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Familial and environmental influences on longevity in a pre-industrial population

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ABSTRACT

Data from historical populations provide an adequate context for the examination of the familial and environmental components of longevity. We have investigated the relation between sibling survivorship and longevity through French-Canadian children of a completed fertility cohort born between 1625 and 1704. The Cox regression model was used to analyze the effects of sibling survivorship on the survival time of these early Canadian inhabitants. Other covariates such as regional variation, secular trends (i.e. period effects), parental and spousal survival were also taken into consideration. Our findings show that individuals with at least one sibling surviving beyond 85 years of age had a life-long sustained mortality advantage over the general population. The risks of death after age 50 was 55% and 60% lower for females and males, respectively, having a long-lived sibling. In comparison, the parental of origin effects were negligible. Only the mother-son association in age at death was found significant among the four possible parent-child pairs. Overall, the various models provided better fit to male than to female data. Biological as well as social explanations are explored in order to account for the various results.

INTRODUCTION

The dramatic increase in life expectancy witnessed over the last two centuries in developed nations has no precedent in human history. The improvement has been general and has involved most strata of society. As populations continue to age, the need to understand mortality differentials, whether environmental or genetic in nature, becomes an imperative. Paradoxically, however, the actual conditions prevailing with regard to mortality at advanced ages in our modern medicalized societies may not furnish the ideal setting to address those differences. In modern populations, genetic and other effects can be masked by the artificial prolongation of life through public health and medical technology (Desjardins 2001; Oshlansky and Carnes, 2002).

An alternative to the study of current mortality patterns at the older ages is to focus on genealogical records from populations of the past. One of the most salient issues throughout the literature has been the examination of familial transmissions of traits from one generation to the next. Such studies have addressed the strength of the association in parent-offspring pairs, and have produced heritability estimates that range from zero to a moderate value of 0.33 (Cournil et al. 2000, Kerber et al. 2001). The extent and the significance of the association in each of the possible parent-offspring pairs (i.e., father-son, father-daughter, mother-son, mother-daughter, parent-son, mother-offspring, and so on) reported in many studies are not clear, and show considerable variation from one to the next. Some have found a stronger paternal-offspring resemblance over that of a maternal-offspring origin of effect (e.g. Gavrilov and Gavrilova 2001), while others have found a stronger maternal effect (Mitchell et al 2001). Bocquet-Appel & Jakobi (1990) believed that the paternal-child resemblance exceeded that of the maternal-child one

because of the high levels of maternal mortality in historical times. Otherwise, the influence of both parents was thought to be almost identical. To further complicate matters, sex-specific parental-offspring resemblance may vary over time, as shown in the 19th Century British Peerage Data (Westendorp & Kirkwood 2001), or by socioeconomic status, as revealed by a comparison of Rural Finns to the European Aristocracy from 1600-1889 (Korpelainen 2000).

Other enquiries focused on sibling pairs, which provided additional clues on the biological and environmental bases of survival to advanced ages. Familial resemblance in survival patterns should be more apparent among siblings. There are a single set of biological parents, whereas the son or daughter may have many siblings. Further, high death rates throughout the lifespan could also make the parental ages at death almost random. Perls et al. (2002, 2003) found that siblings of centenarians in New England had approximately a 50% lower risk of death than the general population. First-degree relatives of siblings surviving to the 95th percentile were approximately twice as likely to survive to the 95th percentile as controls among the Mormons in Utah (1870-1907) (Kerber et al. 2001) and in the Icelandic population (1870-1900) (Gudmunsson et al. 2000). A strong resemblance among brother-brother pairs and a significant but weaker correlation for sister-sister pairs were also reported for the Pennsylvanian Amish born prior to 1890 (Mitchell et al. 2001). All of the studies including siblings contained similar findings, and any differences between them may be attributed to the use of different statistical methods (Perls et al 2002). Overall, the phenotypic resemblance between siblings is expected to be higher than between the parents and their children because of

dominance interaction (Fisher 1930) and of a shared environmental component in the families of origin (Mitchell et al. 2001).

The above studies provide excellent insight into familial longevity, but they are not without limitations. First, they generally do not control for spatial effects on longevity. In 17th Century Britain, for instance, medical practitioners were aware of differential mortality in cities and rural areas. The upper classes or nobility with access to medical practitioners usually fled to the countryside when the epidemics spread through the cities (Stub 1982). Second, the findings may be biased when not accounting for secular trends due to the modernization process and the solidification of Western capitalism. The type of data used in longevity studies usually consists of genealogical records from historic populations with information on births, deaths, marriages, parents, offspring, siblings, and so forth, but not on the major social, economic, and epidemiological transformations that affect survival chances.

Historical data covering periods prior to industrialization (pre 19th Century), i.e., covering the pre-transition/'pre-antibiotic' stage, would provide an excellent situation for the analysis of familial longevity. In historical Québec (1608 - 1800), for instance, environmental conditions were in a 'pure state', unhindered by the effects of modernization and the subsequent development of medical technology, public health, preventative health measures, industrial and agricultural pollution (e.g. industrial smog, sulphur dioxide, pesticides and herbicides), which may have confounding effects on survival. Social and environmental conditions were considered homogeneous and benefited the majority of inhabitants. Settled along the Saint-Lawrence Valley and its principal tributary shores, the vast majority of the first inhabitants were farmers who had

open access to most subsistence resources. Rather than allegiance to feudal lords, familial support and motivation to clear the forest were the key factors in the access to farmland and accumulation of subsistence resources. Water and air were clean, fish and game abundant, and crop failures infrequent. Most importantly, there was a low population density and a slower means of transportation, which aided in limiting the spread of contagions. For this reason, mortality rates were much lower than those prevailing in Europe at the same epoch (Charbonneau et al. 1993). Even so, demographic selection was in full momentum and weeded out frail individuals throughout infancy, childhood and adulthood. The lack of modern knowledge to manipulate mortality conditions (or to "cheat death") ensured in general, that individuals living in favorable social conditions and were endowed with positive genetic and frailty traits had the best chance of survival into the advanced ages.

From 1663 to 1681, however, an influx of French immigrants settled New France and infectious diseases prevalent in Europe traveled along with them (Charbonneau et al. 1993; Desjardins 2001). A strong natural movement brought about increasing population densities, and the epidemics that swept across much of Europe became more prevalent in Quebec. Originating from Québec City, the main port of entry, and spreading along the St. Lawrence River, smallpox, typhus, influenza/pneumonia and measles began to take much higher toll. As a result, the second waves of Canadian born were submitted to higher mortality rates than their predecessors.

The purpose of this paper is to examine the socio-demographic, environmental and biological correlates of longevity in families that lived, reproduced, and died prior the great societal transformation brought about by the industrial revolution. We used data

from the *Registre de population du Québec ancien*, a computerized database made up of biographical files for all individuals of European descent that lived in the Saint-Lawrence valleys in the 17th and 18th Century (Desjardins 1998). A previous study from Charbonneau and Desjardins (1990) has shown a positive, although weak, correlation in the age at death between settlers married before 1665 and their children. They also noticed that the dispersion in age at death was smaller among siblings than among all possible pairs of unrelated individuals, which they took as an additional support for family component in longevity. This conclusion was further supported by the fact that the correlation among spouses was null. More recently, Blackburn et al. (2004) undertook a similar analysis, but with a much larger sample, as the database has continued to grow from its implementation. All previous conclusions on the different correlations were shown to hold, except the one between spouses, which was now positive and significant.

The present paper builds on the last two studies, and on that of Perls et al. (2002). We tested, whether, siblings of long-lived individuals experienced a mortality advantage in post-reproductive years, using a series of Cox proportional hazard models. Although the focus was on siblings, we examined the possibility of coincidental associations by including a set of control variables such as parental and spousal ages at death. As mortality varied across time and space, we included additional variables that took into account the secular trends in mortality, urban/rural residence and geographic location. Since we found evidence that secular trends were conditional upon geographic location, we included the nominal predictors as a cross product term. The temporal/regional variables (and their interaction) account for a large part of differential mortality, which was largely regulated by population density in the pre-transition stage (Desjardin 1996).

DATA AND METHODS

Database and selection of cases

The data for the study was obtained from the *Registre de population du Québec ancien*, created by the *Programme de recherche en démographie historique* (PRDH) at the University of Montreal. This database includes documentation from the parish registers of Old Quebec from the first settlement in 1608 up to 1800 (Légaré 1988, Charbonneau et al. 1993). Individual and familial biographies were reconstituted by linking individuals to their baptismal, marriage and burial certificates. Overall, the database is made up of approximately 690,000 certificates dated prior to the 19th century that have come to us from 153 parishes, missions, and institutions. All individuals and their parents have a unique identification number and through various means, the entire sibship can be identified through either of the parents (however, individuals were not matched with half siblings through a remarried parent).

We avoided problems of right censoring by selecting 15,563 individuals born between 1625 and 1704. The cut-off date of December 31st, 1704 was chosen because the most recent death certificates available while writing this paper were recorded in the first decade of the 19th Century. This allowed an observation of survival to approximately 100 years of age. Further, individuals who did not belong to completed fertility families (mothers who had all their children before 1705) were removed because their inclusion would have biased the estimates toward the mortality patterns of lower birth order

individuals. This resulted in a cohort of 1,680 mothers with a total of N = 11,610 children.

The end of the female reproductive cycle (around age 50) also served as an additional cutoff age for inclusion in the study (N = 4,587 with 2,168 Males and 2,419 Females). A 'minimum life span' is necessary in historical populations because infant mortality is under registered. Further, the major causes of death (i.e. pestilence and famine) during the pre-transition stage would have the largest effect on the survival of young individuals (Oshlansky & Ault 1986). Newborns had a high risk of death from exogenous causes (i.e. accidents and infections) throughout infancy and childhood. Excess deaths from maternal mortality, accidents among males, and epidemics for both sexes created a high risk of death throughout adulthood, as well. Consequently, the life expectancy would be biased downwards ($e_0 = 43.72$ years for the children of the completed fertility cohort). Generally, mortality at younger ages is considered "unrelated to factors influencing longevity" and could conceal significant survival patterns occurring at the older ages (Vaupel 1988).

Another selection criterion involved the individuals who survived beyond 85 years of age (128 males and 190 females). Because we focus on sibling mortality, the above-defined "long lived individuals" without an older sibling themselves were excluded from the analysis. In families with two siblings surviving beyond 85 years of age (N = 100), the eldest of the two was considered the proband. If there were 3 siblings, the eldest and youngest were considered the probands. In total, 59 individuals greater than 85 years of age remained in the analysis (26 males and 33 females). Any individuals without a determined region of death (44 males and 15 females) were also removed from

the study. Additionally, we removed individuals without at least one sibling of either sex surviving beyond 50 years of age (141 males and 162 females). Lastly, we imputed the age at death for missing parents and spouses by taking the difference between the last time the mother or spouse were present and the first mention of their death in the records. We set the acceptable range at 15 years for the interval between these two dates to avoid overly biased estimates. Fifty-five missing parents, as well as 24 wives and 83 husbands, were thus added in the sample. The final portion of the children that remained in the study was N = 3,667, consisting of 1,736 males and 1,931 females.

Covariates

Urban/rural residence and geographic location were included in the model to control for regional, density and period effects of mortality. The Geographical Areas were divided into 3 districts based on the divisions by Gagnon & Heyer (2001). The *Western Area* includes: 1) North of Montreal; 2) Montreal Island &; 3) South of Montreal. The *Central Area* includes: 4) Trois-Rivières; 5) Portneuf and; 6) Lotbiniere. The *Eastern Area* includes: 7) Quebec City; 8) Orleans Island; 9) Beaupré and Charlevoix and; 10) Bas St-Laurent and Beauce. Rural residence and the Western Area were designated as the reference groups, respectively.

The individual's year of birth was entered into the model to control for secular changes and period effects caused by the numerous epidemics. Birth year was collapsed into 4 groups that consisted of individuals born between: 1) 1625 and 1669; 2) 1670 and 1679; 3) 1680 and 1689 and; 4) 1690 and 1704. The main reason for the coding scheme was to capture the effects of living through or being born during the times when there

was a high risk of death from periodic contagions. The 1625 to 1669 birth group was designated as the reference category.

Maternal and Paternal ages at death were entered as continuous covariates in the model to analyze the possible combination of genetic and/or shared environmental effects and their relation to the lifespan of their children. Additionally, spousal age at death was also included as a continuous predictor to control for cohabitational or shared environmental effects between marriage partners and other family members.

The core variable is the survival of siblings. If the ego had a brother or sister that lived beyond a certain age, then does it mean that he/she also has a better chance of survival than the general population? In this case, a sibling, regardless of sex, living beyond 85 years of age is considered as the threshold age of an extremely long-lived person. These individuals may have inherited exceptional vitality characteristics that would have slowed their rate of senescence. The sibling variable was entered as a categorical variable with the distinction of whether or not the ego had at least one sibling surviving beyond 85 years. Individuals with a sibling surviving over age 85 served as the reference group. Table 1 summarizes the coding scheme of the categorical predictors and gives the number of males and females in each category.

TABLE 1 ABOUT HERE

This leads to the test variable that was also included in the model as a predictor. Characteristics shared by the siblings include the inheritance of common frailty traits, longevity enhancing genes or favorable social conditions at birth. These characteristics should become apparent when the sibship is aggregated into a whole unit. If there is a large degree of heterogeneity within the sibship, then little or no similarity should be

apparent. In a natural fertility population, families are typically large and the chances that some children will survive past infancy, childhood and the ages associated with high external risks of death. Thus, we suspect that any familial resemblance in longevity can be captured more successfully through the sibship. When there are multiple survivors, ego's age at death can be compared to the aggregate age at death of the surviving members of the sibship. The average age at death of ego's same-sex siblings can be computed as follows:

$$A_{g50} = \frac{\sum_{\substack{g=sex \\ i=ego}}^{\infty} S_g - d_i}{C_g - E_i} \quad ;$$

and for the opposite-sex siblings: $$A_{g50} = \frac{\sum_{g=sex}^{\infty} S_g}{C_g} \quad ;$$

and for the total sibship: $$A_{T50} = \frac{\sum_{\substack{T=Total \\ i=ego}}^{\infty} S_T - d_i}{C_T - E_i} \quad .$$

The above formulas give each ego a unique average for the same-sex siblings, total sibship and a general average for the opposite-sex sibship members. The S_g represents each sibling's age at death, d_i is ego's age at death, C_g is the total number of surviving siblings at age x and $E_i = 1$ (i.e., ego). Aggregate sibship mortality may serve as a proxy variable to measure the degree of homogeneity between same-sex, opposite-sex siblings and the total sibship unit.

Cox regression models

A series of Cox regression models were fit, to assess whether the predictors had any influence on egos' survival time. The Cox regression model expresses a transformation of the hazard as a linear function of the predictors. A continuous hazard function is a rate with no upper bound (∞) and thus, the logarithm of the hazard is treated as the outcome variable (Singer & Willet 2003: 514):

$$\log h(t_i) = \log h_o(t) + [\beta_1 X_1 + \beta_2 X_2 + ... + \beta_i X_i].$$

The log hazard ($\log h(t_i)$) equals the baseline function ($\log h_0(t)$) or when the covariates equal 0 plus a weighted linear combination of predictors (β) that measure the effect of the covariates on $\log h(t_i)$. The interpretation of the 'time constant effect' involves: a) the partial coefficients, β's effect on log cumulative hazard (absolute amount) and; b) the hazard ratio, $e^{(\beta_1)}$ that measures the effect of a covariate on the raw cumulative hazard (relative amount).

There are two main assumptions involved in the Cox regression model. First, there is a log-linear relationship between the covariates and the underlying hazard function. Second, there must be a multiplicative relationship between the underlying hazard function and the log-linear function of the covariates (Blossfeld et al. 1989). This is also known as the proportionality assumption. It is assumed that the hazard function of any two individuals with different values of the covariates have parallel age (time) patterns (Namboodiri & Suchindran 1987, Elandt-Johnson & Johnson 1980). In other

words, the effect of β or $e^{(\beta_i)}$ will be at a constant distance from the baseline function over the entire lifespan or the hazard ratio should not be time dependent. Any potential violations of the proportionality assumption were checked with log[S(*t*] plots of the categorical variables and Schoenfeld residual plots of all covariates. The covariates showed no deviation from time invariance. Additionally, there were no significant correlations between the Schoenfeld residuals and time (age at death) for each of the covariates. Thus, all of the covariates appeared to meet the proportionality assumption.

RESULTS

The 17th and 18th Century inhabitants of the St. Lawrence Valley had quite remarkable survival characteristics. In our sample, 48.1% of males and 51.2% of females could expect to survive to age 50, while 2.8% and 4.0%, respectively, would survive to age 85. Males had an average of 1.87 brothers and 2 sisters that survived beyond 50 years of age (μ = 3.87). Likewise, females had an average of 1.78 brothers and 2.03 sisters that survived beyond 50 years of age (μ = 3.81). Among males and females, the mean age at death of brothers that survived beyond age 50 was 69.23 years, while the corresponding figure for surviving sisters was 70.95 years. In terms of sibship composition, 16.27% (N = 306) of males had either no brothers at all or none that survived to age 50, and 13.88% (N = 261) had either no sisters at all or none that survived until age 50. Similarly, 18.13% (N = 378) and 13.96% (N= 291) of females had no surviving brothers and sisters to that age, respectively. Lastly, 23.3% (N = 438) of males

and 20.9% (N = 435) of females had at least one sibling who survived beyond 85 years of age.

Figures 1 and 2 show the Kaplan-Meier survival curves of the entire male and female cohorts, respectively. The plots clearly demonstrate that those with a long-lived sibling were at a lower mortality risk over the age span than the rest of the population. For French Canadian males, median survival was 50.91 years for with those with a long-lived sibling and 45.07 years for men without one. Similarly, females with a long-lived sibling had a median survival time of 54.76 years and ones without an aged sibling had a median of 47.40 years. These estimates are likely biased upward because of under registration of infant mortality. The constant group difference over time may either indicate alternate: 1) socio-environmental conditions; 2) familial frailty characteristics or; 3) genetic endowments. Notice that the female curve has a slight degree of concavity throughout the reproductive span. High levels of maternal mortality must have had quite an impact on female survival patterns. In fact, a mortality crossover occurred between the ages of 25 and 40 years, when female mortality hazards surpassed those of their male counterparts (not shown here). Such concavity would likely create problems in the estimation of the parameters. This aspect served as a reason for selecting 50 years as the minimum age span (see data section).

Tables 2 and 3 show the overall model fit (χ^2) and hazard ratios ($e^{(\beta)}$) of the Cox regression models for the children belonging to completed fertility cohort born between 1625 and 1704. Overall, the Cox models fit the male data better than that of the female data. The differences could be the result of unobserved heterogeneity arising from the

lack of covariates on fertility patterns. Fertility patterns are an important contributor to female longevity and are studied in detail in an accompanying paper.

The first four models are nested and will be described accordingly. Model I shows the conditional relationship of the birth groups and geographical regions (Birth Group and Area product terms), while Model II only includes the family predictors. Model III and IV consider all variables entered simultaneously. Variables pertaining to familial effects produced a larger χ^2 difference from the null hypothesis model than those pertaining to period and geographical effects in Models I & II, although both models were significant ($p < .001$).

FIGURE 1 AND 2 ABOUT HERE

Model III had the best overall fit as can be seen by the larger χ^2 difference from the base model ($p < .001$). We also computed the models with robust standard errors and found that the magnitude and significance of the coefficients remained stable under those circumstances. Men living in urban dwellings had a 22.4% higher risk than those living in rural dwellings, whereas women had no significant mortality difference between residential states. In general, mortality was higher in the Eastern regions of the colony, and in the more recent birth groups. Males born between 1690 and 1704, who resided in or around Quebec City, had a mortality risk that was 42.0% higher than that of the earliest settlers of the Western regions ($e^{(\beta)} = 1.420$; $p<.001$). Similarly, females who lived more recently in the Central and Eastern Areas had a higher mortality risk than the oldest settlers living in the Western Area ($e^{(\beta)} = 1.569$ and $e^{(\beta)} = 1.343$, respectively; $p<.001$). The estimation of female parameters in the Central Area, however, could be biased upward because of the fewer residents in that region.

In contrast with results reported in other studies, father's age at death was not a significant predictor in any of the models and will not be dealt with further. On the other hand, mother's age at death had a significant effect on their sons' survival, although this effect was small. Sons had a 0.3% lower risk of death for each additional year that their mother survived, holding the other covariates constant. This effect remains small even when considering that the maternal age at death ranges from 21.55 to 99.41 years. For instance, a man whose mother died at age 83.18 (i.e., 20 years after the average) had a 6% lower hazard ratio than one whose mother died at age 63.18 (the average).

Model III also shows that, in the first years of the French Canadian colony, the presence of a long-lived sibling proves to be the strongest predictor of longevity for both men and women ($p < .001$). Men and women without a long-lived sibling had a 59.7 % and a 58.4% greater risk of death than those with a long-lived sibling. The magnitude of the coefficients are consistent and stable across all models, with or without the period, other familial effects and whatever the sample size. However, for theoretical reasons, we additionally considered another variable capturing the resemblance among siblings. In contrast, Model IV contains the aggregate sibship variable and Model V shows the aggregated deaths partitioned by sex, controlling for period and other familial effects. Aggregate sibship mortality is highly significant ($p < .001$). An annual increase in the sibship age at death gave men a 2.1% lower risk and females a 1.7% lower risk of death. Over a decade, this would cumulate to approximately a 21% lower risk among men and for women a 17% lower risk of death.

Notice the smaller number of cases in Model V (1222 Males and 1326 Females). There are three primary reasons for the different sample sizes. First, a greater proportion

of females surviving to the older age groups is normative in most populations. Secondly, many males or females had either no brothers or sisters or no siblings of either sex that survived until 50 years of age. Third, sibships may consist of either one sex or the other, or an even or uneven number of males and females.

TABLES 2 AND 3 ABOUT HERE

Despite a smaller sample size, the partitioned sibship covariate was significant among both sexes ($p < .001$). Men with sisters that survived an additional year had a 1.4% lower risk and with older brothers a 1.2 % less risk of death. Women, similarly, had a 1.0% and a 1.3% lower risk as their sisters and brothers aged, respectively. Over a decade, men would accumulate a 14% lower risk with older sisters and 12 % with older brothers. Likewise, women would experience a 10% and 13% lower risk of death with an aging sibship of sisters and brothers, respectively. Model IV is more suitable than Model V because the total sibship is taken into consideration and there are less missing cases.

Note, that the maternal age at death was not significant in Model V ($p > .05$). It appears that the smaller number of cases and the inclusion of mothers dying from pregnancy related complications (i.e. we did not left truncate mothers' age at death) may reduce the significance of the mother-son coefficient. Model VI and VII illustrate the effects of the inclusion of spousal age at death. This particular covariate decreased the model fit to the female data, for which the age at death of their husband had no significant effect. With respect to Model III, the sample size is smaller because of missing information on the spousal ages at death. Nevertheless, we believe that the reduced model fit does not explain the lack of influence of husbands' survival on their wives' ages at death. With about the same proportional reduction in sample size, the

model fit of the male data largely improves. Increasing the age at death of a wife by one year meant a reduction of .8% in the mortality risk of her husband (or an 8% lower risk for a 10 year increase). This effect is quite relevant when considering that the spousal age at death ranges from 19.10 to 96.03 years. Husbands whose wives survived to age 87.31, for example, had a 16% lower mortality risk than those with wives that died at the average of 67.31 years.

DISCUSSION

Our study on familial longevity in historical Québec has shown a number of significant findings. Some of our findings confirm, while others contradict the previous studies. Perhaps, the distinct population, historical period, the statistical methods, and the minimum lifespan account for the differences between the studies. For instance, the Cox regression model used in this analysis differs from the conventional ordinary least squares methods in that it can take the event occurrence into account. We did not attempt to estimate the heritability component of survival because there are limited ways to distinguish between genetics and the confounding effects of the environment. We will, nevertheless, discuss the two types of influences in light of our results.

Environmental influences

There was an attempt to isolate one major environmental influence and that was the epidemics. The main port of entry of New France was Québec City (Eastern Area), where epidemics would originate and sweep through the colony along the St. Lawrence

River, exposing long established French Canadian families as well as recent settlers to various parasitic and infectious diseases. Desjardins (1996) has shown how death tolls were modulated by distance, population size and density in the smallpox epidemic of 1702-1703. In some cases, the infection quickly spread to Montreal (Western area), largely sparing the smaller town of Trois-Rivières (Central area) along the way. However, in most instances, the time lag in the rate of spread allowed the inhabitants of the Western region to alter their behavior in preparation for the contagion. Furthermore, the mortality impact was less severe in the West because of the lower population density.

Epidemics also varied temporally to some degree, but this variation was largely conditional upon geographic location, justifying the inclusion of an *area* x *birth group* interaction term (models including separate, main effects for these variables had lower fit; not shown here). Those belonging to the last two birth groups (1680-1689 & 1690-1704) had a higher risk of death because they lived during the time of the epidemics (i.e. 1687, 1699, 1700, 1703, 1708, 1711, 1715, 1728 & 1733). Inhabitants also endured harsh winters and poor crop yields during some of those years. In contrast, when epidemics were spreading throughout the colony birth groups (1625-1669 & 1670-1679) were either already extinct or in adulthood, i.e., in the age strata with the lowest risk. Women from these birth groups in their reproductive ages, however, would not have escaped the higher death tolls.

Charbonneau et al. (1993) indicated that the first generations born in New France had no natural immunity to many of the viruses that came along in the 17th century (e.g. smallpox of 1702-1703). Females seemed more affected by these periodic epidemics, especially during their reproductive ages (Desjardins, 1996). The poor harvests and the

resulting vitamin deficiencies might have had more of an impact on females than males because of the greater susceptibility to cross-infections with their children. Furthermore, being under a natural fertility regime, French Canadian women expended a huge amount of energy on gestation and lactation, rather than on somatic maintenance (Doblhammer & Oeppen 2003). This aspect may have had adverse health effects lasting throughout late adulthood. Males may also show a similar pattern of lowered immunity from high-energy costs, but physical expenditure in this case would be more likely due to the extreme manual labor that was involved in land clearing and farming. Simply put, in comparison with contemporary conditions, the energy expended by both males and females was not consistently replenished because of a lack of nutrients. Thus, the parasitic and infectious diseases that were prevalent at the time may have overwhelmed the immune systems of some of the inhabitants.

Familial influences

In face of such adverse conditions, some could have benefited from a stronger vitality, inherited from their parents. In this regard, we initially suspected that our findings would parallel the other Quebec studies, as well as their European counterparts because Quebec originated as a colony of France. With regards to parent-offspring associations, only the mother/son pair proved to be significant in our analyses, although the magnitude of the effect was relatively small. This is in sharp contrast with the findings of Blackburn et al. (2004), who reported all significant parent-offspring associations.

Although the two studies are based on the same original data, the analyses are not based on the same selected samples. Our highly selective criteria (necessary for the

inclusion of controls) resulted in a smaller sample, and this could explain the lack of significance for the parental effects in our analysis. As an example, when using a larger sample by relaxing selection criteria, the effect sizes did not change for the paternal-daughter pairs, although they became significant at the .05 level. We believe that, significant or not, the strength of the association between parental and children' ages at death can hardly be strong in historical data.

Other studies support this argument. Both French studies (Cournil et al. 2000, Bocquet-Appel & Jakobi 1990) reported no maternal-offspring associations, however, an association appeared in the later cohorts of the British Peerage (Westendorp & Kirkwood 2001). Their study, however, covered a historical period when maternal mortality was not one of the strongest determinants of female survival. Similarly, perhaps the periodic effects of the epidemics and the high level of maternal mortality masked the effects of any possible parent-daughter resemblance. In other words, environmental effects exerted such a strong force on the female risk set that any genetic effects were negated to a considerable degree. Admittedly, this may affect the estimates, leaving one with a false impression that there was no inheritance of frailty.

More generally, it is also conceivable that the results largely depend on the choice of the minimum age at death for inclusion in the analysis. The minimum lifespan in this study was 50 years of age, while it ranged from 15 to 55 in other studies. A larger minimum lifespan tends to coincide with varying familial effects and this could partly account for the contradictory findings. Cournil et al. (2000) made an insightful comment on this issue in their study on the French Jura. They indicated that a parent-daughter resemblance surfaced when using 55 years as the minimum lifespan. However, when

taking into account the entire age span, there was a larger parent-son heritability component. We checked the Québec data for the patterns discussed by Cournil et al. (2000) and found a weak paternal-offspring resemblance using a minimum lifespan of 15 years instead of 50 years (not shown here). The effect was similar to the mother-son effect in the post-reproductive models presented here, but the effect vanished when the sibship and demographic variables were included in the analyses.

One can hardly argue for the complete inexistence of the transmission of longevity through genetic inheritance. But other, more important predictors may limit the effectiveness of a parental transmission. We found, for instance, that individual survival was strongly dependent on sibling survival. In agreement with the New England, Utah and Icelandic studies (Perls et al. 2002, Kerber et al. 2001, and Gudmunsson et al. 2000), individuals with long-lived siblings had a lower hazard than the general population. A remarkable consistency of the coefficients pertaining to this variable was observed in each of our models. Several interpretations may be furnished to account for such a large sibling effect.

Firstly, there is the possibility of a shared genetic component. The constant lower mortality risk of individuals with long-lived siblings, would favor an interpretation of positive genetic traits endowed at birth (see figure 1). This genetic variation would include a lack of predisposition to degenerative diseases, a stronger disease resistance or a slower rate of senescence (Perls & Terry 2003, Perls et al. 2002, Vaupel 1988). Additionally, dominance interactions within loci may be effective in siblings, but would not be shared by a parent and a child, for whom only the additive genetic variation is shared. That is, each parent transmits one copy of the two alleles at each locus and thus,

the child will never share the two alleles that act multiplicatively in the parent (Herskind et al. 1996, McGue et al. 1993). This aspect could explain why parent-offspring associations in ages at death are relatively small. One must be cautious, however, in drawing quick conclusions without a deeper consideration of environmental and socio-demographic processes.

Long-lived siblings may have shared positive social and sanitary conditions. Risk levels may cluster in families because family members tend to congregate in mutually supportive groups. In Québec, for instance, the family was considered a 'collective and egalitarian unit' (Bouchard 1994). There was a high degree of solidarity and, during the colonization phase, the family unit tended to migrate together and establish farms within a close proximity. These production units were usually established around the *vieux bien* and thus, the family members may have had a tendency to remain close to their immediate family. Simply, the norm of reciprocity and the mutual exchange of material resources among kin may have benefited some families.

A closer look at the socio-demographic aspects of survival and longevity suggests further theoretical perspectives. A common characteristic of individuals living past age 85 was their large families. The complete family size among mothers with such a long-lived child was 9.40 (SD = $\pm$ 3.49), while it was 7.09 (SD = $\pm$ 4.01) for mothers without any long-lived children. We believe that this factor creates a distinctive attribute between genetic effects and familial support networks. Large sibling units may mechanically take more time to die off than smaller ones simply because of a larger number of "trials". If the availability of siblings at older ages also improves survival prospects through the accumulation of resources and mutual sharing among kin, then an association between

family size and longevity may surface in the data. In this scenario, a “pushing effect” through “safety in numbers”, would limit the genetic inheritance of frailty.

Against this interpretation, one could argue instead for an association between fecundity and longevity: large sibling units surviving to advanced old ages would have inherited advantageous frailty characteristics (including longevity enabling genes) from highly fertile and healthy mothers. No study has examined the correlation between the crude fertility *of the parents* and the longevity *of their children*. In any case, this association would have to be fairly strong in order to explain the important agglutination of later ages at death in large sibships. Nevertheless, the inclusion of fertility related covariates and duration of environmental exposure may account for some of the heterogeneity of these women.

Males in our study were more dependent on the survival of their spouses and sibships than on that of their mothers. From these results, it would seem that spousal relations are not reciprocal to the extent that both the husband and wife would benefit equally. The lower mortality risk among males with longer lived wives may indicate the presence of a cohabitational effect (cohabitation here, is broadly defined to include geographical proximity as well, as actual cohabitation). In a patrilocal society, the close proximity of the paternal and fraternal kin, the strong kinship ties and mutual aid among males remained a constant buffer against hardships among men and changing roles over the life course. Conversely, women lived under the conditions and norms of their husband’s family, and thus their life experiences may have diverged from that of their biological relatives.

In terms of familial resemblance, females were only dependent on the survival of their brothers and sisters. Thus, siblings are exposed to shared social conditions from birth and the susceptibility to diseases tends to vary with age. This factor can possibly account for the closer relation of men and women to their siblings (i.e. similar social conditions). Further, these women survived the high risk stages during the reproductive span (e.g. lower immunity, cross infections and maternal mortality). The observed survival characteristics would make the French Canadian women a highly selective group and from this aspect alone, male cohabitants may have not had any bearing on their survival.

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TABLES

Table 1. Categorical coding scheme and number of individuals in each group

Residence	**Males**	**Females**
Rural (ref.)	*n= 1402*	*n= 1361*
Urban	*n= 334*	*n= 570*
Birth Group		
1625-1669 (ref.)	*n= 337*	*n= 388*
1670-1679	*n= 502*	*n= 583*
1680-1689	*n= 532*	*n= 590*
1690-1704	*n= 510*	*n= 524*
Area		
Western Area (ref.)	*n= 763*	*n= 818*
Central Area	*n= 331*	*n= 329*
Eastern Area	*n= 787*	*n= 938*
Family		
Sibling >85 (ref.)	*n= 438*	*n= 435*
Sibling <85	*n= 1443*	*n= 1650*

ref.: Reference category

Table 2. Cox Hazard Ratios for Québec males (1625-1704)

Model	Model I	Model II	Model III	Model IV	Model V	Model VI	Model VII
Sample size	*n=1736*	*n=1736*	*n=1736*	*n=1736*	*n=1222*	*n=1495*	*n=1495*
χ^2	38.89***	72.24***	107.22***	79.36***	66.43***	117.62***	93.83***
Urban	1.211**	n/a	1.224***	1.233***	1.158*	1.205**	1.219**
	[0.062]		[0.062]	[0.062]	[0.072]	[0.072]	[0.067]
Birth Group x Area							
1625-1669 x Western	ref.		ref.	ref.	ref.	ref.	ref.
1670-1679 x Central	1.197$^{n.s}$	n/a	1.216$^{\wedge}$	1.246*	1.230$^{n.s}$	1.245$^{n.s}$	1.285*
	[0.109]		[0.109]	[0.109]	[0.129]	[0.113]	[0.113]
1670-1679 x Eastern	1.058$^{n.s}$	n/a	1.096$^{n.s}$	1.074$^{n.s}$	1.104$^{n.s}$	1.057$^{n.s}$	1.042$^{n.s}$
	[0.079]		[0.079]	[0.079]	[0.097]	[0.087]	[0.087]
1680-1689 x Central	1.197$^{n.s}$	n/a	1.201$^{n.s}$	1.185$^{n.s}$	1.129$^{n.s}$	1.178$^{n.s}$	1.162$^{n.s}$
	[0.117]		[0.117]	[0.149]	[0.133]	[0.129]	[0.129]
1680-1689 x Eastern	1.367***	n/a	1.337***	1.348***	1.390***	1.404***	1.405***
	[.079]		[.079]	[.079]	[.093]	[.085]	[.085]
1690-1704 x Central	1.049$^{n.s}$	n/a	1.047$^{n.s}$	1.023$^{n.s}$	1.101$^{n.s}$	1.089$^{n.s}$	1.049$^{n.s}$
	[.116]		[.116]	[.116]	[.135]	[.131]	[.131]
1690-1704 x Eastern	1.470***	n/a	1.420***	1.376***	1.470***	1.388***	1.332***
	[.083]		[.084]	[.084]	[.108]	[.090]	[.091]
Family							
No Sibling >85	n/a	1.602***	1.597***	n/a	n/a	1.599***	n/a
		[0.061]	[0.061]			[.066]	
Maternal Death	n/a	.997*	.997*	.997*	.997$^{n.s}$	.996*	.996**
		[0.002]	[0.001]	[0.001]	[0.002]	[.002]	[0.002]
Paternal Death	n/a	1.001$^{n.s}$	1.002$^{n.s}$	1.000$^{n.s}$	1.002$^{n.s}$	1.002$^{n.s}$	1.000$^{n.s}$
		[0.001]	[0.002]	[0.002]	[0.002]		[0.002]
Spousal Death	n/a	n/a	n/a	n/a	n/a	.992***	.992***
						[.002]	[.002]
Aggregate Male Sibship	n/a	n/a	n/a	n/a	.988***	n/a	n/a
					[0.004]		
Aggregate Female Sibship	n/a	n/a	n/a	n/a	.986***	n/a	n/a
					[0.004]		
Aggregate Total Sibship	n/a	n/a	n/a	.979***	n/a	n/a	.979***
				[0.004]			[0.004]

p <.001***, p <.01**, p <.05*, p <.10$^{\wedge}$
n.s; not significant
ref.; Reference Category
n/a; not applicable
[ase]; asymptotic standard errors

Table 3. Cox Hazard Ratios for Québec females (1625-1704)

	Model I	Model II	Model III	Model IV	Model V	Model VI	Model VII
Sample size	*n=1931*	*n=1931*	*n=1931*	*n=1931*	*n=1326*	*n=1621*	*n=1621*
χ^2=	37.31***	59.45***	95.10***	65.10***	54.10***	68.96***	42.76***
Urban	1.017n.s [0.051]	n/a	1.023n.s [0.051]	1.011$^{n.s}$ [0.062]	1.020$^{n.s}$ [0.062]	.994$^{n.s}$ [0.057]	.988$^{n.s}$ [0.057]
Birth Group x Area							
1625-1669 x Western	ref.		ref.	ref.	ref.	ref.	ref.
1670-1679 x Central	.962$^{n.s}$ [0.120]	n/a	.966$^{n.s}$ [0.120]	.943$^{n.s}$ [0.120]	.900$^{n.s}$ [0.135]	.978$^{n.s}$ [0.129]	.961$^{n.s}$ [0.129]
1670-1679 x Eastern	1.102$^{n.s}$ [0.072]	n/a	1.100$^{n.s}$ [0.072]	1.087$^{n.s}$ [0.072]	1.030$^{n.s}$ [0.089]	1.065$^{n.s}$ [0.078]	1.050$^{n.s}$ [0.078]
1680-1689 x Central	1.334* [0.120]	n/a	1.343* [0.120]	1.339* [0.120]	1.662*** [0.139]	1.226$^{n.s}$ [0.139]	1.225$^{n.s}$ [0.131]
1680-1689 x Eastern	1.251** [.073]	n/a	1.258** [.074]	1.214** [.074]	1.236* [.088]	1.214* [.080]	1.159^ [.081]
1690-1704 x Central	1.537*** [.118]	n/a	1.569*** [.118]	1.549*** [.118]	1.406* [.144]	1.531*** [.129]	1.511*** [.129]
1690-1704 x Eastern	1.385*** [.074]	n/a	1.345*** [.075]	1.329*** [.075]	1.244* [.095]	1.317*** [.084]	1.291** [.084]
Family							
No Sibling >85	n/a	1.544*** [0.060]	1.550*** [0.060]	n/a	n/a	1.544*** [.067]	n/a
Maternal Death	n/a	1.000$^{n.s}$ [0.001]	1.000$^{n.s}$ [0.001]	1.000$^{n.s}$ [0.001]	1.000$^{n.s}$ [0.002]	1.000$^{n.s}$ [0.001]	1.000$^{n.s}$ [0.001]
Paternal Death	n/a	.997$^{n.s}$ [0.002]	.998$^{n.s}$ [0.002]	.997$^{n.s}$ [0.002]	.996$^{n.s}$ [0.002]	1.000$^{n.s}$ [0.002]	.998$^{n.s}$ [0.002]
Spousal Death	n/a	n/a	n/a			.997$^{n.s}$ [0.002]	.998$^{n.s}$ [0.002]
Aggregate Male Sibship	n/a	n/a	n/a	n/a	.987*** [0.003]	n/a	n/a
Aggregate Female Sibship	n/a	n/a	n/a	n/a	.990*** [0.004]	n/a	n/a
Aggregate Total Sibship	n/a	n/a	n/a	.983*** [0.004]	n/a	n/a	.984*** [0.004]

p <.001***, p <.01**, p <.05*, p<.10^
n.s; not significant
ref.; Reference Category
n/a; not applicable
[ase]; asymptotic standard errors

FIGURES LEGENDS

Figure 1. Kaplan-Meier survival functions for males with and without a long-lived sibling (Québec, 17th and 18th centuries)

Figure 2. Kaplan-Meier survival functions for females with and without a long-lived sibling (Québec, 17th and 18th centuries)

FIGURES

Figure 1.

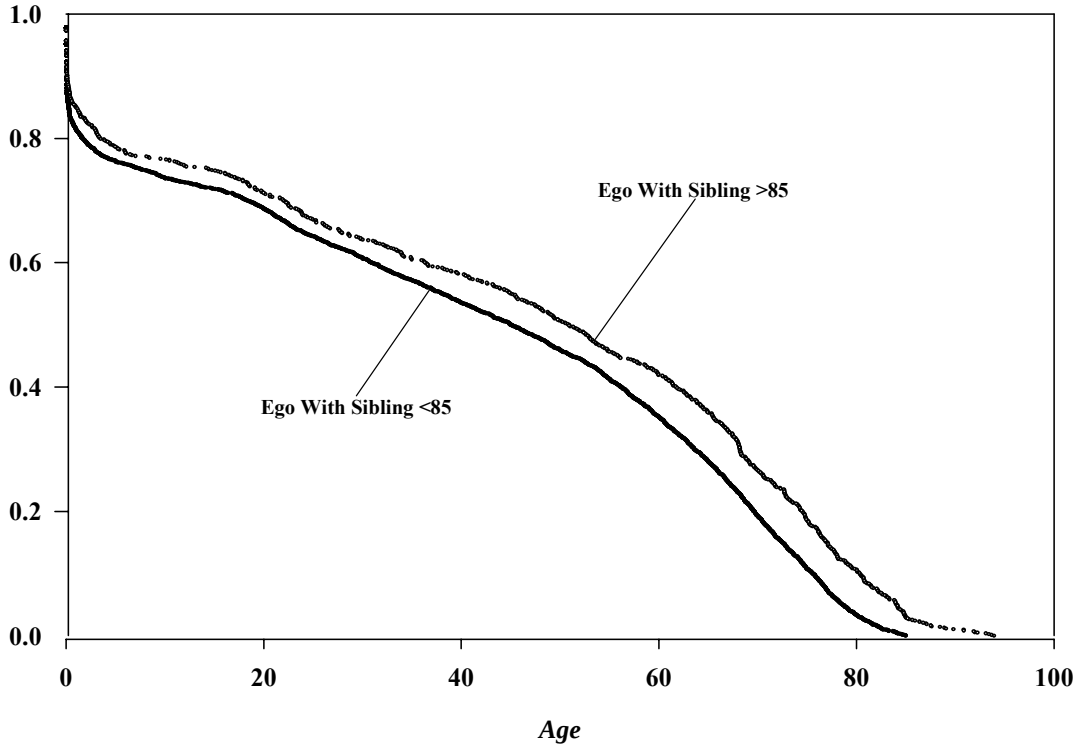
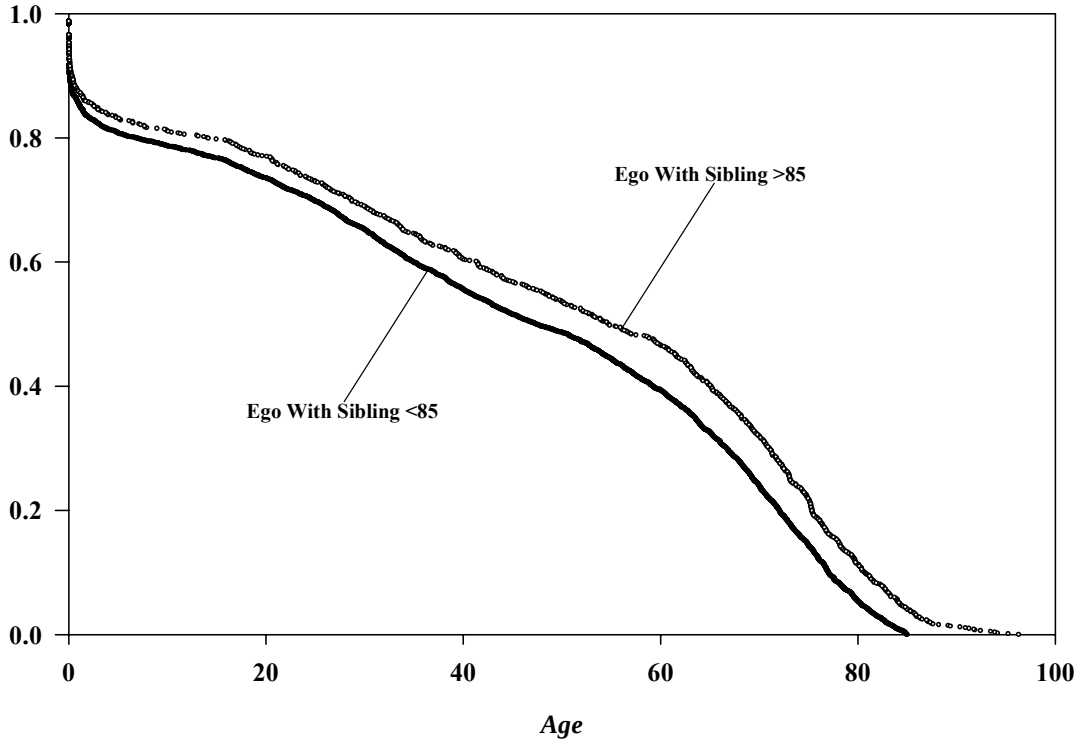


Figure 2.


1.0
0.8
0.6
0.4
0.2
0.0
0
20
40
60
80
100
Age
Ego With Sibling >85
Ego With Sibling <85