PSC Discussion Papers Series

Volume 21 | Issue 4 Article 1

9-2007

The Measles Epidemic of 1714-1715 in New-France

Ryan Mazan
University of Western Ontario, rmazan@uwo.ca

Alain Gagnon
University of Western Ontario, agagnon4@uwo.ca

Bertrand Desjardins
Université de Montréal

ISSN 1183-7284

The Measles Epidemic of 1714-1715 in New-France

by
Ryan Mazan
Alain Gagnon
Bertrand Desjardins

Discussion Paper no. 07-04

September 2007

On the web in PDF format: http://sociology.uwo.ca/popstudies/dp/dp07-04.pdf

Population Studies Centre
University of Western Ontario
London CANADA N6A 5C2

The Measles Epidemic of 1714-1715 in New-France

Ryan Mazan, Population Studies Centre, Department of Sociology, University of Western Ontario, London, Canada

Alain Gagnon, Population Studies Centre, Department of Sociology, University of Western Ontario, London, Canada

Bertrand Desjardins, Département de démographie, Université de Montréal, Montréal, Canada

Paper presented at the *XIVème COLLOQUE NATIONAL DE DEMOGRAPHIE*

Organized by la *Conférence Universitaire de Démographie et d'Étude des Populations* (CUDEP), Bordeaux, May 21 – 24, 2007

Number of pages (including this one): 14

Number of tables: 5

Number of figures 5

Corresponding author:

Ryan Mazan
Dept. of Sociology, University of Western Ontario
London Ontario, Canada
E-mail: rmazan@uwo.ca

Introduction

Spread along the Saint Lawrence Valley, the first French Canadians had a unique epidemiologic profile for the time. Several conditions protected the colony from becoming a perpetual host population of many pathogens that were endemic in Europe at that time. During much of the 17th century, the easy access to subsistence resources would help retain a strong immune system to protect against the increased mortality caused by such outbreaks (i.e. inhibiting factors). More importantly, the population was sparsely dispersed over a large area making it harder and slower for viruses to penetrate the vast frontier. The low density would impede the formation of endemic viruses because of their need for large host populations to survive. Yet, over time the population went through a stage of rapid growth due to the excess births over deaths (natural increase). The lack of frequent exposure to epidemics made the new generations highly susceptible, as the Canadian born had no acquired immunity to many of the infectious diseases that were prevalent. In addition, the increasing population density would create many contact patterns through which a virus could be transmitted more efficiently. As a result, the prevalence of epidemics increased during the 18th century and probably had a large health, social and economic impact on the population living through that particular time.

Unlike historical Europe, however, mortality crises such as that of excess mortality resulting from epidemics have not been extensively documented or analyzed in the Canadian context. Fortunately, detailed Parish registers containing baptisms and burials are available foe Québec from the outset of the European population. Because of this information, we have the potential to conduct detailed analyses on the periodic outbreaks in pre-transitional Quebec. So far, only a couple of studies have undertaken such a task (Desjardins1996: Dechene and Robert 1993).The former analyzed the spread and impact of the *smallpox* epidemic of 1702-03 while the latter was a study on the impact of the 1832 *cholera* epidemic. These studies are very informative and provide insight into the origin, severity and diffusion (regional variations) of the specified epidemics.. The undocumented epidemics consist of different types of diseases such as *measles*. Measles affects the population structure differently than smallpox and cholera and thus, the structure of a measles epidemic in Historical Quebec remains to be examined through demographic analysis.

The objective of the study is to analyze the general pattern of the suspected measles epidemic of 1714-1715 in historical Quebec and add to the sparse existing body of literature on the subject. Particularly, we focus on the *origin*, *path*, and *duration* and *mortality patterns* of the measles epidemic. During this time, Quebec was a natural fertility population (average of 9.2 children) and had a natural rate of increase of about 2.5% with a doubling time of approximately 30 years (Charbonneau et al. 2000). The increasing population density that took place at the turning of the 18th century, combined with the high levels of fertility and the increasingly frequent crop failures probably made the population highly susceptible to any contagions that would reach the colony. The above enabling factors would help create unsanitary and nutrient deficient conditions (especially in urban centers), which probably led to a general weakening of the inhabitants' immune system and thus, a higher susceptibility of death from disease. These conditions are an ideal breeding ground for an infectious agent such as measles. In sum, this exploratory study serves to identify epidemic mortality conditions by period, age, and region and will help determine the appropriate areas of study in following analyses on the consequences of epidemics at the familial and individual levels.

Data and Methods

The data used in this study originates from the *Registre de population du Québec ancien*, compiled by the *Programme de recherche en démographie historique* (PRDH) at the Université de Montréal (Légaré 1988; Charbonneau et al. 1993). The database contains, for individuals that lived in the Saint-Lawrence Valley in the 17th and 18th centuries, the date and place of birth, death and marriage(s), names of parents and spouse(s) and secondary information on places of residence and of origin. The population remained quasi-closed until the 19th century because of particular historical and geographical circumstances, and thus the usual problem of missing observations because of migration is greatly reduced (Charbonneau et al. 1993; Desjardins 1996). As the development of the database is still in progress, the available information varies in time according to the date of the events and the period of birth and marriage of the individuals. Births are matched with individuals and their parents up to the year 1776, and deaths up to around 1850 (relating to people born before 1750). All the ancestors of every individual who married before 1800 can be traced back to the founders of the population.

Data Quality

The Quebec data is highly reliable and accurate. Records were well organized and duplicates of the vital events were kept (Charbonneau et al. 2000), so relatively few parish records were lost. However, some information may be suspect and may bias estimates. This includes the under registration of infants dying before baptism and other circumstances such as people who died outside of the parish areas (e.g. voyagers) and the neglect of young children . These losses were evaluated at approximately 10% (Desjardins 1996). For under registration, the problem could be greater because of the possibility of administrative disorganization during an epidemic. However, Desjardins (1996) examined the data and found that numbers of those who were not baptized and born during epidemics were no different than the figures during 'normal' years. Thus, it can be concluded that the clergies kept good records at all times.

Regions

We selected only well established parishes at the time of the epidemic. Charbonneau et al. (2000) indicate that records of established parishes were globally more complete than those from only recently established parishes, where the absence of a priest in residence for example caused a greater proportion of under reporting.. Including population counts from those parishes would bias mortality estimates downwards because the denominator would be inflated. To alleviate the problem, we only considered 63 well-established parishes (excluding missions), which also included all of the recorded deaths during the year of the measles epidemic. The individuals inhabiting these parishes would be considered the only ones with a chance of being exposed to the epidemic. Further, we divided the parishes into broad regions based on geographical proximity, the small number of events (deaths) and population sizes of the individual parishes. Quebec City and Montreal are presented as their own separate regions or as the major urban centers. Otherwise, the remaining 61 parishes were divided into 4 broad regions or rural areas to increase the population size and number of events (deaths) so that rates could be estimated (or to reduce the high random variation due to the small number of events). Table 1 shows the division of the 63 parishes into 6 broad regions.

Table 1. The Designated Urban Towns and Rural Regions of Quebec: 1711-1716

Region	Name
1	North of Montreal, South of Montreal & Remainder of Montreal Island
2	Montreal
3	Trois-Rivières, Portneuf & Lotbinière
4	Quebec City
5	Parishes surrounding Quebec City
6	Orléans Island, Beaupré/Charlevoix, Bas-St-Laurent/Beauce

Study Population

The study population consists of the inhabitants residing in the 63 established parishes (6 regions) between 1711 and 1716. Selection of the inhabitants was based on 3 criteria in order to derive an estimate of the population size of the regions. We selected Canadian born individuals and immigrants with a registered date of birth, death and a known parish of death. The Canadian born and immigrants were selected on condition that they died in one of the established parishes either during or some years after the measles epidemic. Essentially, we have to assume that all individuals resided in the parishes where they died (non-transient) even if it was many years after the time of the epidemic. This assumption may be partly unrealistic (causing biased population estimates) because of internal migration, but it is the only way to derive a population estimate of the selected regions. Table 2 shows the population estimates and vital events of the 6 regions between 1711 and 1716.

Table 2. Population Estimates and Vital Events of the Regions: 1711-1716

Region	1711	1712	1713	1714	1715	1716
1	3,426	3,575	3,682	3,733	3,812	3,919
2	2,867	2,971	3,026	3,007	3,036	3,041
3	2,685	2,780	2,842	2,890	2,940	3,018
4	2,372	2,490	2,576	2,545	2,535	2,593
5	2,390	2,484	2,557	2,535	2,541	2,590
6	3,511	3,675	3,762	3,766	3,780	3,903
Total	17,251	17,975	18,445	18,476	18,644	19,064
Not Established	3,979	4,231	4,438	4,653	4,902	5,163
Births	801	855	861	949	837	884
Deaths	539	378	391	918	669	464

Results: The Measles Epidemic of 1714-1715

In the following section, we analyze the origin, spread, duration and mortality patterns of the epidemic by age and regions. We defer from analyzing sex differences due to the small number of vital events when the colony is partitioned into regions. We checked for sex differences and found that there were differences between males and female death rates. As in many other measles epidemics, females at the younger age groups tend to have higher mortality rates (Garenne 1994). Additionally, we only focus on infants and children up to 15 years of age because 79% of the recorded deaths occurred in those age groups. An elevated risk of death was observed among adults, which indicates that measles was not endemic in the population. Sex differences and adult mortality are addressed in a forthcoming study.

Background of the Measles Virus

Measles is one of the most contagious diseases known. It is an acute viral illness caused by a virus in the *paramyxovirus* family. The measles virus is spread by airborne droplets (i.e. nasal or throat secretions produced by coughing or sneezing) through close personal contact with infected persons (WHO). Almost all non-immune children contract measles if exposed to the virus with approximately 99% of susceptibles contracting the disease after first contact with an infective. The development of infectiousness (the latent period) is anywhere from 5-10 days, while the infectious period lasts for about another 7 days thereafter (Murray & Cliff, 1977). Individuals usually do not die directly from the measles virus, but from its complications. The most serious complications include blindness, encephalitis (a brain infection causing inflammation), severe diarrhea (with the potential of dehydration), ear infections and severe respiratory infections such as pneumonia, which is the most common cause of death associated with measles. Individuals who recover from the measles virus are immune for the rest of their lives. Epidemics are therefore self-limiting and due to the depletion of susceptibles, subsequent epidemics can occur (in the absence of migration) only after a new group of susceptibles are born into the population (Giesecke, 2002; Finkenstadt et al., 1998).

Origin, Spread, Duration and Mortality Patterns

The origin of the measles epidemic of 1714-1715 appears different than the majority of outbreaks during the late 17th and early 18th centuries. Most epidemics began in the Eastern areas or around Quebec City. This particular outbreak, however, may have begun in Montreal and its surrounding rural parishes (region 1). Figures 1 through 6 show the quarterly mortality rates from 1711 to 1716 by the six regions. It appears that the epidemic started near the end of the 2nd quarter of 1714 in March or April. The rates in Montréal and the surrounding parishes (regions 1 and 2) were above normal rates in previous years and those of the Central and Eastern Regions. By August (3rd Quarter), the measles virus spread east into Quebec City and the Eastern rural parishes. As can be seen by the sharp peaks, the quarterly mortality rates were much higher in the Eastern part of the colony. These elevated rates are clearly distinguishable by the large peaks among the post-neonatal, toddler, early childhood and adolescent mortality.

Table 3 shows the age specific relative risks of death by region in 1714 and 1715. These ratios consist of an ‘annualized’ quarterly rate compared to a ‘normalized’ rate. The observed quarterly rates are annualized to adjust for differences in the number of the days in each quarter. The normalized rate for each age group was estimated with a cubic spline fit to the quarterly rates between 1710 and 1720. This base serves as a measure of normal mortality conditions and allows for comparisons between regions and for each quarter during the epidemic. Caution must be exercised when interpreting the table due to the small number of events. Small changes in the number of events can produce extremely large risks. Therefore,

the large fluctuations in the ratios could be a combination of random variation and excess mortality from the epidemic. However, the risk table should serve as a complement to the graphs and allow one to gauge an approximate level of risk relative to 'normal' mortality conditions.

The risk table shows that neonatal mortality varied little from the normalized rates. The trend in the line graph shows a more random pattern with the exception of Montreal, Quebec City and the Eastern rural parishes (region 6) during the 3rd and 4th quarters of 1714. The parishes surrounding Quebec City (region 5) had the highest risk at 3.3 times, followed by Quebec City (region 4) and Montreal (region 2) at around 2.8 and 2.7 times the normalized rate, respectively. Most of these deaths probably occurred among infants closer to one month old. Post-neonatal mortality peaked in the 3rd quarter of 1714 in the Eastern area with the rural parishes of region 5 being 7.0 times the normal rate and Quebec City having a 6.0 times higher risk of death. The approximate level of risk in the Western area (regions 1 and 2) ranged from 1.8 times to a high in Montreal at 3.9 times the normalized rate between the 2nd and 4th quarters. By 1715, the level of risk varied little from the normal rate with the exception of the Western region, where the risk was over 2.2 times higher in Montreal and the surrounding parishes during the 3rd quarter. For infants, it appears that the measles virus lingered in the Western area for a longer period, but at a lower rate than in the East (3 quarters in 1714 and 1 quarter in 1715). Conversely, the measles epidemic had a shorter duration in the East but at a more severe rate (2 quarters in 1714 and 1 quarter in 1715). The parishes of Trois-Rivières, Portneuf, and Lotbinière (region 3) were largely spared from the epidemic during infancy and childhood. This trend could be the result of greater isolation from the other regions, lower contact density or better nutrition.

Figure 1. Quarterly Neonatal Mortality Rates by Region: 1711-1716

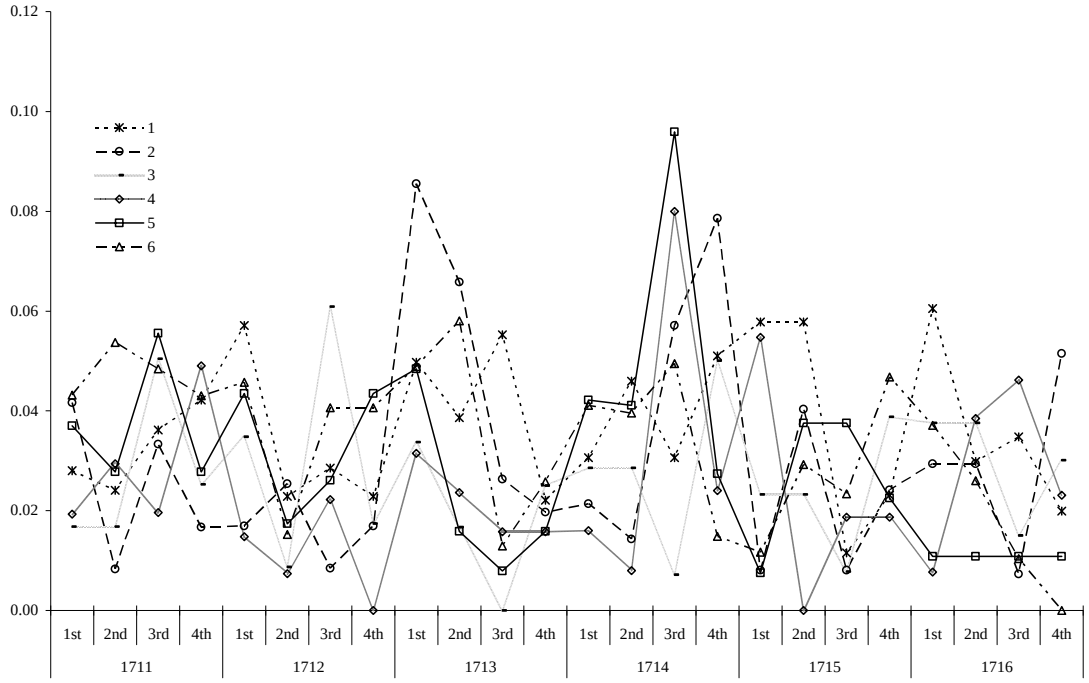


Figure 2. Quarterly Post-Neonatal Mortality Rates by Region: 1711-1716

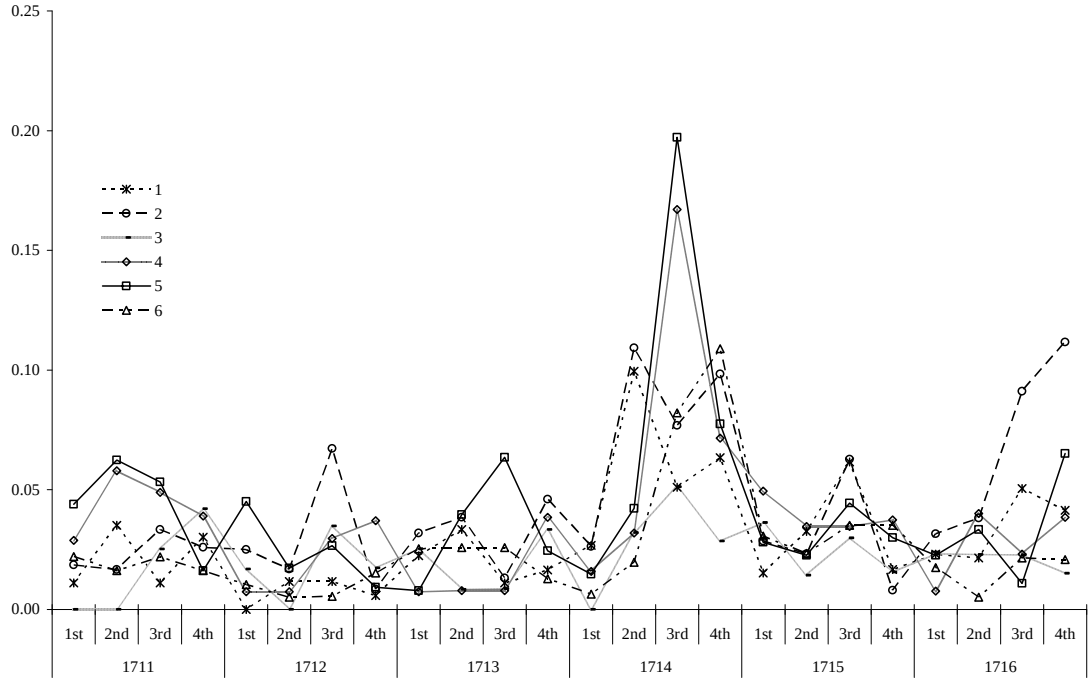


Figure 3. Quarterly 1 Year Old Mortality Rates by Region: 1711-1716

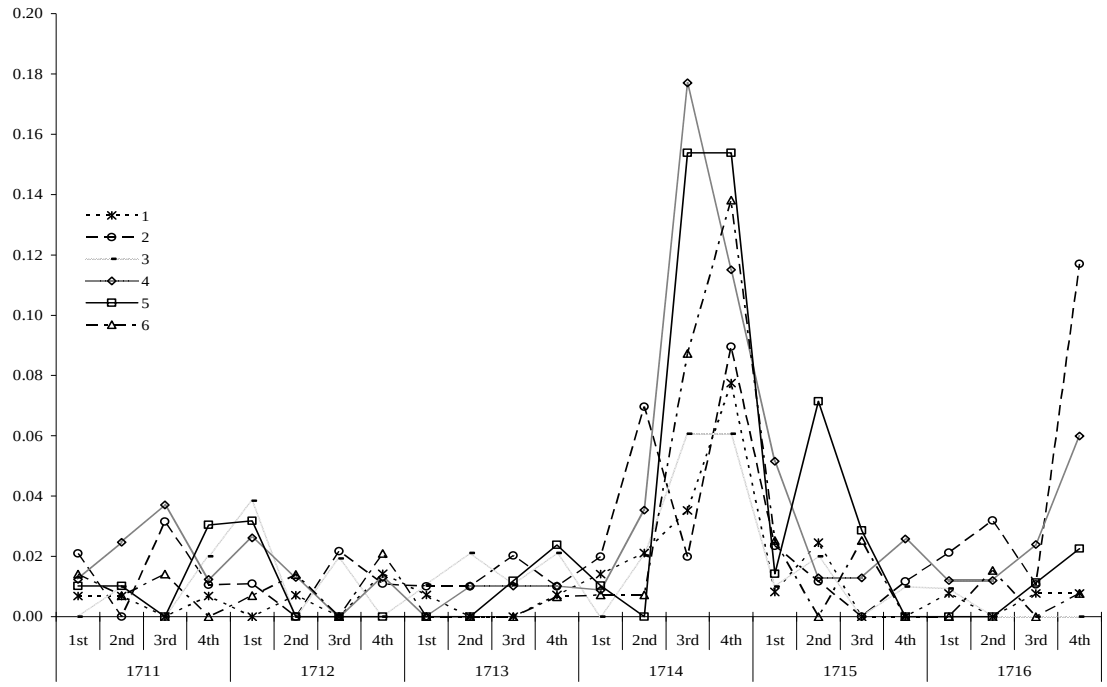


Figure 4. Quarterly 2 to 5 Year Old Mortality Rates by Region: 1711-1716

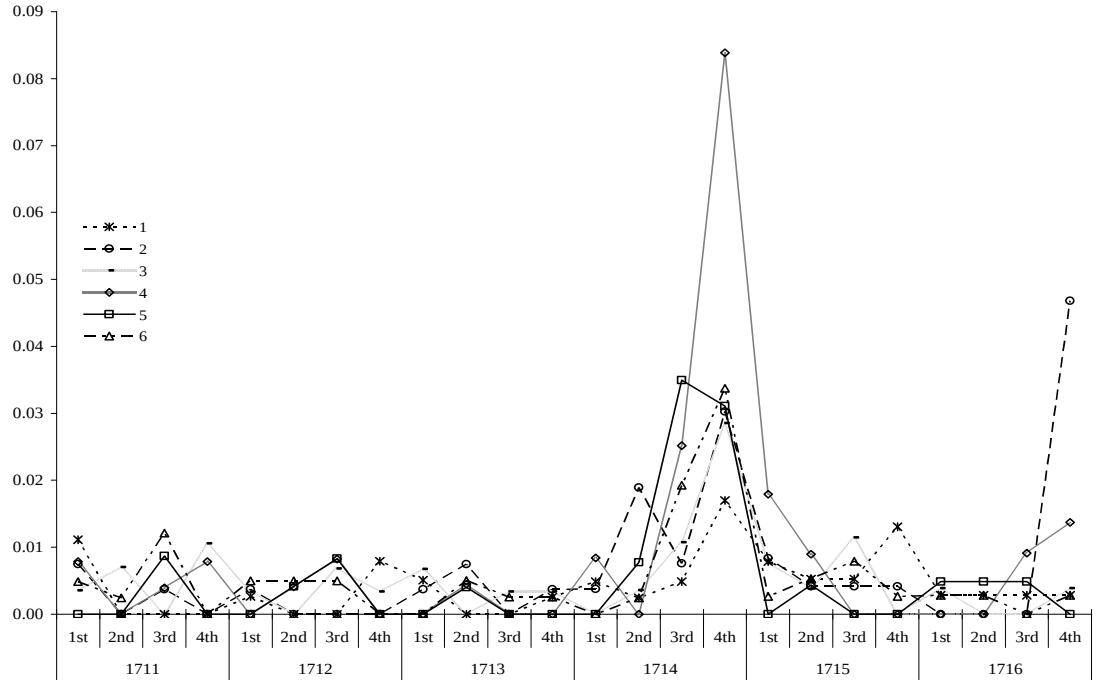
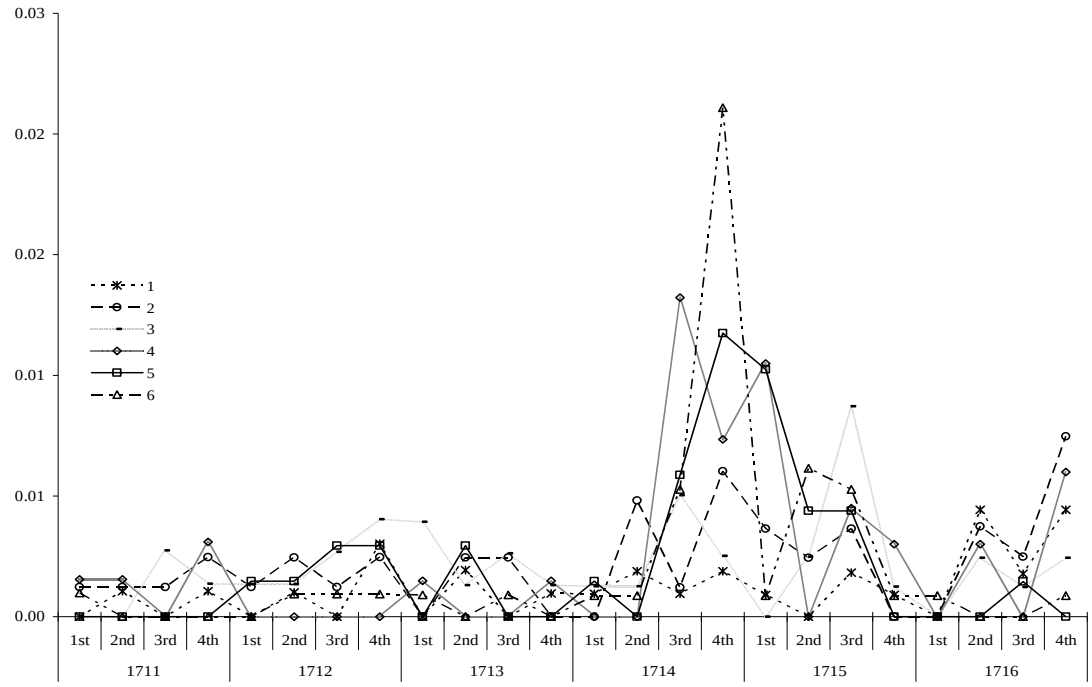


Figure 5. Quarterly 5 to 15 Year Old Mortality Rates by Region: 1711-1716



The elevated risk or peaks in the graphs among toddlers and young children also first appeared in Montreal and its surrounding rural parishes during the 2nd quarter of 1714. However, toddlers, younger and older children in all regions were especially hit hard during the 3rd and 4th quarters, most notably in the Eastern Area; mortality peaked during the 4th quarter among these age groups. During the 3rd quarter, the ratios ranged from a low of 1.7 in Montreal to a high of 15.3 times the normal rate in Quebec City. Likewise, in the 4th quarter the level of risk ranged from a low of 5.2 in the Central parishes (region 3) to a high of 13.3 times in the parishes of region 6. The same mortality pattern occurred among 2 to 5 year olds and 5 to 15 year olds. For children aged 2 to 5 years, the risk level during the 4th quarter ranged from 5.0 in region 1 to a high of 24.4 times the normal rate in Quebec City. As for the 5 to 15 year olds, the ratio ranged from 1.4 to a high of 15.4 times the normalized rate in the rural parishes of region 6. Even though the measles epidemic began in the Western area, the risk of death was consistently higher in the Eastern area during the 3rd and fourth quarters of 1714. Fade-out seems to have occurred in the Eastern area sometime in late autumn or the 4th quarter of 1715. For infants, the epidemic faded out after the 4th quarter of 1714, while it became more intermittent for older children after the 1st quarter of 1715. Thus, the epidemic lasted approximately from the 2nd quarter or the spring of 1714 to the 4th quarter or late autumn of 1715 or with an overall duration of around 20 months.

Table 3. Mortality Ratios Comparing "Annualized" Quarterly Mortality Rates to the Normalized Annual Rate by Age and Region

Age	Year	Quarter	Region					
			1	2	3	4	5	6
0-28 Days	1714	1st	1.1	0.7	1.0	0.6	1.5	1.4
		2nd	1.6	0.5	1.0	0.3	1.4	1.4
		3rd	1.1	2.0	0.2	2.8	3.3	1.7
		4th	1.8	2.7	1.7	0.8	0.9	0.5
	1715	1st	2.0	0.3	0.8	1.9	0.3	0.4
		2nd	2.0	1.4	0.8	0.0	1.3	1.0
		3rd	0.4	0.3	0.3	0.6	1.3	0.8
		4th	0.8	0.8	1.3	0.6	0.8	1.6
28-365 Days	1714	1st	1.0	0.9	0.0	0.6	0.5	0.2
		2nd	3.5	3.9	1.1	1.1	1.5	0.7
		3rd	1.8	2.7	1.8	6.0	7.0	2.9
		4th	2.3	3.5	1.0	2.5	2.8	3.9
	1715	1st	0.5	1.0	1.3	1.8	1.0	1.1
		2nd	1.2	0.8	0.5	1.2	0.8	0.8
		3rd	2.2	2.2	1.1	1.2	1.6	1.3
		4th	0.6	0.3	0.6	1.3	1.1	1.3
1 to 2 Years	1714	1st	1.2	1.8	0.0	0.8	0.9	0.6
		2nd	1.8	6.1	1.8	3.1	0.0	0.6
		3rd	3.0	1.7	5.2	15.3	13.3	7.5
		4th	6.7	7.7	5.2	9.9	13.3	11.9
	1715	1st	0.7	2.1	0.9	4.5	1.3	2.2
		2nd	2.1	1.0	1.7	1.1	6.2	0.0
		3rd	0.0	0.0	0.0	1.1	2.5	2.2
		4th	0.0	1.0	0.9	2.2	0.0	0.0
2 to 5 Years	1714	1st	1.4	1.1	0.0	2.5	0.0	0.0
		2nd	0.7	5.6	1.1	0.0	2.3	0.7
		3rd	1.4	2.2	3.1	7.3	10.2	5.6
		4th	5.0	8.8	8.3	24.4	9.1	9.8
	1715	1st	2.3	2.5	2.3	5.3	0.0	0.8
		2nd	1.5	1.2	1.1	2.6	1.3	1.6
		3rd	1.5	1.2	3.3	0.0	0.0	2.3
		4th	3.8	1.2	0.0	0.0	0.0	0.8
5 to 15 Years	1714	1st	0.7	0.0	0.9	0.0	1.1	0.7
		2nd	1.4	3.6	0.9	0.0	0.0	0.6
		3rd	0.7	0.9	3.7	9.7	4.3	3.9
		4th	1.4	4.4	1.8	5.4	8.6	15.4
	1715	1st	0.7	2.7	0.0	7.8	7.7	0.7
		2nd	0.0	1.8	1.8	0.0	3.2	4.5
		3rd	1.3	2.7	6.4	3.3	3.2	3.9
		4th	0.7	0.0	0.9	2.2	0.0	0.6

To give a more complete depiction of the measles epidemic of 1714-1715, we present the above results in a more summarized form. Table 4 shows the annual age specific mortality rates of each region in 1714 and 1715 and Table 5 shows the relative risks of the age specific mortality rates of each region compared to the normalized rate. The measles epidemic appeared to be most severe among on infants and toddlers. The infant mortality rate was highest in the rural parishes of region 5, Quebec City and Montreal. The IMR was 539 deaths

per 1000 live births in region 5, 482 deaths per 1,000 in Montreal and 414 deaths per 1000 in Quebec City.

Table 4. Age-Specific Mortality Rates (per 1,000) by Region

Year	Age	Region						All
		1	2	3	4	5	6	Regions
	0-28 Days	158	171	114	128	207	145	154
	28-365 Days	241	311	112	286	332	217	250
1714	1-2 Years	148	199	141	336	318	240	230
	2-5 Years	29	60	43	117	74	55	63
	5-15 Years	6	12	10	21	19	28	16
	0-28 Days	150	81	93	92	105	111	105
	28-365 Days	126	123	96	156	125	124	125
1715	1-2 Years	33	47	40	103	114	51	65
	2-5 Years	31	21	23	27	4	19	21
	5-15 Years	4	10	12	18	19	13	13

Table 5. Mortality Ratios Comparing Annual Mortality Rates to the Normalized Rate

Year	Age	Region						All
		1	2	3	4	5	6	Regions
	0-28 Days	1.1	1.4	1.1	1.5	1.7	0.9	1.3
	28-365 Days	2.1	2.7	1.0	2.5	2.9	1.9	2.2
1714	1-2 Years	3.2	4.3	3.1	7.3	6.9	5.2	5.0
	2-5 Years	2.1	4.4	3.1	8.6	5.4	4.1	4.6
	5-15 Years	1.0	2.2	1.9	3.8	3.5	5.2	2.9
	0-28 Days	1.1	0.7	0.9	1.1	0.9	0.7	0.9
	28-365 Days	1.1	1.1	0.8	1.4	1.1	1.1	1.1
1715	1-2 Years	0.7	1.0	0.9	2.2	2.5	1.1	1.4
	2-5 Years	2.3	1.5	1.7	2.0	0.3	1.4	1.5
	5-15 Years	0.7	1.8	2.3	3.3	3.5	2.4	2.3

The risk tables shows that neonatal mortality varied little from the normalized rate, whereas post neonatal ranged from a low of 1.0 (no difference) in the Central parishes to a high of 2.9 times the normalized rate in region 5. Toddlers suffered most in Quebec City and the surrounding parishes of region 5. The mortality rate was 336 deaths per 1000 population in Quebec City and 318 deaths per 1000 in the surrounding parishes of region 5. The relative risk of the two regions was 7.3 and 6.9 times the normalized rate, respectively. The largest increases in risk also occurred among 2 to 5 year olds in Quebec City. For instance, 2 to 5

year olds in Quebec City had an 8.6 times higher risk of death as compared to the normal rate. For 5 to 15 year olds, the risk ranged from a low of 1.0 (no difference) in region 1 to a high of 3.8 times the normal rate in the rural parishes of region 6. By 1715, as the epidemic was fading out, the annual mortality rates also started approaching the level of the normal mortality conditions, with the exception of 5 to 15 year olds. Accordingly, the level of risk deviates less from the normal rate as compared to those of 1714. Overall, the virulence of the epidemic peaked in the 3rd and 4th quarters of 1714, where the Eastern regions of the colony were most severely hit by the epidemic.

Discussion

There are limited historical accounts on the existence of an epidemic between 1714 and 1715. Nevertheless, in the spring of 1714, there was a sudden increase in the level of mortality throughout the colony that would indicate a severe event or crisis. Additionally, we do not have detailed cause of death records for that period, which would enable us to easily identify this as a measles epidemic. However, there are some precipitous events, which help confirm the mortality crisis was probably the result of the measles virus. The most obvious one is that a measles epidemic was reported in Boston, Massachusetts in 1713. The epidemic was also reported New England and by February of 1714 it had spread to New York, New Jersey and Pennsylvania (Duffy 1953). This epidemic probably spread into New France and exposed many susceptible individuals to the virus. It is also a good indication of why the virus originated in the Western area of the colony. Montreal and the surrounding parishes are closer to the political border of the United States (e.g. New York).

Further, there is always a 'triggering event', which precedes any crisis (Palloni, 1990). In this case, the triggering event could have been climatic conditions and poor farming practices. Duncan et al. (1997) found that, low spring and autumn temperatures were directly associated with measles epidemics and mortality. Further, Canada has always been well known for its frequent cold snaps and generally, as a nation with a cold climate. These trends coincide with the seasonal patterns of the measles virus. In the Northern Hemisphere, measles epidemics usually occur and peak during the spring, autumn and early winter months (contact density increases). These are the precise seasons where the mortality rates of most regions were well above the normalized rate. In turn, the above triggering events could lead to poor harvests and those may lead to shortages of food containing essential vitamins. In New France, "poor to disastrous harvests" were reported between 1714 and 1717 (Crowley, 1991). There was no indication on the exact regions affected or whether the entire colony experienced poor harvests. Of course, any crop failure could then have a large impact on the general level of nutrition of the population. Even though, there was probably large-scale food availability in the colony, foods with certain vitamins essential to the development of a healthy immune system among children may have been lacking in all or certain areas of the colony.

Essentially, the poor harvests in Quebec may have led to malnutrition and particularly a widespread Vitamin A deficiency. Generally, Vitamin A helps regulate the immune system, which prevents or fights off infections by producing white blood cells to destroy harmful bacteria or viruses. Infants that are breastfed may incur a higher level of resistance to the measles virus or lower risk of death through their mother because Vitamin A is concentrated in breast milk. However, if the mother is also malnourished, the level of Vitamin A will also be lower in the breast milk. Inadequate nutrition in pregnancy is known to cause low birth weight in infants with a greater susceptibility to infectious diseases (Berman, 1991). This seems not to be the case in Quebec because neonatal mortality did not deviate much from the normalized rate. Vitamin A deficiency leads to lowered resistance and thus an individual will be at a greater risk of death from measles complications. Vitamin A also reduces the risk of

death in infants infected with the virus. In areas of the world where vitamin A deficiency is widespread or where at least the case fatality rate is 1%, the WHO recommends giving high doses of vitamin A supplements to children with the infection. Arguably, improved diet and vitamin A supplementation leads to a marked fall in the mortality of the disease (Barclay et al., 1987; Berman, 1991).

In modern times, the fatality rate from measles for otherwise healthy people in developed countries is low at approximately 1 death per 1,000 cases. Furthermore, reports from practitioners in the 18th century England showed that most healthy children rarely died from measles complications. Rather, fatalities occurred in infants of 'weak constitution', particularly among the working class in the most populous centers (Duncan et al. 1997). On the other hand, complications from measles are more severe in malnourished children, particularly those with vitamin A deficiency. In developing countries, the case-fatality rate may be as high as 30%. Mortality also increases among infants greater than one month old and among children 1 to 5 years old. Young children are especially sensitive to crises triggered by food shortages because they depend more on solid foods and their immune systems are not completely developed. Infants, on the other hand, may incur some protection from breastfeeding. Thus, the combination of food scarcity and infectious diseases has devastating consequences on 1 to 5 year olds (Palloni, 1990).

These are the precise patterns we observe in the Quebec Data. Even though we cannot estimate a precise fatality rate because we don't know who was exactly infected, it appears that the case fatality rate would be around 20 to 30 % during the epidemic of 1714-1715, as well. The above scenario provides strong support that there was widespread malnutrition and Vitamin A deficiency throughout most regions the colony (with the exception of the central region). Overall, the triggering events and the observed mortality pattern during the crisis would suggest the event that took place between the spring of 1714 and the autumn/early winter of 1715, was in fact, the measles virus. As with most measles epidemics in pre-transitional populations, the virus had devastating effects on many families by killing off their infants and young children.

References

Barclay A. J. G., Foster A., Sommer A., 1987, "Vitamin A supplements and mortality related to measles", *B.M.J*, 294, pp. 294-296.

Berman S., 1991, "Epidemiology of acute respiratory infections children in developing countries", *Rev. Infect. Dis.*, 13, pp. S454-S462.

Charbonneau H., Desjardins B., Guillemette A., Lardry Y., Légaré J., Nault F.,1993, *The first French Canadians: Pioneers in the St. Lawrence Valley*. Newark, University of Delaware Press.

Charbonneau H., Desjardins B., Légaré J., Denis H., 2000, "The population of the St. Lawrence Valley, 1608-1760", in *A population history of North America*, Haines M. R., Steckel R .H., (ed.). Cambridge, Cambridge University Press, pp. 99-142.

Crowley T., 1991, "Violence and protest", in *Economy and society during the French regime, to 1759*, Cross M. S., Kealey G. S., (ed.). Toronto, McClelland and Stewart, pp. 120-151.

Dechene L., Robert J-C., 1993, "Le cholera de 1832 dans le Bas-Canada: mesure des inegalites devant la mort", in *The great mortalities: methodological studies of demographic crises in the past*, Charbonneau H., Larose A., (ed.). Belgium, Oridona Editions (IUSSP), pp. 229-256.

Desjardins B., 1996, "Demographic aspects of the 1702-1703 smallpox epidemic in the St-Lawrence valley", *Canadian Studies in Population*, 270, pp. 49-67.

Duffy J., 1953, *Epidemics in colonial America*. Baton Rouge, Louisiana State University Press.

Duncan C. J., Duncan S. R., Scott S., 1997, "The dynamics of measles epidemics", *Theoretical Population Biology*, 52, pp. 155-163.

Finkenstadt B., Keeling M., Grenfell B., 1997, "Patterns of density dependence in measles dynamics", *Proc. R. Soc. Lond.* B, 265, pp. 753-762.

Garenne M., 1994, "Sex differences in measles mortality: a world review", *international Journal of Epidemiology*, 23, pp. 632-642.

Giesecke J., 2002, *Modern infectious disease epidemiology*. London, Arnold.

Légaré J., 1988, "A population register for Canada under the French regime: Context, scope, content, and applications", *Canadian Studies in Population*, 15, pp. 1-16.

Murray G. D., Cliff A. D., 1977, "A stochastic model for measles epidemics in a multi-region setting", *Transactions of the Institute of British Geographers*, 2, pp. 158-174.

Palloni A., 1990, "Assessing the levels and impact of mortality in crisis situations", in *Measurement and analysis of mortality: new approaches*, Vallin J., D'Souza S., Palloni A., (ed.). Oxford, Clarendon Press, pp. 195-228.