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

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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 longevity. Recently, more attention has been given to the role that very early conditions (including *in utero*) of childhood have on adult health outcomes ([1-3]). 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, andadolescence.

In this paper, we focus our attention on key family circumstances that may set the stage for affecting mortality risks decades later. We begin with 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 associated with all known blood relatives.

Apart from parental survival, parents affect resources and opportunities to their children that may have lasting consequences for their progeny. Specifically, parental fertility patterns can create wide ranging family structures for their offspring. The fertility patterns that we consider are the individual's sibship size (parental parity) 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 also of potentially great importance. Parental ocioeconomic resources may affect childhood nutrition, housing, and risk of childhood illnesses. In our sample, we are able to consider both the effects of parental SES but we also examine simultaneously the role of religion in childhood (and the social integration and lifestyle effects it represents) and their influence on adult mortality.

While our focus in this paper is aimed at childhood family conditions, we also consider natural extensions of our earlier work on the impact that fertility, religion, and socioeconomic status as an adult has on the adult's own mortality past age 50 ([4]). Our intent here is to assess how the effects of childhood life conditions, as described here, compare to the influences of adulthood circumstances (fertility, religion, and SES) 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. In an effort 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. While unobserved heterogeneity is important to consider for this analysis, their significance on mortality risks directly and their influence on our general results regarding the effects of observed heterogeneity are minor.

Background

Familiality of Longevity

In humans, the familial component of age at death has been examined repeatedly over the last century by biologists, population geneticists, evolutionists, and demographers[5-15]. Reported *heritability* estimates of age at death vary widely, ranging from nearly zero [9] to 0.33 [15], in part because of differences in the types of paired relationships examined, the time periods and number of generations considered, and the quality of data among source populations. These estimates are normally derived from familial correlations; as such, they are always elevated by non-genetic factors shared by families, but that vary within and between populations. These non-genetic factors (e.g., nutrition, housing, lifestyle) may link parental and offspring longevity together.

Parental Death in Childhood

There is reason to predict that for dependent children, the death of a parent will have adverse effects on these children later in their lives ([16-18]). Younger surviving spouses encounter the psychological strains of bereavement, the loss of social and economic support, the challenges of being a single-parent, and ultimately an excess risk of poor health and premature death ([19]). Additionally, widowhood for younger or middle-aged individuals more often arises unexpectedly, thereby minimizing the ability of the surviving spouse to prepare for the impending death ([20]). Younger children of widowed households are, therefore, likely to experience comparable socio-emotional and economic deprivations as those encountered by their surviving parent. Studies of historical populations showed how important parental death was

for survival of children and differences in the roles played by fathers and mothers [21]

Sibling Size

The effect of sibling size on adverse health, socioeconomic, and behavioral outcomes is well established. Sibship size is positively associated with increased cancer risk, lower educational achievement, and unhealthy lifestyle choices ([22-27]). Little literature exists on adult mortality [and health] in relation to sibship size. However, children from large families in historical populations and in developing countries experience higher infant and childhood mortality rates than children from smaller families ([28-31]). In part this may be due to greater exposures to infectious diseases and maternal depletion, associated with shorter birth intervals. However, in families with more children, the effect of sibship size on adult health and mortality remains to be seen. Large families have historically lived in more crowded conditions. In turn, children from large sibships may have a greater risk of contracting an infectious disease, which can then influence their adult health ([24]). Alternatively, others have argued that children who survive widespread infectious diseases are strengthened and go on to live significantly longer lives then their counterparts (Meindl, 1982). The resource dilution model ([22, 27, 32, 33]) posits that parents have finite levels of resources (both economic and physical), which are divided among siblings. The larger the sibling group, the greater the dilution of resources. Overall, we do not know whether larger families translate into adverse health and longevity effects into adulthood.

Birth Order

The literature on birth order effects is vast with less consistent results. However, few studies have examined birth order in relation to longevity or mortality risk. A notable

exception to this is Modin who found that later-born siblings generally (especially girls) have higher mortality risks than firstborn siblings [27]. The mortality rate was particularly high for later-born girls, who had four times the mortality rate of firstborn females. Overall, this study concluded that birth order and mortality risk are positively associated. First-born children may benefit from more parental time, attention, and resources. Perhaps as a consequence, first-born children are over-represented in college populations ([34]) and reach higher levels of educational and occupational achievement than their siblings ([27, 35-38]). Later-born children have been found to enjoy greater social success and score higher on measures of social skills ([39]) and that they tend to be more accepting of change than first-born children ([38]).

Parental Age

A consistent association has been shown between parental age and Down syndrome, birth defects, and schizophrenia, and a suggested association between parental age and longevity ([25, 40, 41]). Older mothers have older ova (eggs) that give rise to more birth defects such as Down Syndrome ([42, 43]). Still others contend that longevity is affected by the number of mutations accumulated in germ line (ova and sperm) cells ([41]). For human females, the estimated number of cell divisions between egg and zygote (the product of the fusion of an egg and a sperm that develops into an embryo) is twenty-four while for human males the higher incidence of cell divisions between sperm and zygote increases dramatically with a man's age ([41]). 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 ([40]). However, 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 ([44]). Older parents are more mature and are more likely to have greater socioeconomic resources. However, older parents share fewer years with their children than other parents. The adverse effects of early (teenage) parenthood in terms of economic and educational outcomes, childbearing and mental health characteristics have also been demonstrated ([45, 46]).

Socioeconomic Status in Childhood and Adulthood

Socioeconomic status (SES) has long been positively associated with longevity [47]but there is some debate regarding when SES matters in the course of an individual's life. Several studies have explored this topic but differing results with respect to the strength of the association between SES in childhood and later life health [48-50].

Own Fertility

While women who bear a large number of children are associated with excess post-reproductive mortality[4, 51], women who are able to bear children in mid- to late-life (e.g., after age 45) are possibly aging more slowly than women who are unable to bear children at the same advanced reproductive age Several recent studies have indeed shown that late fertile women have lower rates of later-life mortality([4, 52]).

Materials and Methods

Data

The analyses are based on information obtained from the Utah Population Database (UPDB), one of the world's largest and most comprehensive computerized genealogies. In the 1970s, approximately 170,000 Utah nuclear 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 across generations; in some instances, the records span seven generations. The UPDB now holds data on migrants to Utah and their Utah descendants (not only Mormons) that number more than 1.8 million individuals born from the early 1800s to the mid-1900's and that are linked into multi-generation pedigrees. 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). The UPDB is an active genealogy: new families and their members are continually being added as the UPDB is linked to other sources of data, including birth and death certificates. Additional information on these families comes from sources such as drivers' license records and the Utah Cancer Registry. Because these records include basic demographic information on parents and their children, fertility and mortality data are extensive with coverage up to 2002.

For this study, we consider individuals (egos) from sibships born between 1850 and 1900. This historical period is advantageous for this study given that the parents of these children lived during a time when effective modern contraceptive methods were nonexistent or very limited. Accordingly, their family formation patterns reflect natural fertility conditions where reproduction has influenced less by choices made by couples and more by biological and

environmental factors. It was during this era that the elderly of the 20th century were born and their mortality risks may have been shaped in fundamental ways based on the circumstances of their childhood.

The sample selected for analysis relies on the deep multigenerational structure of the UPDB. For our purposes, we have identified parents who have completed their childbearing during the 50 year interval spanning 1850 and 1900. It is the mortality experiences of their children that are the focus of our attention. To assess factors that may affect the later-life mortality of these offspring, we rely on information about the offspring 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 (frailty), we have selected same-sex sib pairs. Families that had either 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 samples. This selection rule means that we exclude families with only children or sibships where a single son or daughter is present. For this population, this latter restriction eliminated less than 10% of all sibships and thus a small bias could arise due to this constraint.

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. Using all sibling egos from all eligible parents will be explored in future analyses; fitting our survival models that allow for shared or correlated frailty with large sibship sizes and a large number of sibships is computing intensive and improving the efficiency of these techniques is

something we are currently investigating (with Dr. Terry Therneau, Mayo Clinic). The first born/last born sib-pair 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 also later-born sisters).

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. The born from 1850 to 1900 that survived to age 50 numbered 12,366 sons (6,184 brother pairs) and 11,896 daughters (5,948 sister pairs).

Methods

All models are based on variations of the Cox proportional hazards models (PHM) where we model time between age 50 and death. The sample comprises an extinct cohort of individuals where all have observed death dates. We conducted analyses on four types of survival models. The first model is a "naïve" model in which we estimate a Cox PHM that ignores the clustered sib-pair data and does not attempt to model shared unobserved heterogeneity or frailty that is common among siblings. This approach assumes there is a sample of men and a sample of women and that they are unrelated and independent:

$$h_i(a) = h_o(a) \exp(X_i b)$$

where i indexes individuals, a measures age, X are observed covariates, and b are regression parameters.

The second model extends the Cox PHM by taking into account the fact that the siblings in a family are not statistically independent. This is done by modeling robust variances of the

regression parameters ([53]) but which generates the same regression parameters as the naïve model.

The third method models the Cox PHM by allowing for shared frailty. 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. This model treats frailty, f, as a Gamma distributed random variable:

$$h_{ij}(a) = h_o(a) \exp (X_i b + f_j)$$

where j indexes families (sibships).

Finally, we provide estimates of the Cox PHM that assumes that the association between siblings' hazard rate for mortality that 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:

$$h_i(a) = h_o(a) \exp (X_i b + f_i),$$

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

where K is a kinship matrix. One random effect per subject 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 for age-attainment models are also provided to assess how childhood conditions affect an individual's chances of reaching specific age thresholds. Two dependent variables examined here are dichotomous and, conditional on survival to age 50, measure whether an individual lives to the top 10% or top 5% of the sex-specific age-at-death distribution. The comparison group for all three variables is whether the individual died at an age that marks the

75th percentile of the sex-specific age-at-death distribution. For example, one dependent variable measures whether an individual lived past the top 5% age at death (=1) versus not living past the 75th percentile age at death (=0). The effects of our set of covariates on each dichotomous dependent variable are estimated using logistic regression.

Measures

Mortality – For the hazard rate models, the outcome is the hazard rate for all-cause mortality starting at exact age 50. The age-attainment models are based on dichotomous outcomes that equal one of an individual survives to 75, 85, or 95 and equals zero if an individual does not survive to 75. These three age thresholds represent greater exceptional survival for men than women. However, using comparable percentile cutoffs for survival (e.g., live past an age for the top 5% of sex-specific age at death) did not change the qualitative patterns of the results shown. The rationale for using a single comparison/control group is to sharpen the differences between increasingly extreme ages and controls. Age 75 represents the approximate median survival age for males and females in the sample (median(males)=73.3 and median(females)=76.2).

Parental Longevity – The age at death of mothers and fathers have been categorized into four groups each: died before the 75th percentile (father died<81, mother died<82) , 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), after the 99th percentile (father≥94, mother≥96).

Familial Excess Longevity – 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 potential confounders (gender, birth year, affiliation with the Church of Jesus Christ of Latter-day Saints). Expected longevity ($\hat{y}$) is estimated from an accelerated failure time model in the following manner:

$$\hat{y} = e^{\alpha+\beta_1 \cdot gender+\beta_2 \cdot birthyear+\beta_3 \cdot religious\ affiliation}$$

where α is the intercept, β_1, β_2, β_3 are slope coefficients, and the 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 subject was alive). Given that our longstanding interest is in longevity among the elderly, our approach here is to focus on only those persons who reached the age of 65. Excess longevity is then extended to pedigree members (blood relatives) for each individual. Averaging the excess longevities of all family members 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) [54]:

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

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 kth member of K, and f(i,k) is the kinship coefficient [54], the probability that i and k share a given gene identical by descent

from a common ancestor. On average, persons born prior to 1900 had 233 kin who lived to age 65 on whom their FEL measure was based.

A very small fraction of individuals (17 persons of over 24,000) did not have sufficient data on their kin to reliably estimate their FEL. A separate dummy variable is used to identify these individuals. These persons are then assigned the sample-wide mean value of FEL are included in the analysis.

Childhood Family Conditions

Sibship Size – The total number of siblings for egos (including ego) averaged 7.70. In preliminary examination of our models, we observed a small but positive association between number of siblings and mortality risk. We discovered that this association was largely attributable to whether ego had only one sibling or not. 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 persons represented in the data. We also assessed whether it is the number of sisters or brothers that affects survival but found no evidence to support this.

Birth Order – The average birth order for egos is 4.2. Birth order is naturally affected by sibship size since a child cannot be of high birth order unless there are many siblings born previously. To include birth order effects but that take this problem into account, we use a dummy variable that specifies whether an ego in a sib pair was the first born in the pair or not.

Parental Age at Child's Birth – The parental age when an ego was born is measured separately for mothers and fathers. For maternal age, four age categories are used: under 20 years, 20-29 years (reference category), 30-34 years, and 35 years or older. For fathers, age at

birth was more variable and more categories were used: under 20 years, 20-29 years (reference category), 30-39 years, 40-49 years, 50-69 years, and 70 years or older.

Parental Age at Death during Ego's Childhood – We determined whether an ego's parents died while ego was a child or adolescence. 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.

Religion in Childhood – The UPDB contains the dates of baptism for all egos when a baptism occurs, 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 any other age or never baptized are presumed not to have been raised in an LDS household.

Socioeconomic Status of Father – For fathers who died in Utah and for whom we obtained a Utah death certificate, we capture their usual industry and occupation from the death certificate. This is possible because we have access to all death certificates that have been issued in the state of Utah (1904-2002). Industry and occupation data have been converted to a socioeconomic index developed by Nam and Powers ([55]). Higher scores are associated with higher SES. Approximately 18 percent of fathers in the sample did not link to a Utah death certificate. These individuals are identified by a dummy variable and are assigned the group mean for the Nam-Powers socioeconomic index.

Early and Middle Adulthood Family Conditions

Fertility – Ego's own fertility behavior is assessed using two measures: parity and age at last birth. Based on an earlier birth cohort in the UPDB, we found previously that these two measures of fertility were strongly associated with later life mortality[4]. Age at first birth or age at first marriage was not a strong predictor of post-reproductive mortality and was not introduced here. The UPDB includes a large fraction of fertility data from individuals born from 1850-1900. However, we are continuing to identify and link births to the UPDB for these individuals. At this time, approximately one-third of the sample has incomplete fertility information, largely because these are persons bearing children outside Utah during the 20th century. To include these individuals, we have constructed a set of dummy variables: parity of 1-2 (reference group), parity 3-5, parity 6-8, parity 9-11, parity 12 or higher, and fertility information missing. For age at last birth, we constructed four categories: under 35 years, 35 to 44 years, 45 years or older, and fertility information missing (same dummy as the variable for parity).

Religion in Adulthood – The UPDB contains dates of baptism but it also contains dates of endowment. Individuals with an endowment date are adult Mormons who have made a conscious pledge or covenant with God to conduct their lives that is guided by the doctrine of the LDS Church. This typically occurs as a young adult and later for converts. In general, individuals who have made an endowment are considerably more likely to abstain from tobacco and alcohol as well as participate actively in church and religious activities. Persons with an endowment date prior to age 40 are treated as devout members of the LDS Church.

Socioeconomic Status – As with the fathers of egos, we obtain data on ego's usual industry and occupation from their death certificates. Industry and occupation data are again

converted to the Nam and Powers socioeconomic index. Approximately 38 percent of egos in the sample did not link to a Utah death certificate, reflecting the higher rate of Utah out-migration of egos relative to their fathers. Egos lacking a Utah death certificate (and hence occupation and industry data) are identified by a dummy variable and are assigned the group mean for the Nam-Powers socioeconomic index.

RESULTS

The results are organized by ego's gender. The descriptive statistics for the brother-pair and sister pair samples are shown in Table 1. A few details of the data deserve brief comment here. The mean for the familial excess longevity (FEL) is 2.98 years. This figure indicates that across the entire sample, egos have blood relatives who live approximately three years longer (rather than zero) than expected. For the full UPDB, the mean FEL equals zero. This feature of the data is a function of the survival selection of the sample (e.g., sib pairs who both survived to age 50). The fertility data (parity, age at last birth) have a high fraction of missing data reflecting the fact that some egos from our birth cohort left the state of Utah and bore children in other states. This limits our ability to update their fertility information with Utah birth certificates. Given the fertility information from either genealogical data or in-state birth records, we show that males and females have similar levels of parity but that the ages at last birth differ substantially. Approximately 17 percent of all 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, where we rely on Utah death certificates for industry and occupation for socioeconomic data, we are able to generate a Nam-Power score for 83% of the fathers of egos but 62% of male egos. This again reflects the higher rate of Utah out-migration of male egos in relation to their fathers since missing SES data generally means a death outside Utah.

Table 1

Table 2 lists results for sex-specific birth-year/parental-longevity adjusted estimates regarding the association between our measures of childhood and early adulthood conditions on later-life mortality. Each variable is considered in isolation in order to provide a contrast with the full, multivariate models. These results are provided for reference and are not discussed here. When the full models are estimated, we estimate several types of Cox proportional hazard rate models that range in the treatment of frailty effects and adjustments to parameter variances due to the sib-pair construction of the sample. The results shown in Table 2 are based on the simplest version of the Cox proportional hazard models that make no adjustments for the paired data structure or frailty.

Table 2

The effects of early life conditions on later-life mortality based on the fully-adjusted model are shown for males and females in Tables 3 and 4 respectively. We focus on the results from the basic Cox proportional hazards models and describe important differences with the results generated from the variance-corrected or frailty-based extensions to the Cox model.

Table 3 and 4

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. This result per se may arise for a variety of reasons including inheritance of genetic variants associated with slower rates of aging and a shared environment between parent and offspring during egos' childhood. 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. We note that the interpretation of the effect that FEL has on ego mortality is less problematic. Specifically, FEL considers the influence that the longevity of numerous relatives 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. This is not to say that ego and extended family members do not share life circumstances in some ways. Our observation is simply that FEL is the single strongest predictor of ego survival but the association is based less on a shared environment argument (given the manner in which FEL is constructed) than the association between parent and offspring. Indeed, once we adjust for the influence of 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, including grandparenting their grandchildren as well as providing direct economic and psychosocial support to egos.

We also consider FEL to be a potentially important and 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 frailty that include FEL, again with sib-pair data. When FEL is excluded, we find significant effects of frailty (not shown) suggesting that there are shared factors among siblings that contribute to a common excess risk of mortality. When FEL is added to the model, FEL becomes the strongest predictor of mortality and frailty effects disappear. This finding suggests that whatever factors link siblings' survival, an important component is the familiality of longevity within their extended family, suggesting that they share alleles affecting survival.

Childhood Family Conditions

Female Survival

The types of 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. Women's family structure in childhood has little effect on later-life mortality. Their birth order has no effect on their survival but their sibship size does, albeit with a small impact. 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. Two exceptions to this exist, one small and one remarkable. First, girls born to young mothers (<20) experience excess mortality risk but its effect is small (RR=1.06, p=0.08). Converesely, exceptionally old fathers (over age 70) have daughters whose mortality rate is 40% higher than control fathers (fathers bearing children 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 the social and economic resources represented by church membership and higher socioeconomic standing 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 sibling size or birth order on male late-adult mortality. This result held for sister pairs as well.

Male survival is sensitive to maternal age at birth but not paternal age. Boys born to very young (under age 20) and older mothers (age 35 or older) have significantly higher mortality than comparison males with maternal ages of 20-29 years of age. 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).

The experience of 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 (perhaps given their small numbers) but, interestingly, loss of one parent to death is associated with *lower* later-life mortality for males. 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), we suggest that children reaching age 50 are a select subset of egos who are more robust and have adapted in ways that confer a small (RR=0.946) survival advantage in later adulthood.

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 enjoy better survival chances 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). Conversely, women whose age at last birth was after age 45 had lower mortality risks in relation to women who last child was born before age

35 (RR=0.894). When women have missing fertility data, they are more apt to have moved out of Utah before they began bearing children since we were less able to secure birth records from other states. These out-migrant women have significantly higher mortality risks than women who remain in Utah with 1 to 2 children (the reference category). 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.

Socioeconomic Status (males only)

Adult male socioeconomic status 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 no-farmers and that increasing levels of SES were associated with lower later-life mortality.

Religion

Both males and females who, as adults, make a conscious pledge and commitment to God and to abide by the LDS faith in terms of spiritual beliefs and lifestyle, have lower rates of mortality. LDS males enjoy a large and significantly lower mortality risks than other (non-LDS or gentile) men (RR=0.82, $p<.0001$)). For LDS women, however, they have only a slightly lower mortality hazard rate than gentile 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, aspects of Mormonism that do not hold for women. 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 health and longevity advantage to Mormon males. This differential is far less likely to occur between Mormon and gentile 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.

Living to the Top 5th Percentile

We briefly describe the results for logistic regressions where we examine how childhood and middle adulthood conditions affect the prospects of living to the top 5th percentile in the age-at-death distribution (Table 4 and 5). For women, sibship size, paternal SES, and parity influence survival to extremes age as before although now we find that women who lost their fathers as children had significantly smaller chances of experiencing exceptional survival. FEL and parental longevity are the strongest predictors of remarkable longevity, both for males and females.

Table 5 and 6

For males, the only childhood factor contributing to exceptional survival is the age of their mother at birth. Boys born to older mothers (age $\geq$ 35) faced 30% lower odds of reaching an advanced age in relation to boys born to twenty-something mothers. The strong protective influences of having been a farmer and being a member of the LDS Church persist.

Summary and Discussion

We have examined how important indicators of family structure and well-being variables present in childhood and early/middle adulthood affect the mortality risk of adults after age 50. 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 adult mortality and to control for the possible effects of shared unobservable variables within a sibship.

The impact of parental longevity on offspring survival cannot be underestimated. Our analysis raised questions about what parental longevity represents as a causal mechanism. After introducing familial excess longevity (FEL) , a genealogically-based measure that assesses an individual's propensity for exceptional survival, we found attenuated but significant 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 may be helpful for demographers to get observable measures of frailty and that one way of procuring this information (in the absence of a UPDB resource) 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) in relation to our measures of familial aggregation of longevity. 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. 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. These figures are provided 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 at this point. We are investigating the medical and vital records of these individuals to shed some light on this question. This result also raises some questions about fertility in contemporary society where a growing proportion of children are conceived by older couples

In pervious 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 weaker effects. This maybe a function of this sample having lived in a qualitatively different era (dropping fertility rates) that also coincided with the Great Depression (for the large fraction of the sample born 1890-1900) when fertility rates dropped further, especially for those over 40.

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.

Our findings are based on a historical population when fertility and infant mortality was high. It remains to be seen whether the early life condition that we examined here will have comparable effects for contemporary populations. In particular, countries like the U.S. 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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Table 1. Descriptive Statistics of All Variables by Gender

	Males			Females		
Label	Mean	Sum	N	Mean	Sum	N
Age at Death	73.307	906512	12366	76.253	907107	11896
Birth Year	1877.755	23220318	12366	1878.234	22343476	11896
Ego lives to top 15 pct	0.137	1465	10730	0.142	1448	10234
Ego lives to top 5 pct	0.066	656	9921	0.065	610	9396
Pa Dage 75-90pct	0.149	1836	12366	0.154	1837	11896
Pa Dage 90-95pct	0.051	636	12366	0.054	641	11896
Pa Dage 95-99 pct	0.040	500	12366	0.041	486	11896
Pa Dage >=99 pct	0.010	127	12366	0.012	140	11896
Ma Dage 75-90pct	0.150	1854	12366	0.158	1878	11896
Ma Dage 90-95pct	0.049	604	12366	0.050	598	11896
Ma Dage 95-99 pct	0.042	524	12366	0.042	500	11896
Ma Dage >=99 pct	0.011	134	12366	0.010	122	11896
Familial excess longevity	2.980	36851	12366	2.975	35385	11896
FEL not estimable	0.001	7	12366	0.001	12	11896
Has One Sib	0.107	1318	12366	0.117	1394	11896
1st born of Sib pair	0.500	6182	12366	0.500	5947	11896
Committed to LDS	0.542	6700	12366	0.567	6743	11896
Baptized as Child in LDS	0.725	8969	12366	0.751	8931	11896
ALB 35-44	0.318	3927	12366	0.410	4872	11896
ALB 45+	0.169	2094	12366	0.032	379	11896
Nulliparous/Unk FertHx	0.414	5119	12366	0.380	4521	11896
Parity=3-5	0.182	2249	12366	0.181	2149	11896
Parity=6-8	0.197	2440	12366	0.201	2395	11896
Parity=9-11	0.088	1085	12366	0.101	1205	11896
Parity 12+	0.038	470	12366	0.046	550	11896
Maternal Age <20	0.106	1305	12366	0.103	1227	11896
Maternal Age 30-35	0.158	1953	12366	0.162	1932	11896
Maternal Age 35+	0.288	3565	12366	0.281	3347	11896
Paternal Age <20	0.007	80	12366	0.008	94	11896
Paternal Age 30-39	0.325	4013	12366	0.333	3962	11896
Paternal Age 40-49	0.237	2935	12366	0.228	2708	11896
Paternal Age 50-69	0.098	1210	12366	0.096	1137	11896
Paternal Age 70+	0.004	49	12366	0.003	36	11896
Dad Died before R was 20	0.168	2076	12366	0.172	2042	11896
Mom Died before R was 20	0.112	1379	12366	0.113	1344	11896
Orphaned before R was 20	0.024	294	12366	0.025	296	11896
Father SES (Nam-Power)	43.374	536362	12366	43.175	513611	11896
Father SES not estimable	0.178	2205	12366	0.185	2206	11896
Own SES (Nam-Power)	48.408	598608	12366			
Male ego farmer	0.277	3429	12366			
Own SES not estimable	0.390	4817	12366			
Sp died by Egos age 50	0.402	4974	12366	0.389	4630	11896
Ego-sp age	-3.522	-43558	12366	4.107	48857	11896

Table 2. Hazard Rate Ratios from Cox Proportional Hazard Rate Models.
Birth Year, Maternal and Paternal Longevity are included in all models.
All other models then add each variable set without any other statistical controls.

	Males		Females	
Variable Label	Hazard Ratio	P	Hazard Ratio	P
Birth Year	1.00	<.0001	0.99	<.0001
Pa Dage 75-90pct	0.87	<.0001	0.91	0.000
Pa Dage 90-95pct	0.79	<.0001	0.90	0.009
Pa Dage 95-99 pct	0.81	<.0001	0.78	<.0001
Pa Dage >=99 pct	0.59	<.0001	0.81	0.013
Ma Dage 75-90pct	0.94	0.012	0.88	<.0001
Ma Dage 90-95pct	0.97	0.449	0.82	<.0001
Ma Dage 95-99 pct	0.85	0.001	0.76	<.0001
Ma Dage >=99 pct	0.66	<.0001	0.60	<.0001
Familial excess longevity	0.94	<.0001	0.94	<.0001
FEL not estimable	1.44	0.333	0.81	0.460
Has One Sib	1.00	0.882	1.07	0.014
1st born of Sib pair	1.00	0.955	1.01	0.553
Committed to LDS	0.78	<.0001	0.93	0.000
Baptized as Child in LDS	1.00	0.971	0.95	0.022
ALB 35-44	0.94	0.008	0.92	0.001
ALB 45+	0.86	0.010	0.89	0.002
Nulliparous/Unk FertHx	1.14	0.001	1.06	0.113
Parity=3-5	0.99	0.694	0.94	0.113
Parity=6-8	1.03	0.378	1.02	0.656
Parity=9-11	1.08	0.082	1.03	0.595
Parity 12+	1.17	0.004	1.10	0.121
Sp died by Egos age 50	1.01	0.622	1.11	<.0001
Ego-sp age	1.00	0.064	1.00	0.926
Maternal Age <20	1.10	0.003	1.07	0.047
Maternal Age 30-35	1.01	0.661	1.00	0.991
Maternal Age 35+	1.06	0.014	1.01	0.790
Paternal Age <20	1.18	0.146	1.04	0.679
Paternal Age 30-39	1.00	0.935	1.00	0.917
Paternal Age 40-49	1.01	0.772	0.99	0.595
Paternal Age 50-69	1.01	0.807	0.96	0.240
Paternal Age 70+	1.10	0.505	1.49	0.017
Dad Died before R was 20	0.96	0.069	1.04	0.097
Mom Died before R was 20	0.98	0.552	0.95	0.062
Orphaned before R was 20	1.03	0.640	0.93	0.243
Father SES (Nam-Power)	1.00	0.990	1.00	0.081
Father SES not estimable	1.00	0.882	0.96	0.058
Own SES (Nam-Power)	1.00	<.0001		
Male ego farmer	0.87	<.0001		
Own SES not estimable	1.07	0.003		

Table 3. Hazard Rate Ratios from Full Cox Proportional Hazard Rate Model. Females Only

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Variable Label	Hazard Ratio	P	Chi-Square
Birth Year	0.99	<.0001	124.52
Pa Dage 75-90pct	0.95	0.065	3.41
Pa Dage 90-95pct	0.96	0.353	0.86
Pa Dage 95-99 pct	0.81	<.0001	19.28
Pa Dage >=99 pct	0.87	0.107	2.59
Ma Dage 75-90pct	0.92	0.001	11.50
Ma Dage 90-95pct	0.86	0.000	13.21
Ma Dage 95-99 pct	0.82	<.0001	18.65
Ma Dage >=99 pct	0.66	<.0001	20.82
Familial excess longevity	0.94	<.0001	156.15
FEL not estimable	0.71	0.245	1.35
Has One Sib	1.07	0.022	5.27
1st born of Sib pair	1.02	0.536	0.38
Committed to LDS	0.97	0.126	2.34
Baptized as Child in LDS	0.96	0.126	2.34
ALB 35-44	0.96	0.114	2.50
ALB 45+	0.89	0.052	3.77
Nulliparous/Unk FertHx	1.14	0.000	12.74
Parity=3-5	0.99	0.749	0.10
Parity=6-8	1.04	0.350	0.87
Parity=9-11	1.08	0.094	2.81
Parity 12+	1.16	0.008	7.08
Maternal Age <20	1.06	0.078	3.11
Maternal Age 30-35	1.01	0.651	0.20
Maternal Age 35+	1.03	0.391	0.74
Paternal Age <20	1.01	0.893	0.02
Paternal Age 30-39	1.01	0.616	0.25
Paternal Age 40-49	0.98	0.507	0.44
Paternal Age 50-69	0.96	0.297	1.09
Paternal Age 70+	1.40	0.049	3.89
Dad Died before R was 20	1.05	0.066	3.37
Mom Died before R was 20	0.96	0.216	1.53
Orphaned before R was 20	0.95	0.435	0.61
Father SES (Nam-Power)	1.00	0.067	3.35
Father SES not estimable	0.96	0.106	2.61
Sp died by Egos age 50	1.00	0.933	0.01
Ego-sp age	1.00	0.046	3.99

Table 4. Hazard Rate Ratios from Full Cox Proportional Hazard Rate Model. Males Only.

	Full Male Sample			Males Age at Last Birth<50		
Variable Label	Hazard Ratio	P	Chi-Square	Hazard Ratio	P	Chi-Square
Birth Year	1.00	<.0001	19.11	1.00	<.0001	17.83
Pa Dage 75-90pct	0.88	<.0001	23.30	0.88	<.0001	21.71
Pa Dage 90-95pct	0.83	<.0001	18.73	0.83	<.0001	17.56
Pa Dage 95-99 pct	0.86	0.001	10.30	0.87	0.004	8.55
Pa Dage >=99 pct	0.66	<.0001	20.69	0.67	<.0001	18.31
Ma Dage 75-90pct	0.98	0.359	0.84	0.97	0.310	1.03
Ma Dage 90-95pct	1.04	0.368	0.81	1.03	0.521	0.41
Ma Dage 95-99 pct	0.91	0.030	4.69	0.91	0.045	4.00
Ma Dage >=99 pct	0.75	0.001	11.19	0.75	0.001	10.18
Familial excess longevity	0.95	<.0001	135.81	0.95	<.0001	125.39
FEL not estimable	1.42	0.354	0.86	1.36	0.419	0.65
Has One Sib	0.99	0.612	0.26	1.00	0.890	0.02
1st born of Sib pair	1.03	0.316	1.01	1.01	0.613	0.26
Committed to LDS	0.82	<.0001	92.58	0.83	<.0001	72.14
Baptized as Child in LDS	1.02	0.496	0.46	1.02	0.407	0.69
Maternal Age <20	1.06	0.073	3.22	1.07	0.055	3.67
Maternal Age 30-35	1.01	0.796	0.07	1.00	0.978	0.00
Maternal Age 35+	1.08	0.015	5.87	1.06	0.053	3.75
Paternal Age <20	1.09	0.459	0.55	1.06	0.635	0.23
Paternal Age 30-39	1.00	1.000	0.00	1.00	0.873	0.03
Paternal Age 40-49	0.99	0.815	0.05	1.00	0.931	0.01
Paternal Age 50-69	1.01	0.806	0.06	1.02	0.646	0.21
Paternal Age 70+	1.07	0.627	0.24	1.10	0.515	0.42
Dad Died before R was 20	0.95	0.039	4.25	0.95	0.088	2.90
Mom Died before R was 20	0.99	0.631	0.23	0.99	0.668	0.18
Orphaned before R was 20	1.02	0.785	0.07	1.05	0.409	0.68
Father SES (Nam-Power)	1.00	0.336	0.93	1.00	0.322	0.98
Father SES not estimable	1.01	0.818	0.05	1.02	0.548	0.36
Own SES (Nam-Power)	1.00	0.000	14.58	1.00	0.001	11.51
Own SES not estimable	1.03	0.139	2.19	1.03	0.155	2.03
Male ego farmer	0.90	<.0001	15.19	0.92	0.001	10.14
Sp died by Egos age 50	1.10	<.0001	24.44	1.06	0.010	6.59
Ego-sp age				1.00	0.241	1.37
ALB 35-44				0.98	0.375	0.79
ALB 45+				0.95	0.196	1.67
Nulliparous/Unk FertHx				1.05	0.229	1.45
Parity=3-5				0.94	0.132	2.27
Parity=6-8				1.02	0.677	0.17
Parity=9-11				1.01	0.824	0.05
Parity 12+				1.10	0.116	2.47

Table 5. Odds Ratios from Logistic Regression for Living to the top 5th Percentile for Age at Death. All Variables Included. Females Only.

Variable Label	OR	95% CI		P	Chi-Square
Intercept				<.0001	83.86
Birth Year	1.04	1.03	1.05	<.0001	75.84
Pa Dage 75-90pct	1.10	0.87	1.40	0.441	0.59
Pa Dage 90-95pct	1.11	0.78	1.58	0.557	0.35
Pa Dage 95-99 pct	1.71	1.20	2.44	0.003	8.77
Pa Dage >=99 pct	1.16	0.60	2.25	0.667	0.19
Ma Dage 75-90pct	1.23	0.97	1.55	0.086	2.96
Ma Dage 90-95pct	1.48	1.04	2.10	0.029	4.78
Ma Dage 95-99 pct	1.85	1.31	2.62	0.001	11.98
Ma Dage >=99 pct	3.11	1.79	5.43	<.0001	16.07
Familial excess longevity	1.21	1.16	1.26	<.0001	82.36
FEL not estimable	2.41	0.30	19.44	0.410	0.68
Has One Sib	0.74	0.55	0.99	0.040	4.20
1st born of Sib pair	0.97	0.76	1.24	0.791	0.07
Committed to LDS	1.14	0.94	1.39	0.194	1.69
Baptized as Child in LDS	1.23	0.96	1.57	0.096	2.78
ALB 35-44	1.21	0.96	1.51	0.109	2.56
ALB 45+	1.40	0.83	2.36	0.207	1.60
Nulliparous/Unk FertHx	0.73	0.52	1.02	0.065	3.42
Parity=3-5	1.05	0.76	1.44	0.782	0.08
Parity=6-8	0.91	0.66	1.27	0.579	0.31
Parity=9-11	0.81	0.54	1.21	0.302	1.07
Parity 12+	0.56	0.31	1.01	0.053	3.75
Maternal Age <20	0.83	0.58	1.17	0.286	1.14
Maternal Age 30-35	0.85	0.64	1.12	0.245	1.35
Maternal Age 35+	0.97	0.73	1.28	0.803	0.06
Paternal Age <20	1.18	0.42	3.35	0.757	0.10
Paternal Age 30-39	1.07	0.84	1.36	0.579	0.31
Paternal Age 40-49	1.06	0.78	1.44	0.708	0.14
Paternal Age 50-69	1.18	0.81	1.71	0.387	0.75
Paternal Age 70+	0.57	0.07	4.41	0.591	0.29
Dad Died before R was 20	0.70	0.53	0.92	0.010	6.70
Mom Died before R was 20	1.05	0.79	1.39	0.733	0.12
Orphaned before R was 20	1.36	0.83	2.25	0.227	1.46
Father SES (Nam-Power)	1.01	1.00	1.02	0.039	4.27
Father SES not estimable	1.23	0.97	1.55	0.088	2.90
Sp died by Egos age 50	0.90	0.73	1.11	0.328	0.96
Ego-sp age	0.99	0.97	1.01	0.275	1.19

Table 6. Odds Ratios from Logistic Regression for Living to the top 5th Percentile for Age at Death. All Variables Included. Males with Age at Last Birth < 50.

Variable Label	OR	95% CI		P	Chi-Square
Intercept				<.0001	33.61
Birth Year	1.02	1.01	1.03	<.0001	28.23
Pa Dage 75-90pct	1.55	1.23	1.96	0.000	14.02
Pa Dage 90-95pct	1.68	1.20	2.37	0.003	8.88
Pa Dage 95-99 pct	1.47	1.01	2.15	0.044	4.06
Pa Dage >=99 pct	2.99	1.69	5.30	0.000	14.10
Ma Dage 75-90pct	0.93	0.73	1.19	0.574	0.32
Ma Dage 90-95pct	0.85	0.57	1.26	0.407	0.69
Ma Dage 95-99 pct	1.18	0.81	1.70	0.391	0.73
Ma Dage >=99 pct	2.18	1.25	3.81	0.006	7.50
Familial excess longevity	1.20	1.16	1.25	<.0001	81.27
FEL not estimable	<0.001	<0.001	>999.999	0.966	0.00
Has One Sib	1.04	0.78	1.37	0.811	0.06
1st born of Sib pair	0.85	0.67	1.08	0.184	1.77
Committed to LDS	1.67	1.36	2.05	<.0001	24.17
Baptized as Child in LDS	1.04	0.82	1.30	0.772	0.08
ALB 35-44	1.21	0.95	1.54	0.130	2.29
ALB 45+	1.33	0.95	1.86	0.100	2.70
Nulliparous/Unk FertHx	0.94	0.65	1.36	0.738	0.11
Parity=3-5	1.34	0.95	1.89	0.096	2.77
Parity=6-8	1.02	0.72	1.46	0.897	0.02
Parity=9-11	0.98	0.64	1.51	0.924	0.01
Parity 12+	0.55	0.27	1.09	0.085	2.97
Maternal Age <20	0.82	0.59	1.14	0.240	1.38
Maternal Age 30-35	0.84	0.64	1.10	0.196	1.67
Maternal Age 35+	0.71	0.54	0.94	0.015	5.89
Paternal Age <20	1.15	0.35	3.79	0.813	0.06
Paternal Age 30-39	0.99	0.78	1.25	0.902	0.02
Paternal Age 40-49	0.99	0.74	1.32	0.929	0.01
Paternal Age 50-69	0.95	0.66	1.36	0.774	0.08
Paternal Age 70+	0.37	0.05	2.78	0.332	0.94
Dad Died before R was 20	1.22	0.95	1.57	0.117	2.46
Mom Died before R was 20	0.92	0.69	1.23	0.576	0.31
Orphaned before R was 20	1.04	0.56	1.92	0.904	0.01
Father SES (Nam-Power)	1.00	1.00	1.01	0.601	0.27
Father SES not estimable	0.88	0.69	1.13	0.325	0.97
Sp died by Egos age 50	0.93	0.75	1.15	0.480	0.50
Own SES (Nam-Power)	1.00	1.00	1.01	0.142	2.16
Own SES not estimable	1.30	1.02	1.65	0.032	4.62
Male ego farmer	1.02	0.82	1.27	0.863	0.03
Ego-sp age	0.99	0.96	1.02	0.521	0.41