

Does Having Children Extend Life Span? A Genealogical Study of Parity and Longevity in the Amish

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Background. The relationship between parity and life span is uncertain, with evidence of both positive and negative relationships being reported previously. We evaluated this issue by using genealogical data from an Old Order Amish community in Lancaster, Pennsylvania, a population characterized by large nuclear families, homogeneous lifestyle, and extensive genealogical records.

Methods. The analysis was restricted to the set of 2015 individuals who had children, were born between 1749 and 1912, and survived until at least age 50 years. Pedigree structures and birth and death dates were extracted from Amish genealogies, and the relationship between parity and longevity was examined using a variance component framework.

Results. Life span of fathers increased in linear fashion with increasing number of children (0.23 years per additional child; $p = .01$), while life span of mothers increased linearly up to 14 children (0.32 years per additional child; $p = .004$) but decreased with each additional child beyond 14 ($p = .0004$). Among women, but not men, a later age at last birth was associated with longer life span ($p = .001$). Adjusting for age at last birth obliterated the correlation between maternal life span and number of children, except among mothers with ultrahigh (>14 children) parity.

Conclusions. We conclude that high parity among men and later menopause among women may be markers for increased life span. Understanding the biological and/or social factors mediating these relationships may provide insights into mechanisms underlying successful aging.

PREGNANCY and childbirth exert biological stresses on the human body. The long-term consequences of these events are uncertain, although increasing parity has been correlated with development of a number of adverse health outcomes, including obesity (1), diabetes (2), and cardiovascular disease (3). From an evolutionary perspective, it has been proposed that a tradeoff exists between increased fertility and decreased human life span because resources allocated to fertility and child raising in the early to middle part of life are diverted from somatic maintenance functions that are required for extended longevity (4,5). Actual data relating number of offspring and life span in humans, though, are conflicting (6–13). However, few of these studies have considered populations with large family sizes. Many have additionally been limited by their inclusion of persons with variable social status and wealth, factors that are correlated with both parity and life span.

If a relationship does exist between parity and longevity, we reasoned that it should be easiest to detect in a population with natural fertility patterns, large family sizes, and little variation of possibly confounding variables. We therefore evaluated this issue in the Old Order Amish (OOA) popu-

lation of Lancaster County, Pennsylvania—a population characterized by large family sizes and close knit familial units. Still today, divorce and the use of modern birth control are rare. Importantly, the Amish lifestyle is relatively homogeneous in terms of religious belief systems and socioeconomic status, and has been relatively unchanged across generations. Alcohol consumption, cigarette smoking (especially among women), and sedentary lifestyles are almost nonexistent. Adult mortality patterns among the OOA have been markedly constant, at least for the cohorts born in the 18th and 19th centuries (14). Thus, the Amish provide a well-suited population for a study of this type.

In addition to considering the overall relationship between parity and life span, the rich genealogical information available on the OOA provides the opportunity to evaluate several related issues, including the relationships between age at first and last birth on life span, possible differential effects of sons and daughters on life span, and the relationships between these variables on mothers and fathers separately. Our analyses revealed increasing numbers of offspring to be correlated with increasing life span in both men and women, although in women, life span

decreased, rather than increased, after 14 children. In women, but not men, accounting for age at last birth obliterated the correlation between parity and life span.

METHODS

The OOA maintain extensive genealogical records that indicate that the current OOA population of Lancaster County, numbering about 25,000–30,000 individuals, all descend from approximately 300 founders who immigrated to central Pennsylvania from central Europe beginning in the early 1700s (15). Our recent analysis of Amish genealogies and Y chromosome markers indicates that more than 95% of the OOA gene pool is derived from fewer than 100 founders (T. Pollin, unpublished data, 2004). The genealogy used for this study was constructed by reconciling all the nuclear families in the OOA Lancaster-specific genealogy prepared by King and colleagues (16) with the larger Anabaptist Genealogy Database (AGDB) version 3.0 (17,18). Both the King and AGDB genealogies originated from digitized versions of published information contained in the Fisher Family History (19), with supplemental information added after the 1988 publication of the printed version. We improved the accuracy of the King database by filling in dates, correcting dates, and adding some children who were in the AGDB but missing from the King database. The version of the King genealogy for this study includes 52,620 individuals born between the years 1736 and 1990. Many individuals with life span of at most a few days are included in the genealogy, but it is likely that some infant deaths and stillbirths were not recorded. Birth dates and death dates recorded in the genealogy were used to calculate life span and parental ages at birth of offspring.

The study population for this report was restricted to all individuals represented in the genealogical database who survived to their 50th birthday, who were born prior to 1912, and who had one or more children ($n = 2015$ individuals). We restricted our analysis to those individuals who survived to their 50th birthday to allow women the opportunity to have reached full reproductive opportunity. The purpose of excluding individuals born after 1912 was to maximize the possibility that everyone in the cohort would have died. Thirteen individuals were born prior to 1912 and did not have a recorded death date. However, none of the 13 had children and therefore were excluded from our analysis. The 2015 individuals with nonmissing data were linked into a single pedigree and were analyzed using pedigree-based analytic methods.

The goal of our primary analysis was to evaluate whether a father's or mother's number of offspring was correlated with his or her age at death. We also evaluated whether age at last birth was associated with age of death after accounting for total number of children. The initial analyses were carried out under a linear regression framework, modeling life span as the dependent variable and including parity and selected covariates as the independent variables. Because there is a familial resemblance to human life span (14,20–23), we anticipated that the simple linear regression approach would provide us with unbiased estimates of effect measures, but inflated variances of these parameters. We

therefore repeated all analyses using a variance component modeling framework that allowed us to account for the residual correlations in age at death potentially existing among related individuals (24). Briefly, the variance component approach models the correlations between the independent and dependent variables conditional on the residual correlations among individuals implied by the pedigree structure. Specifically, the covariance between each pair of individuals within the pedigree is estimated as a function of their degree of relationship, the trait heritability, and the phenotypic variance of the trait. The model is thus defined as:

$$Y = x\beta + g + e,$$

where Y is a vector of values correlating to each individual's age at death, x is a matrix of fixed covariates, and β are the effects of interest. The g term is a covariance component that is distributed multivariate normally with a mean of zero and a covariance equal to two times the kinship matrix times the expected variance due to the additive effect of genes. The e term is a normally distributed error component. The likelihood of the pedigree data was then computed under the assumption of multivariate normality. As expected, the point estimates obtained under the simple linear regression and variance component frameworks were similar, although a larger variance for the regression coefficients (and hence wider confidence intervals [CI]) was estimated under variance components. All parameter estimates and significance testing presented in this report are based on the more conservative variance component modeling.

Polynomial regression models were constructed to identify nonlinear relationships between variables (25). The results of the polynomial models were used to determine inflection points for piecewise linear regression models using the above framework. These piecewise linear models were created to aid in the interpretation of the results. Analyses were conducted for men and women separately and were performed using the SOLAR software package (24). All p values reported are two sided.

RESULTS

The study included 2015 individuals (937 mothers and 1078 fathers) born between 1749 and 1912. Summary characteristics of these individuals are shown in Table 1 according to birth cohort and sex. Women had on average (standard deviation) 7.2 (3.5) offspring during this time period, and men 7.4 (3.6). Among women, mean age at last birth was 38.6 (5.2) years, and mean age at death was 76.4 (11.0) years. The corresponding estimates for men were 41.1 (6.3) years and 75.2 (10.6) years for mean age at last birth and age at death. Year of birth was not correlated with number of offspring in either men or women but was positively correlated with life span, with this correlation achieving statistical significance in men only. Results of subsequent analyses were essentially unchanged when the analyses were repeated with year of birth as an additional covariate.

Of the 937 women in our study, 88 (9.4%) had husbands who died prior to their (the wife's) 50th birthday. Because these women might have had limited reproductive

Table 1. Descriptive Characteristics of Old Order Amish Men and Women With Children, by Birth Cohort (Lancaster County, Pennsylvania)*

Year of Birth	Number	Number of Children Mean (SD)	Age at First Birth, Years Mean (SD)	Age at Last Birth, Years Mean (SD)	Age at Death, Years Mean (SD)
Women (N = 937)					
1749–1799	14	8.1 (2.2)	23.2 (2.7)	40.0 (5.5)	75.9 (8.3)
1800–1849	159	6.9 (3.1)	23.7 (3.9)	38.8 (4.7)	75.7 (11.2)
1850–1899	581	7.1 (3.6)	24.5 (4.2)	38.4 (5.4)	76.4 (11.1)
1900–1912	183	7.6 (3.5)	23.6 (3.9)	38.9 (4.8)	77.2 (10.5)
Men (N = 1078)					
1749–1799	14	8.9 (2.0)	23.8 (2.0)	43.2 (3.8)	76.1 (10.2)
1800–1849	182	7.4 (3.4)	26.2 (3.9)	42.3 (6.3)	72.6 (10.4)
1850–1899	654	7.4 (3.6)	26.1 (4.3)	41.0 (6.6)	76.1 (10.9)
1900–1912	228	7.3 (3.8)	25.8 (4.0)	40.2 (5.6)	74.6 (9.6)

Notes: *All men and women lived at least 50 years and were born between 1749 and 1912. SD = standard deviation.

capability, as only seven of the women went on to have children with other men, analyses were performed again with those 88 women removed from the sample. Results of analyses based on exclusion of these women were essentially unchanged.

Heritability

We have previously reported in the OOA that there is significant familial aggregation for both age at death (14) and number of offspring (26). The familial correlations for age at death recalculated in these data correspond to heritability estimates of 31% in men and 25% in women; for number of offspring, the heritability estimates are 22% and 27% in men and women, respectively ($p < .0001$ for all). These estimates represent the proportion of variance in life span (or number of offspring) due to the additive effect of genes, i.e., narrow sense heritability. We then re-estimated the heritability of life span both with and without covariates. The heritability was not appreciably changed for either men or women upon controlling for number of children, age at last birth, or both, suggesting that genes influencing longevity are largely independent of those that influence parity and the ability to reproduce later in life.

Number of Children

Figure 1 shows the distribution of age at death among the 1078 fathers surviving until age 50 or older according to number of children. Longevity increased in linear fashion with the number of children; there was an average 0.23-year increase (95% CI, 0.05–0.40; $p = .01$) in life span with each additional child.

Among the 937 women with children, an association was also observed between number of children and life span, with life span increasing with increasing number of children up to 14 children and then decreasing thereafter. Figure 2 shows results from both a smooth polynomial model and piecewise linear regression. Both the positive association between life span and higher parity prior to the 14th child and the negative association seen afterward were statistically significant. The piecewise regression predicts an increase in

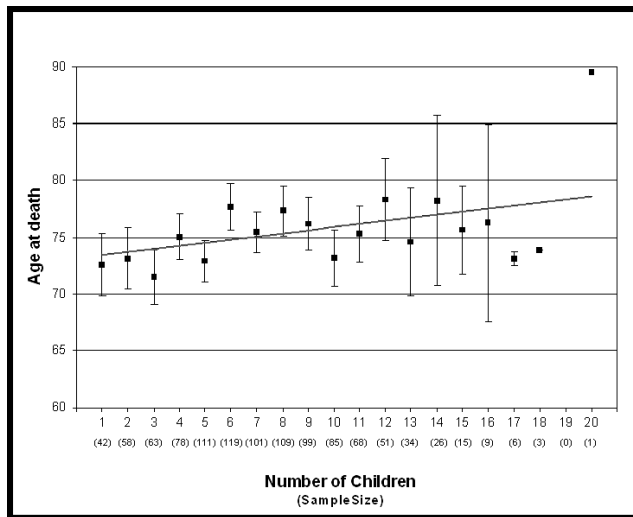


Figure 1. Average life span and 95% confidence intervals for Old Order Amish fathers who lived to at least age 50 years and were born between 1749 and 1912 by the number of children, Lancaster County, Pennsylvania. Average age of death and 95 percent confidence interval by number of children. Trend line given by unadjusted linear regression. Secondary axis gives sample size.

life span of 0.32 years (95% CI, 0.10–0.54 years; $p = .004$) for each additional offspring until child number 14. For each additional child after the 14th, a woman’s life span was predicted to decrease by 4.01 years (95% CI, 1.81–6.20 years; $p = .0004$). Thirty-nine women in the cohort gave birth to 14 or more children.

Age at Last Birth

Older ages of both the mother and father at birth of the last child were significantly associated with increased life

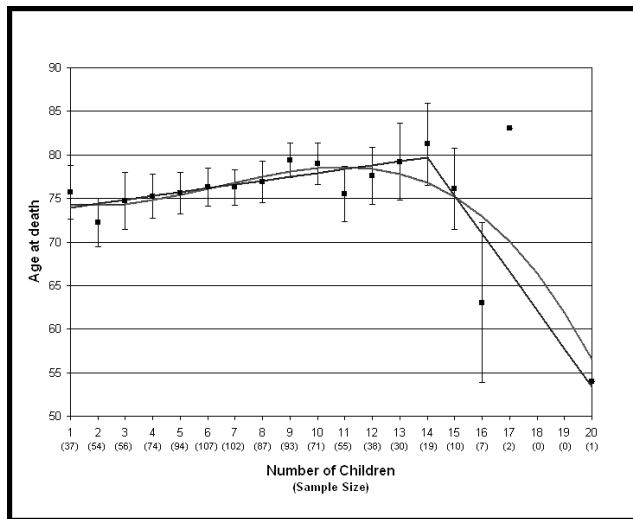


Figure 2. Average life span and 95% confidence intervals for Old Order Amish mothers who lived to at least age 50 years and were born between 1749 and 1912 by the number of children, Lancaster County, Pennsylvania. Average age of death and 95 percent confidence interval by number of children. Trend lines represent piecewise linear regression with one knot and smooth polynomial model as described in text. Secondary axis gives sample size.

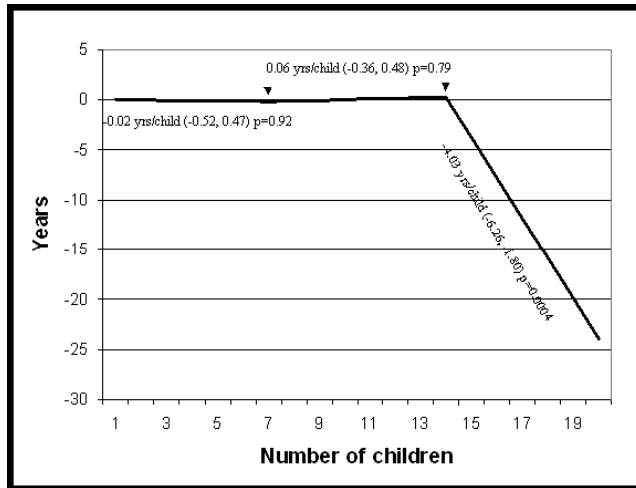


Figure 3. Predicted effect on average life span conditional on age at last birth for Old Order Amish mothers who lived to at least age 50 years and were born between 1749 and 1912 by the number of children, Lancaster County, Pennsylvania. Piecewise linear regression was fit with two inflection points, at 7 and 14 children.

span, with the association particularly strong for the mothers. In men, the effect of age at last birth was completely obliterated after accounting for the number of children fathered, with each additional year of age at last birth associated with an average increase in life span of only 0.0007 years ($p = .91$). This result suggests that the number of children fathered, rather than the timing of the births, is the better predictor of life span.

The reverse was seen among women. After adjusting for the number of children, the age at last birth remained strongly associated with life span, with each additional year of age at last birth associated with an average increase in life span of 0.29 years ($p = .001$). Moreover, after accounting for age at last birth, the correlation observed in women between number of children and life span was eliminated. Figure 3 shows the association between number of children and postreproductive life span after accounting for age at last birth. Age at last birth accounts for the positive association seen in Figure 2 between the birth of a woman's first child and to her 14th. Over a large range of parity values, 1–14 children, parity has little or no association with life span in the presence of a strong positive association between life span and later age childbirth.

Sons and Daughters

Of the 6711 children born to women in our cohort, 3455 (51.5%) were sons and 3254 (48.5%) were daughters. Two children were of unknown sex. Table 2 shows the effects of sons and daughters on life span when modeled separately. We assessed the hypothesis that sons and daughters have differential effects on the life span of their parents by including separate variables for the number of sons and number of daughters into a single model. We then performed a log likelihood ratio test constraining the two effects to be equal (e.g., effect associated with birth of a son equals that associated with birth of a daughter). For neither fathers nor

Table 2. Mean Difference (95% CI) in Life Span of Old Order Amish Men and Women Born Between 1749 and 1912, by Number of Sons and Number of Daughters (Modeled Separately) (Lancaster County, Pennsylvania)

Life Span	Birth of a Son Mean Difference (95% CI)	Birth of a Daughter Mean Difference (95% CI)	Equivalence <i>p</i> Value*
Paternal	0.34 (0.07, 0.61)	0.22 (−0.06, 0.51)	.48
Maternal	0.14 (−0.17, 0.46)	0.32 (0.09, 0.55)	.41
Maternal, controlling for age at last birth	−0.24 (−0.60, 0.12)	0.02 (−0.35, 0.39)	.33

Notes: *Equivalence *p* value determined by likelihood ratio test as described in the text.

CI = confidence interval.

mothers was there significant evidence for a differential effect between birth of a son and birth of a daughter.

DISCUSSION

The OOA provide an excellent opportunity to study the relationship between parity and longevity. Amish families continue to be characterized by large family sizes, with average family sizes remaining relatively constant over the 163-year duration of our study period. Moreover, the very high parity characteristic of some of these families allows us to evaluate the impact of ultrahigh parity on life span. The egalitarian nature of Amish culture offers a further advantage as it reduces or eliminates much of the variation among social lines, such as that attributable to differences in income or access to health care, which could confound the relationship between longevity and family size.

Our analyses revealed a correlation between increasing parity and increasing life span in both women (among those with less than ultrahigh parity) and men. Notably, the correlation observed in women, but not men, was largely due to a later age at last birth, as the parity–life span correlation was essentially eliminated when differences in this variable were taken into account. The correlation observed among women in our study between older age at last birth and longer life span has been reported by others (7,10,27–30).

What are some possible explanations for the parity–life span correlation observed in this population? One likely possibility is that highly parous parents may represent a healthy subset of the population, whose favorable genetic constitutions and/or healthy lifestyles lead them to be both more fertile and to live longer lives. According to this speculation, parity itself may have no direct relationship to life span, but rather high parity may be merely a reflection of men and women who are destined to live long lives.

Notably, the correlation of life span with parity disappears after accounting for age at last birth among women but not men. Possibly, late childbirth in OOA women may be a marker for delayed menopause, which, in turn, could reflect a slower rate of biological aging. In this context, the delayed reproductive aging of these women may be associated with reduced risk or delay of cardiovascular and other diseases in later life. Although attractive, this hypothesis must be

viewed as speculative because historical measures of menopausal status are unavailable from women in our study.

Social factors may also influence the parity–life span relationship. One possibility, for example, is that large family sizes may simply reflect happier marriages, which may in turn be associated with extended life span. Alternatively, large offspring sizes might directly lead to extended parental life span insofar as an increased number of offspring may provide stronger social networks for the parents in their older ages. Thus, the correlation between parity and increased life span might be mediated through social factors that act indirectly by increasing both parity and life span or directly by strengthening familial networks that are valuable for survival into old age.

Our data suggest that life span is reduced among women of ultrahigh parity (>14 children). The reason for this is not evident. Possibly, any social and/or biological benefits associated with multiparity and/or late childbirth are overwhelmed by detrimental effects incurred by repeated pregnancies and childbirths. Several studies [recently reviewed in (31)], have highlighted the risk of adverse maternal and fetal outcomes associated with very high parity, and have concluded that there was “possible evidence” of increased maternal risk (e.g., diabetes, essential hypertension) in these women. However, few studies appear to have directly assessed the long-term survival of ultrahigh parous women.

There are significant heritable components to both fertility and life span, and it is therefore intriguing to speculate whether genes favoring increased parity might also favor increased life span. In our data, we observed no appreciable change in the heritability of life span whether adjusting or not for differences in parity and/or age at last birth; the data provided no strong support for the presence of pleiotropic effects influencing both parity and life span. This finding does not rule out the possibility that such genes might exist, or that genes positively affecting longevity, parity, and delayed menopause, although perhaps different, may all maintain a survival advantage and may be selectively inherited through generations.

We have considered in this article the relationship between parity and life span. Our sample included very few individuals who could be characterized as experiencing extreme longevity, e.g., only five individuals survived to age 100 years. There is some debate as to whether correlates of extended life span will also predict survival to extreme longevity. We found that the oldest old had reproductive profiles similar to those of the rest of the sample (data not shown).

The OOA are an unusual population in terms of their social and cultural heritage. Generalization of our findings to other populations is therefore limited. For example, societies lacking the extensive social network of the Amish might not realize the same potential benefits that may accrue from having large families. It is equally possible that non-Amish families might realize the same or greater potential benefits from having large families in the face of less extensive community social ties.

Analyses for this study were restricted to women (and men) aged 50 years and older to allow women to have achieved their full reproductive potential. Only one woman

in our study had a child after her 50th birthday. This woman went on to live to 77 years of age. Thus no deaths occurring during childbirth were included in our analysis.

Our study has several additional limitations. Foremost among these is that the available genealogical records may not accurately reflect infant deaths occurring during the first year. Thus, our definition of parity should be interpreted in the context of births surviving at least into early childhood. The lack of information regarding pregnancy history precluded our ability to look at additional variables such as total pregnancies, all live births, etc. We also based our analysis on number of children (not number of births) as the independent variable. There were 58 women in our sample (6%) who gave birth to at least one set of twins or triplets. Results were unchanged when mothers with twins were removed from the analysis.

In summary, our data reveal a positive correlation between number of offspring and life span. The correlation may be an indirect one, arising from the fact that healthy individuals are more likely to have large numbers of offspring and to experience a longer life span. There might also be social and/or biological attributes associated with high parity that promote longer life. The positive correlation observed is mediated by factors associated with the age at last birth among women but not men. Further understanding of these factors may improve our understanding of the biological and social mechanisms underlying successful aging.

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Received March 21, 2005

Accepted September 1, 2005

Decision Editor: Luigi Ferrucci, MD, PhD

Editor Nominations

The Gerontologist

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The position will become effective January 1, 2007. The Editor-in-Chief makes appointments to the journal's editorial board and develops policies in accordance with the scope statement prepared by the Publications Committee and approved by Council (see the journal's General Information and Instructions to Authors page). The Editor-in-Chief works with reviewers and has the final responsibility for the acceptance of articles for his or her journal. The editorship is a voluntary position. Candidates must be dedicated to developing a premier scientific journal.

Nominations and applications may be made by self or others, but must be accompanied by the candidate's curriculum vitae and a statement of willingness to accept the position. **All nominations and applications must be received by March 31, 2006.** Nominations and applications should be sent to the Publications Committee, Attn: Patricia Walker, The Gerontological Society of America, 1030 15th Street, NW, Suite 250, Washington, DC 20005-1503.