

## PSC Discussion Papers Series

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Volume 22 | Issue 5

Article 1

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9-18-2008

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### Recommended Citation

Gagnon, Alain; Smith, Ken R.; Tremblay, Marc; Vézina, Hélène; Paré, Paul-Philippe; and Desjardins, Bertrand (2008) "Is There a Trade-off between Fertility and Longevity? A Comparative Study of Three Large Historical Demographic Databases Accounting for Mortality Selection," *PSC Discussion Papers Series*: Vol. 22 : Iss. 5 , Article 1.

Available at: <https://ir.lib.uwo.ca/pscpapers/vol22/iss5/1>

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# Is There a Trade-off between Fertility and Longevity? A Comparative Study of Three Large Historical Demographic Databases Accounting for Mortality Selection

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Discussion Paper No. 08-05

Sept 18, 2008

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On the web in PDF format: <http://sociology.uwo.ca/popstudies/dp/do08-03.pdf>

Paper presented at the IUSSP International Seminar on Trade-offs in Female Life Histories: Raising New Questions in an Integrative Framework, Bristol, United Kingdom, 23-25 July 2008 organized by the IUSSP Scientific Panel on Evolutionary Perspectives in Demography, in collaboration with the University of Bristol, and with support from the British Society for Population Studies and the Galton Institute.

Preliminary version: please do not cite without contacting and asking the authors.

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## ABSTRACT

Frontier populations provide exceptional opportunities to test the hypothesis of a trade-off between fertility and longevity. In such populations, mechanisms favoring reproduction usually find fertile ground, and if these mechanisms reduce the chances for survival in old age, demographers should observe higher post-reproductive mortality rates among highly fertile women. We test this hypothesis using complete female reproductive histories from three large demographic databases: the *Registre de la population du Québec ancien* (Université de Montréal), which covers the first centuries of settlement in Quebec; the BALSAC database (Université du Québec à Chicoutimi), including comprehensive records for the region of Saguenay-Lac-St-Jean (SLSJ) in the 19<sup>th</sup> and 20<sup>th</sup> centuries; and the Utah Population Database (University of Utah), including all individuals who experienced a vital event on the Mormon Trail since the early 1800s. From these databases, we extracted, respectively, 5447, 1610, and 11395 women who survived married to age 50. Together, the three samples allow for comparisons over time (Old Quebec versus more recent Quebec and Utah) and space (Quebec versus Utah), and represent the largest data collection used to assess the impact of female reproduction on post-reproductive survival in a natural fertility context. Using survival analyses controlling for observed and unobserved factors we found a negative influence of *parity* and a positive influence of *age at last child* on post-reproductive survival in the three populations, with remarkably similar effect sizes in the three samples. However, we found little evidence of early fertility effects. We used Heckman's two-stage procedure to assess the impact of mortality selection during reproductive years, with no appreciable alteration of the main results. We conclude our empirical investigation by discussing the needs and the advantages of collaborative and comparative approaches.

## INTRODUCTION

Ten years ago, Westendorp and Kirkwood (1998) offered evidence for the disposable soma theory (Kirkwood 1977), according to which it is selectively advantageous to limit maintenance of somatic cells to accelerate development and reproduction, with the downside effect of faster post-reproductive deterioration and death. Using data from the British aristocracy, they reported the expected positive association between age at first birth and longevity and a reduction in the number of progeny for women who died after age 80. About a year later, Gavrilov and Gavrilova (1999) retorted with a critical review of the account, arguing that it failed to adjust for the obvious confounder of age at marriage (“the most important explanatory variable for both the number of children and for the age at first child”) and that the data were in fact inappropriate for such a study (incomplete genealogies with many underreported records). This critic found echoes in Le Bourg’s review (2001) and in a number of more recent contributions (see for instance Gagnon and others 2008).

Westendorp and Kirkwood’s empirical study was not the first of its kind, nor is the disposable soma theory the sole evolutionary interpretation of the empirical relationships between reproduction and senescence. The “antagonistic pleiotropy” theory – according to which deleterious mutations having a late age of onset freely accumulate over time if these mutations favor vigor and reproduction at younger ages – also lead to the prediction that delayed and less intense reproduction should be associated with longer lifespan (Williams 1957). Some had also previously tested the association, either by explicitly referring to the underlying action of evolutionary mechanisms (Le Bourg and others 1993) or not (Bideau 1986; Knodel 1988). Published in *Nature*, however, Westendorp and Kirkwood’s peerage

study revived the field by sparking immediate and sustained criticisms and by prompting new enquiries that attempted a replication (Le Bourg 2007). Unfortunately, the succession of investigations has resulted in more confusion than clarification, as inconsistent and contradicting results piled-up in the field. The two most recent literature reviews on the subject (Hurt and others 2006; Le Bourg 2007) are quite revealing in this regard. On reading, one is left with the impression that “it all depends”, that in some contexts, large parity would embody robustness and longevity while in other contexts it would instead compromise health and survival prospects. As an illustration of the divergence of findings, three studies found a positive relationship between total parity and post-reproductive survival (Müller and others 2002; Sear 2007; Voland and Engel 1986), three studies found the opposite (Doblhammer and Oeppen 2003; Gagnon and others 2008; Smith and others 2002), and four other studies showed no significant relationship at all (Bideau 1986; Helle and others 2002; Knodel 1988; Le Bourg and others 1993).

Ironically, among these references, the same data on the population of Old Quebec were used to support each of the three claims! This internal inconsistency refers to the eventual influence of total parity on longevity, but we could also report many contradicting results from the studies addressing the effects of ages at first or last birth. The choice of the relevant measure to assess the evolutionary significance or the health consequence of reproduction also varies quite extensively. Regarding the timing of reproduction, for instance, some studies identified delayed (Westendorp and Kirkwood 1998) or optimal (Mirowsky 2002; Mirowsky 2005) age at first birth as the relevant indicator, while other focused instead on age at last birth as a proxy for the timing of menopause and the overall rate of aging (McArdle and others 2006; Perls and others 1997; Smith and others 2002). Some also argued for the alternative position that the very fact of having children at a late

age could foster better health and survival chances in late life (Mueller 2004; Yi and Vaupel 2004).

It is likely that the lack of consensus in the literature is attributable to the adoption of different research protocols, varying data availability, choice and number of relevant measures (age at first birth, parity, etc.), population characteristics (e.g., with or without fertility control), and sample selection criteria (Grundy and Tomassini 2005; Hurt and others 2006). In addition, critically small samples may have led some (e.g., Helle et al. 2002) to report insignificant parity effects where lack of statistical power was probably the real issue. Following work initiated by Smith et al. (2002), and pursued in Gagnon et al. (2008), the present comparative study attempts to alleviate these problems by using the same sample selection criteria and performing the same statistical methods in the three large historical populations. The *Registre de la population du Québec ancien* at the Université de Montréal, the BALSAC database at the Université du Québec à Chicoutimi, and the Utah Population Database at the University of Utah allow for an observation at different time points (Historical Quebec on one hand versus the more recent Quebec and Utah on the other) and at different geographic locations (Quebec versus Utah). Yet, the three databases share one important characteristic: they comprise frontier populations that yield the most favorable conditions to study the relationship between fertility and longevity. In such conditions, there are very few intentional checks on reproduction, and if increased fertility reduces the chances for survival to old ages, higher post-reproductive mortality rates should be observed among highly fertile women. If late fertility can be reasonably taken as a sign of late menopause, and if late menopause occurs because of a slower rate of aging, than we should also see increased survival chances at advanced ages among women who were older at the time of their last birth.

To address these questions, we first estimate a series of proportional hazard models with a Gompertz specification of the risk of female mortality after age 50, controlling for a number of factors such as the interval between marriage and the first birth (as a proxy for the biological capacity to reproduce), the fraction of all children who died as infants, the year of birth, spousal age at death, residence in the case of Quebec, and religion in the case of Utah. To these control variables, we add random effects capturing unobserved factors shared by individuals from the same families (sisters). Since health selection during reproductive years may affect the true relationship between fertility and longevity (Doblhammer and Oeppen 2003), we use a two-stage Heckman sample selection procedure in the second part of our analysis to correct for the possibility that women surviving to age 50 are a non-random (i.e., more robust) subsample of reproductive-age women. Phenotypic correlations may indeed suppress fertility trade-offs if the healthier have both a high fecundity and a long life span (Helle and others 2002). To conclude, we briefly discuss the advantages of a comparative approach in a collaborative effort to unveil post-reproductive aging patterns in natural fertility populations. An accompanying paper from E. Grundy (===) in this issue and an earlier study (Doblhammer 2000) demonstrate the benefits of using a comparative strategy in contemporary populations.

## **DATA AND METHODS**

### ***The three populations***

The three sources of data used in the present study provide a unique opportunity to study the relationship between reproduction and longevity in a natural fertility context. The first retraces the completed fertility histories of women who lived in the St. Lawrence Valley in Quebec (born between 1599 and 1730; average 1703); the second also covers fertility



histories of women from Quebec but born on average one century and a half later (born between 1809 and 1870; average 1853) in the Saguenay-Lac-St-Jean (SLSJ) region; the third database includes women who were the contemporaries of the SLSJ women but who settled in Utah (born between 1753 and 1870; average 1852). All women included in the three samples have a known death record.

The Old Quebec (St Lawrence Valley) data were taken from the *Registre de la population du Québec ancien* (1608–1799), designed by the *Programme de recherche en Démographie Historique* at the University of Montreal (Charbonneau 1993; Légaré 1988). The database rests on the approximately 803,900 marriage, baptism, and burial certificates that were registered in 153 catholic parishes of pre-industrial Quebec from the onset of settlement (Desjardins 1998). It covers the vital events and kinship network of every individual of European ancestry who lived in the St. Lawrence Valley from before 1779, and the genealogy of every individual who married before 1800. As the population remained quasi-closed until the nineteenth century, the usual problem of missing observations due to inter-parish migrations is greatly reduced and the date of death is known for 88 – 89% of all those who married in the colony (Desjardins, 1999). The deaths records of individuals who died after the age of 50 were retraced up to 1850 for all individuals born before 1750, whose ages at death could thus be assessed to the age of 100. We removed women born after 1730 from our analyses since their reproductive histories are not yet completed. Fertility was particularly high among the settlers of New France. Data from the present study show an average of 9.97 children for each fertile woman who survived married to the age of 50 (i.e., for women whose family was “complete”; see Blum and Henry 1988; Henry 1968). As a result, the population increased at a very fast pace – from about 1,000

individuals in 1650 to 20,000 in the early 18<sup>th</sup> century, and 200,000 a century later (Charbonneau and others 2000).

The Saguenay-Lac-Saint-Jean region (SLSJ; current population 273,000) is located in the province of Quebec, approximately 200 kilometers north of Quebec City. Settlement in this region began during the second quarter of the 19th century (Roy and others 1988). Until the 1930s, the population was mainly rural, with fertility levels as high as in the old Saint Lawrence Valley (average of 9.79 children in complete families). All births, marriages, and deaths that occurred in this region since the beginning of settlement until 1971 were transcribed and linked in the BALSAC Population Register (Bouchard 1992; Bouchard and others 1995). This register presently contains 660,000 records from the SLSJ population and more than 1.7 million records from the other regional populations of the province of Quebec, covering the 19th and 20th centuries (BALSACProject 2008). The data for this study consist of all women who were born between 1829 and 1870 and who were married (only once) in the region.

Finally, our third sample of women was drawn from the Utah Population Database (UPDB), also one of the world's most comprehensive computerized genealogies. In the mid-1970s, over 170,000 three-generation families were identified on "Family Group Sheets" from the archives at the Utah Family History Library, each with at least one member having had a vital event on the Mormon Pioneer Trail or in Utah (Smith and others 2002). These families have been linked into multigenerational families. The genealogy provides data on migrants to Utah and their Utah descendants for more than 1.6 million individuals born from the early 1800s to the mid-1970's. The UPDB includes individuals who have lived in other states and countries and represent families with and without an affiliation to the Church of Jesus Christ of Latter-day Saints (LDS or Mormons). New families and their

members are continually being added as the UPDB adds other sources of data, including Utah birth and death certificates. Because these records include basic demographic information on parents and their children, fertility and mortality data are extensive with coverage up to the present. As for the SLSJ, we selected from the UPDB a sample of women who were born between 1753 and 1870 for comparability and in order to avoid potential biases originating from fertility control. There is, in either population, little evidence that women born before that effectively limited their fertility (Bean and others 1990)

### ***Data selection and multivariate analyses***

In order to maximize the comparability of our results, we used the same criteria to select our samples from the three populations, and applied the same statistical procedures in each case. In the first series of analyses, we selected all women who were married only once at a maximum age of 35, had at least two children (so that ages at first and last births could be computed and birth intervals would have a useful meaning), had a first birth interval larger than 8 months (to avoid confounding from premarital conceptions) and smaller than 5 years (very large first birth intervals could occur because the first birth has been lost), and who survived married until the age of 50 (the husband had to be alive when the woman reached age 50). From the three samples, we performed survival analyses with a Gompertz hazard rate. No right censoring occurred because all three cohorts were followed to extinction. The key independent variables were the age at first and at last birth, the interval between marriage and first birth, and the total number of children ever born or total parity.

We also included a series of control variables to check for coincidental associations, the most important being the fraction of infants who died before their first birthday as a general proxy for health (a woman who lost a large fraction of her infants probably had bad

rearing and health practices). In the tables, women with less than 20% of infant deaths were classified as women with “Low infant mortality”. Other control variables common to all three populations include the mother’s year of birth to capture secular trends in mortality and the age at death of the spouse to indirectly capture environmental conditions in the household. Other variables specific to the studied populations include the immigrant status (immigrants appear to have better survival in Old Quebec and SLSJ, a known effect of selection called the «healthy immigrant effect»), the urban/rural status (mortality in urban areas was higher in Old Quebec but lower in SLSJ), the region of residence (mortality was lower in the Western part of Old Quebec but did not vary appreciably by region in SLSJ and Utah) and religion (an important determinant of life-style and health in Utah but with little relevance in the “all Catholic” samples from Quebec).

Since individuals are clustered within families, we introduce a random effect accounting for interfamilial variation in the risks of deaths (Cleves and others 2004; Vaupel and others 1979). In other words, all siblings (sisters) from a same family are assumed to have the same value for this random (frailty) effect. Following a model estimation that assumes a gamma or an inverse Gaussian distribution of frailty, we obtain the variance of the random effect, “theta”, which reflects the interfamilial variation in the hazard. Exponentiating the square root of this theta and subtracting it from one gives the average variation in mortality between families expressed as a convenient hazard ratio (Pankratz and others 2005). For example, with a theta of 0.25, we may say that typically, the hazard is approximately 65% higher or lower than the baseline risk ( $\exp(0.25^{1/2}) - 1 = 0.65$ ). Defined for each familial cluster, such theta will account for a latent common group effect shared by family members.

Frailty models are a major advance in the study of time to event data. If the familial clustering is not taken into account then the estimated models typically lead to downward biases in parameter estimates and p-values (Garibotti and others 2006). Frailty models rely on *a priori* assumptions about the distribution of the random effect (gamma, inverse Gaussian, etc.). In some cases, however, it is possible to have a clue of the mechanisms that generate variations in frailty. Some processes occurring prior to entry into the observation period can generate a heterogeneity component that may be captured if the necessary data are available. Hardship endured during reproductive years in a natural fertility context, which often lead to maternal mortality, certainly qualifies as a strong selection mechanism. Could this mortality selection affect our parameter estimates tracing the effect of fertility on post-reproductive survival? The residuals – or the components left unexplained after having described such a selection process – could be interpreted as unobserved health factors. If these factors correlate with the residuals of our model predicting post-reproductive survival, then the two processes are not independent and we may say that selection affects our results from the survival analysis of post reproductive mortality.

Accordingly, economists and political scientists have recently proposed appropriate sample selection models in the context of time to event data (Boehmke and others 2006; Prieger 2002). Results from these models, however, are not immediately estimable using standard statistical software and do not cover the full range of correlations in the residuals. In order to alleviate the potential problem of selection, we use instead a Heckman two-stage modelling strategy (Heckman 1979). In the first stage, a probit model is used to assess potential factors leading to sample selection among all women who were married once (before age 35) and who had at least 2 children. Here, the dependent variable is dichotomous where women who survived to age 50 still married are coded as one and zero

otherwise. This probit selection equation generates the inverse Mills' ratio (IMR), which can be interpreted as the hazard of not being selected in the sample on which the Gompertz survival model is based (i.e., the hazard of not surviving married to the age of 50). Our independent variables include some variables already entered as controls in the survival model (year of birth, immigrant status, urban/rural status, and region of residence), as well as other variables more closely related to reproduction (age at marriage, first birth interval, mean birth interval, and whether the mother experienced a low rate of infant mortality). Note that variables such as total parity and age at last birth could not be entered in this selection model because of obvious endogeneity: a woman who has a late age at last birth (say age 48) is more likely to reach age 50 almost by definition. Similarly, women who had many children tended to have a later age at last birth than women who had fewer children, and including parity in the selection model would lead to a comparable tautological misspecification of the model.

In the second stage, the survival Gompertz model is estimated again, except that it now includes the inverse Mills' Ratios (IMR) originating for each individual from the estimates of the probit model in the first stage. A similar sample selection correction strategy was adopted in a study of the determinants of the timing of leaving home among young adults (Billari and Liefbroer 2007). Table 1 gives the descriptive statistics for all these variables in the three populations. Values from the larger sample of all married women are in the upper panel while values for the restricted sample of women who survived married to the age of 50 are in the lower panel.

**TABLE 1 ABOUT HERE**

## RESULTS

### *Models without correction for selection*

Table 2 presents the parametric survival analyses of post-reproductive mortality for our three populations. Entries are hazard ratios, meaning that coefficients  $>1$  are associated with an increase in mortality rates. It is immediately apparent that the parameter estimates of interest are remarkably similar in the three populations. While the first birth interval and the age at which this birth occurs do not affect post-reproductive survival (perhaps with the exception of age at first birth in Utah, which is marginally significant), age at last birth and parity have a remarkably similar significant effect in the three populations. Each additional year in the age at last birth represents, respectively in Old Quebec, SLSJ, and Utah, a reduction in the post-reproductive mortality rate of 1.9, 2.6, and 1.8% (that is the complement to one of, respectively, 0.981, 0.974, and 0.982). A ten years difference as for two women whose ages at last birth are 35 and 45 years would mean a difference of approximately 20-25% in the hazard. The effect of total parity is also consistent among the three populations, although somewhat more variable than that of age at last birth, and marginally significant in the case of SLSJ. The parameter estimate for parity is reduced when the models are not adjusted for the age at last birth – and vice versa (not shown here). The reason is quite simple: women who had a very late birth also tend to have large parities and since these two variables have opposing effects on survival, the estimate of one is suppressed when the other is not accounted for.

The results concerning the dummy variable “Infant mortality low” is not significant for the Utah sample but quite similar in the two Quebec samples, where the absence of notable infant mortality in a woman’s progeny can be taken as a sign of maternal health. A

woman who lost no more than two children out of ten (remember that the average total fertility in complete families is around 9-10 children) could hope for a 15% reduction in hazard rate, in comparison with other women. Using the percentage of infant deaths as a continuous rather than a categorical covariate leads to the same interpretation, although the parameter estimates are somewhat less consistent. At any rate, in evaluating the effect of parity, it is of prime importance to control for the fraction of infant deaths, as the two are positively correlated. Otherwise, one could take a large negative impact of large parity as a detrimental consequence of high reproductive costs, where in fact large parity could simply result from a high infant mortality load, indicative of poor environmental health conditions (e.g., the maternal depletion “vicious circle”).

#### **TABLE 2 ABOUT HERE**

Less influenced by biological constraints than fertility, the parameters for our control variables display more variation from one population to the other. Here, environmental, historical, and social factors are keys. Age at death of the spouse has a clear positive influence among women from Old Quebec and Utah but not among their 19<sup>th</sup> and 20<sup>th</sup> century counterparts of SLSJ. It is also interesting to note how 150 years of socio-sanitary transformations in Quebec has brought upon a complete reversal of urban influences on health and mortality. In Old Quebec, city dwellers endured a mortality rate 1.2 times higher than that of their rural counterparts (the hazard ratio for urban residence is 1.194). In SLSJ, just the opposite is true (the hazard ratio is 0.788). Note also the apparently more favorable conditions in the Western part of the colony of the Saint-Lawrence Valley, where epidemics were less severe in the earlier days of the Quebec population (Gagnon and Mazan 2006). Individuals active in the Church of Latter Days Saints (LDS or Mormons) enjoyed a 7% reduction in the risks of deaths in relation to all others. This survival benefit is most likely to



occur among LDS members because they are proscribed from the use of alcohol and tobacco and they enjoy a high degree of social integration and support with their religion (Mineau and others 2004).

As shown by the highly significant and sizeable theta parameter accounting for shared frailty among siblings, our set of controls has left aside significant portions of the variation in risks, especially in Utah. The theta parameter of 0.224 for Utah means that, in this population, the log of the risk typically varies by 47% (the square root of 0.224) around its mean from one group of sisters to the other. The exponential of this number provides a measure of per family risk expressed as a hazard ratio: on average this risk is about  $1 - \exp(0.47) = 60.5\%$  larger or smaller than the overall risk of death. The figure for Old Quebec is also significant, even though the parameter estimate is smaller. Still, it represents a 20% variation in the per-family risk of death, reflecting a sizeable degree of familial aggregation unaccounted for by the listed predictors. The parameter for SLSJ is of the same order than that of Old Quebec but is not significant. This could be explained by the smaller average sibship (sisters) size in this data, which is due to the larger fraction of immigrants. Removing the immigrants and running the same analysis for SLSJ yielded a significant theta value of 0.14 ( $p=0.044$ ), with no fundamental alteration of the other parameter estimates of the model. The hazard ratios of the 757 women native to the region for the variables age at last birth and parity were, respectively, 0.965 ( $p=0.029$ ) and 1.059 ( $p=0.028$ ), in good agreement with the results reported in Table 3.

### ***Correcting for sample selection***

Table 3 reports, in the upper panel, Gompertz proportional hazard models with the same covariates as in Table 2, except that these models now include the inverse Mills' ratios estimated from the probit model in the lower panel. The goal of this two-stage estimation

process is to account for the possibility of mortality selection bias. Women who survived to the age of 50 may not be representative of all the women with regards to their health and fertility outcomes. Consequently, the effect of these outcomes on post reproductive mortality rates may be biased if selection is not accounted for.

The first set of results to consider is in the lower panel of Table 3, which includes the probit sample selection estimates. Note that the sample sizes are larger in the selection models than in the Gompertz survival models. This is because all once married women are included in the selection model, not only those who survived married to age 50. Most of the variables included in the probit models have significant effects on the selection process, especially in Old Quebec, where close to half of the women did not survive married to the age of 50 (maternal mortality was of course more important in the older cohorts of the Quebec population). As expected, age at marriage and survival to age 50 are positively associated, which reflects the higher maternal mortality levels among women with a very low age at first birth. This time, the first birth interval has a clear effect, at least in the Old Quebec cohorts. The value of -0.068 for the probit estimate means that the odds of surviving to age fifty are decreased by approximately 13% with a one year increase in the interval ( $= 1 - e^{-0.068 \times 1.7}$ ). Long intervals could reflect low fecundability that could be a sign of poor health. The positive estimates for the average birth interval in the three populations also confirm the detrimental effect of short birth intervals, which originate through maternal depletion cycles, and are also reflected in the positive effect for the variable "infant mortality low". Note again the similarity of the coefficients for the three samples of cohorts. Overall, the effect sizes for the selection model are smaller in the case of Utah and larger in the case of old Quebec, with those pertaining to SLSJ being at an intermediate level; this was expected as a consequence of the varying percentage of women selected out

prior to age 50, respectively 17.5% (Utah), 31.9% (SLSJ), and 45.8% (Old Quebec). Indeed, the descriptive statistics of Table 1 show that women from Utah had better infant survival rates than their Quebec counterparts, which explains the differences in maternal mortality. Admittedly, the parameter estimates for the environmental controls are less coherent. But this was also expected.

### **TABLE 3 ABOUT HERE**

Turning to the Gompertz proportional hazard model in the upper panel, note the inverse Mills' ratio (IMR), obtained from the probit model of selection. The parameter is smaller than one, meaning that, because of some unobserved health factors, some women with a large inverse Mills' ratio (i.e., women who, according to the probit model, were not predicted to survive to age fifty) not only made it to age 50 but also had lower mortality risks thereafter. One has to exercise caution with this sort of interpretation, however, as it has been shown that selection models are quite sensitive to the choice of the covariate in the selection equation (Stolzenberg and Relles 1997; Winship and Mare 1992). Any misspecification may lead to errors in estimation and inference. Additionally, the parameter for the IMR is significant in Utah, marginally significant in Old Quebec and not significant in SLSJ.

Yet, although mortality selection, as specified here, does not fundamentally alter the parameters of the Gompertz models, most effect sizes increase with the introduction of IMR, just as one would expect for any time to event model that adds a few relevant predictors (Garibotti and others 2006). Of interest is the large increase in the estimate for parity in the three cases (which can be seen by comparing figures 2 and 3), through which health selection could indeed essentially operate (Doblhammer and Oeppen 2003). Our sample would contain a certain proportion of these more robust women who would have

been able to afford costly reproductive lives. At the same time, frail women who would have had more children than their frailty might have permitted would have failed to survive to age 50, meaning that our post reproductive sample would contain, among the frail women, mostly those whose reproductive life would have been less costly. The two mechanisms of selection “from above” and “from below” would combine to dampen the effect size of parity when selection was not accounted for.

Also of interest is the coefficient for the first birth interval, which is now marginally significant in the contemporary samples of SLSJ and Utah. While this variable clearly influenced the chances for survival to age 50 in the Old Quebec cohorts, it had no influence in SLSJ and Utah. In the Gompertz model, the opposite situation prevails. More favourable early adult life conditions among the more recent cohorts of SLSJ and Utah may have helped a larger fraction of frail individuals to reach age 50 than among the older cohorts of the Saint Lawrence Valley. The downside of the “reprieve” for these women – who may not have survived their reproductive period in the earlier days of the Quebec colony – is a higher post-reproductive mortality rate. The intensity of prior selection on subsequent mortality rate is also reflected in the gamma parameter, the exponential rate of increase of mortality. The reader may have noticed that gamma is much larger in Utah (0.123) than in Old Quebec (0.088) or SLSJ (0.089). On the other hand, the baseline level of mortality or the constant of the Gompertz model (not shown in the tables) at age fifty is lower in Utah than in Old Quebec or SLSJ. The smaller gamma in SLSJ (in comparison with Utah) could also be explained by the differential in selection during reproductive years, as we can see from the probit model. In comparison with Utah, higher selection in SLSJ would leave hardier cohorts at age 50 with a lower exponential increase in mortality.

The inverse association between initial and subsequent mortality has been explained, in part, by the process of differential selection or culling: when mortality risks are relatively low at the beginning of the risk period, more individuals (including the frail) are present in the population which, in turn, elevates the rate of increase in mortality of that population in the ensuing years (after age 50). Conversely, populations with high environmental threats have greater mortality risks at early ages resulting in a more robust subset of individuals who survive to age 50. These individuals are the select survivors who go on to reveal a slower rate of increase in mortality with age. This general finding was first demonstrated by Strehler and Mildvan (1960) and has been recently replicated in comparative mortality analyses between human and non-humans (Hawkes and others 2008).

## **DISCUSSION**

Empirical research on the relationship between fertility and longevity has furnished, over the last three decades or so, a series of results that are at best inconsistent. One is left with the impression that the relationship is largely context-dependent, and that no unifying framework can account for the variations across populations. Yet, it is hard to believe that such an important component of the life history as reproduction would have such variable effects, especially in natural fertility contexts. Since women's reproductive lives rest on a set of strong biological constraints, their reproductive (and, presumably, longevity) outcomes should be stable from one population to another. Unchecked, fertility should lead to the same outcomes or reveal the same clues about variations in women's robustness, whatever the context. Provided that researchers use similar sampling procedures and methods, the results should be replicable. If the issue has remained unresolved so far, it is perhaps

because of the extreme variation in the research protocols that were used to address it. The present work shows that when using the same research protocol, results are, after all, reassuringly consistent.

Is there a trade-off between fertility and longevity? Given the results of this comparative study, the answer is “yes”. Our results were robust to the introduction of numerous control variables, as well as to specification of shared frailty among siblings, and to mortality selection occurring during the reproductive years. Clearly, a large number of children in itself may be detrimental to a woman’s survival prospects in older ages. However, the trade-off is perhaps not as important as Westendorp and Kirkwood previously envisioned it: fertility may need to be fairly high for the trade-off to be revealed in these natural fertility populations. It would be interesting to replicate another comparative study in other natural fertility populations but with lower fertility levels. Analyses of contemporary Malthusian populations (i.e., with fertility control) accounting for socioeconomic differentials and other control variables also revealed the presence of a trade-off but at much lower levels of fertility (Doblhammer 2000; Grundy and Tomassini 2005).

Timing of the first birth, on the other hand, does not seem to bear strong influence on longevity. Age at first birth is not significant for the two Quebec samples, and in Utah, the effect size for this variable is relatively small in comparison with that of age at last birth or parity. The relative lack of influence of age at first birth is not surprising from an evolutionary perspective. In most human populations, the age at which women bear their first child depends primarily on the age at which they marry, which in turn depends on kinship systems, inheritance rules or demographic pressure (Laslett and Wall 1972; Wall 1983). Polymorphism on some fertility loci may well account for a significant part of the variation in age at menarche. This variation, however, seems to have no direct effect on the

timing of nuptiality. Hence, there may be relatively little selection on the timing of first reproduction within a fairly large range of ages (Helle and others 2005). It is possible, however, that a genuine beneficial effect of delayed first birth was blurred in the two Quebec samples since delayed first marriage – and delayed first pregnancy – may be the result of poor health (Gagnon and others 2008).

Variations in health can also be involved at the other end of reproductive life to explain the positive relationship between age at last child and post-reproductive survival; an early cessation of reproduction could be the result of poor health, which would of course be associated with an increase of mortality. However, women with a late child delivery were also advantaged in comparison with women with a median or average age at last child. Indeed, the effect of this variable is linear with the log of postreproductive hazard throughout the whole range of ages at last births in the three populations. This replicated result is consistent with the hypothesis of a slower rate of aging among women with late child bearing, for which we find additional support in the fact that brothers of women with a late childbearing also tend to survive longer (Smith and others 2008). Recent research has also hypothesized a genuine causal effect of late pregnancy on postreproductive survival. Yi and Vaupel (2004) suggested for instance that extended periods of endogenous estrogen production, later pregnancy, birth delivery, and breastfeeding could stimulate biological systems and positively affect survival and health. They also suggested that late fertility could foster better survival chances through adoption of healthy behaviors or through social support in older ages from younger children. Whether late fertility is a sign of good health or of a slower rate of aging, or whether it should be seen as a “life prolonging event” (Mueller 2004) or a combination of all these factors, the fact that our results are consistent and of the same order of magnitude in the three population is already quite encouraging.

More generally, before concluding on the mechanisms underlying fertility and longevity associations, it seems necessary to use similar methods and variables so that empirical investigations may be comparable. Many interesting theoretical issues remain irresolvable and subject to debate, definitively because of variations in data sources and methods. As a result, instead of replacing or complementing each other in a progressive march toward a consensus, alternative theories simply melt into an untidy bundle. Many of the current explanations for the associations between fertility and longevity may indeed account for a certain part of the variation, but lack of comparability and coordination in our empirical effort of validation makes it difficult to delineate the respective roles of each factor. As experimental research tracking the effect of fertility on longevity is obviously not possible with human subjects, comparative analyses using data from various populations is the best alternative. We hope that this modest initial step will encourage others to adopt collaborative approaches.

## **ACKNOWLEDGEMENTS**

We wish to thank the Huntsman Cancer Foundation for database support provided to the Pedigree and Population Resource of the Huntsman Cancer Institute, University of Utah. The *Registre de la population du Québec ancien* has received continuous support from the Université de Montréal. This work was supported by the Social Sciences and Humanity Research Council of Canada (AG) and by a NIH grant AG022095 (The Utah Study of Fertility, Longevity, and Aging; KRS). We also thank Dr. Michèle Jomphe and Manon Bouchard from the BALSAC project and Dr. Geraldine P. Mineau and Alison Fraser at the Pedigree and Population Resource of the Huntsman Cancer Institute for valuable assistance in providing the data.

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## TABLES

Table 1. Descriptive statistics of the three study populations

Variable	Old Quebec			Saguenay-Lac-St-Jean			Utah		
	Mean	Min	Max	Mean	Min	Max	Mean	Min	Max
<b>Total Sample</b>									
Age at marriage	21.9	11	35	22.1	11	34	20.4	12	35
First birth interval	1.20	0.67	4.98	1.18	0.67	4.98	1.27	0.60	4.99
Age at first birth	23.0	13	38	23.2	12	38	21.7	14	37
Age at last birth	38.5	16.4	49	39.5	17	49	39.9	18	50
Total parity	9.0	2	23	9.0	2	19	8.5	2	21
Mean birth interval	2.03	0.75	20.44	2.10	0.70	8.18	2.60	0.82	21.00
Infant mortality low	16%			71%			83%		
Birth year	1703	1599	1729	1853	1809	1869	1852	1753	1870
Immigrant	6%			52%					
Urban	18%			77%					
West	52%								
LDS							62%		
Age at death of spouse	66.9	23	99	71.7	22	101	70.9	23	103
Age at death	60.6	16	99	65.9	18	102	73.9	20	108
<b>Selected sample (age at death &gt;=50)</b>									
Age at marriage	22.2	11	34	22.3	13	34	20.5	13	34
First birth interval	1.19	0.67	4.95	1.18	0.67	4.98	1.27	0.60	4.99
Age at first birth	23.4	13	38	23.5	14	38	21.7	14	37
Age at last birth	41.1	19	49	41.5	17	49	40.5	18	50
Total parity	10.0	2.0	23	9.8	2	19	8.7	2	21
Mean birth interval	2.12	0.91	20.44	2.15	0.70	8.18	2.62	0.82	21.0
Infant mortality low	18%			74%			83%		
Birth year	1702	1599	1729	1852	1809	1869	1852	1753	1870
Immigrant	7%			53%					
Urban	16%			77%					
West	54%								
LDS							63%		
Spouse age at death	72.2	36	99	75.3	40	100.0	74	41	103.3
Age at death	71.4	50	99	74.4	50	102.5	76	50	108.0

Table 2. Gompertz proportional hazard models of post-reproductive mortality risks in three natural fertility populations (entries are hazard ratios)

Variable	Old Quebec		SLSJ		Utah	
	Hazard ratio	P-value	Hazard ratio	P-value	Hazard ratio	P-Value
<u>FERTILITY</u>						
First birth interval	1.011	0.622	1.073	0.098	1.001	0.385
Age at first birth	1.005	0.327	1.009	0.354	0.992	0.050
Age at last birth	0.981	<0.001	0.974	0.007	0.982	<0.001
Total parity	1.016	0.030	1.031	0.057	1.029	<0.001
Infant mortality low	0.885	0.001	0.855	0.009	1.030	0.642
<u>CONTROLS</u>						
Birth year	0.997	<0.001	1.000	0.899	0.996	<0.001
Age at death of Spouse	0.995	<0.001	1.002	0.502	0.996	<0.001
Immigrant status	0.938	0.323	0.886	0.046		
Urban residence	1.194	<0.001	0.796	0.000		
Live in the Western region	0.873	<0.001				
LDS					0.930	0.003
Gamma	0.088	<0.001	0.089	0.004	0.123	<0.001
Theta	0.033	0.023	0.028	0.193	0.224	<0.001
N	5,477		1,610		11,395	

Table 3. Gompertz post-reproductive relative hazards of death (upper panel) in Old Quebec, Saguenay-Lac-St-Jean (SLSJ), and Utah accounting for sample selection bias (lower panel) and using the two-stage Heckman sample selection procedure

	Old Quebec		SLSJ		Utah	
<b>Gompertz model</b>	<b>Hazard ratio</b>	<b>P-Value</b>	<b>Hazard ratio</b>	<b>P-Value</b>	<b>Hazard ratio</b>	<b>P-Value</b>
First birth interval	1.030	0.214	1.086	0.066	1.003	0.044
Age at first birth	1.004	0.350	1.011	0.289	0.986	0.002
Age at last birth	0.971	<0.001	0.963	0.020	0.972	<0.001
Total parity	1.036	0.005	1.054	0.081	1.056	<0.001
Infant mortality low	0.847	0.052	0.797	0.023	0.839	0.063
Birth year	0.998	0.003	1.001	0.680	0.995	<0.001
Age at death of Spouse	0.995	<0.001	1.002	0.487	0.996	<0.001
Immigrant	0.891	0.098	0.891	0.056		
Urban residence	1.281	<0.001	0.791	<0.001		
Live in the West	0.838	<0.001				
LDS					0.813	<0.001
Inverse Mill's ratio (IMR)	0.587	0.055	0.555	0.383	0.061	0.003
Gamma	0.088	<0.001	0.089	0.000	0.123	0.000
Theta	0.032	0.027	0.029	0.186	0.226	0.000
N	5,477		1,610		11,395	
<b>Selection model</b>	<b>Probit</b>	<b>P-Value</b>	<b>Probit</b>	<b>P-Value</b>	<b>Probit</b>	<b>P-Value</b>
Age at marriage	0.033	<0.001	0.030	<0.001	0.016	<0.001
First birth interval	-0.068	0.001	-0.046	0.314	-0.001	0.697
Mean birth interval	0.244	<0.001	0.279	<0.001	0.061	<0.001
Infant mortality low	0.130	<0.001	0.241	<0.001	0.179	0.006
Birth year	-0.002	<0.001	-0.003	0.196	0.001	0.073
Immigrant	0.158	0.002	-0.026	0.679		
Urban	-0.223	<0.001	0.025	0.697		
West	0.121	<0.001				
LDS					0.126	<0.001
_cons	2.240	0.008	5.023	0.277	-2.262	0.105
N	10,114		2,365		13,739	