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Dr. Natalia S. Gavrilova et al.

Center on Aging
NORC/University of Chicago

Address for correspondence:
Dr. Natalia S. Gavrilova, Center on Aging
NORC/University of Chicago
1155 East 60th Street, Chicago, IL 60637
Fax: (773) 256-6313; Phone: (773) 256-6359
E-mail: nsgavril@midway.uchicago.edu

Section 37 "Life Span and Cumulative Risk Approaches to Mortality"

Mechanisms of Familial Transmission of Human Longevity: Comparison of Maternal and Paternal Contributions into Offspring Lifespan

Natalia S.Gavrilova[1,3], Leonid A.Gavrilov[1,2],
Galina N.Evdokushkina[3], Victoria G.Semyonova[3]

[1] Center on Aging, NORC and The University of Chicago, 1155 East 60th Street, Chicago, IL 60637.
[2] A.N.Belozersky Institute at Moscow State University, Moscow 119899, Russia
[3] Institute for Systems Analysis, Russian Academy of Sciences, Moscow, Russia

Abstract. The purpose of this paper is to determine whether human longevity is inherited more strongly along the maternal line (consistent with cytoplasmic mitochondrial inheritance) or there is a predominance of paternal longevity influence (expected as a result of hemizygosity of genes on sex chromosomes in males). For this purpose the genealogical longevity data on European noble families were analyzed for adults (30 years and above) born in 1800-1880. Employing data on more than 800 noble families, we apply multiple linear regression model using 3 independent predictors for offspring life span: paternal age at death, maternal age at death and sex-specific cohort life expectancy. It is found that both paternal and maternal longevity have a positive statistically significant effect on offspring life span. The paternal effect (regression slope) is higher than maternal one at any level of parental longevity and is particularly strong for sons, supporting the hemizygosity hypothesis.

Introduction

In order to understand the mechanisms of human longevity it is important to know the mode of its inheritance. For example, specific transmission of human longevity from father to son only might indicate that human longevity genes are located in Y chromosome. In the case of specific transmission of human longevity from father to daughter only, one may suggest that longevity genes are located in X chromosome since only daughters inherit this chromosome from the father. Strong maternal effect for inheritance of human longevity might indicate the involvement of mitochondrial DNA, since it is inherited from maternal side only. Thus, it is very important to find out what is the relative contribution of maternal and paternal longevity into sons' and daughters' life span and to compare these observations with different models and hypotheses of longevity inheritance.

Studies of familial effects on human longevity have a long history. Next year the scientific community will celebrate the 100th anniversary for the first systematic studies on familial determinants of human longevity. In 1899 the founder of biometrics, Karl Pearson (1857-1936) and his student, Mary Beeton published the very first study on the inheritance of human longevity (Beeton and Pearson, 1899). They analyzed the correlation of parent/child ages at death, based on English genealogies going back to the 17th century (three series of data were taken from the English Peerage and Landed Gentry). Owing to the limitations of their data, Beeton and Pearson dealt with only the adult males age 20 and over. Their second study (Beeton and Pearson, 1901) was based upon more extensive pedigree records of the members of the English Society of Friends and of the Friends' Provident Association (these data included both males and females of all ages). Using such data, Beeton and Pearson (1901) measured the correlation for ages at death not only for parent/child pairs, but also in the siblings. As a result of their studies, Beeton and Pearson concluded that "the expectation of life is seriously modified by the ages of death of the relatives."

The problem of familial transmission of human longevity was also examined by such outstanding investigators as the telephone inventor Graham Bell (1918) on genealogical data of about 3,000 members of the Hyde family in New England and by one of the founders of biodemography, Raymond Pearl (Pearl, 1931; Pearl, Dewitt, 1934; Pearl and Pearl, 1934) who initiated the famous Baltimore Longevity Study.

Following these initial studies on familial transmission of human longevity early in the century, a number of scientists have since devoted their attention to this topic (Wilson and Doering, 1926; Holmes, 1928; Yuan, 1931; Preas, 1945; Dublin et al., 1949; Jalavisto, 1951; Cohen, 1964; Hawkins et al., 1965; Abbott et al., 1974; 1978; Murphy, 1978; Philippe, 1977; 1978; 1980; Welter, 1978; Wyshak, 1978; Glasser, 1981; Crawford and Rogers, 1982; Swedlund et al., 1983; Vandenbroucke et al., 1984; Desjardins and Charbonneau, 1990; Bocquet-Appel and Jakobi, 1990; 1991; Brand et al., 1992; Mayer, 1991; Robine and Allard, 1997; Tallis and Leppard, 1997).

In addition to traditional familial longevity studies, one of the most powerful approaches for assessing genetic and cultural contributions to inter-individual variation in human life span has also been done – the evaluation of the relative longevity of twins (Kallman and Sander, 1948; 1949; Kallman, 1957; Jarvik et al., 1960; Harvald and

Hauge, 1965; Wyshak, 1978; Hrubec and Neel, 1981; Carmelli, Andersen, 1981; Carmelli, 1982; Hrubec et al., 1984; Hayakawa et al., 1992; McGue et al., 1993; Herskind et al., 1996; Yashin and Iachine, 1997).

Studies on the longevity of adopted children were also made and have demonstrated the importance of longevity of the biological parents in predicting offspring longevity (Sorensen et al., 1988; Sorensen, 1991; Nielsen et al., 1992).

Does this brief historical review of scientific literature on familial longevity suggest that all of the concepts, methodology and conclusions have already been established? Surprisingly this seems not to be true, since there is still no consensus even for the most fundamental issues regarding familial longevity. For example, the role of genetics in familial longevity resemblance was challenged by some authors (Murphy, 1978; Philippe, 1978; Jacquard, 1982) who have found very weak familial resemblance and emphasized the importance of social explanations. The mode of longevity inheritance in humans is also not yet determined and there is still a controversy on the relative importance of the maternal versus paternal longevity influence on offspring life span. Is human longevity inherited more strongly along the maternal line (consistent with cytoplasmic, mitochondrial inheritance – Sont and Vandenbroucke, 1993; Wallace, 1995) as would appear to be demonstrated in many studies (Pearl, 1931; Jalavisto, 1951; Abbot et al., 1978; Brand et al., 1992)? Or, on the contrary, is there a predominance of paternal longevity influence on offspring life span as suggested in other studies (Bell, 1918; Cohen, 1964; Philippe, 1978; Welter, 1978; Bocquet-Appel and Jakobi, 1990)?

There are at least two possible flaws in previous studies on this topic that could lead to existing controversies and biased estimates of life span heritability. First, in some studies the birth cohorts were not extinct by the date of data collection (Beeton and Pearson, 1899; 1901; Bell, 1918; Hawkins et al., 1965; Abbott et al., 1973; Murphy, 1978). For this reason the data was biased in favor of shorter-lived persons, because the life span data for longer-living persons was not yet available by the time of data collection (longer-living persons were still alive). The importance of this problem has already been discussed in the scientific literature (Yuan, 1932). Moreover, for those family members whose birth date is close to the truncation date, only the short-lived persons will be included in the analysis, thus producing a spurious correlation between ages at death for short-lived relatives. For this reason, in our study we analyzed only extinct birth cohorts (for people born before 1880).

Another possible flaw of previous studies is the lack of proper control for historical changes in human life expectancy. When the data for families (parents and their children) that lived in different historical periods are mixed in one sample and analyzed together, a spurious artifact correlation between relatives' life span is produced (since life expectancy in early historical periods was low both for parents and their children relative to more recent time periods). That is why the estimates of familial aggregation of human longevity are biased (overstated) in earlier studies not controlled for secular effects (Beeton and Pearson, 1899; 1901; Bell, 1918; Jalavisto, 1951). Moreover, since the secular trends for male and female life span are different, a spurious gender difference in familial transmission of human longevity could be produced in such uncontrolled studies. Some authors tried to regress out the secular effects by introducing the calendar year of death as a covariate in a multivariate analysis (Wyshak, 1978; Bocquet-Appel and Jakobi, 1990). Since the calendar year of death tends to be higher for longer-lived people (the

more you live the later you die), this procedure could decrease not only the noise (secular effects), but also the signal itself (human longevity). Attempts were also made to use the calendar year of birth to control for secular effects, but this variable made virtually no contribution into regression fitting (Wyshak, 1978). In order to resolve this problem we propose to use a novel and what we believe is an improved method for data analysis that allows for the control of strong and complex historical changes in cohort life expectancy (Gavrilov and Gavrilova, 1997b; Gavrilov, Gavrilova et al., 1997b; 1998a; 1998b; Gavrilova et al., 1997a; 1997b; 1998). Our new method is based on the idea of an internal control variable: the sex-specific life expectancy for each birth cohort is calculated and included in the analysis as an independent covariate, so that any historical trends and fluctuations in human life expectancy are regressed out (Gavrilov, Gavrilova et al., 1998a; 1998b; Gavrilova et al., 1997b; 1998).

Data and Methods

Data Sources. There are two mutually exclusive constrains that should be taken into account in familial longevity studies. First of all, the data for those born in 20th century should be excluded from the analysis in order to avoid false familial resemblance caused by life span bias in truncated, not extinct birth cohorts. At the same time, the data collected in the pre-antibiotic era and particularly in the pre public health era is considered to be irrelevant to current low mortality populations (Cohen, 1964; Smith, 1993), since everything was quite different in the past (high mortality from infectious diseases and tuberculosis in particular, under-nutrition and sometimes even starvation (Fogel and Costa, 1997), high poverty rate and poor sanitary conditions, high seasonal mortality in winter period).

There is however one fortunate exception that fits the purpose of this project – socially elite royal and nobility families. We have found that the modal age at death for parents in these socially elite families in 19th century is remarkably high (70-75 years for fathers and 75-80 years for mothers) and comparable with modern low mortality populations.

Another important advantage of this kind of data is its high reliability, accuracy and completeness, since data on noble families was recorded in great detail for many centuries. Also, since this privileged social group lived in rather favorable conditions for many centuries, one could expect less influence of adverse social factors (poverty, for example) on life span and hence lower bias caused by these factors. This kind of data allows us to minimize the social heterogeneity of the population under study and to avoid overstating the familial component of longevity when a mixture of families with different social status is analyzed. Thus, although the sample analyzed in this study is biased towards higher social status and does not represent the whole human population, it is the best possible sample where the effects of population heterogeneity are minimized with regard to social status. Also, all the cases of familial inbreeding are well documented for noble families, so these cases could be studied separately or excluded from the analysis.

In this study we have computerized and analyzed genealogical data on longevity in European noble and royal families published in "Genealogisches Handbuch Des Adels" (1980-1994) and in other professional genealogical sources, listed elsewhere (Gavrilov, Gavrilova et al., 1996).

Offspring longevity was analyzed for adults (those who survived by age 30) in order to eliminate the possible bias in longevity estimates associated with high infant mortality rates and high proportion of premature deaths at young ages due to infectious diseases and violence observed in the 19th century.

Extinct Birth Cohorts. The major bias of genealogical data is observed for those birth cohorts that are not extinct by the date of data collection. Such data is biased in favor of shorter living persons because the lifespan data for longer living persons are not yet available by the time of data collection (longer living persons were still alive).

The shorter the time interval between the birth year for the particular birth cohort and the year of data collection, the stronger is the bias (because of higher truncation rate). Thus, for more recent birth cohorts the mean life span level is more and more underestimated and this increasing bias will decrease the estimate for real historical improvement in human life expectancy – perhaps even imitating a catastrophic life shortening for the most recent generations. Moreover, for those family members whose birth date is close to truncation date, the short-lived persons only will be included into analysis, thus producing spurious correlation between ages at death for short-lived relatives. In our study the data for offspring born in the 20th century were excluded from the analysis in order to have unbiased estimates of longevity for extinct birth cohorts. The data for offspring born before the 18th century were also excluded in this study in order to minimize the heterogeneity of the study population. Thus, in order to avoid possible biases described above, the data for the offspring born in 1800-1880 only were used in this study.

Adjustment for Historical Trends and Fluctuations in Human Mortality. A new improved method for data analysis has been used in this study that allows us to study familial determinants of human longevity controlling for strong and complex historical changes in life expectancy for birth cohorts (Gavrilov and Gavrilova, 1997b; Gavrilov, Gavrilova et al., 1997b; 1998a; 1998b; Gavrilova et al., 1997a; 1997b; 1998). In previous studies by other authors the historical changes in life span were often ignored or controlled in a crude way by assuming a strictly linear trend for life expectancy as a function of proband's calendar year of birth or death. Our method is based on the idea of an internal control variable: the sex-specific life expectancy for each birth cohort is calculated and included in the analysis as an independent covariate, so that any historical trends and fluctuations in human life expectancy are regressed out (Gavrilov, Gavrilova et al., 1998a; 1998b; Gavrilova et al., 1997b; 1998).

For each birth cohort the sex-specific mean expectation of life at age 30 was calculated and used as a dependent variable in multiple linear regression to control for cohort and secular effects on human longevity.

Coping with sex bias. In most genealogical books and databases the sex bias is usually observed - the number of complete records (both birth and death dates available) for males is higher than for females (biased sex ratio). Since genealogical records are focused on family names which are transmitted by males only, women could be lost by genealogists when they marry and change their family names (Wyshak, 1978). In the present analysis of longevity, there is no reason to believe that women about whom information is not recorded differ from those whose records have been traced. In addition, the main focus of the analysis was based on comparisons, thus eliminating problem that may relate to representativeness (in the statistical sense) of the data.

Nevertheless a special efforts were made in this study in order to minimize the sex bias and its possible influence on final conclusions: the data for different families were computerized and cross-checked in order to find complete records (death dates in particular) for those women who changed their family names and were lost.

Characteristics of multiple linear regression. Data on European royal and noble families was analyzed for adults (30 years and above) born in 1800-1880. For each birth cohort the mean sex-specific expectation of life at age 30 was calculated and used as an independent variable in multiple linear regression to control for cohort and secular trends and fluctuations in human longevity. The parameters of multiple linear regression were calculated using SPSS statistical package (Norusis, 1985). Offspring longevity for each particular sex (8,228 records for males and 3,222 records for females) was considered as a dependent variable in multiple linear regression model and a function of 3 independent predictors: paternal age at death, maternal age at death and sex-specific cohort life expectancy. The data analysis was made for 6 overlapping ranges of parental (both paternal and maternal) ages at death: 30 years and above (30+), 40+, 50+, 60+, 70+ and 75+ (see Tables 1-4).

Results and Discussion
The results of the study on the dependence of sons' life span as a function of paternal and maternal life span are presented in Table 1. There are 8,228 cases of life span data for combinations "son-father-mother" (both parents lived at least 30 years) that allowed to calculate the multiple regression slopes for paternal effect ($+0.089 \pm 0.012$) and maternal effect ($+0.030 \pm 0.010$) on sons' longevity. Note that the regression slopes are rather small - 10 additional years of paternal life span will increase the mean life span of sons by only 0.89 ± 0.12 years, while maternal effect is even weaker: 0.3 ± 0.1 year of additional life span (for sons) for 10 additional years of maternal life span.

This weak resemblance between offspring and parental life span is consistent with results obtained by other authors (Murphy, 1978) and could explain partially the previous controversial results for maternal and paternal effects on human longevity. Since the familial resemblance is rather weak, while life span itself is characterized by high variability, quite different results and conclusions could be obtained just because of random fluctuations (unless very large sample size is studied). However in this study the sample size is large enough (8,228 cases) to find out that the paternal effect is 3 times higher than the maternal one and this difference is statistically highly significant ($p<0.001$, see Table 1).

It might be interesting to check whether the obtained result and conclusion is robust to sample variation and to parental life span ranges in particular. For this purpose those cases when at least one parent died prematurely (before age 50) were excluded from the analysis and the remaining 6,211 cases were re-analyzed (Table 1). In this case the paternal effect on sons' life span ($+0.113 \pm 0.018$) is almost twice higher than the maternal one ($+0.063 \pm 0.017$) and this difference is statistically significant ($p<0.05$, see Table 1). Studies of other sub-samples (with other ranges for parental life span) have demonstrated that paternal effects are always higher than maternal ones, although this difference is sometimes not statistically significant, probably because of smaller sample size (Table 1). Further studies in this direction are planned that will allow to increase the

sample size significantly and to check whether for longer lived parents the preferential paternal inheritance of sons' life span is also observed.

A similar data analysis made for daughters (3,222 cases) has produced somewhat different results (Table 2). The paternal effect (+0.047 ± 0.020) is virtually the same as maternal one (+0.045 ± 0.017), so no preferential paternal inheritance of life span is detected in the case of daughters. This mode of life span inheritance is consistent with polygenic model of life span inheritance when genes affecting life span are located on autosomes rather than sex chromosomes. There is however one interesting exception in our results that is not consistent with autosome mode of life span inheritance - in those cases when each of the parents lives 70+ years (863 cases), the paternal effect (+0.344 ± 0.081) is more than 5 times higher than maternal one (+0.062 ± 0.079) and this difference is statistically significant (p<0.05, see Table 2). Further studies on larger sample sizes will allow to clarify whether it is a real phenomenon or a result of random fluctuation in this particular sub-sample. It should be noted however that in all 6 sub-samples studied the maternal effect was never significantly higher than the paternal one (Table 2).

The results obtained in this study do not support the hypothesis of preferential maternal inheritance of human longevity - in fact the opposite phenomenon is observed for sons (preferential paternal inheritance). This might be a true biological (or social) phenomenon or an artifact caused by gender differences in human longevity (women live longer, so the maternal life span has wider range for variation than the paternal life span). In order to control for gender differences in life span variation the variables should be normalized and the standardized regression slopes (beta-coefficients) should be calculated (Norusis, 1985). The results of this kind of analysis are presented in Tables 3 and 4.

Normalization of the data did not change the results for sons' life span - the standardized paternal effect (+0.079 ± 0.011) was as twice as higher than the standardized maternal effect (+0.032 ± 0.011) and this difference was statistically highly significant (p<0.01, see Table 3). For each of 6 sub-sample studies the standardized paternal effect was higher than the standardized maternal one, although further increase in sample size is important to check the statistical significance of this difference in the case of longer lived parents.

In the case of daughters the normalization of data also does not change significantly the previous results and conclusions (see Table 4). Again for the sample of 3,222 cases the standardized paternal effect (+0.038 ± 0.016) is essentially the same as the standardized maternal effect (+0.046 ± 0.017) - the difference between paternal and maternal effects is statistically insignificant (Table 4). Normalization of the data also did not change the exceptional result obtained for parents lived 70+ years: the standardized paternal effect (+0.135 ± 0.032) is 5 times higher than standardized maternal effect (+0.025 ± 0.032) and this difference is statistically significant (p<0.05, see Table 4).

The method of multiple linear regression used in this study is based on the assumption that the paternal and maternal effects on offspring longevity are linear and independent on each other (additive effects). Although the assumption of linear additive effects is common in quantitative genetics and has theoretical justification in polygenic models (Falconer, 1989; Khoury et al., 1993), it is worth to check whether these assumptions are also valid in this particular case. For this purpose the method of contour plots was used in this study (see Figures 1-2).

Contour plots are the maps for the levels of offspring life span (graded from white color for low life span levels to dark black color for high life span levels) as a function of paternal life span (X-axis) and maternal life span (Y-axis). The levels of offspring life span are calculated as deviations from the sex-specific cohort mean levels. Areas with equal levels of offspring life span are connected by isolines. If offspring life span is determined by maternal life span only, one should expect that isolines will have horizontal orientation with more dark color on the top of the plot. On the contrary, if offspring life span is inherited on paternal side only, the isolines will have vertical orientation with more dark color on the right side of the plot. In the case when both paternal and maternal life span have independent effects on offspring life span, the isolines will have diagonal orientation so that white color (low levels for offspring life span) will be concentrated in the bottom left corner of the plot, while dark black color will be concentrated in the top right corner of the plot. In particular case when paternal and maternal effects are strictly additive, the slope for diagonal isolines will be equal to minus one, connecting the areas where the mean parental life span is constant. Although contour plots are semi-quantitative by their nature and are limited in their ability to check for statistical significance of the observed differences, they are very efficient for visualization of the possible complex interactions between paternal and maternal life span effects on offspring longevity. The results of this approach are presented at Figures 1-2.

Contour plot for sons (Figure 1) demonstrates strong vertical orientation of isolines in the area of paternal life span above age 70. This result is consistent with earlier analysis based on multiple linear regression model that has also detected the preferential paternal inheritance of sons' longevity (see above, Tables 1,3). However, in the area of lower levels of paternal life span (below age 70), the isolines are organized in a horizontal manner indicating preferential maternal inheritance for sons' life span. In other words, the isolines are organized in rectangular rather than diagonal manner that indicates strong interaction between maternal and paternal life span effects (Figure 1).

The observed rectangular organization of isolines could explain many of previous controversies on the relative importance of paternal and maternal life spans as predictors for offspring longevity. Those authors who studied populations with low paternal life span (below age 70) should observe preferential maternal inheritance of human longevity. On the contrary, in populations with significant number of cases of high paternal life span (above age 70) the preferential paternal inheritance of human longevity is observed (the case of this study).

Contour plot for daughters (Figure 2) is different from the plot for sons (Figure 1). In the case of daughters the isolines are organized more in diagonal rather than rectangular manner indicating relatively equal importance of both paternal and maternal effects as was also found through the multiple linear regression analysis (Tables 2, 4). However, the isolines are not strictly diagonal (rectangular shape fragments are also observed) and the contour plot does not support the model of additive independent effects of paternal and maternal life span. In particular, if maternal life span is less than 65 years (common situation for earlier historical periods with high female mortality), virtually no paternal inheritance of life span is observed. On the contrary, if maternal life span is in the range between 65 and 80 years, the paternal inheritance of life span is particularly high. Finally, when maternal life span is above 80 years, the daughters' life span has weaker response to paternal life span again. Thus, the effect of one parent depends on the effect of another

parent indicating strong interaction between paternal and maternal effects on daughters' longevity (Figure 2).

Although the contour plots for sons (Figure 1) and daughters (Figure 2) are different, they have also some features in common. In particular, when the same-sex parent (father for sons, mother for daughters) has long life span, the opposite-sex parent has little to contribute into the offspring life span (some kind of longevity saturation is observed). On the contrary, if the same-sex parent has short life span, the effect of the opposite-sex parent is either absent (daughters) or greatly delayed (sons) indicating some kind of longevity invalidity for the offspring born to short-lived same-sex parent (Figures 1-2).

The most interesting result of this study is that the maternal longevity effect does not exceed the paternal one - in fact the opposite tendency is observed (for sons in particular). This observation is surprising since mother has many different additional influences on the offspring through specific inheritance of mitochondrial DNA, strong maternal-child interaction during in utero development and later during the formative years of the child. For this reason the maternal effect on offspring traits is usually higher than the paternal one (Falconer, 1981). It is interesting that human longevity is an exception from this common observation and in fact the paternal effects tend even to exceed the maternal ones.

Paternal longevity seems to be a more important predictor of offspring life span than maternal longevity both for sons and in some cases for daughters (see Tables 1-4). Since this preliminary observation has important implications for testing different theories of aging and longevity, it deserves to be studied more thoroughly on larger data sets.

In order to explain the paradoxically stronger paternal effect on human longevity, we suggest the following hypothesis. In the case of mothers all recessive mutations could affect their longevity in those rare cases only when they are in homozygous form. That is why the long-lived mothers may carry essentially the same mutation load as the short-lived mothers and the only difference for long-lived mothers might be more heterozygous pattern for their mutation load. On the other hand in the case of fathers the mutations affecting longevity could not be compensated (complemented by other alleles) if they are located on X or Y chromosome. Thus, the relationship between genes and phenotypic longevity should be more straightforward and simple for fathers than for mothers. That is why the paternal longevity might be a better predictor for offspring longevity than the maternal one.

According to our suggested explanation, the stronger paternal longevity effect is related to male hemizygosity of genes on sex chromosomes and their higher selection in males. There is one interesting prediction from this hypothesis that should be checked in future studies - in families having centenarian grandfather from maternal side (transmission of X chromosome) both daughters and sons could live longer while in families with centenarian grandfather from paternal side (transmission of Y chromosome) the sons should have more "familial longevity benefits" than the daughters.

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Table 1. Sons' life span as a function of paternal and maternal life span.
Parameters of the multiple linear regression model.

Parental age at death (range)	Number of cases in regression	Linear regression slope ± standard error		Difference between paternal and maternal effects	Model Summary	
		Paternal effect	Maternal effect		R Square	F Ratio
30+	8,228	$0.089 \pm 0.012^{***}$	$0.030 \pm 0.010^{**}$	$0.059 \pm 0.016^{***}$	0.018	50.2
40+	7,426	$0.103 \pm 0.014^{***}$	$0.040 \pm 0.013^{**}$	$0.063 \pm 0.019^{***}$	0.019	48.2
50+	6,211	$0.113 \pm 0.018^{***}$	$0.063 \pm 0.017^{***}$	$0.050 \pm 0.025^{*}$	0.019	39.5
60+	4,508	$0.115 \pm 0.026^{***}$	$0.086 \pm 0.025^{**}$	0.029 ± 0.036	0.015	22.5
70+	2,114	$0.136 \pm 0.053^{*}$	0.046 ± 0.052	0.090 ± 0.074	0.014	9.8
75+	1,057	0.165 ± 0.095	0.024 ± 0.090	0.141 ± 0.131	0.010	3.5

[*] - statistically significant at 0.05 level; [**] - significant at 0.01 level; [***] - significant at 0.001 level.

Table 2. Daughters' life span as a function of paternal and maternal life span.
Parameters of the multiple linear regression model.

Parental age at death (range)	Number of cases in regression	Linear regression slope ± standard error		Difference between paternal and maternal effects	Model Summary	
		Paternal effect	Maternal effect		R Square	F Ratio
30+	3,222	$0.047 \pm 0.020^{*}$	$0.045 \pm 0.017^{**}$	0.002 ± 0.026	0.122	148.4
40+	2,904	$0.061 \pm 0.023^{**}$	$0.062 \pm 0.021^{**}$	-0.001 ± 0.031	0.118	129.9
50+	2,415	$0.086 \pm 0.029^{**}$	$0.091 \pm 0.028^{**}$	-0.005 ± 0.040	0.128	117.8
60+	1,757	$0.153 \pm 0.043^{***}$	$0.115 \pm 0.042^{**}$	0.038 ± 0.060	0.123	82.1
70+	863	$0.344 \pm 0.081^{***}$	0.062 ± 0.079	$0.282 \pm 0.113^{*}$	0.140	46.6
75+	432	$0.272 \pm 0.138^{*}$	$0.324 \pm 0.136^{*}$	-0.052 ± 0.194	0.146	24.5

* - statistically significant at 0.05 level; ** - significant at 0.01 level; *** - significant at 0.001 level.

Table 3. Sons' life span as a function of paternal and maternal life span.
Standardized parameters of the multiple linear regression model.

Parental age at death (range)	Number of cases in regression	*Standardized* linear regression slope ± standard error (Beta coefficients)		Difference between paternal and maternal *standardized* effects	Model Summary	
		Paternal effect	Maternal effect		Adjusted R Square	Significance
30+	8,228	$0.079 \pm 0.011^{***}$	$0.032 \pm 0.011^{**}$	$0.047 \pm 0.016^{**}$	0.018	0.000^{***}
40+	7,426	$0.085 \pm 0.012^{***}$	$0.036 \pm 0.012^{**}$	$0.049 \pm 0.017^{**}$	0.019	0.000^{***}
50+	6,211	$0.080 \pm 0.013^{***}$	$0.046 \pm 0.012^{***}$	0.034 ± 0.018	0.018	0.000^{***}
60+	4,508	$0.065 \pm 0.015^{***}$	$0.051 \pm 0.015^{**}$	0.014 ± 0.021	0.014	0.000^{***}
70+	2,114	$0.055 \pm 0.021^{*}$	0.019 ± 0.022	0.036 ± 0.030	0.012	0.000^{***}
75+	1,057	0.053 ± 0.031	0.008 ± 0.030	0.045 ± 0.043	0.007	0.016^{*}

* - statistically significant at 0.05 level; ** - significant at 0.01 level; *** - significant at 0.001 level.

Table 4. Daughters' life span as a function of paternal and maternal life span.
Standardized parameters of the multiple linear regression model.

Parental age at death (range)	Number of cases in regression	*Standardized* linear regression slope ± standard error (Beta coefficients)		Difference between paternal and maternal *standardized* effects	Model Summary	
		Paternal effect	Maternal effect		Adjusted R Square	Significance
30+	3,222	$0.038 \pm 0.016^{*}$	$0.046 \pm 0.017^{**}$	-0.008 ± 0.023	0.121	0.000^{***}
40+	2,904	$0.046 \pm 0.017^{**}$	$0.053 \pm 0.018^{**}$	-0.007 ± 0.025	0.118	0.000^{***}
50+	2,415	$0.056 \pm 0.019^{**}$	$0.062 \pm 0.019^{**}$	-0.006 ± 0.027	0.127	0.000^{***}
60+	1,757	$0.080 \pm 0.023^{***}$	$0.062 \pm 0.023^{**}$	0.018 ± 0.033	0.122	0.000^{***}
70+	863	$0.135 \pm 0.032^{***}$	0.025 ± 0.032	$0.110 \pm 0.045^{*}$	0.137	0.000^{***}
75+	432	$0.088 \pm 0.045^{*}$	$0.107 \pm 0.045^{*}$	-0.019 ± 0.064	0.140	0.000^{***}

* - statistically significant at 0.05 level; ** - significant at 0.01 level; *** - significant at 0.001 level.