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Early Life Conditions and Adult Mortality In the St Lawrence Valley, Canada 1700-1850

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1 Introduction and background

What determines human mortality? Are only the current conditions important for our mortality or are the conditions in the beginning of our lives as important as the current conditions? Of course, it is impossible to argue against the fact that current conditions matter for our health and mortality and – ultimately – for our longevity. Current conditions may be even more important than one might think.[1] However, it seems also clear that our mortality partly is determined already when we are infants or even as early as during intra-uterine life, which would implicate that conditions in the uterus and first year or first years of life are vital determinants of morbidity and mortality in adult ages.[2] A number of studies of various modern populations have concluded that improved conditions in early life matters for morbidity and mortality in adult life.[3] It has also been suggested that one should rather take a life-course perspective on health and not be too focused on either current conditions or early life conditions, and that our mortality rather is determined by a combination of past influences and current conditions.[4] In either case, it is hard to dismiss the fact that many studies also of historical populations based on aggregated data has shown that conditions in early life affected mortality,[5] as well as based on individual data; both in adult life and in childhood,[6] so regardless of if using data at the micro level or macro level and of if looking at modern or historical populations, there seem to be substantial empirical proof of that conditions in early life affect mortality in later life.

In this study, we focus on early life conditions and mortality in historical populations. Such studies are interesting from at least two perspectives. One is if looking at it from a modern perspective: detailed data on historical populations can help us to test the validity of the early life hypothesis or 'Barker' hypothesis with empirical data, since data covering a long-enough time period to be able to carry out an empirical investigation of any effects from early life to adult and/or old age mortality is not common. The other is if we look at it from a history-oriented perspective, we still know very little of why mortality differed between different populations before the 20th century.[7] In fact, it is not even entirely clear why there was a general decline in most European and North-American populations during the 18th and 19th century, or why the mortality decline patterns differed between different countries, or sub-populations within countries.[8] At least to some extent, the general decline in mortality can be explained by changes in

[1] Gjonca, Brockmann & Maier (2000), Vaupel, Carey & Christensen (2003:1679-1680).
[2] Barker (1998:5-41, 2001:69-88).
[3] Elo & Preston (1992:204-205), Fogel & Costa (1997:56-60), Marmot (1997:3-8), Barker (1998:5-41, 151-167, 2001:69-88), Wadsworth (1999:44-48), Doblhammer (2004:149-167).
[4] Kuh & Ben-Shlomon (1997).
[5] Kermack, et al (1934:702-703), Preston & van de Walle (1978:290-291), Fridlizius (1989:3, 16-17).
[6] Bengtsson (1997:15-19), Gavrilov & Gavrilova (1999:365-366), Bengtsson & Lindström (2000:273-275, 2001:10), Bengtsson, Broström & Lindström (2002:1-4, 20-24), Alter & Oris (2000), Gavrilova, Gavrilov, Evdokushkina, & Semyonova (2001:2, 10-13), Johansson (2004:207-212).
[7] Fogel (1988:369, 376), Schofield & Reher (1991:7-17).
[8] Fridlizius (1975b:148-151, 1984:107, 1989:6), Perrenoud (1984:41), Fogel (1986:440, 1994b:369, 376), Schofield & Reher (1991:1-2), Chesnais (1992:78-85), Easterlin (1996:7-9, 69-82, 1999: 266-275), Bengtsson (1998:85), Riley (2001:6-31), Johansson (2004; Chapter 2).

early life conditions, which resulted in lower mortality for later-born cohorts.[9] Thus, both from a modern perspective relating to the empirical testing of the early life or 'Barker' hypothesis, and from a historical perspective relating to explaining historical mortality differences and understanding the general mortality decline, the study of early life conditions and mortality in historical populations can help us understand mortality in general and early life condition effects in particular.

Most investigations of the relation between conditions in early life and mortality in later life in historical populations have dealt with populations living in a European environment during the general mortality decline.[10] Hence, it would be interesting to investigate if there were early life condition effects in other historical populations. The populations and environments in the studies above, and the conditions the investigated populations lived in, have often not been very different from each other. They have mostly been European peasant populations in environments where land and food have been scarce, and – as most of Europe's populations – rather poor populations, starving or more or less starving all the time.[11] What about where food and land was not scarce, as in the New World at the other side of the Atlantic?[12] Did early life conditions also affect mortality in the American and Canadian populations at about the same point in time? In this paper, we will investigate if early life conditions affected adult mortality in the population of St Lawrence Valley, Canada in the 17th and 18th centuries.

The St Lawrence Valley area is interesting for several reasons. Relating to what is stated above, it is a population in a non-European environment and it is an area where the population had very different living conditions than in Europe. Land was not scarce, and the area was not over-crowded. The weather was colder than in many European areas, but even though a bit hostile, not overwhelming harsh.[13] The population was also different, since it was an immigrant population. The settlers who formed it were almost exclusively from France; especially North-western France,[14] so even though it is a North-American population, it is a population with European origin. Further, the immigrants who came from France to Canada did not constitute a random sample from the French population, but was rather individuals with quite specific characteristics. Thus, the St Lawrence Valley population was by no means an ordinary population: it is a population of European origin, experiencing very different environmental conditions, regarding pollution, access to land, food supply, crowding, and so on, and who's ancestors was not a random selection of the French population but rather highly selected.

Due to such differences in living conditions, it is interesting to see if early life conditions also are important, as the investigations of European populations have shown. There must have been a strong selection effect among the settlers and the environment was different, giving them

[9] Fridlizius (1989:3, 16-17), Johansson (2004:33-41).
[10] Kermack, et al (1934:702-703), Preston & van de Walle (1978:290-291), Fridlizius (1989:3, 16-17), Alter & Oris (2000), Gavrilov & Gavrilova (1999:365-366), Bengtsson & Lindström (2000:273-275, 2001:10), Bengtsson, Broström & Lindström (2002:1-4, 20-24), Gavrilova, Gavrilov, Evdokushkina, & Semyonova (2001:2, 10-13).
[11] Fogel (1994:371-374).
[12] Fogel, Easterlin
[13] Charbonneau *et al* (2000:99).
[14] Charbonneau *et al* (1993:66-70, 2000:108-109).

better possibilities to feed themselves compared to the European counterpart populations in the same era, which should also mean to have a better resistance against diseases. Further, the effects of lineage and thus genetics might play a strong role in the Canadian case. Investigations of the St Lawrence Valley family lineage shows that a large part of the population in the end of the 18th century was related to a very small number the first immigrant couple settlers.[15] Hence, genetics might be an important determinant of mortality differences in the St Lawrence Valley population.

Investigating any early life condition effects on the population of the St. Lawrence Valley is interesting also for other reasons, as for example an increased understanding of contemporary mortality. Any investigation of mortality in history is important since better knowledge of what caused mortality in historical environments – often very similar to conditions in contemporary developing countries – can increase our understanding on how to improve the health of contemporary populations in developing countries, which of course in itself is an extremely important issue. If conditions in early life are important in historic populations, this should have important implications for understanding but also predicting adult mortality in today's developing countries, and should be useful for the forecasting of future demographic measures, as populations size.[16] Thus, the aim of this study is to investigate if conditions in early life affected mortality in adult ages for the population living in the St Laurence Valley, Canada during the 17th and 18th centuries.

1.1 The importance of early life conditions for later life health and mortality

So then what are conditions in early life and how are those conditions connected to mortality in later life? The basic idea is quite straightforward but is there any scientific evidence for such a connection, and how does this relationship look like? What are the mechanisms such an effect could work through, and is this something only social scientists claim or has the early life idea any validity also in the fields of medicine or other fields?

The main idea concerning early life condition effects on morbidity and mortality is that unfavourable conditions in early life can result in impaired health later in life. A number of early studies of historic populations based on aggregated data have suggested that early life conditions affect mortality later in life,[17] and recent studies of adult mortality based on micro data and using early life conditions can – at least partly – explain some of the individual mortality difference for young adults, adults, and old age adults during the 18th and 19th century Sweden.[18] Also child mortality in Sweden during this period seems to be affected by conditions in early life.[19] Further, there are also numerous recent biomedical and epidemiological individual-based studies of modern populations showing that a number of diseases such as rheumatic heart disease, respiratory tuberculosis, bronchitis, hepatitis B and liver cancer have a well-established link to circumstances

[15] Charbonneau *et al* (1993:161).
[16] Bongaarts & Bulatao (2000; Chapter 1), Johansson (2004:3).
[17] Kermack, McKendrick & McKinlay (1934:702-703), Preston & van de Walle (1978:290-291), Fridlizius (1989:16-17).
[18] Bengtsson (1997:12-19), Bengtsson & Lindström (2000:270-276, 2001:10, 24-27), Bengtsson, Broström & Lindström (2002:1-4, 20-24).
[19] Johansson (2004:206-211)

in early life,[20] but also mental health and learning is affected by early life conditions.[21] Other studies have shown a relation between birth season and adult life span,[22] birth weight and later life blood pressure,[23] general signs of risk factors measured early in life,[24] and birth weight and later life chronic airflow obstruction.[25] Thus, it seems clear that early life conditions matters for later life mortality, but to understand this better we will take a more detailed look on how conditions in early life can affect health and mortality in later life.

1.2 Early life conditions and later life health and mortality – a closer look

There are two prime risk factors during early life, malnutrition and disease,[26] but also other risk factors are also of importance. Five direct explanations of how conditions in early life could affect later life health will be suggested here, along with one more indirect: undernutrition in early life, high disease load and/or disease virulence in early life, poisoning in early life, indoor air pollution during early life, bad sanitation and hygiene in early life, as well as low socio-economic status during early life.[27] Main focus will be on malnutrition and disease since these have been shown to be of major concern during early life.[28]

1.2.1 Early life nutrition

During the foetal stage, the nutritional status of the foetus depends on how the mother can feed her foetus, which includes delivering proper amounts of nutrition and oxygen, and this in turn depends on her size and body composition, her food intake, and her nutrient stores. If the foetus demands more nutrient than the mother can give, the foetus will be undernourished, and this will affect the growth and body composition of the foetus.[29] Undernutrition can have different effects, depending on when this occurs. Many organs and tissues are formed in early gestation, while in later gestation, the cells in the organs and tissues mostly are enlarged and few new cells are produced.[30] Since foetal development is fast, especially in late gestation, organs and tissues may be damaged if cell numbers are reduced, and after birth there is no possibility for the body to produce cells to catch up.[31] In the short run, undernutrition results in catabolism, while in the long-run effect is slower growth rate to ensure the development of vital organs as the brain, which means that other organs as kidney and liver can get underdeveloped: the physiology and metabolism of the foetus is changed. This programming of the foetus may be the source of

[20] Elo & Preston (1992:205-206).
[21] Jones (1997:135, 147-151), Maughan & McCarthy (1997:166-167), Suomi (1997:175-180), Sylva (1997:185).
[22] Gavrilov & Gavrilova (1999:365-366), Gavrilova, Gavrilov, Evdokushkina, & Semyonova (2001:2, 10-13).
[23] Whincup & Cook (1997:121), Cheung, Low, Osmond, Barker & Karlberg (2000:795-796, 799-800), Alexander (2003:457).
[24] Roseboom, Meulen, Ravelli, Osmond, Barker & Bleker (2001:94-95, 97).
[25] Shaheen (1997:61-65).
[26] Wadsworth (1999:45).
[27] Johansson (2004:43-46).
[28] The following section draws on Johansson (2004:43-74).
[29] Barker (1995:171), Barker (2001:69).
[30] Hales (1997:115), Perry (1997:149), Barker (2001:69).
[31] Barker (2001:70-71).

several diseases in later life, for example coronary heart disease and the related disorders stroke, diabetes, and hypertension.[32] Thus, there is a relationship between nutrition in the foetal stage and during infancy, and morbidity and mortality. This nutrition-morbidity/mortality is not related to contemporary health but rather to health in later life.[33] Postpartum, the development of the infant is still dependent on the mother since it is dependent on the quantity and quality of milk the mother gives during lactation, but of course also the food quantity, food quality, and food diversity after breastfeeding has ended.

1.2.2 Early life disease

Regarding the other major risk factor during early life, disease, it is clear that infections during the foetal stage, the prenatal stage, and the postnatal stage can affect the anatomical structure and the development of the immune system, which can lead to disease or increase the risk of disease susceptibility in later life; for example influenza and rubella. Disease in early life depends on both foetal and maternal factors such as nutrition, genetic makeup, foetal development stage, and anatomical factors. On relation is between disease in early life and morbidity later in life is Hepatitis B in childhood and primary liver cancer in adult life.[34] Thus, there is a connection between infection in childhood and disease in later life. Even though the relationships between early infectious disease and later lung diseases are complex and poorly defined, events in critical periods of the prenatal and postnatal development of the lung and immune system may influence susceptibility to later infectious, allergenic, or toxic challenges to the airways.[35] Insults from infectious disease in very early life are shown to have a large effect on chronic disease and disability in later life.[36] Disease in early life might also affect the development of the immune system.[37] This would mean that individuals exposed to a high disease load during early life would have higher morbidity and higher mortality in later life. This has been also been shown in several investigations of Swedish mortality.[38]

1.2.3 Other links from early life conditions to later life mortality

There are also a number of other possible links from early life to adult or old age. There are for example reasons to believe that air pollution in early life is important, and indoor air pollution is more important than outdoor pollution.[39] In the 18th and 19th centuries, wood-fired heating and cooking stoves were used, and studies on contemporary developing countries show that the use of such stoves increases respiratory infections in both infants and children.[40] Also poisoning in early life can affect later life health: during food intake, the body absorbs not only healthy substances. In the 18th and 19th centuries, it was, for example, common to get lead poisoning from jars and pots used in food preparation, and milk used to feed children was often

[32] Barker (1997:96).
[33] Barker (1998:7-9, 13-20, 46-50, 145).
[34] Hall & Peckham (1997:10-11, 17-19, 21-22).
[35] Strachan (1997:113-114).
[36] Fogel (2004:4).
[37] Fridlizius (1989:3, 8), Moore, Cole, Poskitt, Sonko, Whitehead, McGregor & Prentice (1997:434).
[38] Bengtsson & Lindström (2000, 2001), Johansson (2004).
[39] Graham (1994:184), Strachan (1997:113).
[40] Morris, Morgenlander, Coulehan, Gahagen & Morgenlander (1990:105-106).

highly contaminated with bacteria, causing mainly diarrhoea.[41] Inadequate hygiene and sanitation was equally common, which also has been shown to have an early life condition effect on adult mortality.[42] Finally, there is also the effect of socio-economic status in early life, even though this could be argued to be a summary measure for generally unhealthy conditions. Both the main risk factors in early life, malnutrition and infection, are associated with poor social conditions.[43] This might also be the reason why empirical evidence is not clear-cut when it comes to socio-economic status and later life mortality. Some studies support the hypothesis,[44] while in some studies, especially when taking the effects also of adult socio-economic status into consideration, are varying.[45]

The conclusion is that several investigations show that malnutrition and infection in early life, and possibly also interactions between infection and disease in early life, affect later life health. Suggested links are constrained nutrition in early life as well as disease in early life, which – separately or in combination – lead to underdevelopment of organs, changes in body composition, body size, and metabolism. It can also damage to the immune system, thereby raising the susceptibility to infectious diseases. Infection and malnutrition aggravate each other,[46] but nutrition does not affect all infections in the same way.[47] The combined effect of disease and low nutrition makes it harder for the body to absorb nutrition when in fact extra nutrition is needed because of the sickness.[48] Moreover, it is possible that not only one but also two or more mechanisms can be in operation at the same time.[49]

1.3 Empirical measuring of early life conditions and mortality

It is of course clear that measuring the effects in early life mentioned above is not an easy task, so can any effects of early life conditions on adult health be measured, and – if so – how can they be measured? The early empirical investigations of how conditions in the beginning of life affects morbidity and mortality later in life study differences between individuals of different geographical origin,[50] and many of these show a significant correlation between conditions during early life and health in later life through place of origin.[51] However, place of origin is a rather crude measure, and the precision in the estimates when for example looking at correlation between

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[41] Scrimshaw (1985: 336-7).
[42] Preston & van de Walle (1978:288-291), Edvinsson (2001:252-265).
[43] Wadsworth (1999:45).
[44] Alter & Oris (2000), Nyström-Peck (1994).
[45] Lynch, Kaplan, Cohen, Kauhanen, Wilson, Smith & Salonen (1994:524-526), Blane, Hart, Davey Smith, Gillis, Hole & Hawthorne (1996:1434-1437), Wannamethee, Whincup, Sharper & Walker (1996:1259, 1262-1263), Brunner, Shipley, Blane, Davey Smith & Marmot (1999:757, 761), Barker, Forsén, Uutela, Osmond & Eriksson (2001:1-4), Blackwell, Hayward & Crimmins (2001:1269, 1275, 1278)
[46] Chandra (2002:S73).
[47] Journal of Interdisciplinary History (1983:506); Figure 3.
[48] Scrimshaw (1985:332-36).
[49] Johansson (2004:109-110).
[50] Barker (1998); Chapter 1.
[51] For example, Forsdahl (2002: 304-307, reprint; originally published in 1977), Barker & Osmond (1986a, 1986b, 1987), Barker, Osmond, Golding, Kuh & Wadsworth (1989), Osmond, Barker & Slattery (1990), Barker (1998); Chapter 1.

age at death and birth area with respect to if the area was poor or not is not very high. A more useful measure is birth season. Birth season *per se* is of course not important for the health of an individual but it may have an effect on the health of an individual through the level of nutrition and infection, varying with different seasons. If food is scarce and/or of lower quality and/or less diversified during some parts of the year, this would of course affect the nutritional status of all individuals, but especially the most sensitive ones as newborns or the ones yet not born, via the mother. Historically, lack of food and vitamins was common during winter and especially spring, and also the disease load was worse during these seasons and sometimes but sometimes also hot summers can increase the disease load.[52] Season can thus be used as an indicator of conditions in early life: individuals born during a season with a high disease load and/or low nutrition and less diversified food intake would then raise the mortality for these individuals in later life, compared to individuals born during more favourable seasons. Several studies of birth season and later life mortality have shown such effects, and also that the effect is positive. This means that the affected individuals get weaker by the higher disease load and/or low nutrition, not stronger, which supports the early life hypothesis.[53] Some other studies have shown less clear results when it comes to effects from early life conditions to later life mortality measured via season, but this can be caused by the fact that these investigations also use several other measures for early life conditions simultaneously in the regressions.[54] Another measure that could be used in the same way is to use anthropometric measures as final height, and studies have shown that conditions in childhood are important for adult height.[55]

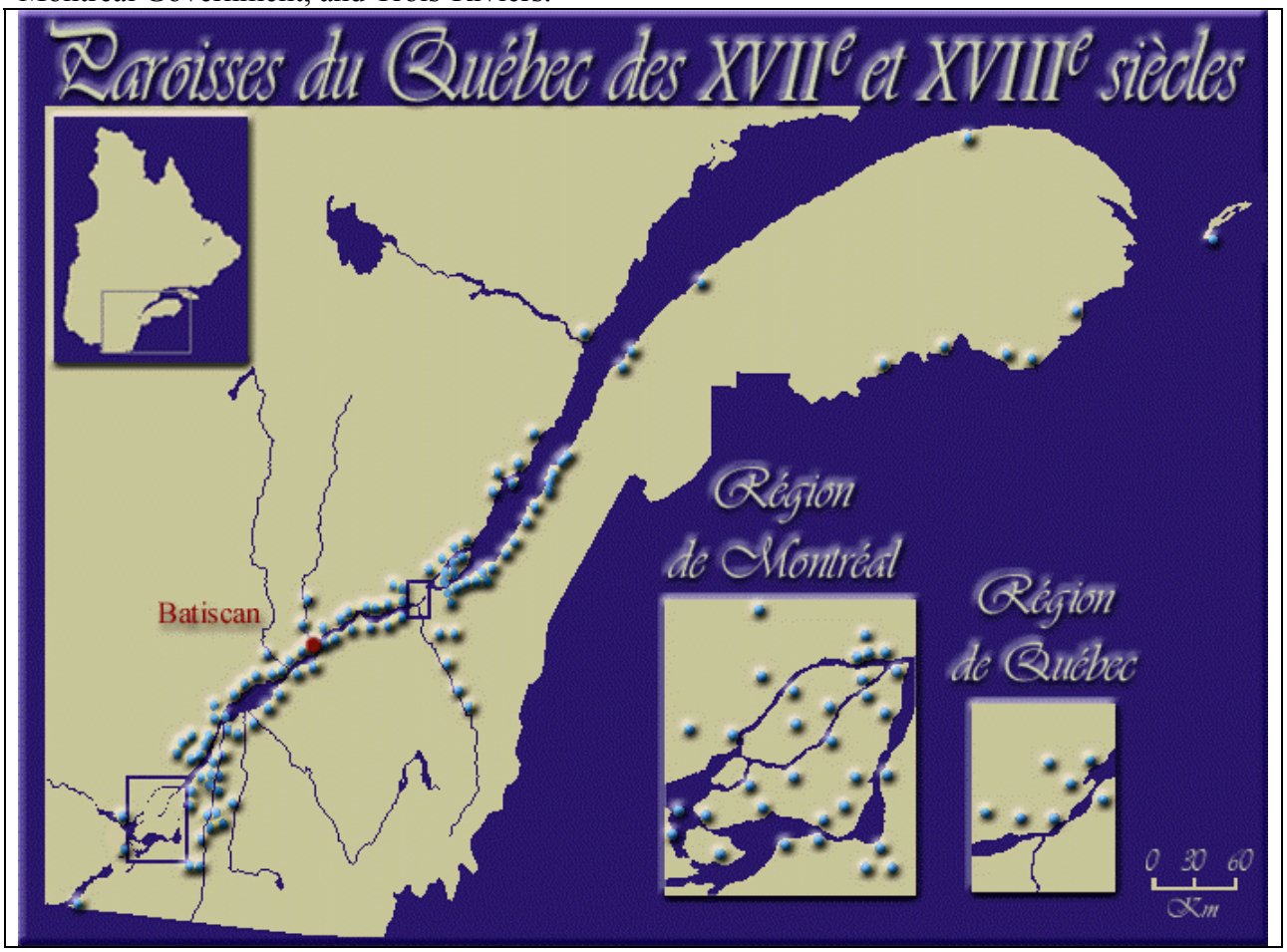
However, both anthropometric measures as height for example and seasonal measures are summary measures and they cannot test any specific link from early life conditions to later life health. To solve this issue, individual's life histories for historical populations have been used for testing the relationship between early life conditions and later life health. To test this link specifically, proxies for disease load and nutrition intake have been used in Cox regressions with community variables in the form of local mortality rates for infants and for adults, and local price of food. Several investigations have shown a link between a high disease exposure in early life and adult mortality in Sweden; both for adults in ages 25 to 55 and for adults in age 55 to 80.[56] Also for children, a study of Sweden 1766-1894 has shown that a high disease load during infancy increased the risk of mortality in age 2-14.[57] Thus, there have been several studies of early life conditions using different approaches to capture the hypothesised link between conditions in early life and mortality in later life, and although using different methods, most of them have shown clear relationship between early life conditions and later life mortality.

[52] Utterström (1954:117-118), Fridlizius (1984:90, 94), Livi-Bacci (1991:75-79).
[53] Doblhammer (1999:4-7), Gavrilov & Gavrilova (1999:365-366), Doblhammer & Vaupel (2001:2938-2939), Evdokushkina, & Semyonova (2001:2, 10-13), Doblhammer (2002:17-20), Gavrilova, Gavrilov, Johansson (2004:194-195)
[54] Bengtsson & Lindström (2001:5, 24), Bengtsson, Broström & Lindström (2002:20, 23).
[55] Nyström-Peck (1994), Alter & Oris (2000).
[56] Bengtsson (1997:16-19), Bengtsson & Lindström (2000:274-275), Bengtsson & Lindström (2001:9-13), Bengtsson, Broström & Lindström (2002:11, 20-23).
[57] Johansson (2004:167-177)

2 The St Lawrence Valley area and population

2.1 Geographical area

The first permanent French settlement in North America was Quebec, founded 1608 in the St Lawrence Valley, and later followed by the settlement of Trois-Rivières in 1634 and of Montreal in 1642. In 1681 there were more than 40 parishes and in 1722 over 80 parishes. The colony was 35000 square meters large and 500 km long.[58] The colony was organised on a North-East to South-West gradient along the shores of the St-Lawrence. It had three administrative regions called 'Governments'. The most North-Eastern was the Quebec Government: Quebec City where ships from Europe docked and then the rural surroundings. At the other end (south-West), the city of Montreal – the centre of fur trading with regions towards the West – is found, and its rural surroundings. Sandwiched in-between, a small town called Trois-Rivieres and its surroundings is found. The large size of the St Lawrence Valley area makes it appropriate to make a distinction between some of the regions. Thus, we have chosen to divide the area into five regions, of which two are city regions, Quebec City and Montreal, and the three rural areas Quebec Government, Montreal Government, and Trois-Riviers.[59]



[58] Charbonneau *et al* (2000:99, 104).
[59] Desjardins

The usual land plot was 150 times 1600 meters, and they were given free of charge, but the tenants had to pay a percentage of their income as fee. Officials from France governed the colony, but since the officials could not live on rental income only, they were – as opposed to their counterparts in the homeland – integrated within the rest of the populations and also involved in business. [60]

2.2 Population

At the time of the settlement, there were only about 60 white inhabitants in the area, while and at the time of the British conquest, the St Lawrence Valley had 70000 inhabitants. This was mostly due to a high fertility combined with low mortality, and even though 1/3 of the immigrants left again, there was an impressive 2.5 % average annual increase in the population. This should be compared to the about 1 % yearly increase in population in the homeland, France, at this point in time.[61] Almost all the immigrants were born in France and foremost came from West of the Bordeaux-Soissons line. Most of them were young single persons, who came alone, especially later on, and immigrant arrival was irregular over the years.[62]

2.2.1 Mortality

Generally, mortality in the colony was relatively low. This is most likely due to a strong selection effect. The women and men who decided to leave their home country – in most cases France – and move to a cold country, far away, must have had both a good physique and mental health. This is what is usually referred to as 'healthy migrant effect'. Also the long voyage at sea must have selected individuals in two stages: first to take the decision to go at all, and then to actually survive the voyage. Malnutrition, disease, crowding, bad hygiene, and poor ventilation was common on the ships, and estimates say that more than 7 to 10 % of the passengers dies during the voyage, and more if there were outbursts of disease.[63] However, this might be exaggerated; some studies of immigration over the Atlantic indicate that mortality during the voyage and afterwards were much lower; maybe about 4 % for adults and twice as much for children.[64] Adult mortality was rather stable over time.[65] Men in the St Lawrence Valley had lower mortality, but looking at age-specific mortality for the two sexes, it is revealed that women experienced fewer deaths before age 60, while after 60 they experienced more deaths.[66]

[60] Charbonneau *et al* (2000:100-101).
[61] Charbonneau *et al* (2000:106).
[62] Charbonneau *et al* (1993:77, 2000:106-111).
[63] Charbonneau *et al* (1993:164-171, 2000:111, 122-125).
[64] Riley (1981), Cohn (1984), Grubb (1987).
[65] Charbonneau *et al* (2000:122-125).
[66] Charbonneau *et al* (1993:164-171).

2.2.2 Fluctuations in mortality

Mortality fluctuated more with time since the population got denser and could sustain epidemics, and some epidemics were especially brutal, as with the smallpox epidemics 1703 and 1733. Before this, there had been several outbreaks of contagious disease, as typhus in 1687-88, which came with the ships arriving in Quebec City. In 1699 there was the only summer epidemic recorded (smallpox) and in 1700 a violent outburst of influenza. After this point in time, the epidemic contagious diseases became endemic, and some years with bad harvests and/or very cold weather had peaks in morality, as the mentioned 1703, 1708, 1711, 1715, and 1728. Some of the years where the particular diseases causing the increased mortality was also 1710 when the yellow fever came with a ship from the West Indies, as well as measles in 1714.[67]

2.2.3 Infant mortality

Looking at the infant mortality rates, it is of course easy to assume that the low mortality in the early period must be due to underregistration of deaths, and this was also assumed for a long time. It is a reasonable assumption, since underregistration generally is not uncommon in early mortality rates for infants. Further the priests were farther from their flock in the first 30 or 40 years of the colony than later and getting around was more difficult. With time, it has been discovered that infant deaths were probably not as under-registered as was thought, and that out-migration of young men is more frequent than first thought; but the period up to 1680 probably had more under-registration than the later periods. Notwithstanding, there is no doubt that the earlier cohorts experienced lower infant mortality because of the selection of their mothers, the healthy living conditions, the low density of population and the absence of epidemics. And with time, Montreal and Quebec increasingly became cities in the traditional sense, with the usual consequences of bad hygiene, higher population density, and thus spread of diseases, which makes an increasing infant mortality very much reasonable. The first epidemic struck the colony in 1703, and there were some bad years for agriculture in the 1730's. Finally, a bourgeoisie developed which used wet-nursing and thus caused extra infant mortality. So the trend in a rise in infant mortality is real, and an expected consequence of this is a higher mortality in the cities.[68]

2.3 Fertility, family, and genetics

Since population of the St Lawrence Valley did not practice any form of birth control, this natural fertility conditions resulted in large families, and the average family had 7 to 8 children – 40 % of the families had at least 10 children.[69] Combined with a low infant mortality, especially in the times of the early settlement, this meant that the families who settled early in the St Lawrence Valley had many children and that most of them also survived to adulthood, and also could have plenty of children. The result is a strong genetic contribution from the first immigrants: the 1500 men and 1100 women who came before 1680 are responsible for two-thirds of the genetic makeup of the French-speaking population of the Quebec today. However, some of the settlers were even more important: one-seventh of the genetic makeup in the same population today

[67] Charbonneau *et al* (1993:175- 179, 2000:106)
[68] Desjardins
[69] Charbonneau *et al* (2000:119).

comes from seventy of the early immigrants. Thus, some pioneers have had a very strong impact on the genetic makeup today. The less importance for some others can of course be due to randomness but also due to socio-economic or biologic features, and when they initiated their fertility. But another component can have been heterogeneity when it comes to infant mortality: the low mortality in the early period worked in favour of the first settlers, and this can be due to selection effects (more robust individuals) as well as healthier environment – the spread of contagious disease is much less in a thinly populated area. But there seems also to be a family component, which means that families with about the same prerequisites could have very different experiences when it comes to infant mortality.[70] Thus, there is what could be called a family effect in infant mortality.[71] Such an effect has also been shown for both infants and children, and in some cases adults, in other historic populations and contemporary populations in developing countries.[72] The conclusion is the St Lawrence Valley population – as in many other aspects – are also special when it comes to their lineage since of their strong genetic bonds to the pioneers, even still today.

[70] Charbonneau *et al* (1993:161, 2000:128-130).
[71] Pavard (2004).
[72] Johansson (2004:116-118); see also later section on theoretical model here.

3 Data

To investigate the impact of early life conditions on adult mortality – say in age 50 to 80 years – at least reliable data on when and where a large number of individuals were born and died, but preferably individual data covering the whole life span of the individuals with information on family relations and individual characteristics is needed. In practice, this would mean individual-bases information, available in digital format for a population covering at least a century. Usually, information with these characteristics is not available until the later part of the 20th century,[73] or further back in time, since there is individual data available for some populations before 1900 that also covers long time periods.[74]

3.1 The PRDH data base

The data in this study comes from the population register created by the Programme de Recherche en Démographique Historique (PRDH) at the University of Montreal.[75] The PRDH data base is based on some 690,000 baptisms, marriages and burials registered in the parishes of the St. Lawrence valley. These records were aggregated to family histories using the family reconstitution technique. Although the data cover in principle the period from 1621 (the beginning of the French settlement) up to 1799 the number of births is quite low before middle of 17th century, which means that for example it is hard to calculate any robust infant mortality rates. More or less the entire population of the region is found in the database and that there was almost no out-migration from the region and if out-migration took place, it is usually known, which makes this area special compared to other areas where this kind of population information also has been gathered.[76] A recent update of the PRDH data base includes mortality information for the period 1800-1850 relating to people born before 1740. It includes 113606 individuals and 45106 unions.

3.2 Sampling frame

3.2.1 Time period

The time period selected for the adult mortality analysis is 1700 to 1850. The birth cohorts used are cohorts born between 1650 and 1740. This is due to that the aim of this study is to adult

[73] It is common that such collections with records of individual data are found for the time period before 1900 as well as after 1960´s or 1970's. However, data bases with data covering the entire 20th century are not found anywhere, but work is in progress in this area – the Research Group in Economic Demography at Department of Economic History, Lund University has started a research project with the aim to cover also this period. It will connect data for a number of parishes in Southern Sweden, where up to now, individual data from 1760-1894 and from 1968 and onwards has been available, but not for the period in-between.

[74] Two examples are the two Swedish historical databases, the SDD and Umeå. The Scanian Demographic Database (Skånes Demografiska Databas) is presented at http://www.ehl.lu.se/database/sdd.htm. Skellefteå is a part of the Demographic Databse (Demografiska Databasen) in Umeå, presented in Sundin (1977) and at http://www.ddb.umu.se.

[75] Charbonneau *et al* (1993, 2000).

[76] Charbonneau *et al* (1993:61-62, 2000:102-104).

mortality between ages 50 and 80, so the first cohort reaches age 50 in 1700.

3.2.2 Data selection

As clear from above, the selected birth cohorts are born between 1650 and 1740, but there are a number of other restrictions used in the data selection. Some are straightforward obvious and not commented, while others are commented below. In the sample are individuals with the following characteristics selected:

- Only those with exact date of birth
- Only those with birth date accurately known and not estimated by age declarations
- Only those who's parental start of marriage is exactly known. This is because it is needed for family identification purposes
- Only live births
- Only those born in Quebec (locality code of 6303 and less): since the early life conditions of in-migrated children are not known, only children actually born in the area are included.
- Those with no information about date of death are excluded. However, right-censoring is permitted but only 284 cases in the entire database are actually right-censored.
- Only individuals who were ever-married. Ever-married individuals have lower mortality than those never-married. Since most of the individuals are ever-married, never-married individuals were not selected to avoid bias from this group of individuals.

4 Model

4.1 Theoretical model

As a theoretical model of adult mortality, a model is used where age at death depends on:

- Sex
- Birth cohort
- Early life conditions
- Genetic endowment
- Family-specific conditions

Sex-differential mortality was and still is common in some populations, and is always important. Birth cohort is important since cohorts born later are assumed to have lower mortality due to technical change (modern populations) and changes in disease environment and/or resistance to disease, as probably accounted for a large part of the general decline in mortality.[77] Early life conditions are – as shown in the first part of this paper – of importance for later life health and mortality, and also the subject of this paper. The genetic endowment also matters for mortality, as well as family-specific characteristics.

4.2 Empirical model

Based on the theoretical model of adult mortality given in previous section, this section will be used to derive an empirical model of adult mortality, ready to be used for analysing the Quebec data.

4.2.1 Sex

The sex variable is straightforward. Men constitute the reference group, so the estimates will be relative to men. The expectations are not clear-cut: generally, women have lower mortality in old age than men, but on the other hand, generally females have higher mortality after age 60 in the St Lawrence Valley population. Thus, these expectations are opposed to each other, so the combined expectation is that it could go either way but reasonably females should have a lower mortality.

4.2.2 Birth cohort

Historically, mortality has decreased over time, so to be born in a cohort later in time – on average – means lower mortality. This decline can be for example due to technical changes or

[77] Fridlizius (1983, 1989), Johansson (2004).

improvements in medicine or hygiene, but during this time, these are not the most reasonable explanations, and in fact, there are no simple explanations for the general decline in mortality during the 18th and 19th centuries. The most reasonable explanation is that it was caused by a decline in natural factors; thus, a decline in disease virulence and/or improved human resistance to diseases.[78] Since a general change in virulence and/or improved resistance is unobservable and not possible to quantify, a negative relationship between mortality and time can be assumed to account for these unobservable changes during the general mortality decline. To include this negative relationship between mortality and time, a cohort factor in form of a time trend will be put into the empirical mortality model. This proxy will represent a general birth cohort effect; hence, an effect of being born at a specific time, shared by everyone born at this point in time. Such a cohort variable has been approximated in the empirical models by a simple time trend in several studies for example of Swedish historical mortality, where the results for different ages and period have varied.[79]

4.2.3 Early life conditions

To capture any relationship between conditions in early life and later life mortality, one or several variables approximating the conditions in early life has to be used. This could be for nutrition in early life and/or disease load in early life. Several studies mentioned earlier have used local price of food and local mortality rates as proxies for early life conditions. Since food price should be less important in a rural setting where anyone who wanted could have land to cultivate, it is less likely that price of food should be related to nutrition intake. Also, most investigations trying such a link has failed to show any connection; probably mostly due to that it is not a good proxy.[80] The same is true for proxies for disease load during the foetal stage.[81] Thus, here, only a proxy for infancy disease load is used, and as a proxy, local infant mortality rates are used. The sample dataset consists of actual life histories for real individuals so data is individual data that can be supplemented with community data. Hence, estimates of local infant mortality rates common to all individuals born in a year are used to investigate if there is a relation between early life conditions and adult mortality.

The St Lawrence Valley area is vast in geographical space, which means – for example – considerable time lags in the spreading of diseases. This makes it is necessary to take geographical information into account when calculating mortality rates. To account for the vast size of this area, regional mortality rates will be used to improve the precision of the mortality rates and therefore the precision of the disease load experienced in early life. Therefore, five different local mortality rates – based on the five regions mentioned earlier in the description of the area – will be used in constructing the infancy disease load variable. Individuals born in one region in a certain year gets the local infant mortality rate value for his or her birth area, while individuals in another gets values from his or her region.

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[78] Johansson (2004:15-41).
[79] Bengtsson (1997:17-19), Bengtsson (2000:143), Bengtsson & Lindström (2000:274, 2001:24-27), Bengtsson & Dribe (2002:25-26) Bengtsson, Broström & Lindström (2002:20-23).
[80] Johansson (2004:166-170).
[81] Johansson (2004:170-176).

The cohort infant mortality rates for the different regions are shown in Figure 3 below. Urban areas have open circles, rural areas filled circles. Quebec areas (City and the rural "Government") are black, Montreal areas are red and Trois Riviere ("rural") is green. First the people settled mainly in what became later Quebec City, then they settled in the rural areas nearby (Quebec Government), and then finally they travelled southwards up the St. Lawrence stream to found Montreal City and again with some time delay they went in sufficient numbers in the countryside around Montreal City (= Montreal Government). And at last the rural area of Trois Riviere (situated actually between Quebec City and Montreal City) reached population size big enough to reach 50 and more births per year. The rural areas (filled circles) had lower infant mortality than urban areas and the three rural areas have very similar level of mortality! Generally, mortality was low in the early periods, but it was then increasing until approximately 1700, from when mortality is still increasing but at a slower rate.

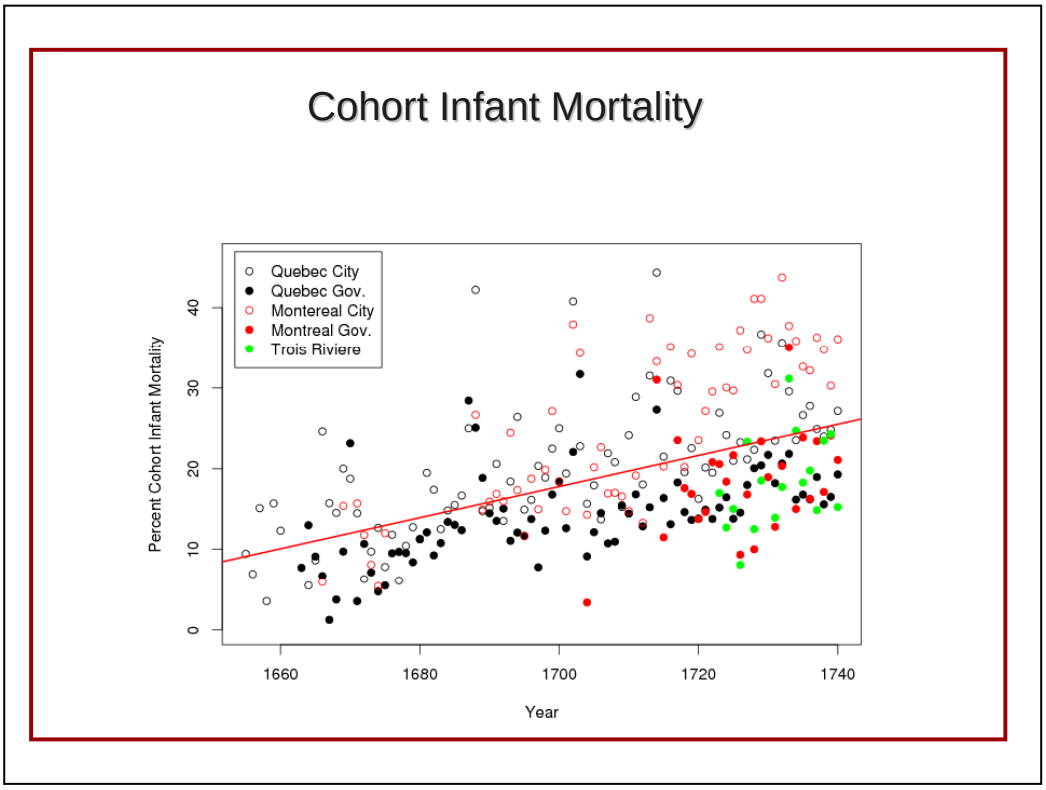


Figure 3

The red horizontal lines show the borders of the categories that are used for the cohort infant mortality rate. These are the quantiles based on yearly mortality rates in all regions, i.e. of all the points you see in the graph. Each of the five areas framed by the red lines comprises 20% of the data points (= infant mortality rates). The lowest category is predominantly present in the early years up to approximately 1680 and the highest category in the later years (from about 1700). The five geographical units differ in their number of cases (Quebec Government contributing by far the most cases, see Figure 7) and in the temporal beginning and development of recorded births.

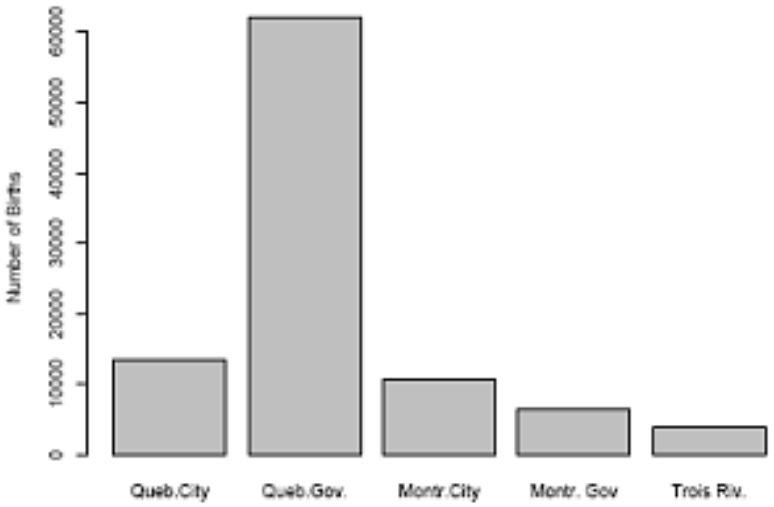


Figure 7

Note, that the different regions start to contribute to the analysis at different times which is the result of the historical development of the population settlement – in terms of founding settlement but also in terms of population growth – a lower bound on at least 50 births have been set to ensure reasonable local infant mortality rate estimates (horizontal line).

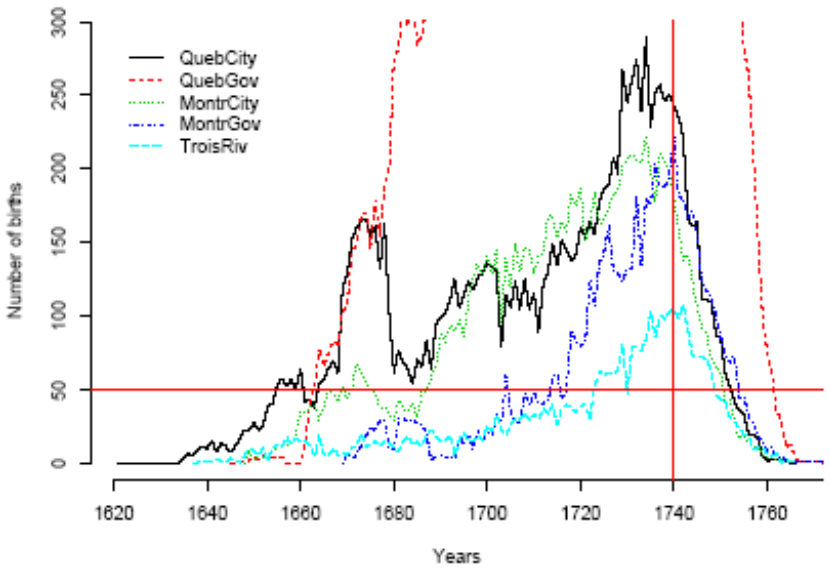
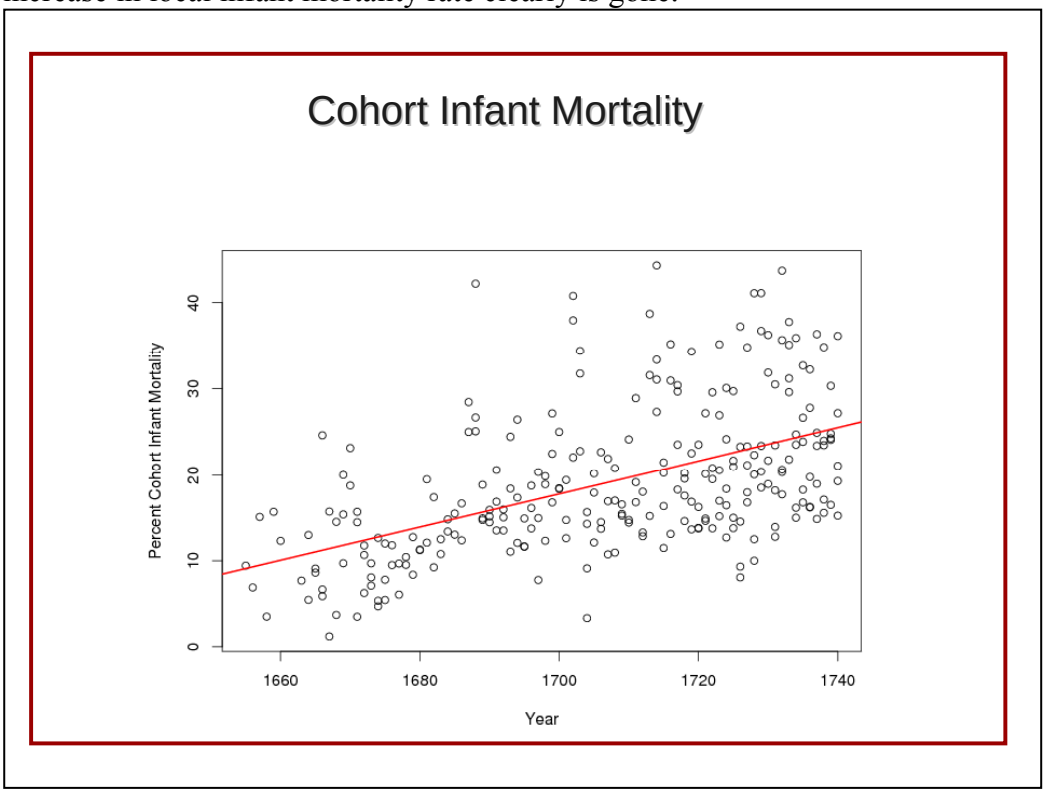
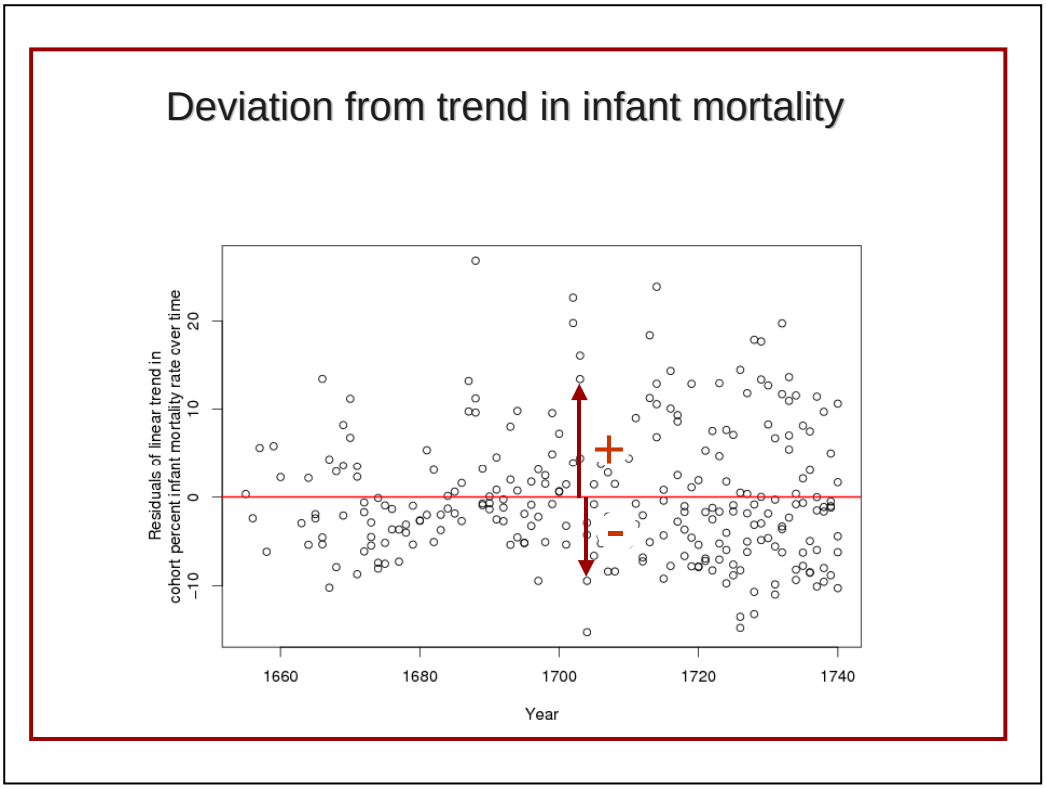


Figure 8

Figure 9 gives the estimates of the local infant mortality rates for the region. Due to small sample, only the local estimates for Quebec city are found in the beginning of the period (see also Figure 8), but after 1660 also Quebec Government and Montreal city reaches more than 50 born per year and makes a contribution, and also the smallest regions contribute in the later part of the period. What is also clear from Figure 8 is that there is an increase in infant mortality (see trend line), and this was not due to a higher mortality in the smaller regions, as mentioned earlier. On the contrary, they had lower mortality than the cities. To avoid that this affects the regression estimates, the short-term deviation from the trend is used. This means that the early life condition effect from disease load is measured as the deviation from what was the common level of disease load in the time when an individual was born. This makes the early life condition proxy comparable over the whole time period, since it means deviation to what was a common level in the times around their birth. The deviation variable is shown in Figure 10, where the constant increase in local infant mortality rate clearly is gone.





The expectations on the infancy disease load variable is that it will have a positive influence on mortality; thus, that a high disease load during early life increases the relative risk of mortality in adult life. This is in accordance with what has been found in several studies before,[82] and also what Barker and others have stipulated: if harsh early events have an impairing effect, we expect higher mortality late in life for the individuals exposed to so such events in early life. This is the opposite of a selection effect, *i e*, if harsh conditions are wiping out the frailer part of a cohort and let only the robust one survive (more or less un-impaired). Then it is possible to expect lower mortality for those individuals exposed to early harsh conditions (and surviving them), but as seen in the first section of this paper, almost all of the studies have shown a positive relationship and rejected a negative relationship.[83] However, a recent study has shown a negative relationship but this study is – contrary to most other studies mentioned in the beginning of this paper – based on aggregated data for the entire Sweden. Thus, the value of this result can be questioned since it is with Swedish aggregated historical data it is not possible to control for anything except for sex. There are also questions to be raised about the how the time series methods are used in the analysis.[84] Furthermore, women and men may be affected differently, or there might be an effect for women and not for men, or vice versa.[85]

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[82] Bengtsson (1997:15-19), Gavrilov & Gavrilova (1999:365-366), Bengtsson & Lindström (2000:273-275, 2001:10), Bengtsson, Broström & Lindström (2002:1-4, 20-24), Alter & Oris (2000), Gavrilova, Gavrilov, Evdokushkina, & Semyonova (2001:2, 10-13), Johansson (2004:207-212).

[83] For example, Bengtsson 1997, Bengtsson & Lindström (2001, 2002), Doblhammer, Doblhammer & Vaupel, Johansson (2004)

[84] Catalano & Bruckner (2006).

[85] Gavrilova, Gavrilov, Evdokushkina, & Semyonova (2001:2, 10-13), Bengtsson & Broström (2006), Catalano & Bruckner (2006).

As is clear from a number of studies mentioned earlier in the paper, birth season can be used as a proxy for conditions in early life. Since food supply was usually less good during winter and spring, and vitamins were scarce during winter and spring, birth season can be used as a proxy for nutrition in early life. But it can also approximate disease load. During the winter and spring, a cold climate made the living conditions characterised by crowding, poor heating, humid air, and bad ventilation in mind, which made increased mortality from respiratory diseases during wintertime was a logical consequence, since people then had to stay indoors because of cold weather.[86] The homes and the clothes of the poor did not give sufficient protection during cold winters, and cramped and unhygienic homes increased mortality. For example, damp houses could increase the number of dead children by 3 times.[87] In the summertime, high temperatures could result in higher mortality risk due to increased contamination of foodstuff and thereby higher risk of infection.[88] Thus, birth season will be used as a proxy for early life conditions; especially early life nutrition. The year is divided into four seasons according to quarters, starting on the 1st of December.[89] The mortality risk of being born during the winter season is expected to be higher than if born during the other seasonal quarters, with the possible exception of spring, which also could be bad in terms of disease load and nutrition. The winter season has been selected to constitute the reference group.

4.2.4 Genetic endowment and family-specific conditions

A number of investigations show rather clear family effects on infant mortality,[90] and other shows clear family effects on child mortality,[91] while other studies only show small or no effects on child mortality.[92] Such contradictory results may be a result of the various methods used in the different studies. The use of family effects in investigations of adult mortality is less common. One study reveals large effects on mortality for different age groups, but also that the family-part of the risk declines with age, and that it is strongest for young adults.[93] Thus, since it has been shown that there is a family effect on mortality in general in several European historical populations, so it is reasonable to assume that it is even more important for the Canadians since of their genetic heritage. As was concluded earlier on, there seem to be a clear effect of biological endowment in the St Lawrence Valley population. Considering that today, one third of the population stems from 60-70 couples that arrived during the 17th century, it is not surprising then that there is a strong genetic component when it comes to health, and that mortality would depend also on genetics. Biological endowments should then also be taken into consideration when analysing mortality, but also effects that have their origin in the family but not are effects that stem from genetics must be taken account for. This is for example family values and preferences experience during childhood.

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[86] Fridlizius (1984:90).
[87] Utterström (1954:117-118), Fridlizius (1984:90, 94),
[88] Livi-Bacci (1991:75-79).
[89] This has been used for example in Johansson (2004), where it was based on the observations by Berg (1879:96-99).
[90] Das Gupta (1990:499-503, 505), Lynch & Greenhouse (1994:127-131), Ronsmans (1995:458-461).
[91] Curtis, Diamond & McDonald (1993:33, 41-42), Zenger (1993:486-487), Rogers (2002:13-15, 17-20), Johansson (2004:187-189).
[92] Guo & Rodriguez (1992:969-970, 974-975), Guo (1993:27-30), Sastry (1997:258-259).
[93] Alter, Broström & Edvinsson (2001:12-14).

Biological endowment is, however, not directly measurable but siblings have similar biological endowments because of their genetic background, which makes them share a part of the mortality risk.[94] Also, the actual family values and preferences of parents are unknown and thus not possible to quantify and explicitly state in an empirical model. Hence, family values and preferences must be approximated in an empirical model, just as family knowledge. Studies have shown that there were differences in knowledge and ability to utilize information, even in times where knowledge on disease, hygiene, and nutrition was generally very low.[95] The family values and preferences, the genetic part, and family knowledge could be called components shared by siblings or just shared components. Since family belonging thus is an important carrier of information, it is important to account for heterogeneity between different families, regardless of whether this may stem from family values and preferences, genetics, or knowledge; even though unobserved. In demography, this is usually referred to as frailty, which means that some individuals are weaker than other individuals and tend to have higher mortality without any apparent or observable reason.[96] These unobserved characteristics result in a different mortality pattern when compared to other individuals with the same observable prerequisites. Here, frailty is considered to be biological endowments shared with siblings, while frailty originally refers to individual frailty. Thus, these unobserved characteristics are supposed to be shared by siblings and are measured as frailty derived from their family. In the model, all the siblings share this family effect, and it is estimated as shared frailty, assuming a Gaussian distribution.

4.3 Statistical tools

With longitudinal data as in the Quebec data set, it is possible to test hypotheses with multivariate statistical survival analysis tools. This allows for simultaneous control of such characteristics as sex and birth cohort; for example within a proportional hazards regression model as the Cox model. Further, if the data set is supplemented with other data at the community level, variables common to a fraction or all of the individuals in a community can be put into a statistical micro-analysis model.[97] This technique has been used in various studies of mortality and general economic demography.[98] For example, the combined micro and macro analysis model is used in several publications from the EurAsia Population Project.[99] It is especially useful in the analysis of early life condition effects on later life mortality, since it makes it possible to attach values for conditions in early life to the data records of the individuals, which can be used as proxies for

[94] Schultz (1984:216-220).
[95] Dribe & Nystedt (2003:25-28).
[96] Vaupel, Manton & Stallard (1979:389-397), Vaupel (1988:277-287), Andersen, Borgan, Gill & Keiding (1997:660-674).
[97] The technique is developed in Bengtsson (1993:239-258) and, for example, used in Bengtsson (1997, 1999, 2000), Bengtsson & Dribe (2002), Bengtsson & Lindström (2000, 2001), Bengtsson, Broström & Lindström (2002), Dribe (2000), and Johansson (2004).
[98] For example, it is used in Bengtsson (1993, 1997, 1999, 2000), Bengtsson & Dribe (2002), Bengtsson & Lindström (2000, 2001), Bengtsson, Broström & Lindström (2002), Dribe (2000), and Johansson (2004). The EurAsia Population Project is described in Bengtsson & Campbell (1998:115-125) and in Bengtsson & Saito (2000:12-13). EurAsia Population Project publications using this approach are, for example, Bengtsson (2000), Alter & Oris (2000a), Campbell & Lee (2000), and Bengtsson, Campbell, Lee, *et al* (2004).
[99] Bengtsson, Campbell, Lee, *et al* (2004).

early life conditions. Thus, using for example the local mortality rate as a proxy for the general disease load during the infancy of an individual, the value for local infant mortality rate during the first year of this individual's life can be used as an ordinary fixed covariate in a survival regression model, as the Cox proportional hazard model.[100] Hence, with this method, it is possible to test if early life conditions have a significant impact on mortality in later life, and at the same time control for other individual characteristics, as sex, birth season, birth cohort, etc. Furthermore, it is possible to extend this combined micro and macro analysis hazard model with a multi level approach, incorporating unobserved characteristics among siblings (shared frailty); thus, a family effect on mortality.[101]

[100] Bengtsson & Lindström (2000, 2001), Johansson (2004).
[101] Johansson (2004:142-144).

5 Results and discussion

The estimates from four versions of the Cox regression for adult mortality in ages 50 to 80 are given in Figure 11 below. The first three are mainly made to check the robustness of the model, and reveals that the model seems robust since adding or subtracting variables does not change the estimates more than very little. Concentrating then on Model 4, it shows that

- Women had lower mortality risk relative to men. It is highly significant, and about 8 % units lower than for men. It is in line with what could be expected, and at least not contrary what was expected here.

- Birth season is as expected: the winter season born individuals has higher relative risk compared to the individuals born in summer or fall. Also the spring-born have higher risk compared to the ones born in summer and fall, and it is actually higher than the winterborn children. However, none of the estimates are significantly different from zero, even at the 5 % level. Hence, it is not possible to show any early life condition effects with the births season variable.

- The birth cohort variable also shows the expected effect, but in this case it is also highly significant from zero. The exponent of the estimated coefficient is smaller than one, which means that for every year later born, an individual has a relatively lower mortality compared to the individuals born in previous cohorts. The effect is not very large but since it is for every year later born, it definitely has an impact. Also, it is not expected to be huge, and it is in line with other studies of adult mortality using a time trend for the general decline in mortality.

- The other early life variable, measuring local disease load, is also in accordance with what was expected. It is significant at a high level, and it is positive. This means that the estimates support the Barker or Fridlizius hypothesis of bad conditions in early life in the form or a high disease load leading to a higher relative risk of dying in adult ages. It is in accordance with what most studies have shown before. Relative to other individuals born in this area, an individual experiencing a relatively high disease load during infancy has an elevated mortality risk in ages 50 to 80. However, even though significant, the effect is small. This could either mean exactly this; there is an effect but it is small, or that there is an effect but we cannot measure it properly by the proxies used for disease load.

- The family effect is significant at extremely high levels, so there should be no doubts that family matters, but this effects is also small. Since it is an estimate for shared frailty, it is the variance of the frailty component and thus not as easy to interpret as a standard regression coefficient. The variance is estimated to be almost 0.11, and this is considered to be a rather small variance, especially considering the strong genetic bonds in the Quebec area. But it might be the case that one should not expect the variance to be high in an area with strong genetic relationships. Rather, it might be that one should expect the opposite, and then the results from the regressions would be very reasonable: a small but highly significant family effect.

Cohort Infant Mortality

n = 25,221	Model 1 exp(coef)	Model 2 exp(coef)	Model 3 exp(coef)	Model 4 exp(coef)
Sex	0.925***	0.925***	0.922***	0.919***
% Infant Mort.	1.004**	1.004**	1.004**	1.004**
Birth Season				
Winter		1	1	1
Spring		1.015	1.016	1.015
Summer		0.994	0.993	0.990
Fall		0.977	0.973	0.967
Birth Cohort			0.997***	0.997***
Family Effect				
σ^2				0.107***

*** p < 0.001, ** p < 0.01, * p < 0.05, + p < 0.1

Figure 11

The conclusion is that basically the estimates confirms the expectations but the birth cohort variable does not seem to be important, the family effect is small, and the local infant mortality rate supposed to catch disease load in early life is significant but very small.

Non-linear effect?

n = 25,221	Linear exp(coef)	Categor. exp(coef)
Sex	0.919***	0.922***
Infant Mort.	1.004**	
0 to 10 perc.		0.933**
10 to 25		0.978
25 to 75		1
75 to 90		0.981
90 to 100		1.042+
Birth Season		
Winter	1	1
Spring	1.015	1.016
Summer	0.990	0.993
Fall	0.967	0.972
Birth Cohort	0.997***	0.997***
Family Effect		
σ^2	0.107***	x

*** p < 0.001, ** p < 0.01, * p < 0.05, + p < 0.1

A possibility is that the effect is non-linear or that there is a threshold for disease load exposure, that once above this value, the effect is strong but below this, the effect is rather small. Figure 12 shows the effect when the local infant mortality rate variable has been categorised so that the individuals experiencing the 10 % lowest disease load in infancy, and the 10-25 %, as well as 75-90, and 90-100 % are grouped relative a relatively broad 'mid-range' disease load constituting of the 25-75 % disease load group. At least two things are clear from Figure 12: the other estimates are more or less the same as in the first variant where infant mortality was used as a continuous variable, so the estimates seems robust, and there seem to be an effect of very favourable conditions in infancy as well as very unfavourable. The most favourable group with least disease load exposure have an almost 7 % lower and significant risk of morality compared to the mid-group. And the individuals who were unfortunate to be born during the least favourable conditions have a highly significant 4 % higher mortality risk in adult ages. Thus, there seem to be a lower threshold of 10 % most favourable and upper threshold of the 10 % least favourable conditions in early life. Also other studies have shown this, but then only for the least favourable group.[102] As in the previous model, the effects are not very large, but not that small either, and the effects are highly significant. Note that this model has no family component (yet).

The last variant is made to check if there is some evidence of different effects on women and on men, as some studies have suggested. The results are given in Table 13. Please note that this model has no family component (yet) either. The estimates show that a stronger significance for the women but the estimated coefficient is larger for the men. Thus, there seem to be a difference between men and women and that men are more affected than women, but the effects are not very far from each other, and it is a very different result compared to the previous mentioned studies considering sex-differentials in early life conditions, since they show that there are only effects for women.

[102] Bengtsson, Broström & Lindström (2002:1-4, 20-24).

Do the man and women differ?

	All exp(coef)	Men exp(coef)	Women exp(coef)
Sex	0.919***		
% Infant Mort.	1.004**	1.005**	1.003+
Birth Season			
Winter	1	1	1
Spring	1.015	1.008	1.027
Summer	0.990	0.998	0.988
Fall	0.967	0.984	0.963
Birth Cohort	0.997***	0.996***	0.998***
Family Effect			
σ^2	0.107***	x	x

*** p < 0.001, ** p < 0.01, * p < 0.05, + p < 0.1