Is there a trade-off between fertility and longevity? A comparative study of women from three large historical databases accounting for mortality selection

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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 longevity, demographers should observe higher post-reproductive mortality 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 Saguenay-Lac-St-Jean (SLSJ) in Quebec in the 19th and 20th centuries; and the Utah Population Database (University of Utah), including all individuals who experienced a vital event on the Mormon Trail and their descendants. Together, the three samples allow for comparisons over time and space, and represent one of the largest set of natural fertility cohorts used to simultaneously assess reproduction and longevity. Using survival analyses we found a negative influence of *parity* and a positive influence of *age at last child* on post-reproductive survival in the three populations, as well as a significant interaction between these two variables. The effect sizes of all these parameters were remarkably similar in the three samples. However, we found little evidence that early fertility affects post-reproductive survival. The use of Heckman's procedure assessing the impact of mortality selection during reproductive ages did not appreciably alter these results. We conclude our empirical investigation by discussing the advantages of comparative approaches.

INTRODUCTION

Ten years ago, Westendorp and Kirkwood (1998) offered evidence for the disposable soma theory (Kirkwood, 1977), which posits that it is selectively advantageous to limit the maintenance of somatic cells in order to accelerate development and reproduction, with the downside effect of faster post-reproductive deterioration and death. Using data from the British aristocracy, they reported a 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 that Wesdendorp and Kirwood's account failed to adjust for the obvious confounder of age at marriage ("the most important explanatory variable for both age at first child and family size") and that the data used were inappropriate for such a study (incomplete genealogies with many underreported records). This critique was echoed in Le Bourg's review (2001) and in a number of more recent contributions (e.g., Gagnon et al., 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 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 evolutionary mechanisms (Le Bourg et al., 1993) or not (Bideau, 1986; Knodel, 1988). Published in *Nature*, however, Westendorp and Kirkwood's peerage study revived this research area by sparking criticism 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 results piled-up in the field. The two most recent literature reviews on the subject (Hurt et al., 2006; Le Bourg, 2007) are quite revealing in this regard. The reader is left with the impression that "it all depends," and that in some contexts, large parity would embody robustness and longevity while in other contexts it would instead compromise survival prospects.

As an illustration of the divergence of findings, three studies found a positive relationship between total parity and post-reproductive survival (Müller et al., 2002; Sear, 2007; Voland and Engel, 1986), three studies found the opposite (Doblhammer and Oeppen, 2003; Gagnon et al., 2008; Smith et al., 2002), and four other studies showed no significant relationship (Bideau, 1986; Helle et al., 2002; Knodel, 1988; Le Bourg et al., 1993). Ironically, among these references, the same data on Old Quebec were used to support each of the three claims! This internal inconsistency refers to the relationship between parity and longevity, but we could also report many contradicting results from the studies on the effects of ages at first or last birth.

It is likely that the lack of consensus in the literature is attributable to the adoption of different research protocols, varying data availability, choice of 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 et al., 2006). 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 three 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. Together, the three databases allow for an examination at different time points (Historical Quebec versus the more recent Quebec and Utah) and at different geographic locations (Quebec versus Utah). The three databases also share one important characteristic: they trace the demographic history of frontier populations, which probably 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 (described below) and adding random effects capturing unobserved factors shared by 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. The paper concludes on the advantages of comparative approaches. An accompanying paper by E. Grundy (===) in this issue and an earlier study (Doblhammer, 2000) also demonstrate the benefits of a comparative strategy in contemporary populations.

DATA AND METHODS

The three populations

The Old Quebec (St Lawrence Valley) data were taken from the *Registre de la population du Québec ancien* (1608–1850), 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). 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 married individuals (Desjardins, 1999). In the present study, we included women born between 1599 and 1730 (average 1703). 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 18th century, and 200,000 a century later (Charbonneau et al., 2000).

The Saguenay-Lac-Saint-Jean region (SLSJ; current population 273,000) is located approximately 200 kilometers north of Quebec City. Settlement in this region began during the second quarter of the 19th century (Roy et al., 1988) and, 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 et al., 1995). The register presently contains 660,000 records 431,500 births, 91,160 marriages and 122,570 deaths records from the SLSJ population and more than 1.87 million records (mostly marriage records) from the other regional populations of the province of Quebec, covering the 19th and 20th centuries (BALSAC-Project, 2008). The data for this study consist of 3265 women who were born between 1829 and 1870 (average 1853).

Finally, our third sample of women was drawn from the Utah Population Database (UPDB), also one of the world's most comprehensive computerized genealogies. 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 Trail or in Utah (Smith et al., 2002). 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. 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. We selected from the UPDB a sample of women who were born between 1753 and 1870 (average 1852) for comparability with the SLSJ data and in order to avoid potential biases originating from fertility control. There is, in either population, limited evidence that women born before that effectively limited their fertility (Bean et al., 1990).

Data selection and multivariate analyses

In order to maximize comparability, 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 and who bore at least two children (with a first birth interval larger than 8 months and smaller than 5 years). All women survived married until the age of 50 (the husband had to be alive when the woman reached age 50). This sample selection strategy helps tracking the multivariate effects of both *intensity* and *timing* of fertility on longevity, and is not intended to account for the eventual impact of childlessness or very low fertility. In order to use the first birth interval as a proxy for *fecundability* (biological capacity for reproduction), it is customary to exclude women with small or very large intervals because these may either signal a premarital conception or the loss of the record. Be that as it may, results were not appreciably altered when uniparous cases or unusual first birth intervals were reintroduced in the tabulations (not shown here).

From the three samples, we performed survival analyses with a Gompertz specification of the baseline hazard. No right censoring occurred because all three cohorts were followed to extinction (all women have a known, recorded date of death). The key independent variables were the age at first and at last birth, the interval between marriage and first birth, and total parity. Since the relative influence of parity may vary according to the timing of fertility, we tested a series of interaction effects between parity and age at first birth and age at last birth. Only the latter was significant and the results are presented accordingly. Since mortality could be far higher in women with the highest fertility (a j-

shaped effect), we tested a quadratic specification of parity, though this effect was not significant.

We also included a series of control variables, 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 might have had bad rearing and health practices or lived in an inhospitable setting). 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), 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 also used a random (frailty) effect accounting for interfamilial variation in the risks of deaths (Cleves et al., 2004; Vaupel et al., 1979) with a usual *a priori* assumption about the distribution of the random effect (here, "Gamma"). The data allow us, however, to have a clue of the mechanisms that generate variations in frailty. Hardship endured during reproductive years, which often led to maternal mortality, certainly qualifies as a strong health selection mechanism. Could this selection affect our estimates of 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 (Doblhammer and Oeppen,

2003). If these factors correlate with the residuals of our model predicting post-reproductive survival, then the two processes are not independent and selection affects our results regarding post reproductive mortality.

Accordingly, economists and political scientists have proposed appropriate sample selection models in the context of time to event data (Boehmke et al., 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 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, as well as other variables more closely related to reproduction (see Table 3). Note that variables such as total parity and age at last birth could not be entered in this selection model because of 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.

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 with increasing values of the covariate. 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), the main effects of age at last birth, and parity, as well as their interaction are remarkably similar in the three populations. Comparing women with mean parities (i.e., not accounting for the interaction with parity), it can be seen that an extra year in the age at last birth represents, respectively in Old Quebec, SLSJ, and Utah, a reduction in the post-reproductive mortality rate of 2.6%, 3.6%, and 2.2% (the complement to one of 0.974, 0.964, and 0.978).   A similar reduction of 2.8% for each additional year was also

obtained by Helle et al. (2005) for the Sami women in Finland. Concerning the main effect

of parity, an extra child for women with mean ages at last birth represents an increase of

the mortality rate by 1.6%, 3.2%, and 3.1%, respectively for the three populations. The

effect of total parity is also consistent among the three populations, although it is marginally

significant in the case of SLSJ. Being smaller than 1, the parameters for the interaction term

indicate that the relative benefit of a late age at last birth increases at high parities (or that

the relative penalty of increasing parity declines with late ages at last birth). Note also that

the effect of parity appears smaller when the models are not adjusted for the age at last

birth – and vice versa (not shown here): 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 variable "Infant mortality low" are 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 20% of her children

(remember that average fertility in complete families is 9-10 children) could hope for

approximately 15% reduction in hazard rate. It is important to control for infant mortality.

Otherwise, a large impact of parity could be taken as the 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.

TABLE 2 ABOUT HERE

Less influenced by biological constraints than fertility, our control variables display

more variation. For instance, the age at death of the spouse has a clear positive influence

among women from Old Quebec and Utah but not among their 19th and 20th century

counterparts of SLSJ. It is interesting to note how 150 years of socio-sanitary

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transformations in Quebec has brought upon a complete reversal of urban influences on 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 is 1.191). In SLSJ, just the opposite is true (hazard ratio=0.799). Note also the more favorable conditions in the Western part of the Saint-Lawrence Valley, where epidemics were less severe (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 (hazard ratio=0.929). 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 with their religion (Mineau et al., 2004).

As shown by the highly significant theta parameter accounting for shared frailty among sisters, the set of controls has left aside significant portions of the variation in risks, especially in Utah. The theta parameter of 0.222 for Utah means that the log of the risk typically varies by 47% (i.e., $\sqrt{0.222}$) 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 $1-\exp\sqrt{0.222}$ = 60.5% larger or smaller than the overall risk. The figure for Old Quebec is also significant, although smaller, with 20% variation in the per-family risk of death ($1-\exp\sqrt{0.031}=0.193$). The parameter for SLSJ is of the same order but is not significant, perhaps because of a smaller average sibship size. Removing the immigrants (who belong to smaller sibhips) and running the same analysis yielded a significant theta value of 0.14 (p=0.044), with no fundamental alteration of the other parameter estimates of the model.

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. 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 more important in the older cohorts of the Quebec population). As expected, age at marriage and survival to age 50 (i.e., being selected in the sample) are positively associated, which reflects the higher maternal mortality among women with a young age at first birth. This time, the first birth interval has a clear effect, at least in the Old Quebec cohorts, where long first birth intervals could reveal a poor health. The negative value for the probit estimate means that the odds of surviving to age fifty decrease with increasing intervals. The positive probit estimates for the average birth interval in the three populations confirm the detrimental effect of a short birth interval, which may originate with maternal depletion cycles. The estimate for the variable "infant mortality low" (positive) is consistent with this interpretation. 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

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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 may explain the differences in maternal mortality. 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 who were not predicted to survive to age fifty according to the probit model not only made it to age 50 but also had lower mortality risks thereafter. Here, one has to exercise caution, 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). Moreover, the parameter for the IMR is significant in Utah and Old Quebec but not significant in SLSJ.

Provided that our model is correct, we may infer that selection does not fundamentally alter the results of our Gompertz survival model as all effects remain in the same direction. Yet, most effect sizes increase with the introduction of IMR, just as one would expect for any survival model that adds some relevant predictors (Garibotti et al., 2006). Of interest is the sizeable increase in the estimates for parity, through which health selection could indeed essentially operate. The samples would contain a certain proportion of 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 permitted would have failed to survive to age 50, meaning that the post reproductive samples would contain,

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among the frail women, mostly those whose reproductive life would have been less costly (see Doblhammer and Oeppen, 2003 for a similar interpretation). 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 SLSJ and Utah. This factor influenced the chances for survival to age 50 in the Old Quebec cohorts, but not in SLSJ and Utah (see probit model). 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. In general, the inverse association between initial and subsequent mortality has been explained 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. Conversely, populations with high environmental threats have greater mortality risks at early ages resulting in a more robust subset of individuals who survive and who go on to reveal a slower rate of increase in mortality with age. Note that the exponential rate of increase of mortality (or gamma in the tables) is lower in Old Quebec (0.088) than in Utah (0.123). 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 et al., 2008).

DISCUSSION

Empirical research on the relationship between fertility and longevity has furnished 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 reproduction can have such a variable effect on survival, especially in natural fertility populations. Unchecked, fertility should lead to similar mortality outcomes or reveal similar 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 research protocols. The inconsistencies may also partly stem from the fact that fertility influences on longevity are not very large, and can thus be masked by variations in other important environmental influences (e.g., urban versus rural residence). The present work shows that using the same research protocols and yet specifying appropriate measures of environmental influences, leads to results that are, after all, reassuringly consistent.

Is there a trade-off between fertility and longevity? Given the results of this comparative study, the answer appears to be "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 may be detrimental to a woman's survival prospects in older ages. However, the trade-off is not as important as Westendorp and Kirkwood previously envisioned it: fertility may need to be fairly high for the trade-off to be revealed. We also observed an interaction effect between parity and age at last birth that indicates a more

complicated scenario: the detrimental effect of high parity is weaker for women who gave birth to their last child at an older age. Hence, having children late may be taken as a sign of robustness or of delayed aging (see below) and this signal would be stronger for women who already had many children. It would be interesting to replicate another comparative study in other natural fertility populations with lower fertility. Analyses of contemporary populations with fertility control 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). Hence, there may be relatively little selection on the timing of first reproduction within a fairly large range of ages (Helle et al., 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 et al., 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 in older ages. However, the effect of age at last birth was systematic with the log of postreproductive hazard throughout the whole range of

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ages in the three populations. This replicated result is consistent with the hypothesis of a slower rate of aging among women with late child bearing. We find additional support in the fact that brothers of women with a late childbearing also tend to survive longer (Smith et al., 2008).

Recent research has also hypothesized a genuine causal effect of late pregnancy on post reproductive 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. Determining 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, will demand further research. The fact that our results are consistent and of the same order of magnitude in the three populations is already quite encouraging.

More generally, before concluding on the mechanisms underlying fertility and longevity associations, it seems necessary to use similar research protocols and methods so that empirical investigations may be comparable. Many interesting theoretical issues have remained irresolvable and subject to debate 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. Several explanations for the associations between fertility and longevity may 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 remains the best alternative. We hope that this modest initial step demonstrates the usefulness of collaborative and comparative approaches.

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TABLES

Table 1. Descriptive statistics of the three study populations

Variable	Old Quebec			Saguenay-Lac-St-Jean			Utah		
Total Sample	Mean	Min	Max	Mean	Min	Max	Mean	Min	Max
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%		
Spouse age at death	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
N	10,114			3,265			13,739		

Selected sample (age at death >=50)	Mean	Min	Max	Mean	Min	Max	Mean	Min	Max
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	101	74.2	41	103
Age at death	71.4	50	99	74.4	50	102	76.0	50	108
N	5,477			1,610			11,395		

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

Variable	Old Quebec Hazard ratio	P-value	SLSJ Hazard ratio	P-value	Utah Hazard ratio	P-Value
First birth interval	1.011	0.611	1.078	0.080	1.001	0.364
Age at first birth	1.005	0.342	1.009	0.366	0.992	0.055
Age at last birth (a)	0.974	<0.001	0.964	0.001	0.978	<0.001
Total parity (b)	1.016	0.026	1.032	0.047	1.031	<0.001
Interaction a×b	0.998	0.021	0.997	0.059	0.998	0.017
Infant mortality low	0.886	0.001	0.857	0.009	1.030	0.638
Birth year	0.997	<0.001	1.000	0.994	0.996	<0.001
Age at death of Spouse	0.995	<0.001	1.002	0.534	0.996	<0.001
Immigrant status	0.939	0.324	0.893	0.061		
Urban residence	1.191	<0.001	0.799	<0.001		
Live in the Western region	0.873	<0.001				
LDS					0.929	0.003
Gamma	0.088	<0.001	0.089	<0.001	0.123	<0.001
Theta	0.031	0.030	0.030	0.179	0.222	<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	
Gompertz model	**Hazard ratio**	**P-Value**	**Hazard ratio**	**P-Value**	**Hazard ratio**	**P-Value**
First birth interval	1.034	0.157	1.092	0.053	1.003	0.029
Age at first birth	1.004	0.373	1.011	0.282	0.985	0.002
Age at last birth (a)	0.962	<0.001	0.953	0.005	0.966	<0.001
Total parity (b)	1.041	0.002	1.057	0.077	1.063	<0.001
Interaction a×b	0.998	0.010	0.997	0.062	0.998	0.007
Infant mortality low	0.843	<0.001	0.811	0.015	0.822	0.038
Birth year	0.998	0.003	1.001	0.750	0.995	<0.001
Age at death of Spouse	0.995	<0.001	1.002	0.516	0.996	<0.001
Immigrant	0.882	0.072	0.898	0.079		
Urban residence	1.295	<0.001	0.793	<0.001		
Live in the West	0.831	<0.001				
LDS					0.801	<0.001
Inverse Mill's ratio (IMR)	0.527	0.023	0.546	0.382	0.046	0.001
Gamma	0.088	<0.001	0.090	<0.001	0.123	<0.001
Theta	0.030	0.037	0.031	0.173	0.223	<0.001
N	5,477		1,610		11,395	
Selection model	**Probit**	**P-Value**	**Probit**	**P-Value**	**Probit**	**P-Value**
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
Intercept	2.240	0.008	5.023	0.277	-2.262	0.105
N	10,114		2,365		13,739	