

Differential fitness costs of reproduction between the sexes

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Natural selection does not necessarily favor maximal reproduction because reproduction imposes fitness costs, reducing parental survival, and offspring quality. Here, we show that parents in a preindustrial population in North America incurred fitness costs from reproduction, and women incurred greater costs than men. We examined the survivorship and reproductive success (Darwinian fitness) of 21,684 couples married between 1860 and 1895 identified in the Utah Population Database. We found that increasing number of offspring (parity) and rates of reproduction were associated with reduced parental survivorship, and significantly more for mothers than fathers. Parental mortality resulted in reduced survival and reproduction of offspring, and the mothers' mortality was more detrimental to offspring than the fathers'. Increasing family size was associated with lower offspring survival, primarily for later-born children, indicating a tradeoff between offspring quantity versus quality. Also, we found that the costs of reproduction increased with age more for women than men. Our findings help to explain some puzzling aspects of human reproductive physiology and behavior, including the evolution of menopause and fertility declines associated with improvements in women's status (demographic transitions).

maternal depletion | menopause | reproductive effort | biodemography | life-history tradeoff

The idea that reproduction confers fitness costs and benefits is central to evolutionary life-history theory, and evidence for reproductive costs has been found in a variety of species (1–3), including humans (4, 5). The evidence in humans is mixed (6) and controversial (7, 8), and so more studies would help to solve this important question. Also, it is widely assumed that women have higher costs of reproduction than men because of enduring pregnancy, childbirth, and lactation, but evidence is lacking. Studies on women show that high parity and low birth spacing are associated with reduced health (9), postreproductive survival (10–13), and offspring quality (14, 15). There are only a few studies on men, and they suggest that men incur no fitness costs for reproduction (16, 17). Taken together, the evidence supports the idea that women incur greater reproductive costs than men. However, the studies on men were mainly conducted on aristocrats and industrial societies, and men in most societies probably have not been so sheltered from the costs of reproduction. Thus, more studies are needed, especially large, longitudinal studies on couples living in preindustrial societies. Our aim was to examine the fitness costs of reproduction for husbands versus wives in an historical, 19th-century population in North America facing resource limitations, because a previous study suggested that both sexes in this population incurred fitness costs for reproduction (12).

Determining the relative costs of reproduction for the sexes is relevant to understanding the evolution of some puzzling aspects of human physiology and behavior. For example, menopause is difficult to understand from an evolutionary perspective because there are no obvious benefits for reproductive cessation (and menopause is not an artificial consequence of increased longevity from modern medicine, because women in traditional soci-

eties commonly survive long after menopause; ref. 18). Menopause may enable women to stop reproducing around the age when reproduction becomes too risky or harmful for their health (or the quality of their offspring), and the fitness costs begin to outweigh the benefits (19). This “stopping early” or “prudent mother” hypothesis (20) assumes that women incur greater age-dependent costs from reproduction than men, but this hypothesis has not been tested. Another evolutionary puzzle is explaining the declines of human fertility in economically developed countries (demographic transitions) (21). Parents may be trading offspring quantity for quality, as extraordinary levels of investment are often necessary for children to obtain the skills needed to be competitive (4, 22). Yet, fertility declines are most closely linked to improvements in women's education and reproductive autonomy, suggesting that when given the opportunity, women prefer smaller families perhaps to reduce their costs (or perceived costs) of reproduction (23).

To assess the relative fitness costs of reproduction for men and women, we analyzed genealogical data from the Utah Population Database, for which survival information is complete for couples and their offspring. These individuals lacked modern fertility control and couples marrying from 1860 to 1874 were exposed to hardships of migration to the Western frontier, including uncertain food supplies, limited medical care, and physical hazards. Those marrying between 1875 and 1895 experienced the beginning of the fertility transition, were more likely to be native-born Utahns, and encountered fewer hardships because transportation and a developing infrastructure in the West enhanced the quality of life (24). To estimate the association between survival of husbands, wives, and their children and the couple's reproductive history, we used proportional hazards regression models. The large size of our sample (>21,000 couples) provided strong statistical power, which is important because even small fitness differences can have large evolutionary consequences.

Results

We examined how increasing parity affected the survival of couples during the first year and the first five years after the completion of childbearing (age at last birth). Mean age at last birth was 38.5 years (range 20–51). This analysis focuses on the five years after a given couple bears their last child [supporting information (SI) Table 1]. We found that parental survivorship declined with increasing parity for both sexes ($P < 0.001$ for both sexes) during the first year and the first five years after the last birth, and increasing parity had a significantly greater impact on women's survival (Fig. 1a) compared with men's (Fig. 1b).

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Abbreviation: SES, socioeconomic status.

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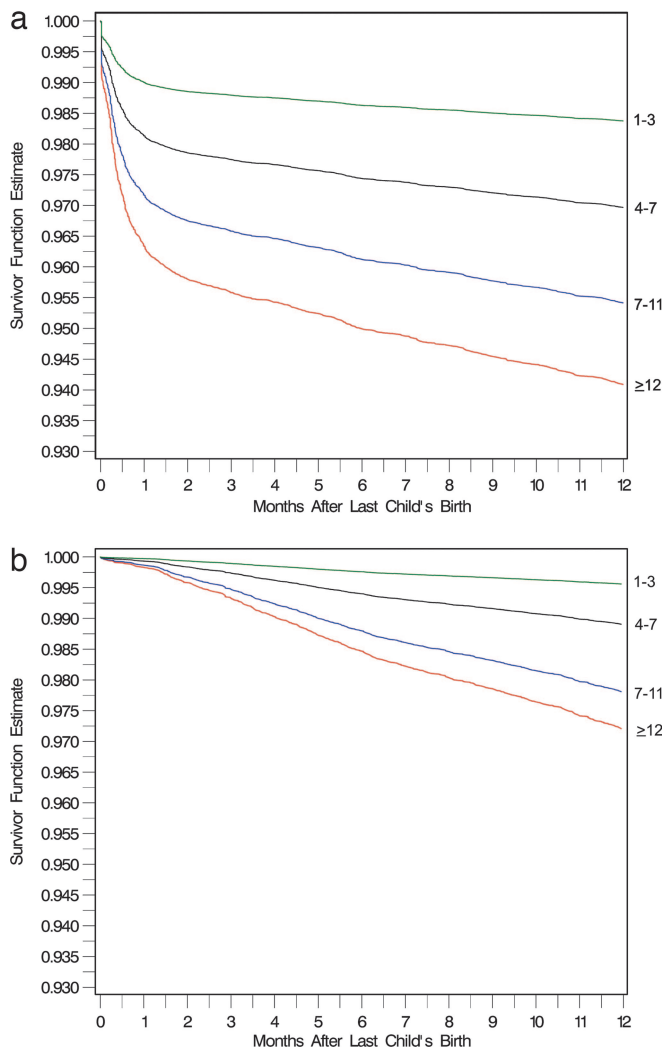


Fig. 1. Survivorship of mothers (a) and fathers (b) during the first year after the last child's birth by parity. The greatest relative mortality risk for women in relation to men occurred at low levels of completed family size [relative risk for mortality of 3.05 ($P < 0.001$) for those with 1–3 children; detail of results in SI Table 2]. This effect occurs because the mortality risk was high for first- or second-time mothers but very low for comparable fathers. If women survived their early reproductive years, their absolute mortality risk increased with additional children, but it rose more sharply for men (in part because men's mortality risks generally rise quicker with age than women's during middle adulthood), although risks of paternal mortality never exceeded those for maternal mortality; those with a completed family size of 4–6, 7–11, 12 or 13, and 14 or more children had relative risks for mortality (between women and men) of 2.65, 2.18, 2.34 (all $P < 0.001$), and 2.74 ($P = 0.016$), respectively, in relation to those with 1–3 children. These sex differences have been adjusted for year of marriage, age at first birth, age at last birth, and child mortality.

Women had significantly higher risks of mortality than men at all levels of parity and worsened with increasing parity (SI Table 2). We also examined parental survivorship after age 50 (postreproductive years for women), and again found a maternal survival disadvantage associated with increasing parity ($P < 0.0001$). The effect of parity on paternal mortality past age 50 was insignificant. To test whether the costs of reproduction increase with age more for women than men, we identified individuals by age at which they had their last child, and then compared the risks of mortality (out to one year) by age at last birth and by sex. Fig. 2 shows that the risk of mortality after childbirth increased with age, and this risk was greater for women than men ($P < 0.0001$).

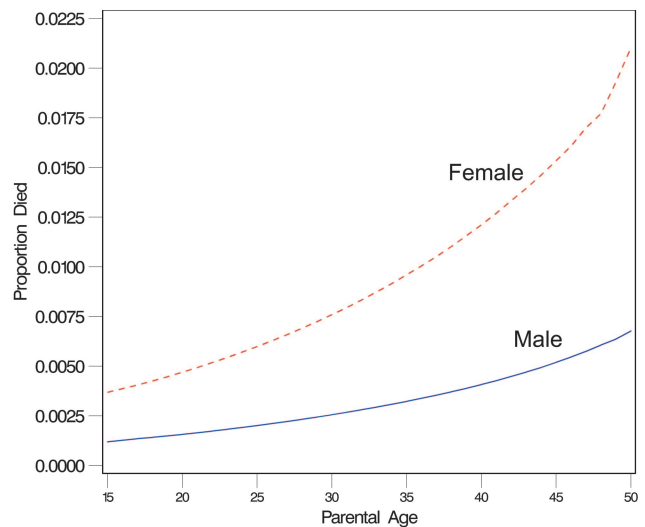


Fig. 2. Age-specific proportions of parents who died within one year after the last birth by sex.

These results indicate that parents of both sexes incurred a survival cost from high parity, but especially for women, and this cost persisted into women's later (postreproductive) life, past age 50.

We addressed whether these findings might be confounded by some other factors not considered in our models. For example, the positive association between parity and mortality could simply be due to couples with low socioeconomic status having high fertility while also having high mortality. To consider this possibility, we expanded our analysis to include socioeconomic status (SES) as an additional covariate (25). The SES measure is based on industry and occupation (25) identified from death certificates after 1904, the first year they were available for Utah death certificates (for males, their own SES is used but for females, their husbands' SES is used instead because women's own SES is not informative for this cohort). This restriction resulted in a smaller sample size of 5,168 couples. Not surprisingly, low SES was associated with increased mortality risk (SI Table 3), which gives us confidence that we are measuring socioeconomic status. When SES is controlled, we found that increasing parity continues to be associated with elevated mortality risks during the five years after the last birth (for women, $P < 0.0001$; for men, $P = 0.21$) and for mortality past age 50 (for women, $P < 0.003$; for men, $P = 0.15$), a result that does not change appreciably whether the model adjusts for SES (SI Table 3). This result suggests that the strong association between parity and parental mortality (primarily for females) is not confounded by SES. To address the possibility that there is still some other factor, besides SES, not considered in our models that explain our findings, we controlled for all unobserved factors that are shared by parents by introducing a random effects term (e.g., frailty) in the Cox models regarding survival during the first five years after the last child's birth. This analysis allows us to test the possibility that some couples are more prone to mortality than others after the time of the last child's birth for reasons other than those included in the model. We found that adverse effects of parity among women in relation to men are unchanged and that the random effect is nonsignificant ($P = 0.81$), which means that the stronger association between parity and mortality for females is not confounded by unobservable factors shared by spouses. Thus, we can be confident our results are not due to SES or other potential confounding factors.

We also tested whether the rate of reproduction (mean birth intervals) affected parental survival while controlling for parity.

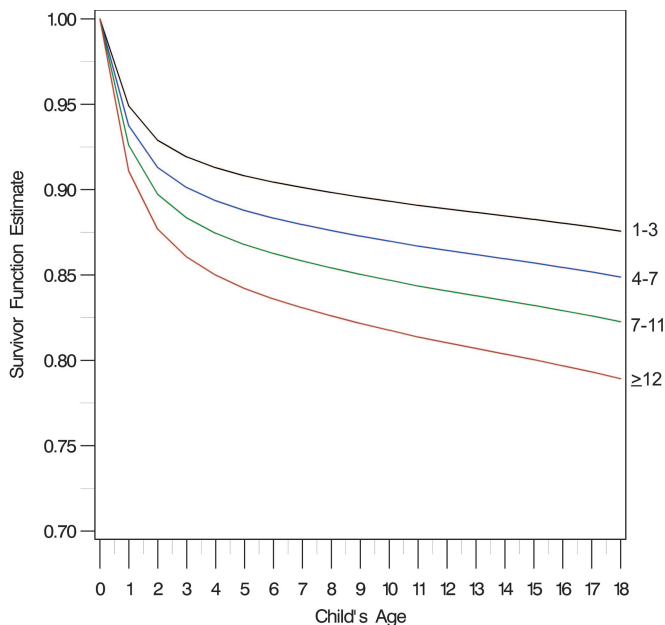


Fig. 3. Child survivorship by sibship size (up to age 18).

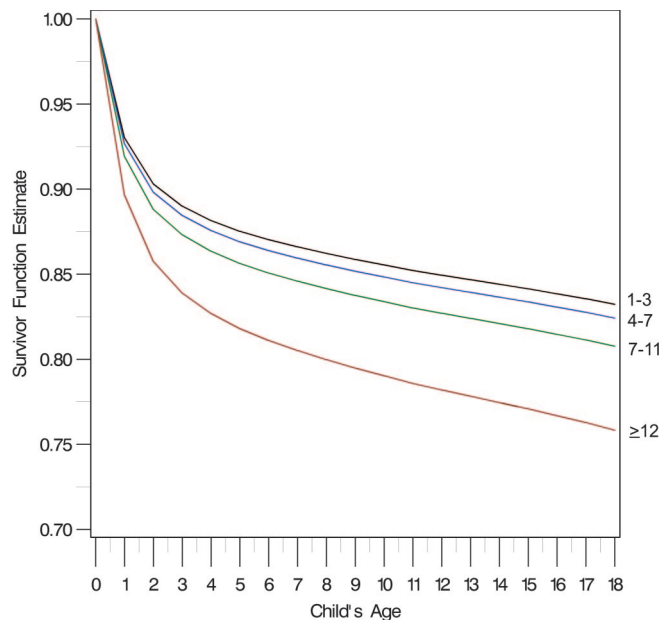


Fig. 4. Child survivorship by birth order (to age 18).

Parental survivorship during the five years after the completion of childbearing declined with lower birth spacing for both sexes, but the adverse effect of short birth intervals was stronger for women than men ($P < 0.001$) (SI Table 4). The excess mortality risk among parents was present both before and after controlling for parity. Some families had short birth intervals because of high infant and child mortality. These child deaths may arise because of adverse living conditions that also imperil the lives of their parents, particularly mothers. Adjusting for infant and child deaths did not substantively alter our previous finding (SI Table 4). Later age at first birth was associated with higher mortality during the first five years after the completion of childbearing, but it did not affect the survival of men and women differently ($P = 0.13$). These results provide further evidence that parents, and especially women, incurred survival costs from reproduction.

We examined whether high fertility affected offspring quality, and found that children with more siblings were less likely to survive to reproductive age (age 18; Fig. 3; $P < 0.001$). Birth order had a significant effect on children's survival ($P < 0.0047$), with later-born offspring in large families (birth order 12 or higher) having the lowest survival (Fig. 4). These results are consistent with a cost of reproduction due to a tradeoff between investing into offspring quantity versus quality.

Because high fertility was associated with reduced parental longevity, we examined how parental mortality affected offspring survival and reproduction. We found that offspring survival was more sensitive to the death of the mother [relative risk of child dying by age 18 was 1.78 ($P < 0.001$) if the mother died before child was age 5] than the father [relative risk = 1.14 ($P = 0.013$) if the father died before child was age 5], indicating that mothers' survival was more important for offspring survival than fathers' ($P < 0.001$) (SI Table 5). We examined how parental mortality affected offspring reproductive success by estimating the differences in the number of grandchildren among couples where a parent dies within five years of the last child's birth. On average, each couple bore 8.04 children (range 1–19) and had 15.35 grandchildren (range 0–102). Mothers who died prematurely were associated with 3.22 fewer grandchildren compared with mothers who did not ($P < 0.0001$). The comparable decline among deceased fathers was 1.71 fewer grandchildren ($P < 0.0001$). This gender difference in offspring repro-

ductive success is significant ($P = 0.0039$) and cannot be explained by offspring mortality. When we restrict the analysis to offspring who survived to sexual maturity, we still observe a reduction in the number of grandchildren if the mother (2.07 decline, $P < 0.0001$) or father dies (0.62 decline, $P = 0.161$) compared with relevant controls, and, again, the gender differences in the number of grandchildren is significant ($P = 0.014$) (SI Table 6).

Premature maternal mortality may have reduced numbers of grandchildren because it reduced assistance with children's reproduction (grandmother effects) or because it reduced offspring numbers or survival to reproductive age (mother effects). The grandmother hypothesis would be supported if maternal mortality still reduced grandchildren numbers for a given level of parity. We found that although mothers who died prematurely (< age 30) produced fewer grandchildren (surviving to age 18), they also had 8.5 fewer surviving offspring than other mothers ($P = 0.0001$), and the effect of maternal mortality on grandchildren is no longer significant when parity is controlled ($P = 0.57$) (SI Table 7). We examined the possibility that there was an interaction, such that the effect of maternal mortality on grandchildren numbers depended on parity. For a given parity, women who died prematurely produced fewer grandchildren than women who lived past 30, which is suggestive of grandmother effects, but this interaction is not significant ($P = 0.13$). We found no such effects from paternal mortality. These results suggest that premature maternal mortality reduced grandchildren mainly by reducing the number of adult offspring.

Discussion

We found evidence that both mothers and fathers incurred fitness costs from reproduction in this population, because their survival declined with both increasing parity and rates of reproduction. This finding is consistent with predictions from life-history theory and evolutionary theories for aging (26). We also found that the costs of reproduction on parental longevity were greater for mothers than fathers. This sex bias was not merely a consequence of childbirth mortality, because it was still significant even after controlling this factor in our analyses. Furthermore, the detrimental effects of high fertility persisted into women's later postreproductive years, indicating greater long-

lasting adverse health effects relative to men (12). We found no evidence that these associations were due to social status or other unobserved confounding factors shared within couples (frailty analyses). Other studies have found that short birth intervals are associated with increased morbidity and mortality for women (27). The physiological mechanisms underlying the impact of reproduction on parental longevity are unclear, although for women, they may be due to insufficient time to recover from the stress from the previous pregnancy (“maternal depletion syndrome” hypothesis) (28). Pregnancy involves calcium loss and other nutritional deficits, oxidative stress, and reduced immunological resistance to infectious diseases (9, 29–31). Mothers stressed from rearing chronically ill children that require enormous investment have short telomeres for their age, suggesting oxidative stress and cellular senescence (32), but it is not known whether fathers incur similar effects. More work is needed on the physiological consequences of reproductive stress for both sexes.

High fertility was associated with reduced parental survival, and, interestingly, we found that premature mortality of mothers was particularly associated with reduced survival and reproduction of offspring. The reduction in grandchildren was not merely due to the death of offspring and might have been due to a reduction in grandmother’s support for grandchildren (the grandmother hypothesis) (33, 34). However, our analyses suggest that the reduction in grandchildren associated with maternal mortality was mainly due to a reduction in the number of adult offspring.

We found that offspring from large families suffered higher mortality than other families, suggesting that natural selection does not necessarily favor maximizing rates of reproduction in preindustrial societies, despite what is sometimes assumed (35). This result provides evidence that parents also incurred a fitness cost from reproduction due to a life-history tradeoff between offspring quantity versus quality, as also observed in more traditional societies (14, 15, 36).

A few caveats regarding our findings should be considered. First, although our results are consistent with the idea that reproduction incurs costs, and particularly for women, they do not demonstrate cause and effect, as causal inference is impossible in studies lacking experimental manipulations. Some field experiments with birds are consistent with our findings (37, 38); however, they were not able to measure parental survival and cannot rule out the possibility that females were more likely than males to disperse from the study site. Second, the gender differences we found may not be a universal feature of humans, and future research should examine variation among populations. Women’s reproductive costs may be lower in matrilineal societies in which living arrangements and institutions are less biased toward men’s interests (matrilineal societies are rare; however, they may have been more common before the spread of intensive agriculture). On the other hand, mothers in this predominately Mormon population may have had unusually strong support from kin and community and, subsequently, incurred relatively low costs of reproduction. Finally, men’s costs of reproduction may be mainly due to mating effort rather than reproduction *per se* or parental effort, which can include extra-pair matings, and our results are limited to the costs of rearing within-pair offspring.

Our findings offer implications for the evolution of senescence (26) and several interesting aspects of human reproductive physiology and behavior. We found that mothers incurred higher costs of reproduction than fathers, these costs increased with age more for mothers than fathers, and mothers’ mortality was more detrimental for offspring survival and reproduction than the fathers’. These findings support the hypothesis that menopause functions to increase the chances that mothers will survive longer to raise their existing offspring (prudent mother hypothesis)

(19). Another explanation for menopause is that natural selection has favored greater longevity in our species so that women can enhance the survival of their grandchildren (grandmother hypothesis) (33, 34, 39). The grandmother hypothesis potentially explains the evolutionary benefits of women’s postreproductive survival, but it does not explain why women cease reproducing or why only women, and not men, undergo menopause. In contrast, these peculiar traits can be explained by sex-specific costs of reproduction. The prudent mother and grandmother hypotheses are not mutually exclusive alternatives because if women have too many children and overshoot their reproductive optimum, they may not survive long enough to help their grandchildren (40). We found that high fertility reduced women’s survival and postreproductive longevity, and premature mortality reduced the number of surviving offspring. This reduction in offspring was sufficient to explain the reduced number of grandchildren, which supports the prudent mother hypothesis, whereas we found no significant evidence to support the grandmother hypothesis. Thus, menopause appears to allow mothers to live longer and rear more offspring to adulthood, and this unusual life history probably evolved in our species because, as we found, offspring are so extremely dependent on their mother’s survival.

Our results also help explain differences in mating preferences between the sexes. Women are thought to be more choosy about sexual partners than men because of their greater investment into reproduction (41), and our findings support this idea. They also help explain why, when seeking potential mates, women emphasize social status, economic resources, and parental investment, whereas men emphasize youth, waist-hip ratio, and other indicators of high reproductive value (42–45). Women’s preferences may help to minimize their costs of reproduction, and men’s preferences may function to obtain a mate who can bear high costs of reproduction. For example, waist-hip ratio indicates a woman’s health and viability, as well as fecundity (44) and, thus, appears to signal a woman’s reproductive value, which includes the ability to bear the costs of reproduction. Furthermore, if there is variation among societies in relative costs of reproduction for the sexes generated, for example, by kinship or other support, then this biological variation might help to explain some of the cultural differences in mating preferences.

We found that high fertility was associated with lower offspring survival and reproduction, which is relevant to understanding the evolution of sibling rivalry and family dynamics. In contrast to the “classical” view that mothers and fathers work harmoniously for the interests of the family (46), evolutionary biology predicts intrafamilial conflicts, especially over the flow of parental investment (47, 48). We found that high fertility was most detrimental for later-born offspring (significant only for birth order 12 or higher), and this result is consistent with other evidence that later-borns sometimes receive low parental investment (49). It is controversial, however, whether birth order has any subsequent effects on the development of behavior (50, 51).

Finally, modern fertility declines associated with increased infant survival and economic development have been difficult to understand from an evolutionary perspective (21). Such declines are most closely associated with improvements in women’s reproductive autonomy. This response may be due to women bearing more economic costs of reproduction than men, but this tradeoff may not be the entire explanation (23). Social scientists have been surprised to find sexual conflicts over reproduction (52), whereas evolutionary biologists expect sexual conflicts in humans and other nonmonogamous species (41, 53, 54). If women have generally incurred greater fitness costs of reproduction, this life history tradeoff could help explain why they generally prefer fewer offspring than their husbands and reduce their fertility when they obtain more reproductive autonomy (23). Conflicts over family size often involve in-laws, and our

findings help to explain why the presence of maternal in-laws lower a couple's fertility, whereas paternal in-laws increase it (55, 56); maternal in-laws have more evolutionary interests in the mother's health and reproductive costs than paternal kin. Thus, sexual asymmetries in reproductive costs and investment may contribute to familial and sexual conflicts (20).

Methods

Population Sample. The genealogical data from the Utah Population Database, established in 1974, contains information from 1.6 million individuals representing $\approx 185,000$ "Family Group Sheets" abstracted from the Utah Family History Library, of which $\approx 65\%$ of the couples were members of the Church of Jesus Christ of Latter-day Saints (the LDS Church or "Mormons"). Since 1974, the Utah Population Database has been enhanced with vital status information from Utah death certificates and the Social Security Death Index. Our sample consisted of 43,368 individuals from 21,684 once-married couples who bore 174,234 children, an average of 8.04 children per couple. We selected these couples based on several criteria. (i) They were married between 1860 and 1895, a time interval that largely excludes the availability of modern contraception. (ii) They were once-married, which limited analytic complications related to fertility spanning multiple spouses. (iii) They had to have at least one child by age 15 or older. (iv) Their marriages ended only by death. (v) Polygynous marriages were excluded because these cases applied only to a small percentage of privileged men ($<10\%$) concentrated during the earliest marriage cohort (57). (vi) Consanguineous marriages, although rare, also were excluded. Among wives, 1,414 died within one year after the birth of their last child, and a total of 2,402 died five years after their last child's birth. For husbands, the number of deaths for these intervals was 613 and 1,696, respectively. By age 18, $\approx 18\%$ ($n = 22,536$) of the children died.

Statistical Analyses. We used SAS (PROC PHREG; SAS Institute, Cary, NC) to estimate Cox proportional hazard rate models for mothers and fathers combined, mothers-only, fathers-only, and children. For the parents' equations, survival spans the first 12 or first 60 months after the last child is born. To test for gender differences in the effects of reproduction on

parental survival, we analyzed mothers and fathers jointly. For the children's equations, survival spans the time between birth and age 18. Cox models are also estimated as before for both parental mortality and offspring mortality except that a frailty (random-effects) component is added by using SPlus. For models assessing the mortality of parents, the random effects control for the shared environment among spouses in a given marriage. For models of child survival, the random effects controls for both the shared early environment among siblings and their shared genetic effects. This means that factors that are shared (either among spouses or among siblings) but are not directly measured in the data are controlled for statistically (58). For all models, the estimated frailty component is not significant. Accordingly, our results are based on Cox models that exclude the frailty correction. The independent variables used in the parents' mortality models are year of marriage, wife's age at first and last birth, parity, whether the last child died, and affiliation with the LDS Church. The independent variables we used in the children's mortality equations were birth year, whether mother or father was alive when their last child was 5 years old, sibship size, and birth order. When estimating linear regressions to assess variations in the number of grandchildren, the independent variables are whether mother or father survived for five years after her last birth, mother's birth year, mother's affiliation with the LDS Church, and mother's age at first birth. In all models, birth cohort effects were treated as categorical or continuous variables; using either approach did not change the results in any appreciable way. Results of all models are shown in [SI Tables 1–7](#). All graphs are based on models that fully adjust for all covariates listed in the tables and are derived from Cox proportional hazards regression.

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