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Influences of Early Life Conditions on Old Age Mortality in Old Québec

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ABSTRACT

Increasingly, bio-demographers are turning to infancy and childhood to gain a better understanding of old age mortality. However, evidence of a link between early life conditions and survival until old age is fragmentary, and the intervening mechanisms remain unclear. Drawing from data on a cohort of French-Canadian children born in the 17th and 18th centuries, we study the effects of infant exposure to infectious diseases (as revealed by the infant mortality rate in the year of birth) on later life mortality. A series of Cox proportional hazard models are used and we control for other familial and environmental conditions prevalent in childhood, as well as in adulthood. Results point to a slight, but not significant effect of a disease load in infancy for females born in years of exceptionally high infant mortality. The results are also not conclusive for males. More generally, a trend of increasing infant mortality over time correlates with general decreases in post-reproductive mortality rates, which are probably due to period improvements in later life conditions. Our study supports the view that period changes have stronger relevance than cohort effects in the study of historical variations in old age mortality.

INTRODUCTION

Despite spectacular improvements in survival and longevity over the past two centuries, we still have limited knowledge about the factors limiting or enhancing old age mortality. Recent research has pointed to early life conditions that have across-the-board associations with chronic conditions and survival prospects in adult life (Lawlor et al., 2004; Poulton et al., 2002; Preston et al., 1998). Fogel (1993) proposed causal mechanisms that connect malnutrition *in utero* and in infancy to chronic diseases in later life. Barker (1992; 1995) suggested that the conditions for coronary heart disease, hypertension, or stroke were initiated prior to birth. War-time malnutrition has been used as a natural experiment to test the effects of extreme exposure, with inconsistent results (Kannisto et al., 1997; Roseboom et al., 2000; Sparen et al., 2004). The month of birth could also influence lifespan through seasonal variations in early life conditions (Doblhammer and Vaupel, 2001). The authors do not indicate which of the prenatal or the early infancy periods are the most influential on later life. Rather, they insist on mechanisms that link birth weight to variations in maternal nutritional status during pregnancy.

Other studies have specifically identified the exposure to epidemics in infancy as a debilitating factor with far-reaching consequences. According to Fridlizius, infectious diseases during the first year affect survival in later life through irreversible damages on the immunological system (Bengtsson and Lindstrom, 2000). One study from 18th-19th century Sweden has shown that individuals born during years of smallpox and whooping cough epidemics had an increased risk of death past age 50 (Bengtsson and Lindstrom, 2000; Bengtsson and Lindstrom, 2003). Two studies in England suggested that respiratory related infectious diseases in the first year of life are associated with later cough, phlegm, and impaired ventilatory function (Barker et al., 1991; Shaheen et al., 1994). Finch and Crimmins (2004) took the issue further and hypothesised that a reduction in the exposure to infectious diseases has made a major contribution to the historical decline in old-age mortality. Analysing birth cohorts since 1751 in Sweden, they reported strong associations between early and old-age mortality in the same cohorts. When reported on a logarithm scale, lines representing probability of death (q_x) for successive cohorts shifted downward by roughly the same amount at all ages. Crimmins and Finch (2006) recently provided further positive associations between mortality at age 1 and 70 of successive cohorts by year of birth in Sweden, France, Switzerland and England. They also proposed a general model linking inflammatory processes and growth in early life to cardiovascular diseases in old age. Using alternative plots on the same data and other works on human mortality (Kannisto, 1994), Barbi and Vaupel (2005) argued for the contrary proposition that period effects were generally more important than cohort effects in the historical decline of old age mortality.

An alternative way of addressing this issue is to study the relationship between early and old age mortality in a context of *increasing*, rather than *decreasing*, infant mortality. Provided that period effects are less important than cohort effects in the balance (as claimed by Finch and Crimmins) we expect that increases in old ages mortality would follow increases in infant mortality of the same cohorts. The pre-transition population of Québec (17-19th centuries) provides an ideal setting for such a “natural experiment”. Founded in 1608 along the St. Lawrence River, Nouvelle-France had a low rate of natural increase for half a century. The colony was sparsely populated over a large area which limited the spread of relatively rare epidemics. These inhibitors led to very low mortality levels for that historical period. As the peopling intensified, however, pathogens found more fertile grounds and, as a result, infant and child mortality began to rise. Did those increases translate in higher mortality rates at older ages in the same cohorts? Or

were they counteracted, at the outset of the 19th century, by incoming period improvements in health and hygiene?

These are some of the questions addressed in this paper. More precisely, our objective is to determine how exposures to epidemics in infancy affected the old age survival prospects of the first French Canadians. To achieve this, we used survival models to analyze data from the *Registre de population du Québec ancien* (Université de Montréal). Since the effect of early life conditions on old age mortality may be mediated by intermediary life events and other factors, we introduced a number of controls such as parity, birth order, parental death, and the number and the average age at death of siblings who survive past age 50. Some of these variables have already been studied and proved to be important factors of old age mortality (Gagnon et al., 2006; Mazan and Gagnon, 2006). Additionally, as mothers in childbearing ages and their young children are often at the highest risk during epidemic outbreaks, we feel that it is necessary to study mothers and children in conjunction. It has been previously shown that maternal death significantly increased pre-reproductive mortality in the past Québec population (Pavard et al., 2005). This stressful event, both physiologically and emotionally, entails serious health consequences that threaten immediate survival. How it would affect the survivors' chances for continued existence to old age is unclear. So far, only the consequences of family disruption (Lundberg, 1993; Schwartz et al., 1995; Tucker et al., 1997) and of breastfeeding (Wingard et al., 1994) on subsequent health and old age mortality have been researched. No investigation has yet addressed the long term effects of a complete lack of maternal care in infancy; for an infant who lost his/her mother during an epidemic outbreak, the effect of exposure to inflammation might prove hard to distinguish from that of missing maternal care. Whether the death of a father during childhood would impinge on longevity has also not received attention.

The use of controls has also relevance in addressing the role of the life cycle in mediating the impact of early life events on old age mortality. Increasingly, researchers are turning to a life course approach to understand the origins of disparities in late life mortality. Biological, behavioural and environmental pathways that operate across life-course, as well as across generations, would influence morbidity and mortality at older ages (Ben-Shlomo and Kuh, 2002; Kuh et al., 2003). For example, a study from the Health and Retirement panel has demonstrated a net effect of childhood conditions on adult morbidity (Blackwell et al., 2001), while another based on the National Longitudinal Survey of Older Men showed few residual childhood effects on old age survival after controlling for adult life mediating factors (Hayward and Gorman, 2004).

Preston and colleagues proposed a useful typology of relations between early and late life mortality risks (Preston et al., 1998) that has yet to be fully integrated into research designs. They presented a two-by-two contingency table with the directions of the relation (positive or negative) as row entries and the causative mechanisms (direct or indirect) as column entries. For example, some conditions or diseases acquired in childhood could permanently impair the survivors, whose elevated mortality risks at older ages would confirm a positive and direct association (Fridlizius's hypothesis). Secondly, a positive but *indirect* association could also surface if those who are born into advantageous conditions are likely to retain the advantages throughout life. In Nouvelle-France, people who were born in the countryside were more likely to live their entire life in the countryside and were thus advantaged in comparison with city dwellers, who endured higher mortality rates at all ages. Not accounting for this differential residence pattern could lead one to falsely attribute the higher mortality rates of city dwellers exclusively to exposure to epidemics in infancy, as infant mortality was higher in urban areas. Familial environments and health behaviours may also correlate across the life cycle and

produce the spurious observation that diseases acquired in childhood leave an indelible imprint on death rates at older age. Thirdly, genuine or "real" positive associations may be reverted by strong selection mechanisms whereby frail individuals do not even survive up to the beginning of the period of observation (here age 50). Consequently, the association between early life conditions and later life mortality is indirect and negative, or inexistent because the sample would mostly consist of robust individuals. Although generally neglected (Preston et al., 1998), some efforts were allocated to track such selection mechanisms (Doblhammer and Vaupel, 2001). We feel that their role could be fundamental, especially in pre-transition populations, where infant mortality was particularly high. How could it be otherwise? The force of selection increases as a direct function of the severity of scourge. Finally, the association may be direct but *negative*: those who were exposed in infancy to a disease such as smallpox (to which immunity can be acquired) would be safer when the disease would surge again. This possibility may look far fetched in modern context, but it is totally sound in a historical context when diseases were not yet endemic and would come back periodically.

DATA AND METHODS

Data were extracted from the *Registre de population du Québec ancien* (RPQA) at the University of Montreal (*Programme de recherches en démographie historique;* PRDH). Vital statistics included in this database comprehensively covers the beginning of New France in the early 1600s to the end of the 18th century. The database includes documentation from the parish registers of Québec from the first settlement. Individual and familial biographies were reconstituted by linking individuals to their baptismal, marriage and burial certificates, indirectly allowing for prospective observation without the usual biases of selectivity in retrospective settings. This basic information is complemented by various socio-demographic characteristics such as socio-professional status, occupation, ability to sign, place of residence and of origin. As the PRDH has recently added the burials of the first half of the 19th century, old age mortality patterns may be observed up to 1850 for people born in the colony prior to 1750. Overall, the database contains 803,900 certificates from 153 parishes (Charbonneau, 1993; Desjardins, 1998). From the outset, priests were entitled by Rome to record baptisms, marriages, and burials. Parishes were organized rapidly and when demographers began microfilming their registers in the late 1960s, they generally found them in very good conditions. Episodes of epidemics, famine and fluctuations of food prices (wheat) are also documented (Dechêne, 1974; Dechêne et al., 1981; Desjardins, 1996).

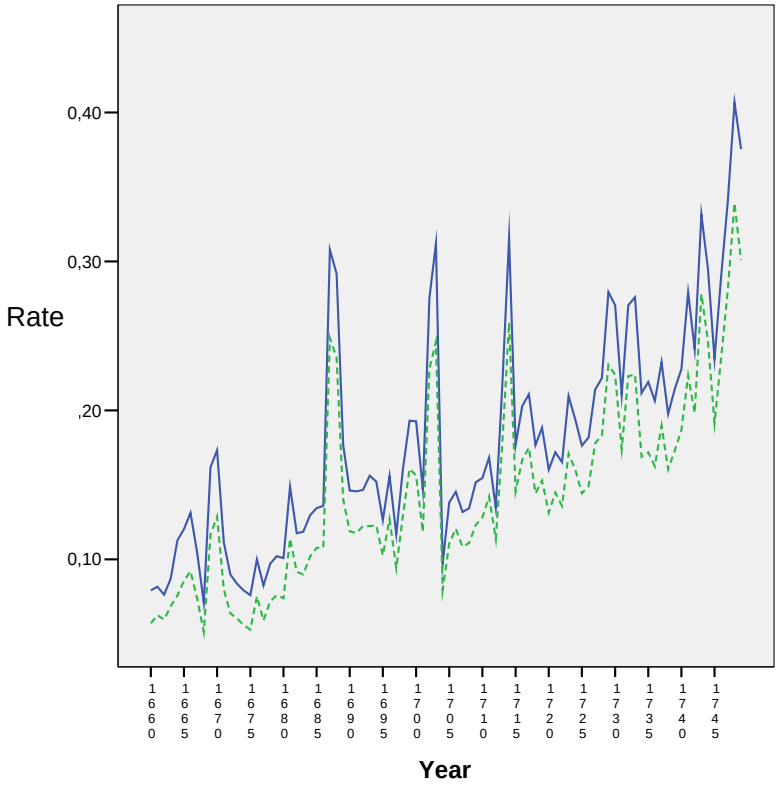
During the French Regime (1608 – 1759), Canada developed from a small colonial trading post to an organized colony with sizable administrative and military apparatus. Growth of the population was initially slow, the colony numbering only around 1,000 individuals in 1650. Only when the French Crown took charge of the peopling of the colony, from 1663 to 1673, did demographic expansion really begin. Thereafter, growth was essentially natural and supported by fertility rates among the highest ever recorded. Women who survived to their 45th birthday bore an average of 9.2 children (Charbonneau, 1975). By the early 18th century, an agricultural population of approximately 20,000 inhabitants has settled along the Saint-Lawrence River. Quebec City and Montreal, the main ports of entry in Nouvelle-France, were emerging as urban centres with important administrative and economic roles. The rapid increases did not level off after the Conquest, and the fertility characteristics of the French Canadian population remained essentially the same under the English Regime.

Due to low population density, which inhibited the spread of diseases, and other favourable conditions such as availability of fresh water, abundance of fish and games, the early colonist experienced quite low mortality for the time (Charbonneau, 1993). Canada had no overcrowded cities and benefited from alternating hot and cold seasons. But it did not manage to escape epidemic outbreaks. The overcrowded ships arriving from Europe periodically introduced contagious diseases such as smallpox, typhus, cholera, and influenza and the native population, which had no natural defences against those diseases, formed an ideal reservoir for the multiplication of the responsible bacteria and viruses. For example, the important smallpox epidemic of 1702-1703 was apparently started by an Indian chief from Orange (now Albany), who died in Quebec City on the 19th of October 1702 (Desjardins, 1996). Contagions also began to take much higher tolls when population increased. Formally sporadic, some diseases became endemic, while others kept on coming back periodically, when the immunity levels in the population were low. Harsh winters or bad crops in some years would have further weakened the inhabitants, offering the ideal breading grounds for contagions (Charbonneau, 1993).

Infant mortality and epidemics

Figure 1 shows the development of infant mortality from 1660 to 1750. Starting from relatively low values, the level steadily increased over time. Two curves are presented. In one case (plain line), we considered all children as deceased for whom no recorded date of death or marriage certificate were available. In the other case (dotted line), we knowsupposed that such children survived their first year of life. The second curve probably underestimates the “true” mortality level as those whose fate is unknown probably died sooner than the others, and most likely during infancy. This doesn’t mean that the first curve is, in return, an overestimation. The rates were tabulated on the basis of recorded births and deaths as they appear in the parish registers. Simply, some births (and subsequent deaths) were not registered because the newborn died before baptism. Using the average interval between birth and baptism, Charbonneau (1975) estimated a loss of around four percent of births. According to Nault et al. (1990), this sort of under-registration was more important before 1680, when many parishes were yet to be established in the colony. From 1680 on, however, the data become highly reliable, and we will thus begin our enquiry with that year. If anything, a sudden increase in mortality during the epidemics would be underregistered, due to the disorganisation of the administrative authorities. However, it appears that these authorities were still accomplishing their duties in a time of crisis (Desjardins, 1996). The fact that the two curves are parallel also attests to the general quality of the records. In addition, we are more interested in the shape of the infant mortality curve than in the “true” level. Working with “apparent” infant mortality is possible provided that underregistration of births and deaths is independent of the variables studied.

Figure 1. “Apparent” infant mortality in the 17th and 18th Century



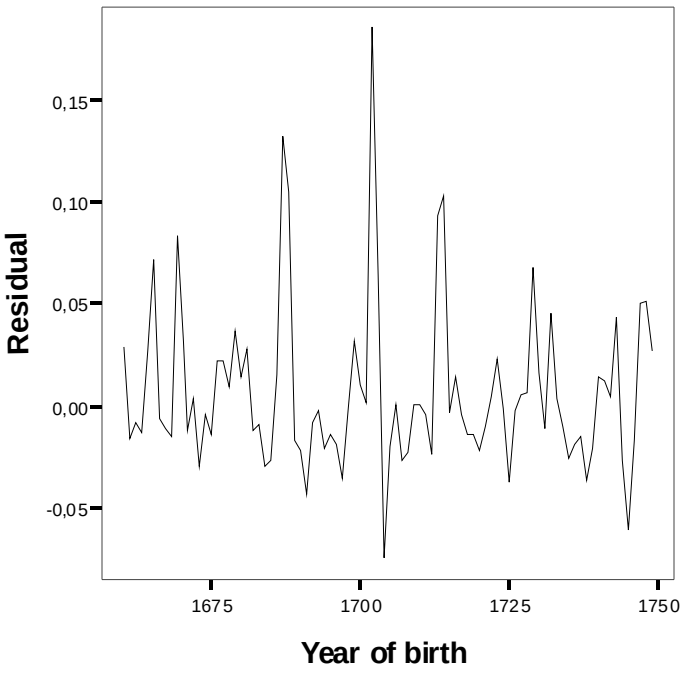
Judging from the noticeable peeks of the years 1669, 1687, 1699, 1703, 1714, 1729, and 1733, one has to acknowledge that priests could register most deaths during the outbreaks. The 1702-03 smallpox epidemic is well known and was studied in detail by Desjardins (1996). The colony experienced again another outbreak 1733. Given contemporary description, the 1687-88 and 1699 epidemics were probably due to typhus (Desjardins, 1996), although the last peak of the 17th century was previously attributed to smallpox (Charbonneau, 1993). When measles hit the colony in 1714, most victims were children; few adults perished, which suggests that they acquired immunity during previous occurrences. Whatever might have cost the lives of a large amount of infants in 1664 and 1669 is unknown. Published information on the matter is for the moment limited, imprecise, if not false or misleading (Desjardins, 1996). Because these events took place before 1680, however, they won’t be considered in this study. Regarding the upper limit of observation, record linkages are for the moment incomplete after 1739. Introducing individuals born after this year would, in such a situation, artificially create a positive association between infant mortality and death rates at older ages because the death of those who died earlier is more likely to be linked to their birth. Overall, our study is based on 8,928 female and 8,660 male births spanning from 1680 to 1739.

Covariates

In order to fully appreciate the impacts of the epidemics, we extracted the general “trend” of infant mortality by using a flanking 5 year moving average, and plotted the residuals from this trend on Figure 2. This figure clearly shows that upward fluctuations are much larger than downward fluctuations; they are the signature of the epidemics. As time passed, however, the residuals began to peek at lesser and lesser values. Since many formerly sporadic contagious diseases

probably became endemic, the general level of infant mortality increased during the first half of the 18th century.

Figure 2 Fluctuations from the “trend” of infant mortality



Instead of using the overall infant mortality rate as a covariate shared by all individuals born the same year (as in Bengtsson and Lindstrom, 2003), we used the infant mortality rate in the region of residence and in the twelve months *following* birth. The regions were defined following Gagnon and Heyer (2001), with additional categories to distinguish Montreal and Quebec City as urban centres, where mortality was higher. The consideration of both time and space is critical in the context of a relatively large but sparsely populated territory such as Nouvelle-France. In the early 18th century, smallpox surged at different places and at different times. For instance, someone born in the region of Quebec City (East) in the spring of 1703 probably escaped exposure to the pathogen in early life. In contrast, another child born the same month on the Island of Montreal (west) would have been at a much higher risk of death. By the time the epidemics reached Montreal, it had completely disappeared from Quebec City (Desjardins, 1996). We also preferred to use the crude level of infant mortality instead of a combination of “trend” and “cycle” (or residual). The level of infant mortality is in itself an indicator of the disease load in infancy, and the fact that this load increases over time should be taken into account, as well as its spatial variation. Moreover, the trend of mortality strongly correlates with the year of birth ($r > .9$). Although the large sample size prevents the typical inflation of standard errors that usually occurs with severe multicollinearity, the interpretation of the coefficients would remain hazardous. When two variables are almost perfectly correlated, there is simply no room (and no relevance) to assess the effect of one of them while the other is maintained constant.

Table 1 furnishes the descriptive aspects of the variables used in this work. Mortality varied over time and space, and also according to a number of familial

conditions. We thus introduced a set of family-related controls. These variables should not only be considered as mere controls without any substantial interest. A number of studies have demonstrated the strong influence of parity and birth order on infant and child mortality (Nault et al., 1990). These early life conditions may also influence old age mortality, although admittedly to a lesser extent. Another event in early life that could affect survival to old age is that of parental death. In Nouvelle-France, the death of the mother entailed pre-reproductive mortality risks that were two times higher than those of the general population (Pavard et al., 2005). This may explain why a very small percentage of individuals who lost their mother in infancy survived through age 50, and points toward the selection mechanisms we were alluding to above. The fact that a similar percentage of individuals lost their fathers at the same age, however, also suggests the presence of inter-familial heterogeneity, in which parental death would indicate higher mortality risks shared by family members. The number of siblings living to age 50 was used as a proxy to capture the effect of mutual support among kin. The average age at death among them would capture this reality as well, but would also be determined by genetic sharing, or biological "robustness". Having many siblings who survived to old ages may increase longevity in a synergic way, so we introduced a cross-product term to capture the eventual interaction.

Table 1 Descriptive statistics of the sample

VARIABLE	FEMALES	MALES
Number of cases	8,928	8,660
Regions		
West North	7%	7%
Island of Montreal	10%	10%
West South	15%	15%
Montreal (City)	9%	8%
Centre	15%	16%
Quebec Region	15%	15%
Quebec city	8%	6%
East North	9%	9%
East South	12%	12%
Mean year of birth	1717.8	1717.8
Mean infant mortality rate in the year of birth, in the region of residence	191 per thousand	
Mean parity (weighted by family size)	9.3	9.5
Mean birth order (rank)	5,3	5.3
Percentage who lost their mother in their first year	0.7%	0.6%
Percentage who lost their mother between age 1 and 15	14.6%	13.9%
Percentage who lost their father in their first year	1.1%	1.1%
Percentage who lost their father between age 1 and 15	17.2%	16.4%
Mean number of siblings alive at age 50	4.05	4.09
Average of the mean age at death among siblings who were alive at age 50	71.1	70.9

Survival Models

We estimated semi-parametric survival models to test our hypothesized effects (Table 2). For each sex, we included three nested models in order to examine whether the effects of disease load during infancy on later life survival between ages 50 and 80 could be altered when controlling for other environmental and familial

variables. More precisely, variables related to the environmental conditions surrounding birth were introduced first (year of birth, level of infant mortality). Proxies for family specific conditions of childhood (parity, birth order, and parental death) were then included. We finally added other family-specific variables related to adult life and to biological endowment (number of siblings alive at age 50, average age at death among those siblings, and the interaction between those two factors). Our evaluation was based on both the magnitude of change and the significance of the parameter estimates when introducing controls. All Cox models were run in Stata, using the Huber-White sandwich estimator of variance. We evaluated the proportional hazard assumption with graphical and goodness-of-fit testing procedures and found it satisfactory. We also ran parametric models with an underlying Gompertz distribution and a shared frailty parameter among family members using a gamma distributed mean of 1 (not shown here). These models, which capture interfamily variation left aside by our set of controls, produced similar parameter estimates. These parametric models were also run in Stata, using the streg module [Cleves, 2004 #45].

RESULTS

Table 2 shows the hazard ratios and the overall model fit of the Cox regression models for males and females, separately. As the entries for the “year of birth” variable show, mortality between age 50 and 80 was lower in the more recent birth cohorts than among the older ones for both males and females. Model I suggests a reduction of 0.3% in the hazard rates for each additional year in the female birth cohorts. Over 10 years, this represents a 3% reduction; over 80 years (e.g. from 1680 to 1750), the reduction would be about 24%. The year of birth has an even higher impact on male survival prospects, with a 7% reduction in hazard rates for each additional year in the birth cohort. This would represent a reduction of 56% over the whole period covered. The parameters remain quite stable with the adjunction of other controls. This yearly reduction in post-reproductive mortality rates sharply contrasts with the steady increase of infant mortality reported above. There is also no firm evidence regarding the alleged role of the disease load in infancy on later age mortality, for both males and females. Although positive, the hazard ratios are not significant at the 0.1 level. Thus, contrarily to what has been found in other studies (e.g., Bengtsson and== 2001, 2003), an increase in region-specific infant mortality level does not result in a significant increase of mortality in later age.

The coefficients are relatively stable after the adjunction of other controls for males (Model II and III), but the effect size and the significance slightly increase in the case of females. We have tested several categorisations of the infant mortality levels, as well as other variables such as deviations from the trends (or residuals) and none proved significant. Be that as it may, the estimated hazard ratio (which represents the increase in hazard rate passing from infant mortality rates of 0 to 100%) remains very small: 1.532. Using categorical specifications with percentiles, the lowest mortality category (lowest 10^{th} percentile) would have benefited from a reduction of about 7-8% (p=.150). The results could become significant with a larger number of cases. But given the already substantial sample size, the interpretation would remain the same. There could be a significant effect, but it would still have to be considered as very small.

TABLE 2. Cox proportional hazard models for survival between age 50 and 80.

FEMALES

	MODEL I		MODEL II		MODEL III	
	hazard ratio	Wald p-value	hazard ratio	Wald p-value	hazard ratio	Wald p-value
Year of birth	0.997	<0.001	0.997	<0.001	0.997	0.003
Infant mortality	1.472	0.178	1.481	0.172	1.532	0.137
Parity			1.006	0.157	1.013	0.009
Birth order			1.001	0.770	1.001	0.802
Age at mother's death						
>15			Ref		ref	
Before 1			1.198	0.217	1.159	0.314
Between 1 and 15			1.049	0.176	1.019	0.587
Age at father's death						
>15			Ref		ref	
Before 1			1.063	0.585	1.074	0.525
Between 1 and 15			1.069	0.041	1.062	0.064
# sibs alive at age 50					0.978	0.002
Mean age at death of siblings					0.974	<0.001
Interaction #sibs * mean age death					0.994	<0.001
N	8,928		8,928		8,928	
Difference in -2LL (Wald chi-square)	14.9		23.9		203.0	

MALES

	MODEL I		MODEL II		MODEL III	
	hazard ratio	Wald p-value	hazard ratio	Wald p-value	hazard ratio	Wald p-value
Year of birth	0.993	<0.001	0.993	<0.001	0.993	<0.001
Infant mortality	1.207	0.514	1.171	0.585	1.168	0.591
Parity			1.000	0.979	1.013	0.012
Birth order			1.000	0.980	1.002	0.676
Age at mother's death						
>15			Ref		ref	
Before 1			1.204	0.245	1.158	0.361
Between 1 and 15			1.077	0.038	1.031	0.392
Age at father's death						
>15			Ref		ref	
Before 1			1.151	0.219	1.085	0.476
Between 1 and 15			1.016	0.625	0.998	0.944
# sibs alive at age 50					0.966	<0.001
Mean age at death of siblings					0.976	<0.001
Interaction #sibs * mean age death					0.994	<0.001
N	8,660		8,660		8,660	
Difference in -2LL (Wald chi-square	80.2		87.6		262.6	

Models II adds three variables pertaining to the effect of family conditions in early life. Although the coefficients for parental death are, as expected, positive, these coefficients do not reach statistical significance. Birth order does not seem to matter either. On the other hand, parity acquires clear significance in Model III, when the number of siblings alive at age 50 is introduced[1]. This was expected: the presence of many siblings in early life could represent an increase in competition for limited resources. Large parity could also result from high infant mortality within families, with the occurrence of maternal depletion and low birth weights as its correlates[2]. The effect is very small, however, with an increase of 1.3% in the hazard rate for each additional sibling. The number of siblings alive at age 50 has a stronger effect, especially for males with a hazard rate *reduction* of about 4.4% for each additional sibling.

The three variables pertaining to the "sibling effect" produce the highest improvement in model fit (Model III). Not only would it be advantageous to belong to a sibship in which the average age at death is high, but this advantage would even be stronger in larger sibships. For example, two more siblings, and five extra years in the average age at death among the whole sibship would mean a reduction of 23 % (= 2 X .044 + 5 X .034 + 2 X 5 X .006) in the hazard rates. With the year of birth, the coefficients and the significance associated to the sibling variables were the most stable among all the variables that we tested so far. Parental ages at death (not included here, but implicitly captured by the age of the child at the death of a parent) also produce significant and stable coefficients, but with smaller effect sizes. The hazard ratios are around .997 for every additional year in the age at death of one or the other parent.

As noted above, the increase of infant mortality over time is associated with a decrease of adult mortality in the same cohorts, which contradicts our main working hypothesis. According to Preston's typology, a negative association between early and late life mortality could be *direct* or *indirect*. A direct mechanism would be through acquired immunity of later cohorts who would have been exposed to an increased presence of pathogens in early life. Rising infant mortality rates could also indirectly create a negative association though selection mechanisms that would leave larger fractions of robust survivors. These mechanisms seem to have no relevance in the early Quebec population. Along with the negative coefficients (i.e., beneficial effect) for the year of birth, our models produced *positive* estimates (i.e., detrimental effect) for the infant mortality levels in infancy, even though the estimates did not reach significance thresholds. To further confirm the absence of selection, we plotted the age-specific probability of death according to the level of infant mortality at birth (fig. 3)[3]. Although the levels are clearly different during the first years of life, the gap largely disappears in adulthood and the curves fully overlap by age 50. If there would have been selection, those who had the highest infant mortality levels would have lower levels in childhood or shortly after (the curves would cross each others).

The most plausible explanation is the occurrence of *period* improvements of mortality at older ages that would have benefited the more recent cohorts. For example, someone born in 1730 could have benefited from the general improvements in health conditions that were already rising at the end of the 18th

[1] Although there is some association between total parity (children ever born) and the number of siblings alive at age 50, the two variables are far from being perfectly correlated (r=0.5).
[2] However, we have tested several variables pertaining to birth spacing and to the status of the previous child (dead or alive) and none of them proved significant (not shown here).
[3] Only males are considered in this figure; female data show similar features. For example, the lowest infant mortality group comprises children who were the lowest 10th percentile of infant mortality when they were born. Their birth may have take place anytime, anywhere in the colony; only the level of infant mortality matters. Three percentile groups are presented, but many more grouping schemes were tested (not shown here).

century (Dechêne, 1974). To substantiate this hypothesis, we plotted the age specific probability of death for the earlier and later cohorts (fig. 4). This time the curve are clearly separated for most years between age 50 and 80, suggesting the primacy of the passage of time over the infant mortality level at birth to explain variations in post-reproductive mortality. Figures 5 and 6 present the variation over time of cohort and period age-specific probability of death, respectively. The comparison of these two figures confirms the importance of period effects over cohort effects. The decrease of mortality at older ages over time is steeper when considering the actual years (fig. 6) than when considering the year of birth (fig. 5). Note also from Figure 5 that none of the cohorts born during years of important epidemic outbreaks (e.g., 1686-87, 1702-03, 1714) show increases age-specific probabilities of death.

Figure 3. Males' age specific probability of death (5qx) according to the level of infant mortality at birth

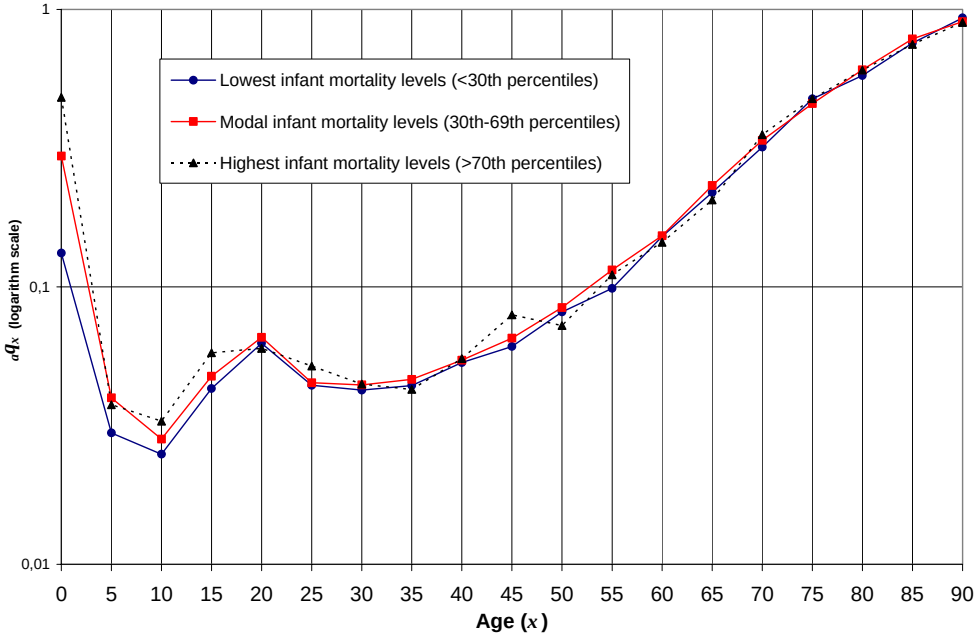


Figure 4. Males' age specific probability of death (5qx) for old and "recent" cohorts

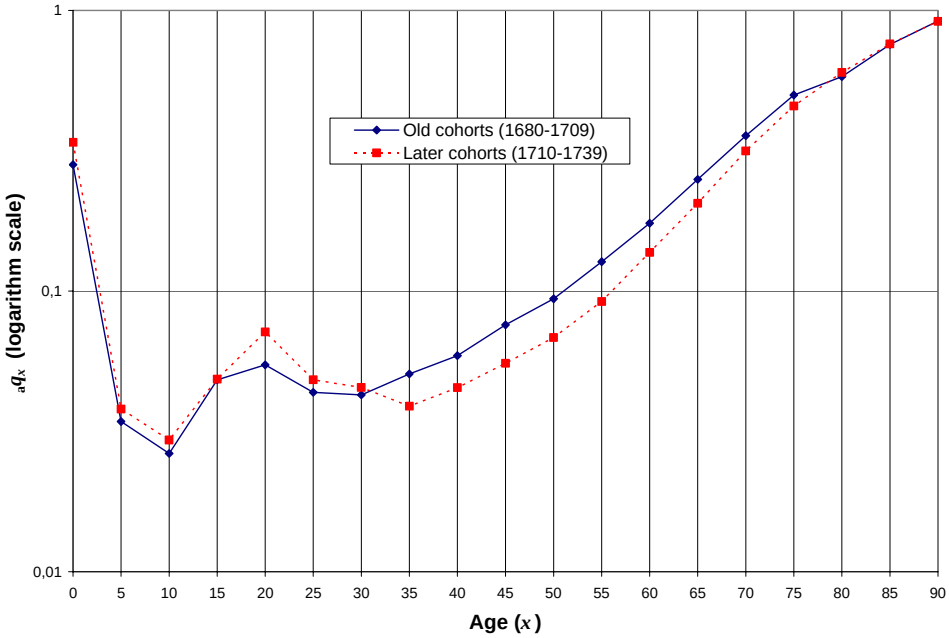


Figure 5. Cohort age specific probabilities of deaths (${}_5q_x$) by year of birth

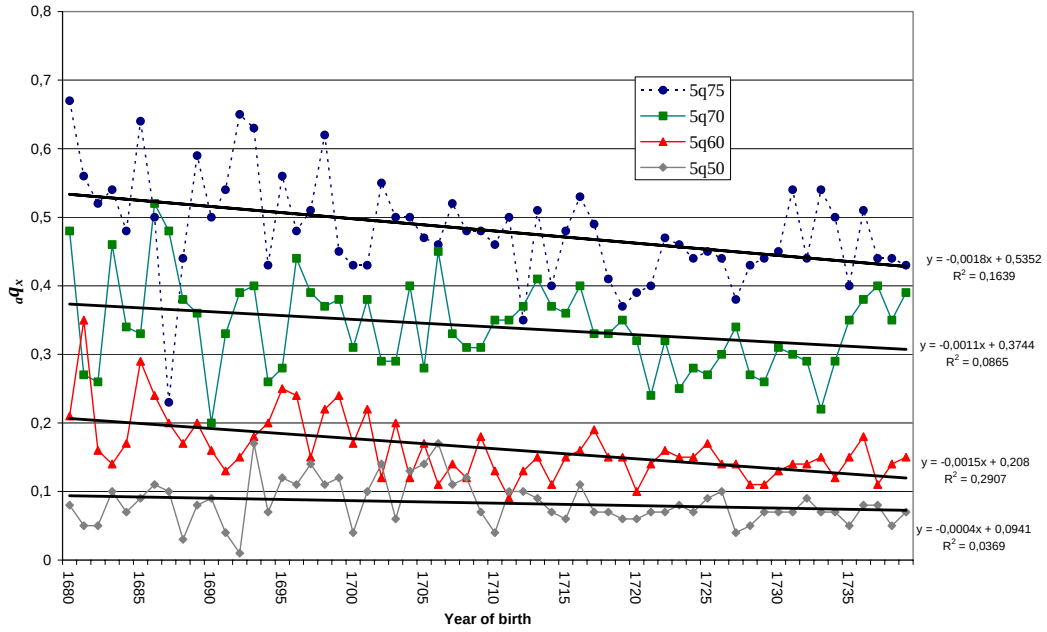
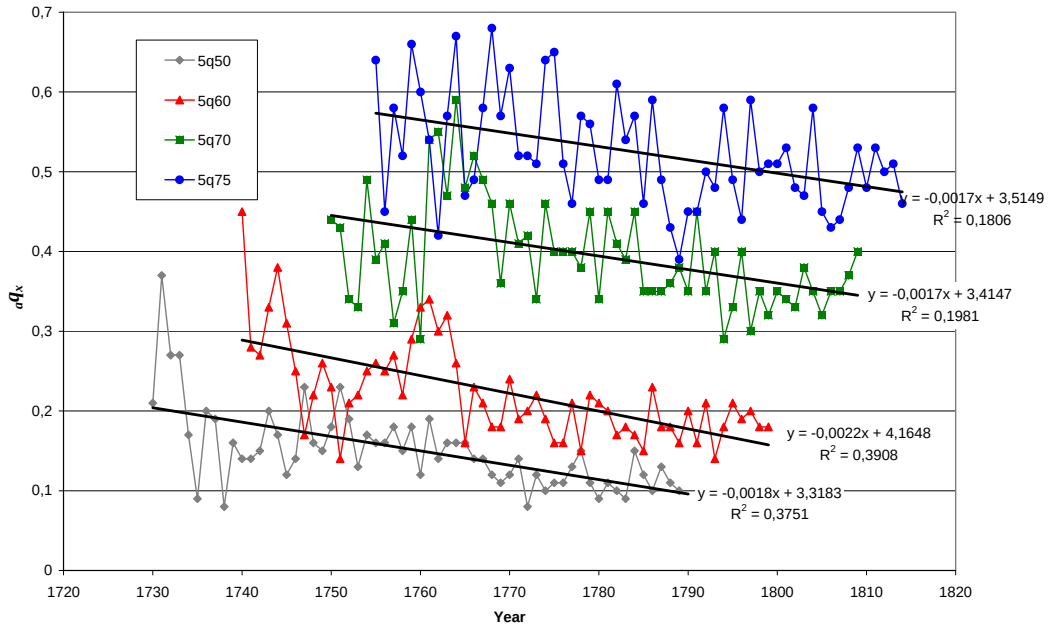


Figure 6. Period age-specific probability of death (${}_5q_x$) at older ages per year



DISCUSSION

Most studies that have addressed the relationship between early life conditions and later life mortality pertain to populations that had already undertaken the demographic and epidemiologic transitions or were about to do so. For example, in the historic sequences presented by Finch and Crimmins (2006; Finch and Crimmins, 2004), none of the cohorts were born prior the year 1751 and most were born in the 19th century. By that time, health conditions have already improved in most European populations (althought there is considerable debate on the timing of the changes). Although the successive decreases in infant mortality in various countries suitably predate the decreases in old age mortality in the same cohorts, the identification of period and cohort effects remains difficult in such a context (Barbi and Vaupel, 2005).

A frontier population such as that of early Quebec allows one to test Finch and Crimmins's hypothesis in a context of increasing infant mortality, prior to the demographic and epidemiologic transitions. A concomitant raise in later life mortality, in such a demographic and socio-sanitary context, would have provided support for the claimed primacy of cohort effects over period effects. However, increasing infant mortality correlates with increasing survival prospect between age 50 and age 80, suggesting instead the occurrence of important period improvements (figs 5 and 6). This counterfactual evidence does not necessarily disprove Finch and Crimmins's findings; perhaps the "morbidity phenotype" of the older cohorts can only be detected when compared with recent cohorts, after a fairly long period of sustained improvement has produced the contrasted low mortality cohorts. However, our study certainly shows how period changes can overshadow cohort effects.

The historical improvements in later life conditions could even have buffered the real, genuine detrimental effects of high disease load in infancy. As a matter of fact, contrarily to what has been found in previous studies (Bengtsson and Lindstrom, 2000; Bengtsson and Lindstrom, 2003), the analysis of the Quebec data provides no firm evidence of a detrimental effect of early life exposure to contagious diseases, even after controlling for the year of birth. The sign of the relationship is, as expected, positive, but the effect sizes are very small. The parameters pertaining to the infant mortality levels in infancy are close to being significant for females, but not males, who benefited from stronger improvements of mortality conditions over time. These stronger period effects could have dampened the manifestation, at older ages, of any detrimental exposure to inflammation in early life. Other times, other realities; studies of post-transitional populations have flooded the scientific communities with accounts of gains in female life expectancy that systematically predate gains in male life expectancy. But males may have benefited first from the earliest improvements in some contexts. Heavily burdened by multiple pregnancies, women would have had to wait for a century or so before taking the lead.

Although not presented, many models were run with different specifications for tracking the disease load in infancy (e.g. infant mortality in the year and the region of birth or over the whole colony, infant mortality in the year preceding birth, etc.) with various categorical specifications of these variables. But no substantial differences in the results, i.e., no consistent tendencies for an association between mortality in infancy and in post-reproductive years, were disclosed. Even the use of "trend" and "cycle" variables did not alter this main conclusion. Only when the cohorts born in the years 1740-1749 were included did some coefficients become significant. But, as written above, including these years introduced a bias because of the incompleteness of record linkage. As we move toward the end of the period of observation, the death certificates of those who died earlier are more likely to be linked to the corresponding birth certificates. Since infant mortality was increasing during the decade, an apparent positive association between earlier death and high

infant mortality during the time of birth surfaces. It is possible that incoming developments of the database (a group of researchers and students is currently working on record linkage of the 1740-50 period) will bring along much larger sample sizes that could provide more power to disclose significant coefficients. The interpretation could however remain the same; we would still have to conclude that if any, the effect is fairly small.

Or wouldn't we? It is possible indeed that the disease load in infancy was not accurately measured in this study. Despite our numerous efforts in that direction (with various sliding windows to capture infant mortality levels, both in time and space), we are still far from being able to declare that a infant born in a given family, in a given parish, in a given time, *was* actually in contact with any of the suspected pathogens, let alone that the infant developed the disease. We are currently developing more accurate methods for tracking the spread of the diseases. For instance, we are following the 1702-03 smallpox outbreak from parish to parish by tabulating mortality rates month by month, flagging children whose date of birth falls in the "outbreak targets". To increase the power of our lens, we give a higher weight when the child lost one family member during the intermission. We believe that such procedure will provide more accurate results in this context of very low density population (with the concomitant sparse and sporadic nature of contageous epidemics).

A further factor that could have altered the results is that of familial conditions. Indeed, family-related variables proved to be the strongest determinants of our response variable, survival between age 50 and 80. The adjunction of these variables did not fundamentally alter the estimation of the parameters pertaining to the year of birth. It did, however, produce a slight increase in the parameter estimates for the infant mortality variable for females. This attests the presence of suppressor effects that could be further removed with stronger control for interfamilial variations. We thus ran several models with parametric (Gompertz) specifications of the hazard rates between age 50 and 80, introducing a frailty parameter (not shown here). Prior the introduction of variables pertaining to sibling's survival, the standard deviation of the frailty random effects were around 0.2 (.19 for males; .20 for females), indicating that typical values of the family-specific risk of mortality were up to 20% larger or smaller than the overall mortality risk, and suggesting an important familial influence in lifespan that was unaccounted by the included covariates. However, the standard deviation of the random effect fell down to a value of .07 when we introduced the "sibling" variables, leaving very few unobserved heterogeneity. All these manipulations did not substantially alter the parameter estimates for the infant mortality levels, suggesting that the options for removing the suppressor effect were exhausted.

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