Paper presented at
The Population Association of America 2000 Annual Meeting
(Session 68 “Health, Longevity, and Mortality”).
March 23-25, 2000
Los Angeles

Biodemographic Study of the Boundaries for Human Longevity

Leonid A. Gavrilov[*], Natalia S. Gavrilova[*],

Galina N. Evdokushkina[**], Victoria G. Semyonova[**]

(*) Center on Aging, NORC/University of Chicago, 1155 East 60th Street, Chicago, IL 60637

(**) Institute of Public Health, Moscow, Russia.

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

BIODEMOGRAPHIC STUDY OF THE BOUNDARIES FOR HUMAN LONGEVITY

Leonid A. Gavrilov, Natalia S. Gavrilova,
Galina N. Evdokushkina, Victoria G. Semyonova.
Center on Aging, NORC/University of Chicago, 1155 East 60th Street, Chicago, IL 60637
lagavril@midway.uchicago.edu.

Abstract

The scientific debates on the future of human lifespan and its possible biological limits has revealed a great need for direct identification of longevity boundaries, if they really exist. The key question posed in this study is - how can we possibly determine the age when human longevity starts? To address this problem, we studied the familial transmission of human lifespan from parents to daughters, since daughters did not have a high incidence of violent causes of death due to military service.

In this study we collected, computerized and analyzed the detailed genealogical records on lifespan of 5,779 adult daughters (30+ years) and their parents, using particularly reliable and complete data on European royal and noble families for extinct birth cohorts (born 1800-1880).

The familial transmission of human lifespan from mother to daughter is essentially non-linear with virtually no resemblance before maternal lifespan of 85 years (regression slope of daughters lifespan on maternal lifespan, $b = 0.002 \pm 0.020$, $n = 4{,}983$ cases, $p = 0.941$, insignificant) and very high familial resemblance (additive heritability) for longer lived mothers ($b = 0.412 \pm 0.204$, $n = 619$, $p < 0.05$). This indicates that maternal age of 85 years could be considered as a demarcation line (lower boundary) for women's longevity. Women who live above this age are fundamentally (genetically?) different from other women in the sense that their daughters live significantly longer. Thus the age of 85 years could be a threshold age when women mortality becomes much more selective and this age threshold in death selectivity should be taken into account in forecasting human life expectancy for women.

A similar study of familial transmission of human lifespan from *father* to daughter revealed a demarcation point at 75 years, suggesting that this age might represent a lower boundary for male longevity. The familial transmission of human longevity from father to daughter is also non-linear with virtually no resemblance before paternal lifespan of 75 years ($b = -0.007 \pm 0.034$, $n = 4{,}011$, $p = 0.829$, insignificant) and very high additive heritability for longer lived fathers ($b = 0.236 \pm 0.078$, $n = 1{,}756$, $p < 0.01$).

These results are also consistent with the predictions of the evolutionary theory of aging and mutation accumulation theory in particular, namely that the additive genetic variance for human life span should increase with parental longevity. In other words, human mortality should be more selective at advanced ages (Gavrilova et al., 1998, *Human Biology*, 70: 799-804). This study was supported by NIA grants AG12857, AG13698-01 and AG16138-01A1.

Introduction

The scientific debates on the future of human lifespan and its possible biological limits has revealed a great need for direct identification of longevity boundaries, if they really exist (Gavrilov, Gavrilova, 1998, *Science*, 281: 1611-1615). The key question posed in this study is - how can we possibly determine the age when human longevity starts? To address this problem, we studied the familial transmission of human lifespan from parents to daughters, since daughters did not have a high incidence of violent causes of death due to military service. If human lifespan is inherited just as any other quantitative trait, then the monotonic linear dependence between offspring lifespan and parental lifespan is expected (Falconer, Mackay, 1996; Lynch, Walsh, 1998). In this case no evidence for any boundaries for human longevity could be detected. On the other hand, if a special age does really exist that corresponds to longevity boundary, this could be detected as a breaking point in the offspring-parent lifespan dependence.

Data and Methods

Main Data Source. In this study we collected, computerized and analyzed the detailed genealogical records on lifespan of 5,779 adult daughters (30+ years) and their parents, using particularly reliable and complete data on European royal and noble families for extinct birth cohorts (born 1800-1880). The main advantage of these data is their high accuracy, reliability and completeness (to be discussed later). Another advantage of this kind of data is the relative homogeneity of this Caucasian population regarding social class and educational background. Since this privileged social group lived in 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. Thus, although the sample analyzed in this study does not represent the whole human population (as laboratory animals do not represent species in the wild), it is one of the best possible samples to test biodemographic hypotheses since the effects of population heterogeneity are minimized with regard to social status.

The database on European royal and noble families (a family-linked database) was developed and already used in our previous studies (Gavrilov, Gavrilova, 1997a; 1997b; Gavrilov et al., 1995; 1997; Gavrilova et al., 1995; 1997; 1998). To develop this database we have used one of the best professional sources of genealogical data available - the famous German edition of the "Genealogisches Handbuch des Adels" (Genealogical Yearbook of Nobility). This edition is known world wide as the "Gotha Almanac" - "Old Gotha" published in Gotha in 1763-1944, and "New Gotha" published in Marburg since 1951 (see Gavrilova, Gavrilov, 1999, for more details). Data from the Gotha Almanach were often used in early biodemographic studies of fertility (see Hollingsworth, 1969, pp. 199-224, for references) and are used now in the studies of human longevity (Gavrilova et al., 1998; Gavrilov, Gavrilova, 1997a).

Each volume of the New Gotha Almanach contains about 2,000 genealogical records dating back to the 14-16th centuries (to the founder of a particular noble genus). More than 100 volumes of this edition are already published, so more than 200,000 genealogical records with

well-documented genealogical data are available from this data source. The high quality of information published in this edition is ensured by the fact that the primary information is drawn from the German Noble Archive (Deutsches Adelsarchiv). The Director of the German Noble Archive (Archivdirektor) is also the Editor of the New Gotha Almanach. Our own experience based on cross-checking the data, has demonstrated that the number of mistakes (mostly misprints) is very low in the "New Gotha Almanac" (less than 1 per 1000 records), so this source of data is very accurate compared to other published genealogies.

The information on noble families in the New Gotha Almanac is recorded in a regular manner. The description of each particular noble genus starts with information on 2-3 generations of founders of male sex only. Then three to four the most recent generations are described in more detail, including information on individuals (e.g., first and last names; event data: birth, death, marriage dates and places; descriptive data: noble degrees, occupation if available, information on death circumstances if available), information on parents (e.g., first and last names; event data: birth and death dates and places), information on spouse(s) (e.g., first and last names; birth and death dates and places; first and last names of parents) and information on children (detailed as for each individual).

The process of data computerization was started from the most recent volumes of the New Gotha Almanac (published in 1990-1994) and reached now the volumes published ten years earlier. The database on European aristocratic families comprises more than 20,000 personal records and is growing further.

Supplementary Data Sources. Some other supplementary sources of data were used in the development of database. These data sources included two computerized data files on European royalty and British peerage (computerized database "Royal92" distributed on the Internet by Brian C. Tompsett at University of Hull, UK, and database on British Peerage distributed on CD by S&N Genealogy Supplies), as well as over 100 genealogical publications on Russian nobility listed elsewhere (Gavrilov et al., 1996). These data were used as a supplement to the main data source since their quality was not as high compared to the Gotha Almanac. Although data on European royalty were recorded in computerized data sources ("Royal92", British Peerage CD, see above) with sufficient completeness, data on lower rank nobility (landed gentry) were less complete and accurate. The same was true for the data on Russian nobility. All supplementary data were matched with the Gotha Almanac data, in order to cross-check the overlapping pieces of information. This cross-checking procedure allowed us to increase the completeness of the database by complementation of information taken from different sources.

The Structure of the Database on European Aristocracy. The database approach used in this study is similar to the approach used for existing family-linked databases, such as the Utah Population Database (Skolnick et al., 1979), Laredo Epidemiological Project (Buchanan et al., 1984) or other historical databases (Gutmann et al., 1989). Initially the information computerized from each volume of the New Gotha Almanac is stored in two files: the Individual File and the Marriage File. Then these two files are merged into one rectangular file with information on up to 4 spouses. Since marriages with 5^{th} and higher orders comprise less than 0.1% of all marriages, the potential loss of information on spouses after data merge is negligible. Then these merged files are linked to the Master File (main database).

In the Master File each record is related to the duration of an individual's life. Each record represents an individual's event data (birth and death dates and places) and individual's

descriptive information (identification number, sex, first and last names, nobility rank, occupation, birth order, cause of death (violent/nonviolent), ethnicity, marital status, data source code number, data source year of publication). Individual information is supplemented by data for parents (identification numbers, first and last names, birth, death and marriage dates, cause of death) and spouses. Thus, the database that is used in this project is organized in the form of triplets (referred to as the "ego" and two parents). This structure of records is widely used in human genetics and is adequate for studies of parent-child relationships. Similar database structure was used in the recent study of kinship networks (Post et al., 1997).

Data Quality Control. Data quality control was an important part of our study designed to develop high quality family-linked databases for longevity studies.

The genealogical data sets were checked for: (1) *completeness* in reporting birth and death dates, which is crucial for calculating individual life span - the variable of particular interest in our study. (2) *accuracy* - whether the percentage of mistakes and inconsistencies between reported dates (such as, for example, birth by the dead mother) is low enough to be acceptable. (3) *representativeness* - whether the characteristics of investigated data sets (distribution by age, sex, marital status, age at death, etc.) is close enough to demographic characteristics of populations in similar geographic areas, historical periods and social groups. In our study we referred to the well-known publication by Thomas Hollingsworth (1962) on British peerage as a standard for European aristocracy, to check for data representativeness.

The completeness in birth and death dates reporting in the New Gotha Almanac was very high: dates of all vital events were reported for nearly 95% of all persons. Such high completeness is not common for many other genealogical data sources. For example, for British Peerage data published in Burke almanac in most cases there are no birth dates for women, which makes the calculation of their life span impossible. In fact, this problem with British aristocratic women was first noticed by Karl Pearson a century ago (Beeton and Pearson, 1899, 1901). He used the British Peerage data to study the longevity inheritance and had to exclude women from his consideration for the following reason: "The limitation to the male line was enforced upon us partly by the practice of tracing pedigrees only through the male line, partly by the habitual reticence as to the age of women, even at death, observed by the compilers of peerages and family histories" (Beeton and Pearson, 1901, pp.50-51).

The accuracy of data published in the New Gotha Almanac is also very high: the frequency of inconsistent records is less than 1 per 1000 records while for many other genealogical data sources it falls within 1 per 300-400 records. Comparison of our data with Hollingsworth's analysis of British peerage (Hollingsworth, 1962) revealed good agreement between his findings and our data on mortality patterns, including male/female gap in life expectancy.

The genealogies for the members of European aristocratic families presented in the "Gotha Almanac" are of descending type, tracing almost all the descendants of relatively few founders. This is an important advantage of this data source over other genealogies that are often of ascending type (pedigrees). It is known in historical demography that the ascending genealogies are biased, over-representing more fertile and longer-lived persons who succeed to become ancestors, and for this reason such genealogies should be treated with particular caution (Jetté and Charbonneau, 1984; Fogel, 1993).

Thus, the genealogical data published in the Gotha Almanac are characterized by high quality and accuracy. We have, however, encountered some problems regarding the data completeness that are discussed below, along with proposed solutions.

Censored, truncated observations and missing death dates. Our study revealed that the percentage of cases with unreported death dates is rather small in our main data sources (Gotha Almanac), and is caused mainly by right censoring of long-lived persons who were still alive by the date of data collection and publication. The percentage of non-reported death dates varies from 0 to 7% in extinct birth cohorts (1800-1880), while it is higher in later birth cohorts (1880-1899) - 23% for women and 8% for men, since some individuals were still alive by the date of data collection and volume publication. Note that women, who live longer, have a higher proportion of right-censored observations. The high proportion of censored observations in genealogies is not desirable, since the exact dates of censoring are often unknown. This uncertainty creates problems for data analysis, so the researchers working with genealogies prefer to use non-censored, extinct birth cohorts in their studies (Mayer, 1991; Pope, 1992; Kasakoff and Adams, 1995). We also used extinct (non-censored) birth cohorts in our study. For this purpose only those birth cohorts were used in the study that were born at least 100 years before the year of data publication (to be sure that the birth cohort under study is almost extinct).

Underreporting of women and children. In many genealogical books and databases non-married women as well as children died in infancy are often missed or reported with less completeness. Since genealogical records are focused on family names which are transmitted by males only, women could be lost in genealogies when they marry and change their family names (Hollingsworth, 1976). Also, in many cases data for women do not contain information on their birth and death dates resulting in biased sex ratio in the sample with complete dates. We have also encountered this problem in our studies although for somewhat different reason. Our analysis revealed that the main cause of the sex bias in the New Gotha Almanac is related to the manner of data representation: more recent generations are presented completely, while the earlier generations are limited mainly to the male ancestors (in order to avoid repetitive publication of individuals already presented in previous volumes). That is why, the sex ratio among early birth cohorts (1800-1860) is biased in favor of males while for more recent birth cohorts (1880-1899) it is within normal range. Since in our study the most recent volumes of the New Gotha Almanac (published after 1980) were computerized and analyzed (in order to avoid censoring), the proportion of males in the database was substantially higher than expected. Thus, the ideal way to overcome the sex bias problem is to ensure complete coverage of all aristocratic genuses and families ever published in the Gotha Almanac. However, it may take a long time to computerize all 100 volumes of the New Gotha Almanac. The alternative way is to computerize complete data on early birth cohorts published in old volumes. In this case the data will be heavily censored since many persons would not have death date (be still alive) by the date of publication. We plan to continue computerization of these genealogies that will allow eventually eliminate the sex bias and potential problems associated with it. Sex bias is an important issue in fertility studies since the fertility levels are understated when daughters are underreported, but in the case of longevity studies this issue is less important when non-censored, extinct birth cohorts are analyzed (Wyshak, 1978). According to Wyshak (1978, p.318), "in the … 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".

The underreporting of children died in infancy may be also a serious problem, especially for studies that include fertility analysis. Fortunately, in the Gotha Almanac the noble families are described with remarkable completeness, especially those families which belong to the higher

nobility rank (kings, princes, earls). In particular, all ever born children are recorded, including those who died the same day. Another indicator of data completeness is the normal sex ratio at birth (101 to 108) observed among these families (according to our sample analysis). In our database over 90 aristocratic genuses belonged to the upper nobility were recorded completely, although data for lower rank nobility were not yet completed. Underreporting of children is not a problem for this particular study which is focused on adult life span for those who survive by age 30 years.

Analytical Methods. Since the data used in this study are characterized by remarkable accuracy and completeness, it was possible to apply simple and straightforward methods of data analysis without making heavy assumptions. In particular, since the length of life is known for every person (there were no right censored observations) it was possible to analyze the duration of life directly as a dependent, outcome variable in linear regression model. There was no need to apply the Cox proportional hazard model and to make a strong assumption about multiplicative effects of covariates on hazard rate. Instead, daughters' lifespan was considered as a linear function of parental lifespan, and this assumption of linear dependence between parental and offspring traits is well justified both in the theory and practice of quantitative genetics (Falconer, Mackay, 1996; Lynch, Walsh, 1998). Two additional features were introduced in this simple linear regression model:

(1) A piece-wise linear regression model was applied to test whether the regression slope coefficients of linear regression of daughters' lifespan on parental lifespan are different, if calculated for different ranges of parental lifespan.

(2) To control for secular changes in life expectancy, an additional internal control variable was included into analysis as an independent predictor of daughters' lifespan. Specifically, the mean lifespan of daughters was calculated for each calendar year of birth (81 cases for years 1800-1880). This variable was then included into linear regression model as a predictor variable for individual lifespan of each daughter matched for the same year of birth. This method was already applied earlier in a similar study (Gavrilova et al., 1998) to regress out the secular changes in lifespan.

Data for graphs (Fig.1-2) were calculated in the following way. First, the data on individual lifespan were centered around the mean lifespan in the same birth cohorts in order to control for secular changes in lifespan. In other words, the residuals were calculated as the differences between individual lifespan and the cohort mean lifespan for the same calendar year of birth. These residuals (deviations from population mean) were then plotted against parental lifespan to see whether the mean of these residuals is close to zero (expected if parental lifespan is of no importance), or whether it is increasing with parental lifespan. The dependence of averaged residuals on parental lifespan was generated, that was then smoothed by 5-year moving average in order to decrease the statistical noise and to reveal the pattern of this dependence. These graphs were used to detect the possible breaking points in linear dependence for subsequent piece-wise regression analysis.

Results and Discussion

Figure 1 depicts the dependence between daughters' lifespan and maternal lifespan. This dependence looks like consisting of two pieces. Daughters born to shorter-lived mothers (died before 85 years) seem to demonstrate no resemblance with maternal lifespan. It does not really matter for daughters' lifespan whether their mothers lived 40 years only, or as long as 80 years - the corresponding increase in daughters' lifespan is below 2 years for 40 years of additional maternal lifespan.

Figure 1 about here

On the other hand, daughters born to longer-lived mothers (died after 85 years) demonstrate remarkably steep dependence of their lifespan on maternal lifespan (Figure 1).

These graphical observations are confirmed by statistical analysis presented at Table 1.

Table 1 about here

The familial transmission of human lifespan from mother to daughter is essentially non-linear (consisting of two different lines) with virtually no resemblance before maternal lifespan of 85 years - the slope coefficient of linear regression for daughters' lifespan on maternal lifespan, $b = 0.002 \pm 0.020$, $n = 4{,}983$ cases, $p = 0.941$, insignificant. For longer-lived mothers (died after 85 years) a very high familial resemblance (additive heritability) is observed: $b = 0.412 \pm 0.204$, $n = 619$, $p < 0.05$. In other words, for each additional 10 years of lifespan of longer-lived mothers, daughters gain additional 4.12 ± 2.04 years of lifespan in average. In quantitative genetics the narrow-sense heritability of any trait is estimated as the doubled regression slope coefficient for offspring-on-parent dependence (Falconer, Mackay, 1996; Lynch, Walsh, 1998). Thus, after maternal age of 85 years the narrow-sense heritability of human lifespan increases from virtually zero to $82.4 \pm 40.8\%$. This indicates that maternal age of 85 years could be considered as a demarcation line (lower boundary) for women's longevity. Women who live above this age are fundamentally (genetically?) different from other women in the sense that their daughters live significantly longer. Thus the age of 85 years could be a threshold age when women mortality becomes much more selective and this age threshold in death selectivity should be taken into account in forecasting human life expectancy for women.

Figure 2 demonstrates the dependence between daughters' lifespan and *paternal* lifespan. This dependence also looks like consisting of two lines but the breaking point between these two lines is observed at earlier parental age - about 75 years. Daughters born to shorter-lived fathers (died before 75 years) do not inherit paternal lifespan. It does not matter for daughters' lifespan whether their fathers lived 40 years only, or 70 years - the increase in daughters' lifespan is negligible despite increase in paternal lifespan by 30 years (Fig.2).

Figure 2 about here

On the contrary, daughters born to longer-lived fathers (died after 75 years) demonstrate rather steep dependence of their lifespan on paternal lifespan (Figure 2).

These graphical observations are also confirmed by statistical analysis presented at Table 2.

Table 2 about here

A study of familial transmission of human lifespan from father to daughter suggests a demarcation point at 75 years, indicating that this age may represent a lower boundary for male longevity. The familial transmission of human longevity from father to daughter is also non-linear (consisting of two different lines) with virtually no resemblance before paternal lifespan of 75 years ($b = -0.007 \pm 0.034$, $n = 4{,}011$ cases, $p = 0.829$, insignificant) and very high additive heritability for longer lived fathers ($b = 0.236 \pm 0.078$, $n = 1{,}756$ cases, $p < 0.01$).

The obtained results are consistent with the predictions of the evolutionary theory of aging and mutation accumulation theory in particular, namely that the additive genetic variance for human life span should increase with parental longevity. In other words, human mortality should be more selective at advanced ages (Gavrilova et al., 1998) as observed in this study. Further studies on larger samples with additional consideration of many other explanatory and confounding variables are planned and may shed light on the mechanisms of human lifespan and the boundaries for human longevity.

Acknowledgments.
This study was supported by NIA grants AG12857, AG13698-01 and AG16138-01A1.

References

Beeton, M., Pearson, K., 1899, "Data for the problem of evolution in man, II: A first study of the inheritance of longevity and the selective death rate in man", *Proceedings of the Royal Society of London.* 65: 290-305.

Beeton, M., Pearson, K., 1901, "On the inheritance of the duration of life and the intensity of natural selection in man", *Biometrika* 1: 50-89.

Buchanan A.V., Weiss, K.M., Schwartz, R.J., MacNaughton, N.L., McCartan, M.A., Bates, S.S., 1984, "Reconstruction of genealogies from vital records: The Laredo Epidemiology Project", *Comput. Biomed. Res*., 17: 326-351.

Fogel R.W., 1993, "New sources and new techniques for the study of secular trends in nutritional status, health, mortality, and the process of aging", *Historical Methods*, 26(1): 5-43.

Gavrilov LA, Gavrilova N.S., 1997a, "Parental age at conception and offspring longevity", *Reviews in Clinical Gerontology*, 7: 5-12.

Gavrilov L.A., Gavrilova N.S., 1997b, "When Fatherhood Should Stop?", *Science*, 277: 17-18.

Gavrilov L.A., Gavrilova N.S., Semyonova V.G., Evdokushkina G.N., Gavrilova A.L., Evdokushkina N.N., Lapshin E.V., 1995, "Human longevity genes are located in X-chromosome", In Knook D.L., Dittman-Kohli F., Duursma S.A. et al. (eds.) *Ageing in a*

Changing Europe. III European Congress of Gerontology: 30 August-1 September, 1995. Utrecht: Netherlands Institute of Gerontology, Abstract No.020.0027.

Gavrilov L.A., Gavrilova N.S., Evdokushkina G.N., Semyonova V.G., Gavrilova A.L., Evdokushkina N.N., Lapshin E.V., 1996, "Determinants of human longevity: parental age at reproduction and offspring longevity", *Longevity Report* (ISSN 0964-5659), 10(54): 7-15.

Gavrilov L.A., Gavrilova N.S., Kroutko V.N., Evdokushkina G.N., Semyonova V.G., Gavrilova A.L., Lapshin E.V., Evdokushkina N.N., Kushnareva Yu.E., 1997, "Mutation load and human longevity", *Mutation Research*, 377: 61-62.

Gavrilova, N.S., Gavrilov, L.A., 1999, "Data resources for biodemographic studies on familial clustering of human longevity", *Demographic Research* [Online], vol.1(4): 1-48. Available: http://www.demographic-research.org/Volumes/Vol1/4/default.htm.

Gavrilova N.S., Semyonova V.G., Gavrilov L.A., Evdokushkina G.N., Gavrilova A.L., Lapshin E.V., Evdokushkina N.N., 1995, "Biomedical basis of sex differential in human life span", In Knook D.L., Dittman-Kohli F., Duursma S.A. et al. (eds.) *Ageing in a Changing Europe. III European Congress of Gerontology: 30 August-1 September, 1995.* Utrecht: Netherlands Institute of Gerontology, Abstract No.020.0028.

Gavrilova N.S., Gavrilov L.A., Kushnareva Yu.E., Andreyev A.Yu., Evdokushkina G.N., Gavrilova A.L., Semyonova V.G., Evdokushkina N.N., 1997, "Testing the evolutionary theory of longevity", In *16th Congress of the IAG [International Association of Gerontology], August 19-23, 1997. Book of Abstracts.* Adelaide, 550.

Gavrilova N.S., Gavrilov L.A., Evdokushkina G.N., Semyonova V.G., Gavrilova A.L., Evdokushkina N.N., Kushnareva Yu.E., Kroutko V.N., Andreyev A.Yu., 1998, "Evolution, mutations and human longevity: European royal and noble families", *Human Biology*, 70: 799-804.

Genealogisches Handbuch des Adels. Genealogisches Handbuch der Adeligen Hauser. 1953, vol.5. Van Hueck W. (ed.). Limburg an der Lahn: C.A.Starke Verlag. Ibid. 1955, vol.9, 11; Ibid. 1956, vol.12; Ibid. 1957, vol.15; Ibid. 1958, vol.17; Ibid. 1959, vol. 20; Ibid. 1960, vol.22, 24; Ibid. 1961, vol.26; Ibid. 1962, vol.29; Ibid. 1964, vol.32; Ibid. 1965, vol.34, 36; Ibid. 1966, vol.38; Ibid. 1968, vol.41; Ibid. 1969, vol.45; Ibid. 1970, vol.46; Ibid. 1971, vol.49; Ibid. 1972, vol.52; Ibid. 1974, vol.57; Ibid. 1975, vol.60; Ibid. 1977, vol.64; Ibid. 1979, vol.71; Ibid. 1980, vol.73; Ibid. 1981, vol.76, 78; Ibid. 1983, vol.81; Ibid. 1984, vol.83; Ibid. 1985, vol.86 87; Ibid. 1986, vol.89; Ibid. 1987, vol.89; Ibid. 1988, vol.93; Ibid. 1992, vol.103.

Genealogisches Handbuch des Adels. Genealogisches Handbuch der Freiherrlinhen Hauser. 1952, vol.4. Van Hueck W. (ed.). Limburg an der Lahn: C.A.Starke Verlag. Ibid. 1954, vol.7; Ibid. 1956, vol.13; Ibid. 1957, vol.16; Ibid. 1959, vol.21; Ibid. 1962, vol.27; Ibid. 1963, vol.30, 31; Ibid. 1966, vol.37; Ibid. 1967, vol.39; Ibid. 1969, vol.44; Ibid. 1970, vol.48; Ibid. 1971, vol.51; Ibid. 1975, vol.59; Ibid. 1976, vol.62; Ibid. 1977, vol.65; Ibid. 1978, vol.68; Ibid. 1979, vol.69; Ibid. 1982, vol.79, 80; Ibid. 1986, vol.88; Ibid. 1992, vol.102; Ibid. 1994, vol.106, 107.

Genealogisches Handbuch des Adels. Genealogisches Handbuch der Furstlichen Hauser. 1951, vol.1. Van Hueck W. (ed.). Limburg an der Lahn: C.A.Starke Verlag. Ibid. 1953, vol.3; Ibid. 1955, vol.8; Ibid. 1956, vol.14; Ibid. 1959, vol.19; Ibid. 1961, vol.25; Ibid. 1964, vol.33; Ibid. 1968, vol.42; Ibid. 1971, vol.50; Ibid. 1978, vol.70; Ibid. 1980, vol.74 75; Ibid. 1984, vol.85; Ibid. 1987, vol.90; Ibid. 1991, vol.100.

Genealogisches Handbuch des Adels. Genealogisches Handbuch der Graflichen Hauser. 1952, vol. 2. Van Hueck W. (ed.). Limburg an der Lahn: C.A.Starke Verlag. Ibid. 1953, vol.6; Ibid. 1955, vol.10; Ibid. 1958, vol.18; Ibid. 1960, vol.23; Ibid. 1962, vol.28; Ibid. 1965, vol.35; Ibid. 1967, vol.40; Ibid. 1970, vol.47; Ibid. 1973, vol.54-56; Ibid. 1976, vol.63; Ibid. 1979, vol.72; Ibid. 1983, vol.82; Ibid. 1991, vol.101; Ibid. 1993, vol.104, 105.

Gutmann M., Fliess K.H., Holmes A.E., Fairchild A.L., Teas W.A., 1989, "Keeping track of our treasures: managing historical data with relational database software", *Historical Methods*, 22(4), 128-143.

Hollingsworth T.H., 1962, "The demography of the British Peerage", *Population Studies*, suppl., 18: 3-107.

Hollingsworth T.H., 1969, *Historical Demography*. Ithaca, N.Y.: Cornell University Press.

Jetté, R., Charbonneau, H., 1984, "Généalogies déscendantes et analyse démographique", *Annales de Démographie Historique*, 45-54.

Kasakoff A.B., Adams J.W., 1995, "The effect of migration on ages at vital events: a critique of family reconstitution in historical demography", *Eur. J. Pop.*, 11: 199-242.

Lynch M., Walsh B. 1998, *Genetics and analysis of quantitative traits*, Sunderland, Mass.: Sinauer.

Mayer P.J., 1991, "Inheritance of longevity evinces no secular trend among members of six New England families born 1650-1874", *Am. J. Hum. Biol.*, 3: 49-58.

Pope C.L., 1992, "Adult mortality in America before 1900. A view from family histories", In: C.Goldin and H.Rockoff (eds.), *Strategic Factors in Nineteenth Century American Economic History. A Volume to Honor Robert W. Fogel*. Chicago and London: Univ. Chicago Press, 267-296.

Post W., Van Poppel F., Van Imhoff E., Kruse E., 1997, "Reconstructing the extended kin-network in the Netherlands with genealogical data: Methods, problems, and results", *Pop. Studies*, 51: 263-278.

Skolnick M., Bean L.L., Dintelman S.M., Mineau G., 1979, "A computerized family history data base system", *Sociology and Social Research*, 63: 506-523.

Wyshak G., 1978, "Fertility and longevity of twins, sibs, and parents of twins", *Soc. Biol.*, 25: 315-30.

Table 1

Heritability of Human Lifespan
(Regression Slope of Daughter's Lifespan on Maternal Lifespan) as a Function of *Maternal* Lifespan Range

	Daughters of *shorter-lived* mothers (30-85 years)	Daughters of *longer-lived* mothers (85-95 years)
Regression slope (daughters' lifespan on maternal lifespan)	***0.002***	***0.412***
Standard error for regression slope	**0.020**	**0.204**
t-ratio	**0.07**	**2.02**
Significance (P-value)	**0.941 Insignificant**	**0.044 Significant**
Number of cases	**4,983**	**619**
Range for maternal lifespan, years	**30-85**	**85-95**

The data are for extinct birth cohorts, born 1800-1880. European royal and noble families.

Table 2

Heritability of Human Lifespan
(Regression Slope of Daughter's Lifespan on Paternal Lifespan) as a Function of *Paternal* Lifespan Range

	Daughters of *shorter-lived* fathers (30-75 years)	Daughters of *longer-lived* fathers (75-95 years)
Regression slope (daughters' lifespan on paternal lifespan)	***-0.007***	***0.236***
Standard error for regression slope	**0.034**	**0.078**
t-ratio	**-0.22**	**3.04**
Significance (P-value)	**0.829 Insignificant**	**0.002 Significant**
Number of cases	**4,011**	**1,756**
Range for paternal lifespan, years	**30-75**	**75-95**

The data are for extinct birth cohorts, born 1800-1880. European royal and noble families.