 sib-pairs using frailty models. We found modest effects of key childhood conditions (birth order, sibship size, parental religiosity, parental SES, and parental death in childhood). Our measures of familial aggregation of longevity were large and suggest an alternative view of early life conditions.

Keywords

UAS; early-life conditions; mortality; socioeconomic status (SES); frailty; life course; middle adulthood conditions

Introduction

How do parents affect the health and longevity of their children? Parents can affect their children's life chances by transmitting a genetic endowment (or liability) for a long life while also providing resources and an environment that enhances (or limits) their children's

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longevity. Recently, more attention has been given to the role that very early conditions (including *in utero*) of childhood have on adult health outcomes across a range of countries, cultures, and historical periods (Barker, 1999; Elo & Preston, 1992; Kuh & Yoav, 1997). These and other investigators have been raising a fundamental question about human aging and whether the risk of mortality in the latter half of life is already “scripted” based on conditions arising during infancy, childhood, and adolescence.

The purpose of this study is to examine the effects of a full range of important family and individual circumstances present in early and mid-life on mortality risks later in life. We give attention to parents and how their life span, either as an indicator of genetic predisposition for longevity or as a measure of support for their children, affects adult offspring mortality. Parents dying prematurely may do so at crucial moments in a child's life producing potentially lasting health effects. Accordingly, we also consider mortality influences of parental death when offspring were still children. We explore not only the role of parental longevity on offspring survival but also the effects associated with the longevity patterns of all known blood relatives.

Parents affect opportunities faced by their children that may have lasting consequences. Specifically, parental fertility can create various family structures for their offspring. Factors we consider are the individual's sibship size and birth order. Along with these early family formation factors, we also examine parental ages at the time of the individual's birth. Some studies have considered each of these early characteristics in isolation with an emphasis on a range of adult outcomes such as personality traits, mental health, and status/educational attainment. As far as we have determined, it is not known how these fundamental family characteristics in childhood affect adult mortality.

The lasting effects of childhood socioeconomic circumstances provided by parents are of potentially great importance. We have created a sample in which we are able to consider both the effects of parental SES and simultaneously the role of religion in childhood (and the social integration and lifestyle effects it represents) and their influence on adult mortality.

While this analysis is aimed at childhood family conditions, we consider extensions of existing work on the impact that fertility, religion, and socioeconomic status as an adult has on the adult's own mortality past age 50 (Smith, Mineau, & Bean, 2002). Our intent here is to assess how the effects of childhood life conditions compare to the influences of adulthood circumstances on post-reproductive longevity.

Childhood is a complex stage in an individual's life where numerous biodemographic factors arise that could affect later-life adult mortality. To adjust for familial factors that are not observable, we exploit data on sib-pairs that allow us to adjust for shared and correlated unobservable features of the family environment.

The factors contributing to early life conditions are manifold. Here we describe the key characteristics suggested by the literature and that form the basis for the empirical analysis undertaken in this study.

Familiality of Longevity

Familial aggregation of longevity has been examined extensively across numerous disciplines (Abbott, Abbey, Bolling, & Murphy, 1978; Bocquet-Appel & Jakobi, 1990; Carmelli, Swan, Page, & Christian, 1995; McGue, Vaupel, Holm, & Harvald, 1993; Philippe, 1978; Vaupel, 1988). Although these estimates vary within and between populations, they are normally derived from familial correlations and are therefore elevated by shared non-genetic factors. These factors (e.g., nutrition, lifestyle) may link parental and offspring longevity together.

Parental Death in Childhood

For dependent children, the death of a parent may have adverse effects later in life (Andersson, 1996; Meza, 1991; Umberson, 1994). Younger children in these families are, therefore, likely to experience deprivations (e.g., the loss of social and economic support) such as those encountered by the surviving parent. Studies of historical populations show how important parental death was for survival of children (van Poppel, 2000).

Number of Siblings

Sibship size has been found to be positively associated with lower educational achievement and unhealthy lifestyle choices (Byrne, Agerbo, Ewald, Eaton, & Bo Mortensen, 2003; Downey, 1995; Hart & Smith, 2003; Modin, 2002; Taubman & Behrman, 1986). Little literature exists on adult mortality in relation to sibship size. However, children from large families in historical populations and in developing countries experience excess infant and childhood mortality rates (Bean, Mineau, & Anderton, 1990; Haines, 1998; Knodel & Hermanlin, 1984). Children from large sibships may have a greater risk of contracting an infectious disease, due to crowding, which can influence adult health (Hart & Smith, 2003). Alternatively, others have argued that children who survive widespread infectious diseases are strengthened and go on to live longer lives (Meindl, 1982). The resource dilution model (Downey, 1995; Guo & VanWey, 1999; Marjoribanks, 2001; Modin, 2002) argues that parents have finite levels of resources, and more offspring means a greater dilution of resources.

Birth Order

Few studies have examined birth order and its influence on longevity. One study found that later-born girls have higher mortality risks than firstborn sisters (Modin, 2002). First-born children may benefit from more parental time and resources. First-born children are over-represented in college populations (Altus, 1966) and reach higher levels of educational and occupational achievement than their siblings (Blake, 1989; Davis, 1997; Modin, 2002; Sullo way, 1997; Travis & Kohli, 1995).

Parental Age

Associations have been found between parental age and Down syndrome, birth defects, and schizophrenia, and longevity (Byrne et al., 2003; Gavrilov & Gavrilova, 1997; Priest, Mackowiak, & Promislow, 2002). Older mothers have older ova that give rise to more birth defects such as Down Syndrome (Muller, Rebiffe, Taillandier, Oury, & Mornet, 2000). Still

others contend that longevity is affected by the number of mutations accumulated in germ line cells (Gavrilov & Gavrilova, 2003; Gavrilov & Gavrilova, 2004). Priest et al conclude that both maternal and paternal age influences offspring's mortality and that maternal age affects daughters more, whereas, paternal age affects/influences sons more (Priest et al., 2002). Given that daughters inherit the paternal X-chromosome and sons do not, daughters may be more adversely affected if born to older fathers.

Parental age may affect offspring longevity for social reasons as well. Children born to older parents enjoy higher educational/occupational attainment (Mare & Tzeng, 1989). Older parents are more likely to have greater socioeconomic resources although they share fewer years with their children than other parents. The adverse effects of teenage parenthood in terms of socioeconomic outcomes and mental health characteristics have been demonstrated (Furstenberg, 1976; Moore & Waite, 1981).

Socioeconomic Status (SES) in Childhood and Adulthood

SES is positively associated with longevity (Kitagawa & Hauser, 1973) but there is some debate regarding the age at which SES matters. Several studies have explored this topic but find differing results with respect to the strength of the association between SES in childhood and later life health (Hayward & Gorman, 2004; Lynch, 1994; Melchior, Moffitt, Milne, Poulton, & Caspi, 2007; Osler, Andersen, Due, Lund, Damsgaard, & Holstein, 2003; Peck, 1992; Schwartz, 1995)

Own Fertility

While high parity women experience excess post-reproductive mortality (Doblhammer, 2000; Smith et al., 2002), women bearing children after age 45 in natural fertility conditions are possibly aging more slowly than women who are unable to bear children at the same advanced ages. Recent studies have shown that late fertile women have lower rates of adult mortality (Perls, Alpert, & Fretts, 1997; Smith et al., 2002)).

Materials and Methods

Data

The analyses are based on the Utah Population Database (UPDB), one of the world's most comprehensive computerized genealogies. In the mid-1970s, over 170,000 three-generation families were identified on "Family Group Sheets" from the archives at the Utah Family History Library, each with at least one member having had a vital event (birth, marriage, death) on the Mormon Pioneer Trail or in Utah. These families have been linked into multigenerational families. The genealogy provides data on migrants to Utah and their Utah descendants for more than 1.6 million individuals born from the early 1800s to the mid-1970's. The UPDB includes individuals who have lived in other states and countries and describes families with and without an affiliation to the Church of Jesus Christ of Latter-day Saints (LDS or Mormons). New families and their members are continually being added as the UPDB is linked to other sources of data, including Utah birth and death certificates. Because these records include basic demographic information on parents and their children, fertility and mortality data are extensive with coverage up to the present.

We analyze individuals (egos) from sibships born between 1850 and 1900. This period is used because parents of these egos lived during a time when effective modern contraceptive methods were nonexistent. Accordingly, their family formation patterns reflect natural fertility conditions. It was during this era that the elderly of the 20th century were born and their mortality risks may have been shaped in ways based on the circumstances of their childhood.

We identified parents of egos who completed their childbearing during the 50 year interval spanning 1850 and 1900. It is the mortality experiences of the egos that are the focus of our attention. To assess factors that may affect the later-life mortality of these egos, we rely on information about the egos themselves, their parents, and their children. We have therefore identified a set of three generational pedigrees from which we will examine the mortality patterns past age 50 for the middle generation (egos).

In an effort to adjust our analysis for unobserved heterogeneity, we have selected same-sex sibling pairs. Sibships that had at least two brothers or two sisters are represented in the sample, provided both lived to age 50. In some cases, where there are (at least) two brothers and two sisters, there will be a total of four siblings from the same family included in our sample. This means that we exclude families with only-children or sibships where a single ego son or daughter is present. This latter restriction eliminated less than 10% of all sibships.

To maximize differences in how siblings experience their childhood familial environment, we select first-born/last-born brother pairs and first-born/last-born sister pairs. This sampling strategy means, for example, that the first born son (who could have been the third born child) is compared to the last born son (who could have later-born sisters). Our survival models allow for either shared or correlated frailty as developed by Therneau and colleagues (Therneau & Grambsch, 2000). This approach has been used successfully in an analysis of correlated and shared frailty in multigenerational pedigrees (Garibotti, Smith, Kerber, & Boucher, 2006).

Both individuals in a sib pair are required to survive to age 50 and were ever-married. The very small fraction of egos who reach adulthood (age 20) who never married by age 50 are excluded. Egos born from 1850 to 1900 that survived to age 50 included 12,366 sons (6,184 brother pairs) and 11,896 daughters (5,948 sister pairs).

Measures

Mortality—The outcome is the all-cause mortality hazard rate starting at exact age 50. In this sample, the mean age at death for males is 73.3 and 76.2 for females.

Parental Longevity—Mothers and fathers age at death have been categorized into five groups each: died before the 75th percentile (father died<81, mother died<82) reference category, died between the 75th and 90th percentile (father 82 died<87, mother 82 died<88), died between the 90th to 95th percentile (father died 87 died<90, mother 88 died<91), died between the 95th and 99th percentile (father died 91 died<94, mother 91 died<96), and equal to or after the 99th percentile (father 94, mother 96).

Familial Excess Longevity (FEL)—One of the earliest of conditions is a familial or genetic propensity for a long or short life based upon a family history of longevity. We incorporate a previously developed measure, Familial Excess Longevity (FEL) (Kerber, O'Brien, Smith, & Cawthon, 2001), to represent this predisposition. At its foundation, the FEL is based on the assumption that family history of longevity follows Mendelian patterns of inheritance. To construct familial excess longevity we first measure individual level excess longevity, defined as the difference between an individual's attained age and the age to which that individual was expected to live according to a model that incorporates basic predictors (gender, birth year). Expected longevity ($\hat{y}$) is estimated from an accelerated failure time (AFT) model and excess longevity (l) is $y - \hat{y}$, where y is the attained age in years (either at death or at the time last confirmed the relative was alive; the estimate of FEL does not change materially when the small percentage of kin born before 1900 (<1%) who were censored are excluded). Expected longevity ($\hat{y}$) is based on the lognormal distribution and the AFT model was used because it provides a simple point estimate for duration and fit the observed data (Kerber et al., 2001). Excess longevity is then extended to blood relatives who reached the age of 65 for each individual. Averaging the excess longevity of all kin over 65 for each ego, with the appropriate weighting scheme, generates a point estimate of familial excess longevity. The kinship coefficient, the probability that an individual shares a particular allele with another individual, is used as a weight in calculating familial (Mendelian) excess longevity (FEL) (Kerber et al., 2001):

$$FEL_i = \frac{\sum_{k \in K} f(i, k) \cdot l_k}{\sum_k f(i, k)},$$

where FEL_i is the familial (Mendelian) excess longevity for subject i , K is the set of all blood relatives of subject i living to age 65, l_k is the excess longevity of the k^{th} member of K , and $f(i, k)$ is the kinship coefficient. On average, persons born prior to 1900 had 233 kin who lived to age 65 on whom their FEL was based.

Only 17 individuals (of over 24,000) did not have sufficient data on their kin to estimate FEL. A dummy variable identifies these individuals who are then assigned the sample-wide mean value of FEL.

Sibship Size: We have observed a small but positive association between number of siblings and mortality risk and that this association was largely attributable to whether ego had only one sibling or not (among all the siblings born). We therefore use a dummy variable that captures this simplified version of sibship size. Recall that our sample comprises sib pairs so there are no only-children “sibships.”

Birth Order: Birth order is naturally affected by sibship size. To account for birth order effects, we use a dummy variable that specifies whether an ego in a sib pair was the first born in the pair or not. When sibship size is controlled for, those coded zero on this dummy will be higher order births in absolute terms. Preliminary analyses using an interval-level version of birth order yielded similar results.

Parental Age at Child's Birth: Parental age when an ego was born is measured separately for mothers and fathers. For mothers, four age categories are used: under 20, 20-29 (reference category), 30-34, and 35 or older. For fathers, more categories were used for age at birth: under 20, 20-29 (reference category), 30-39, 40-49, 50-69, and 70 or older.

Parental Age at Death during Ego's Childhood: Four parental categories comprise the parental mortality variable (assessed at ego's age 20) that yields the following dummy variables: both parents were living (reference category), only father was alive, only mother was alive, and neither parent was alive. We tested the hypothesis that the mortality effects of parental death were the same across age categories (<5, 6-10, 11-20) under age 20 for the offspring and all tests were insignificant.

Religion in Childhood: The religious upbringing of a child may affect that child's later life health. For members of the LDS Church, children are exposed to certain behavioral patterns (e.g., proscription from alcohol and tobacco, social integration) that may yield benefits later in life (Mineau, Smith, & Bean, 2002). The UPDB contains dates of baptism for all egos, usually at age eight and later for converts. When an ego was baptized as a child before age 18 within the LDS Church, we treat this as an indication of being raised as a child in a Mormon household. Persons baptized at older ages, posthumously, or never baptized are presumed not to have been raised in an LDS household.

Father's SES: For fathers who died in Utah and for whom we obtained a Utah death certificate, we capture their usual industry and occupation from a death certificate. This is possible because we have access to all death certificates that have been issued in the state of Utah since 1904. Industry and occupation data have been converted to the Nam-Powers socioeconomic index (Nam, 1983). Nam-Power scores range from 1 to 99 and are based on 1950 census data (the midpoint of the century for which we have data) on income and educational level by occupation and are assigned for each occupation that has a U.S. Census code. Higher scores are associated with higher SES. Approximately 18 percent of fathers in the sample have a death date but did not link to a 1904-2002 Utah death certificate. These individuals are identified by a dummy variable and are assigned the group mean for the Nam-Powers index.

Own Fertility: Ego's fertility is assessed by parity and age at last birth. Age at first birth and first marriage were not strong predictors of post-reproductive mortality and are not introduced here. The majority of egos have fertility information; however, approximately one-third of the sample has some piece of fertility information missing and for these individuals we have constructed a set of dummy variables. Categories for parity are 1-2 (reference group), 3-5, 6-8, 9-11, 12 or higher, and some fertility information missing. For age at last birth, we constructed four age categories: under 35 (reference group), 35 to 44, 45 or older, and some fertility information missing.

Own Religion: The UPDB contains dates of baptism but also dates of endowment. Individuals with an endowment date are adult Mormons who enter into a covenant with God to conduct their lives guided by the doctrine of the LDS Church. In general, individuals who have endowment dates are considerably more likely to abstain from tobacco and alcohol as

well as participate actively in church activities. Persons with an endowment date prior to age 40 are treated as devout members of the LDS Church.

Male SES: Ego's usual industry and occupation are obtained from death certificates and are again converted to Nam-Powers scores. Approximately 38 percent of egos had death dates but did not link to a Utah death certificate, reflecting residence outside of Utah. Egos lacking a Utah death are identified by a dummy variable and are assigned the group mean for the Nam-Powers index. Women's SES was measured via her father's SES since women's SES was generally missing or listed as a housewife on death certificates.

Methods

Cox proportional hazards models (PHM) are used to model survival time between age 50 and death. The sample comprises an extinct cohort of individuals so all have death dates. We estimated four types of survival models. The first is a “naïve” model in which we estimate a Cox PHM that ignores the clustered sib-pair data; this model is provided as it is what is mostly commonly used model for survival analyses:

$$h_i(a) = h_o(a) e^{X_i \beta}$$

Where $h(a)$ is the hazard rate, i indexes individuals, a measures age, X are observed covariates, and β are regression parameters.

The second model extends the Cox PHM by taking into account the fact that siblings in a family are statistically dependent. This is done by modeling robust variances of the regression parameters but which generates the same regression parameters as the naïve model.

The third method models the Cox PHM by allowing for shared frailty and robust variances of the regression parameters (Therneau & Grambsch, 2000). This specification allows us to estimate the degree to which siblings are correlated and provides regression parameter estimates that make the paired observations conditionally independent after adjusting for their shared frailty. Frailty, f , is a Gamma distributed random variable:

$$h_{ij}(a) = h_o(a) e^{X_i \beta + f_j}$$

where j indexes sibships.

Finally, we provide estimates of the Cox PHM that assumes that the association between siblings' hazard rate for mortality is genetic in origin. This approach constrains the covariance between two siblings' frailty to be 0.50, reflecting the fact that on average they share half their genes with each other:

$$h_i(a) = h_o(a) e^{X_i \beta + f_i}$$

$$f \sim N(0, \sigma^2 K),$$

where K is a kinship matrix. One random effect per subject i was considered, with covariance matrix $\sigma^2 K$, where K is a matrix with ones on the diagonal, and 0.5 entries for siblings and zeros for non-siblings in off-diagonal cells.

Results

The descriptive statistics for the brother-pair and sister pair samples are shown in Table 1. The mean for FEL is 2.98 years which means that egos have blood relatives who live approximately three years longer than expected. This feature of the data is a function of the survival selection of the sample (e.g., sib pairs where both survived to age 50). We show that males and females have similar levels of parity but that the ages at last birth differ substantially. Approximately 17 percent of males fathered children after age 45, much higher than the 3.2 percent for females. For males who father children past age 50 (our survival threshold), we do not report results on the effects of age at last birth and parity on male survival for the *full sample* but provide instead results for males who completed their fertility by age 50. With respect to SES estimates, we are able to generate a Nam-Power score for 83% of the fathers of egos and 62% of male egos.

The effects of early life conditions on later-life mortality, from both genetic and socio-environmental sources, based on the fully-adjusted model are shown for males and females in Tables 2 and 3 respectively. We focus on the results from the basic Cox proportional hazards models with robust variances but describe important differences with the results generated from the frailty-based extensions to the Cox model.

Role of Parental and Familial Longevity

For both males and females, paternal and maternal longevity have strong positive associations with ego survival. Each parent contributes to the longevity of their offspring with the longest lived parents providing the greatest survival benefit. Certainly a portion of the association between parent and offspring longevity is “environmental” in origin, some elements of which have been controlled for in the model based on the inclusion of observable social, economic, and biologic factors that existed during egos' childhood.

The effect of FEL on ego survival is quite large for both males and females and its influence is stronger than the effects of having an exceptionally long-lived parent. FEL measures the influence that the longevity of blood kin has on ego survival but a large portion of this influence comes from relatives living in socio-environmental conditions that are not necessarily shared with ego. Our observation is that FEL is the single strongest predictor of ego survival but the association is based less on a shared environment argument than the specific association between parental and offspring longevity. Indeed, once we adjust for

FEL, the effect of parental longevity on ego mortality should reflect more of the social influences that long-lived parents have on their adult offspring's survival. When ego was a child, parents who later turn out to be longevous were alive and available for ego, were more apt to be reasonably healthy, and were able to provide assistance in several key ways.

We also consider FEL to be a candidate for being an *observable* indicator of frailty. This observation is based on a comparison between Cox models (with our full set of covariates) that incorporate frailty but exclude FEL and models that incorporate both frailty and FEL. When FEL is excluded, we find significant frailty effects: for shared frailty, the gamma variance is 0.127 ($p < .001$) and 0.091 ($p < .001$) for males and females, respectively. For the correlated frailty model, the male and female variances are 0.277 ($p < .001$) and 0.202 ($p < .001$), respectively. These results suggest that there are factors that contribute to a shared or correlated excess risk of mortality among siblings. When FEL is added to these models, FEL becomes the strongest predictor of mortality and shared frailty effects are insignificant and the effects of correlated frailty are reduced by 35-40% (down to 0.184 for males, 0.127 for females, both $p < .001$; see Appendices A and B). This finding suggests that whatever factors link siblings' survival, an important component is the familiarity of longevity within their extended family.

Childhood Family Conditions

Female Survival—Childhood conditions affecting later-life mortality vary by gender. For women, there are few childhood conditions that generate substantial shifts in their adult mortality risk. Their birth order has no effect on their survival (see O'Leary, Wingard, Edelstein, Criqui, Tucker, & Friedman, 1996) but their sibship size does: girls raised in two-child households have a small ($RR=1.068$) but significant excess risk of adult mortality in relation to girls with more than one sibling.

Parental ages at birth and parental vital status also have no clear effect on female survival with one exception. Exceptionally old fathers (over age 70) are associated with daughters whose mortality rate is 40% higher than daughters born to fathers in their twenties ($p < .05$).

Women baptized in the LDS Church and women with higher SES fathers experience modest survival benefits but the effects are small with weak statistical significance ($.05 \leq p \leq .12$). These results indicate that women experience some enduring benefits from these characteristics but they are minor in relation to the effects of parental and familial longevity.

We explored a range of models that make adjustments for the presence of correlated survival among siblings and the introduction of shared and correlated frailty. Given the covariates in the model, particularly FEL, we find no significant changes to our results when we do or do not consider the potential correlation in survival between siblings, a result that holds for both brothers and sisters.

Male Survival—For the full sample of brother pairs, we find no impact of sibship size or birth order on male late-adult mortality. Male survival is sensitive to maternal age at birth but not paternal age. Boys born to very young (under age 20) and older (age 35 or older) mothers have significantly higher mortality than comparison males with maternal ages of

20-29 years. The effects are again small (RR=1.06 for being born to a young mother and RR=1.08 for being born to having an older mother).

Losing a parent to death in childhood (under age 20) was considered by examining separate survival effects of losing a father only, a mother only, or both in relation to egos whose parents were both alive when ego was 20. Orphans do not experience significant later-life mortality risks but, interestingly, loss of one parent to death is associated with *lower* later-life mortality for males although this effect is small. The impact of parental mortality is only significant in cases where the father dies when ego was a child. This effect is present over a range of ages at the time of a father's death (ego was less than age 18, less than age 15, less than age 10). Given that paternal mortality is associated with excess childhood mortality (under age 20, results not shown) and younger adult mortality (ages 20-50, results not shown), it is possible that children reaching age 50 are a select subset of egos that are more robust and have adapted in ways that confer at least a small survival advantage in later adulthood. For both males and females, the effects of early parental on mortality risk after age 50 are quite similar whether parental longevity (given its association with early parental death) is included or not.

Effects of Fertility, Socioeconomic Status, and Religion in Adulthood

Fertility—Past age fifty, female mortality is significantly affected by their fertility behavior. Women with fewer children and those able to bear children later in life have better survival than high parity women and those completing their childbearing at younger ages. Women with large family sizes (12 or more children) have significantly elevated mortality risks than women bearing 1 to 2 children (RR=1.16). (If women were lacking some fertility information, their mortality risks increased perhaps reflecting a weaker commitment to the LDS church - that is not captured by our more direct measure of religious affiliation - since active LDS members are more vigilant in maintaining complete family genealogies.) Conversely, women whose age at last birth was after age 45 had lower mortality risks in relation to women whose last child was born before age 35 (RR=0.894). Smaller and statistically insignificant effects of parity and age at last birth were observed for men after restricting the sample to males who have concluded their childbearing by age 50.

SES (males only)—Adult male SES has strong protective effects. Given the historical period in which these men lived, nearly 45 percent of men with a known occupation were identified as farmers (28 percent of the full sample). When both a separate dummy variable was included for farming along with a continuous version of the Nam-Powers socioeconomic index, we find that farmers had significantly lower mortality than non-farmers and that increasing levels of SES were associated with lower later-life mortality.

Religion—Both males and females, who, as adults, were active members of the LDS Church, have lower rates of mortality. LDS males enjoy a large and significantly lower mortality risks than other men (RR=0.82, $p<.0001$). LDS women, however, have only a slightly lower mortality hazard rate than non-LDS women (RR=0.968, $p=0.126$). The greater influence of religion for men in relation to women is attributable to the several possible factors. First, being LDS and male is associated with status and the greater potential

for leadership within the LDS Church and local community. Secondly, the lifestyle differences between LDS and non- LDS men are greater than the comparable differences among women. Specifically, the consumption of alcohol and tobacco are prohibited in the LDS Church. Non- LDS men would be more likely to smoke and consume alcohol while LDS men would not, thereby conferring a longevity advantage to Mormon males. This differential is less likely to occur between Mormon and non-Mormon women. LDS males and females are both likely to benefit from the social integration and participation of church-related activities but similar salutary effects would also exist for persons of other faiths.

Discussion

By using a large set of sib pairs (sister pairs and brother pairs), we have been able to generate stable estimates of the impact of suspected early-life and adult conditions on later-adult mortality that control for the possible effects of shared unobservable variables within a sibship. A key issue raised in this analysis concerns parental longevity and what it represents as a feature of early life conditions. After introducing FEL, we found attenuated effects of parental longevity on late life offspring mortality. To the extent that FEL captures an important component of genetic sources of longevity, the effect of parental longevity may now represent the effect of having parents who were not only present in ego's childhood but through much of ego's adult life. The fact that long-lived parents have beneficial effects on offspring survival may suggest that it is healthy parents who are better able to facilitate offspring survival than parents who are less robust. With respect to the FEL measure, we introduced in this paper the idea that genealogies are helpful for demographers to get observable measures of frailty and that one way of procuring this information (in the absence of UPDB) is to seek a family history of longevity from research subjects.

Despite the growing attention and interest given to early life conditions and their possible role in affecting later life health, we have generally found small to modest effects of childhood conditions (birth order, sibship size, parental religiosity, parental SES, and parental death in childhood) on later life health in relation to our measures of familial aggregation of longevity (see similarly weak effects of early life conditions in (Schwartz, 1995). The analyses are based on the UPDB where the records describe the early pioneers of Utah and their descendants, a feature that limits racial and ethnic diversity and where a majority practice the same religion. However, the founding population of Utah had diverse origins, including migrants from New England and numerous Northern European countries; they came with different languages and customs settling a wide geographic area in what is now modern day Utah.

We are intrigued by the finding that individuals born to older parents (especially daughters) were found in some of our analyses to be associated with excess mortality (Gavrilov & Gavrilova, 1997, 2004; Gavrilov, Gavrilova, Olshansky, & Carnes, 2002). Daughters born to older fathers may receive fewer resources that affect the daughters' survival, a deficit encountered less often by sons. It is worth noting that of the men fathering children after age 70, the majority of their wives were under age 35 at the time of the child's birth. This point is made to show that most men fathering children at exceptionally old ages are not necessarily married to women in the oldest reproductive age group. Whether this association

is attributable to the adverse effects of being conceived from older ova and sperm, with their higher levels of germ-line mutations, or whether it is due to having been reared by older parents, is unclear. This result raises questions about fertility in contemporary society where a growing proportion of children are conceived by older couples.

In previous work based on the UPDB, we reported strong effects of fertility on post-reproductive mortality for women and to a lesser degree for men. In that analysis, we focused on a sample whose reproductive years took place when natural fertility conditions prevailed. In the current analysis, we found significant but somewhat weaker effects. This may be a function of this sample having lived in a qualitatively different era (declining fertility rates) that also coincided with the Great Depression when fertility rates dropped further.

For this historical population, we found strong and enduring influences of religion (LDS versus not) and occupation among men. This suggests that choices and behaviors occurring in early adulthood may have more dramatic effects on later life health than early conditions and that potentially harmful conditions in childhood do not necessarily rule out changes in adulthood that generate positive health effects.

The UPDB provided an opportunity to examine numerous early life conditions for an extinct cohort of individuals. Many of the studies on early life factors looked at a range of outcomes but very few considered longevity owing to the difficulty of gaining access to high quality data of early/mid life conditions combined with comprehensive mortality follow-up. While the data does not include all variables of potential interest, the genealogical structure of the data allowed us to control for shared but unobserved factors that were present among siblings.

Our findings are based on a historical population when fertility and infant mortality was high. It remains to be seen whether the early life conditions that we examined here will have comparable effects for contemporary populations. In particular, countries like the U.S., Canada, and those in Northern and Western Europe are now witness to smaller family sizes where women/couples are delaying childbearing beyond age 35 or 40. A child born to older parents in the year 2000 when that child is the first-born may experience very different survival consequences than a comparable child born to older parents in the late 1800s but who was the tenth or fifteenth born.

As with any study that examines early life conditions and its impact on adult outcomes, more attention needs to be made potential selection biases that arise when the sample is restricted to persons surviving to adulthood. It is likely the case that any mortality selection that occurs in such studies will lend itself to conservative estimates of the impact that adverse childhood effects have on adult mortality. This arises because the children most susceptible to deleterious exposures in childhood will be eliminated from the sample, thereby leaving a more robust and homogeneous subset of adult survivors. We also recognize that the variables used in this analysis do not exhaust the numerous factors in childhood that may be pertinent in the study of later-life mortality.

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Appendix A

Males - Comparisons of Cox Models that Treat Paired Data Differently

	Simple Cox Model		Robust Variances		Shared Frailty		Correlated Frailty	
	Rate Ratio	<i>p</i>	Rate Ratio	<i>p</i>	Rate Ratio	<i>p</i>	Rate Ratio	<i>p</i>
Birth Year	0.997	0.000	0.997	0.000	0.997	0.000	0.997	0.000
Father's Age at Death 75th-85th percentile	0.933	0.002	0.933	0.003	0.933	0.002	0.922	0.002
Father's Age at Death 85th-95th percentile	0.813	0.000	0.813	0.000	0.812	0.000	0.783	0.000
Father's Age at Death >=95th percentile	0.693	0.000	0.693	0.000	0.689	0.000	0.646	0.000
Mother's Age at Death 75th-85th percentile	0.931	0.001	0.931	0.002	0.930	0.001	0.917	0.001
Mother's Age at Death 85th-95th percentile	0.95	0.067	0.95	0.070	0.949	0.062	0.929	0.026
Mother's Age at Death >=95th percentile	0.791	0.002	0.791	0.003	0.788	0.002	0.754	0.002
Familial excess longevity	0.944	0.000	0.944	0.000	0.945	0.000	0.943	0.000
Familial excess longevity not estimable	1.452	0.320	1.452	0.120	1.449	0.330	1.431	0.390
Has One Sib	0.987	0.660	0.987	0.680	0.987	0.670	0.989	0.750
1st born of Sib pair	1.027	0.300	1.027	0.300	1.027	0.310	1.027	0.370
Committed to LDS Church	0.797	0.000	0.797	0.000	0.796	0.000	0.765	0.000
Baptized as Child in LDS Church	1.007	0.750	1.007	0.750	1.007	0.750	1.007	0.780
Maternal Age <20	1.064	0.054	1.064	0.057	1.065	0.052	1.077	0.041
Maternal Age 30-35	1.01	0.740	1.01	0.740	1.010	0.750	1.009	0.800
Maternal Age 35+	1.085	0.007	1.085	0.007	1.086	0.007	1.091	0.012
Paternal Age <20	1.079	0.510	1.079	0.550	1.078	0.510	1.092	0.500
Paternal Age 30-39	1.004	0.860	1.004	0.860	1.004	0.870	1.000	1.000
Paternal Age 40-49	0.996	0.900	0.996	0.900	0.996	0.890	0.992	0.810
Paternal Age 50-69	1.019	0.630	1.019	0.630	1.019	0.630	1.021	0.660
Paternal Age 70+	1.098	0.530	1.098	0.480	1.097	0.540	1.085	0.630

	Simple Cox Model		Robust Variances		Shared Frailty		Correlated Frailty	
	Rate Ratio	<i>p</i>	Rate Ratio	<i>p</i>	Rate Ratio	<i>p</i>	Rate Ratio	<i>p</i>
Dad Died before Ego was 20	0.933	0.014	0.933	0.018	0.932	0.014	0.925	0.017
Mom Died before Ego was 20	0.957	0.160	0.957	0.170	0.956	0.160	0.951	0.170
Orphaned before Ego was 20	0.969	0.620	0.969	0.670	0.970	0.630	0.981	0.790
Own SES (Nam-Power) (males only)	0.999	0.017	0.999	0.014	0.998	0.017	0.998	0.013
Own SES not estimable (males only)	1.102	0.000	1.102	0.000	1.103	0.000	1.129	0.000
Father SES (Nam-Power)	1.001	0.280	1.001	0.260	1.001	0.290	1.001	0.370
Father SES not estimable	1.005	0.830	1.005	0.830	1.006	0.820	1.006	0.820
Frailty (variance of Gamma distributed random effect) with FEL included					0.006	0.260		
Frailty (variance of random Gamma distributed effect) with FEL excluded					0.127	0.001		
Variance of normally distributed random effect f with FEL included							0.184	0.001
Variance of normally distributed random effect f with FEL excluded							0.277	0.001

Appendix B

Females - Comparisons of Cox Models that Treat Paired Data Differently

	Simple Cox Model		Robust Variances		Shared Frailty		Correlated Frailty	
	Rate Ratio	<i>p</i>	Rate Ratio	<i>p</i>	Rate Ratio	<i>p</i>	Rate Ratio	<i>p</i>
Birth Year	0.99	0.000	0.99	0.000	0.990	0.000	0.989	0.000
Father's Age at Death 75th-85th percentile	0.942	0.009	0.942	0.011	0.942	0.009	0.937	0.011
Father's Age at Death 85th-95th percentile	0.883	0.000	0.883	0.000	0.883	0.000	0.865	0.000
Father's Age at Death >=95th percentile	0.82	0.027	0.82	0.020	0.819	0.026	0.792	0.021

	Simple Cox Model		Robust Variances		Shared Frailty		Correlated Frailty	
	Rate Ratio	<i>p</i>	Rate Ratio	<i>p</i>	Rate Ratio	<i>p</i>	Rate Ratio	<i>p</i>
Mother's Age at Death 75th-85th percentile	0.978	0.310	0.978	0.330	0.977	0.310	0.971	0.240
Mother's Age at Death 85th-95th percentile	0.872	0.000	0.872	0.000	0.871	0.000	0.849	0.000
Mother's Age at Death >=95th percentile	0.696	0.000	0.696	0.000	0.693	0.000	0.655	0.000
Familial excess longevity	0.938	0.000	0.938	0.000	0.938	0.000	0.937	0.000
Familial excess longevity not estimable	0.681	0.190	0.681	0.410	0.680	0.190	0.686	0.260
Has One Sib	1.07	0.018	1.07	0.018	1.070	0.018	1.075	0.025
1st born of Sib pair	1.018	0.500	1.018	0.500	1.018	0.500	1.026	0.380
Committed to LDS Church	0.965	0.098	0.965	0.099	0.965	0.099	0.965	0.130
Baptized as Child in LDS Church	0.963	0.120	0.963	0.130	0.963	0.110	0.952	0.063
Age at last birth 35-44	0.956	0.081	0.956	0.072	0.956	0.081	0.953	0.088
Age at last birth 45+	0.891	0.046	0.891	0.038	0.890	0.045	0.878	0.040
Nulliparous/Unk FertHx	1.143	0.000	1.143	0.000	1.143	0.000	1.160	0.000
Children = 3-5	0.991	0.810	0.991	0.810	0.990	0.800	0.982	0.660
Children = 6-8	1.043	0.260	1.043	0.260	1.043	0.270	1.037	0.380
Children = 9-11	1.083	0.066	1.083	0.072	1.082	0.068	1.082	0.097
Children 12+	1.164	0.005	1.164	0.004	1.164	0.005	1.168	0.009
Maternal Age <20	1.059	0.082	1.059	0.097	1.060	0.081	1.067	0.075
Maternal Age 30-35	1.014	0.650	1.014	0.650	1.014	0.650	1.010	0.750
Maternal Age 35+	1.026	0.410	1.026	0.420	1.026	0.410	1.028	0.420
Paternal Age <20	1.019	0.860	1.019	0.860	1.018	0.860	1.010	0.930
Paternal Age 30-39	1.014	0.580	1.014	0.590	1.014	0.580	1.021	0.450
Paternal Age 40-49	0.984	0.620	0.984	0.640	0.984	0.620	0.987	0.700
Paternal Age 50-69	0.968	0.420	0.968	0.450	0.968	0.430	0.973	0.550
Paternal Age 70+	1.458	0.028	1.458	0.025	1.458	0.028	1.561	0.019
Dad Died before Ego was 20	1.037	0.200	1.037	0.190	1.037	0.210	1.035	0.290
Mom Died before Ego was 20	0.965	0.270	0.965	0.270	0.965	0.260	0.957	0.210
Orphaned before Ego was 20	0.939	0.320	0.939	0.330	0.939	0.310	0.930	0.300
Father SES (Nam-Power)	0.998	0.056	0.998	0.062	0.998	0.056	0.998	0.063
Father SES not estimable	0.956	0.069	0.956	0.071	0.955	0.068	0.947	0.048
Frailty (variance of Gamma distributed)					0.003	0.350		

	Simple Cox Model		Robust Variances		Shared Frailty		Correlated Frailty	
	Rate Ratio	<i>p</i>	Rate Ratio	<i>p</i>	Rate Ratio	<i>p</i>	Rate Ratio	<i>p</i>
random effect) with FEL included								
Frailty (variance of random Gamma distributed effect) with FEL excluded					0.091	0.001		
Variance of normally distributed random effect f with FEL included							0.127	0.001
Variance of normally distributed random effect f with FEL excluded							0.202	0.001

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Table 1
Descriptive Statistics of All Variables by Gender

	Males = 12,366		Females = 11,896	
Variable	Mean	Sum	Mean	Sum
Age at Death	73.31	906512	76.25	907107
Birth Year	1877.76	23220318	1878.23	22343476
Father's Age at Death				
75th-90th percentile	0.15	1836	0.15	1837
90th-95th percentile	0.05	636	0.05	641
95th-99th percentile	0.04	500	0.04	486
>=99th percentile	0.01	127	0.01	140
Mother's Age at Death				
75th-90th percentile	0.15	1854	0.16	1878
90th-95th percentile	0.05	604	0.05	598
95th-99th percentile	0.04	524	0.04	500
>=99th percentile	0.01	134	0.01	122
Familial excess longevity	2.98	36851.36	2.97	35384.56
Familial excess longevity not estimable	<0.01	7	0.00	12
CHILDHOOD CONDITIONS				
Has One Sib	0.11	1318	0.12	1394
1st born of Sib pair	0.50	6182	0.50	5947
Maternal Age <20	0.11	1305	0.10	1227
Maternal Age 30-35	0.16	1953	0.16	1932
Maternal Age 35+	0.29	3565	0.28	3347
Paternal Age <20	0.01	80	0.01	94
Paternal Age 30-39	0.32	4013	0.33	3962
Paternal Age 40-49	0.24	2935	0.23	2708
Paternal Age 50-69	0.10	1210	0.10	1137
Paternal Age 70+	<0.01	49	0.00	36
Dad Died before Ego was 20	0.17	2076	0.17	2042
Mom Died before Ego was 20	0.1115	1379	0.11	1344
Orphaned before Ego was 20	0.02	294	0.02	296
Baptized as Child in LDS Church	0.73	8969	0.75	8931
Father SES (Nam-Power)	43.37	536362.2	43.18	513610.9
Father SES not estimable	0.18	2205	0.19	2206

	Males = 12,366		Females = 11,896	
Variable	Mean	Sum	Mean	Sum
ADULT CONDITIONS				
Age at Last Birth 35-44	0.32	3927	0.41	4872
Age at Last Birth 45+	0.17	2094	0.03	379
Nulliparous/Missing Some Fertility History	0.41	5119	0.38	4521
Children = 3-5	0.18	2249	0.18	2149
Children = 6-8	0.20	2440	0.20	2395
Children = 9-11	0.09	1085	0.10	1205
Children 12+	0.03	470	0.05	550
Committed to LDS Church	0.54	6700	0.57	6743
Own SES (Nam-Power) (males only)	48.41	598607.6		
Ego is farmer (males only)	0.28	3429		
Own SES not estimable (males only)	0.39	4817		
Spouse died by Egos age 50	0.40	4974	0.39	4630
Own birth year - Spouse's birth year	-3.52	-43557.92	4.11	48857.26

Table 2

Hazard Rate Ratios from Cox Proportional Hazard Rate Models. All Variables Included. Females Only.

Variable	Hazard Rate	<i>p</i>
Birth Year	0.991	<.0001
Father's Age at Death		
75th-90th percentile	0.952	0.0647
90th-95th percentile	0.962	0.353
95th-99th percentile	0.812	<.0001
>=99th percentile	0.871	0.1073
Mother's Age at Death		
75th-90th percentile	0.915	0.0007
90th-95th percentile	0.855	0.0003
95th-99th percentile	0.816	<.0001
>=99th percentile	0.655	<.0001
Familial excess longevity	0.941	<.0001
Familial excess longevity not estimable	0.713	0.2448
CHILDHOOD CONDITIONS		
Has One Sib	1.068	0.0217
1st born of Sib pair	1.017	0.536
Maternal Age <20	1.06	0.0779
Maternal Age 30-35	1.014	0.6514
Maternal Age 35+	1.027	0.3911
Paternal Age <20	1.014	0.8934
Paternal Age 30-39	1.013	0.6161
Paternal Age 40-49	0.979	0.5071
Paternal Age 50-69	0.958	0.2967
Paternal Age 70+	1.4	0.0486
Dad Died before Ego was 20	1.051	0.0662
Mom Died before Ego was 20	0.963	0.2155
Orphaned before Ego was 20	0.953	0.4345
Baptized as Child in LDS Church	0.964	0.1263
Father SES (Nam-Power)	0.998	0.067
Father SES not estimable	0.961	0.1063
ADULT CONDITIONS		
Age at Last Birth 35-44	0.96	0.1136
Age at Last Birth 45+	0.894	0.0522

Variable	Hazard Rate	<i>p</i>
Nulliparous/Missing Some Fertility History	1.142	0.0004
Children = 3-5	0.988	0.7491
Children = 6-8	1.036	0.3502
Children = 9-11	1.075	0.0936
Children 12+	1.155	0.0078
Committed to LDS Church	0.968	0.1263
Spouse died by Ego age 50	0.998	0.9327
Own birth year - Spouse's birth year	1.004	0.0458

Table 3

Hazard Rate Ratios from Cox Proportional Hazard Rate Models. All Variables Included. Males only.

	Full Male Sample		Males Age at Last Birth<50	
Variable	Hazard Rate	P	Hazard Rate	p
Birth Year	1.00	<.0001	1.00	<.0001
Father's Age at Death				
75th-90th percentile	0.88	<.0001	0.88	<.0001
90th-95th percentile	0.83	<.0001	0.83	<.0001
95th-99th percentile	0.86	0.0013	0.87	0.0035
>=99th percentile	0.66	<.0001	0.67	<.0001
Mother's Age at Death				
75th-90th percentile	0.98	0.3589	0.97	0.3098
90th-95th percentile	1.04	0.3675	1.03	0.521
95th-99th percentile	0.91	0.0304	0.91	0.0454
>=99th percentile	0.75	0.0008	0.75	0.0014
Familial excess longevity	0.95	<.0001	0.95	<.0001
Familial excess longevity not estimable	1.42	0.3536	1.36	0.4187
CHILDHOOD CONDITIONS				
Has One Sib	0.99	0.6122	1.00	0.8904
1st born of Sib pair	1.03	0.3158	1.01	0.6131
Maternal Age <20	1.06	0.0728	1.07	0.0554
Maternal Age 30-35	1.01	0.796	1.00	0.9782
Maternal Age 35+	1.08	0.0154	1.06	0.0528
Paternal Age <20	1.09	0.4589	1.06	0.6349
Paternal Age 30-39	1.00	0.9995	1.00	0.8728
Paternal Age 40-49	0.99	0.815	1.00	0.9307
Paternal Age 50-69	1.01	0.8059	1.02	0.6455
Paternal Age 70+	1.07	0.6272	1.10	0.5146
Dad Died before Ego was 20	0.95	0.0393	0.95	0.0884
Mom Died before Ego was 20	0.99	0.6311	0.99	0.668
Orphaned before Ego was 20	1.02	0.7853	1.05	0.4092
Baptized as Child in LDS Church	1.02	0.496	1.02	0.4074
Father SES (Nam-Power)	1.00	0.3356	1.00	0.3222
Father SES not estimable	1.01	0.8177	1.02	0.5481
ADULT CONDITIONS				

	Full Male Sample		Males Age at Last Birth<50	
Variable	Hazard Rate	<i>P</i>	Hazard Rate	<i>p</i>
Age at last birth 35-44			0.98	0.3754
Age at last birth 45+			0.95	0.1963
Nulliparous/Missing Some Fertility History			1.05	0.2285
Children = 3-5			0.94	0.1321
Children = 6-8			1.02	0.677
Children = 9-11			1.01	0.8244
Children 12+			1.10	0.1162
Committed to LDS Church	0.82	<.0001	0.83	<.0001
Own SES (Nam-Power) (males only)	1.00	0.0001	1.00	0.0007
Own SES not estimable (males only)	1.03	0.1388	1.03	0.1545
Ego is farmer (males only)	0.90	<.0001	0.92	0.0014
Spouse died by Ego age 50	1.10	<.0001	1.06	0.0103
Own birth year - Spouse's birth year			1.00	0.2413