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The Demographic Links Between the 1890 and 1918 Influenza Pandemics in Ontario

Stacey A. Hallman, *The University of Western Ontario*

Supervisor: Dr. William Avison, *The University of Western Ontario*

Joint Supervisor: Dr. Alain Gagnon, *The University of Western Ontario*

A thesis submitted in partial fulfillment of the requirements for the Doctor of Philosophy degree in Sociology

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THE DEMOGRAPHIC LINKS BETWEEN
THE 1890 AND 1918 INFLUENZA PANDEMICS IN ONTARIO

(Thesis format: Monograph)

by

Stacey Alanna Hallman, M.A.

Graduate Program in Sociology

A thesis submitted in partial fulfillment
of the requirements for the degree of
Doctor of Philosophy

The School of Graduate and Postdoctoral Studies
The University of Western Ontario
London, Ontario, Canada

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Abstract

Previous research has shown larger than expected numbers of deaths at the age of 28 during the 1918 Spanish influenza pandemic in Canada. To analyze whether this was related to the Russian influenza pandemic that occurred 28 years previously in 1890, the Western, McMaster, Montreal Influenza Pandemic (WMMIP) database was created. It utilizes the death records of 3,316 individuals who died in Ontario between the ages of 23 and 35 from September to December, 1918, and who were also born in Ontario. These were linked to birth records, the 1901 and 1911 Canadian censuses, marriage records, and attestation papers.

A reconstructed date of birth was created for each individual to analyze date of potential exposure in 1890. Those who were *in utero* in 1890 died in greater numbers than would be expected and those in the first trimester of gestation had an unusual sex-ratio at death. Of the various hypotheses proposed to account for the high young adult mortality, these data most closely support that of antigenic imprinting. There is cautious support for the fetal growth restrictions hypothesis, but these data do not support the scarring mechanism. Further, these data do not support the hypothesized relationship between tuberculosis infection and influenza mortality.

More individuals left agricultural homes of origin among the decedents than among the Ontario population in general. There were proportionally more French Canadians, more catholic individuals, and more people from Eastern Ontario. The decedents also came from larger families than were found in the general population, although this may be an artifact of the records linkage process. This research shows that the mortality pattern in Ontario during the pandemic was similar to what it was prior to the epidemic: mortality continued along the fault lines in society and did not equalize risk in a “democratic” manner.

The extant records are appropriate for historical demographic analyses and the strengths and weaknesses for each are detailed. As expected, individuals from the north, who were

aboriginal, from smaller families, or in transient occupations were harder to link.

Keywords:

Influenza, Pandemic, Epidemic, 1918, 1890, Record Linkage, Historical Demography

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Epigraph

Ne pereat population Scientia absente
(Let not the people perish for a lack of knowledge)

Motto of the Ontario Provincial Board of Health
Chief Officer of Health, John W. S. McCullough (1920)

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Chapter 1

1 Introduction

The influenza pandemic of 1918 has been dubbed “America’s Forgotten Pandemic” (Crosby 1989) since one of its many mysteries was how it eluded the public memory for so long. The height of the second wave of the pandemic (October, 1918) came at the end of four years of global war, with armistice and general celebration occurring on November 11th, 1918, and people were anxious to move on with their lives. As described by Middleton and Landon in 1927, nine years later, individuals were still hesitant to talk about the war, since “the miasma of slaughter is still in the air and folk are anxious to turn their minds away from the grief and horror of it all” (1927:757). They predicted that “twenty years hence the Great War will be a topic of universal and fervent interest” (1927:757). It took more than three times that long, but the once forgotten 1918 influenza pandemic has been a topic of considerable study in recent years. Mortality rates, social and environmental risks, the virus itself, and the effect on later life mortality have all been analyzed for the 1918 flu, and the 1890 pandemic is now beginning to receive similar treatment (for example, Smith 1995, Valleron et al. 2010, Herring and Carraher 2011, Gagnon et al. 2013, Hallman and Gagnon 2014).

The unexpected excess mortality among young adults in 1918 has been commented on by physicians and academics since the time of the pandemic itself. However, often what ages constitute “young adults” is not critically analyzed. Through examination of Canadian death records, a distinct peak at age 28 has been noted (Gagnon et al. 2013, Hallman and Gagnon 2014) which requires explanation. Curiously, there was another influenza pandemic that swept the globe 28 years prior to the 1918 pandemic. This raises the questions: Could there be a relationship between the 1890 influenza pandemic and the 1918 influenza pandemic? If so, what is the nature of this relationship? Did prior experience, either socially, physiologically, or immunologically, influence who in particular died from the 1918 pandemic?

Answering these questions is difficult with existing data sources. It requires detailed knowledge of individuals' lives, including the date of birth, and also socioeconomic conditions throughout the lifecourse. This prompted the creation of a unique data source, the Western, McMaster, Montreal Influenza Pandemic (WMMIP) database. Through collaboration and with assistance from individuals at McMaster University and the Université de Montréal, this dissertation describes a records linkage project that involved the location and transcription of birth records, census records, marriage records, and attestation papers for a group of 3,316 individuals who died in Ontario in the influenza pandemic of 1918 (Chapters 3 and 4). From this newly compiled data, it becomes possible to address the question of the linkages between earlier life experiences and mortality in 1918.

This research is a historical demographic analysis that draws heavily from the tradition of the Annales School of History (Burguière 2009). It calls for an interdisciplinary approach to the study of history, whereby “the former reliance on scholarly authority and textual criticism of sources is to be replaced with the search for vast new data bases amenable to statistical treatment” (Bintliff 1991:5). The Annales school advocates for a problem-based approach to history, rather than the previous great-man or chronological approaches, and for a ‘total’ approach to this problem (Crowley 1988, Bintliff 1991, Burguière 2009). This method gives an “injunction to explore one’s chosen microcosm from as many perspectives and through as many different sources as possible” (Tackett 2009:ix).

The ‘problem’ that is focused on in this research is the unusual young adult mortality during the 1918 influenza pandemic. As addressed in Chapter 2, this pattern of death was unique to the 1918 pandemic and the hypotheses surrounding it have yet to be definitively accepted by researchers. These hypotheses tend to focus on one specific aspect or element that could have led to the elevated young adult mortality. Noymer and Garenne (2000) and Noymer (2009) examine the biomedical implications of co-morbidity with influenza and tuberculosis as a direct cause. A biomedical causation is also posited by the overactive immune response and cytokine storm argument (Crosby 1989, Morens and Fauci 2007), the previous exposure causing immunity hypothesis (Taubenberger and

Morens 2006, Morens and Fauci 2007), and has been explained using the antigenic sin or antigenic imprinting hypotheses of Francis (1953) (Gagnon et al. 2013, Hallman and Gagnon 2014). Those researchers who believe that the high young adult mortality was the result of young men congregating in army camps and trenches of the First World War emphasize the unprecedented social upheaval of the Great War (Oxford et al. 2002, Barry 2004, Humphries 2005). It is unlikely that there is one specific answer to the question of young adult mortality and the ‘total history’ approach considers this problem from many possible lenses. Using historical demographic techniques, this research tests the biological hypotheses of previous exposure as well as tuberculosis comorbidity in 1918 (Chapter 5). It also investigates the mechanisms of socioeconomic difference at over the lifecourse, as a means by which the biological susceptibilities may have been mitigated or exacerbated (Chapter 6). Although unable to identify a unique cause of the increased mortality of young adults, this research analyzes the relationship between exposure in 1890 and mortality in 1918, describes the socioeconomic background of those who died in Ontario in 1918, as well as suggests which mechanism was more likely based on historical circumstance and the unique characteristics of individuals who died (such as place of birth, date of birth, sex, and place of death). The new WMMIP database provides hitherto unknown information about those young adults who were born in Ontario and who died in the province during the pandemic of 1918. Added to previous research, this provides a more complete ‘total’ picture of the experience of Ontarians during the Spanish influenza pandemic.

As explained by Herring and Sattenspiel (2003), analysis of historical epidemics tends to rely mainly on aggregate statistics, reflecting “the difficulty and time-consuming process of data gathering and record linkage that is necessary to develop a detailed micro-social picture of the effects on specific families of the H1N1 virus” (2003:171). Although this research focuses on individual-specific history and not the family, this detailed micro-social picture of a select group of the Ontario population gives a broader picture than can be gained through analysis of mortality statistics alone. According to Gadfield, “quantitative methods are only a means of generating usable evidence; they do not in themselves offer reasons why” (1988:163). It is those reasons why that most concerns this research. William David McPherson, the Registrar-General of Ontario in 1918,

reported that there were 3.45 deaths from influenza per every 1,000 of the estimated population in 1918, not including deaths from pneumonia (Legislative Assembly of Ontario 1919a:5). Aside from allowing comparisons with other regions in the world, this number tells us relatively little about the experience during the pandemic. Looking at the historical background of those who died can help to answer new and interesting questions. Exploring the answers to those questions using a new source of data provides the opportunity to disaggregate mortality statistics and, if historical circumstances are similar enough, allows a means to assess those who are at greatest risk in modern-day epidemics and suggest ways to protect the most vulnerable.

Because all previous hypotheses to explain young adult mortality in 1918 are for various reasons inconclusive, this research will explore their suitability and possible syntheses using a new micro-social database in an Ontario context. Thus, this research seeks to address four specific research questions:

1. *Through a historical demographic lens, are the extant historical records in Ontario suitable for demographic analyses of past infectious disease? (Chapter 4)*
2. *Were all ages among young adults in Ontario at equal risk of death from the 1918 flu pandemic? (Chapter 5)*
3. *Since young adults have already been shown to be at an unusually high risk of death, could this be due to: (Chapter 5)*
 - a. *Previous exposure to influenza, resulting in physiological impairments or immunological conditioning?*
 - b. *Co-morbidity with tuberculosis?*
4. *Among young adults, were there unequal mortality experiences resulting from socio-cultural or demographic differences among individuals or groups? (Chapter 6)*

Chapter 2

2 Background and Literature Review

This dissertation explores extant Canadian vital and census records from the late nineteenth and early twentieth centuries. It questions their validity and reliability foremost, but also examines how they can be used to answer questions about the relationship between the 1890 and 1918 influenza pandemics in Canada as well as about the influence of socioeconomic circumstances throughout the lifecourse on mortality in 1918. This chapter provides the necessary historical background in order to answer the research questions as well as an overview of current theoretical hypotheses. It begins by describing Ontario in the late nineteenth and early twentieth centuries, the time period in which two of the most severe influenza epidemics in recent memory occurred. The 1890 and 1918 influenza pandemics are detailed in both their medical and social contexts, focusing on their global and local impacts. This is followed by an overview of the debate surrounding the relationship between poverty and illness at the turn of the twentieth century, focusing specifically on the role of socioeconomic circumstances and risk of death during influenza pandemics and the socioeconomic gradient in health and mortality.

2.1 Ontario, 1881-1921

The 40-year span from 1881-1921 was chosen as the study period for numerous reasons. It encapsulates both the Russian influenza pandemic of 1890 and the Spanish influenza pandemic of 1918. Further, the young adult subjects of this study, who were between 23 and 35 years of age at the time of their death, were born between the years of 1883 and 1895. As Canada's decennial censuses are taken in the first year of each decade, the censuses of 1881, 1891, 1901, 1911, and 1921 give consistent reference points throughout this period. This section provides a general overview of Ontario from 1881-

1921, including demographics, the social environment, the First World War, and the state of public health and the understanding of disease.

2.1.1 *Demographics*

Ontario has been the largest province by population in Canada since confederation in 1867 (McVey and Kalbach 1995, Baskerville 2005). As a destination for immigration, the population of Ontario was 1.93 million in 1881 and grew to 2.11 million by 1891, then 2.18 million in 1901, 2.53 million in 1911, and finally 2.93 million by 1921 (Dominion of Canada 1925). The capital city Toronto was the largest city in Ontario and the second largest city in Canada during this time period (it surpassed Montréal in size only in the latter-half of the twentieth century [Trovato 2009:466]).

Part of this increase in population is because the boundaries of Ontario were being negotiated throughout this era and its final size was not established until 1912 (Zaslow 1967, Baskerville 2005). The government of Ontario was in contest with the Dominion of Canada over former Hudson's Bay Company land along the Ontario/Manitoba border until 1889. At this point, the disputed area was formally granted to Ontario, which included much of Northwestern Ontario, formerly Keewatin Land, through the *Canada (Ontario Boundary) Act, 1889* (*Canada (Ontario Boundary) Act, 1889*, 52-53 Vict., c. 28 [U.K.]) (Zaslow 1967: 114, Dean 1969). In 1912, the Dominion government ceded the parts of the Northwest Territories south of the 60th parallel to Manitoba, Ontario, and Quebec, which extended Ontario's boundaries to their current position (Zaslow 1967:114, Baskerville 2005:127-8).

The size and boundaries of Ontario were changing and so too were the cities and counties. In 1882, there were 10 cities in the province: Toronto, Hamilton, Ottawa, London, Kingston, Guelph, St Catherines, Brantford, Belleville and St Thomas.¹ By 1895 this number had risen to 13, with the inclusion of Stratford in 1885, Windsor in

¹ The point at which a town could incorporate to become a city was set by law in the *Ontario Municipal Act, 1849*, also known as the *Baldwin Act, 1849*.

1891 and Chatham in 1894 (Legislative Assembly of Ontario 1887a, 1893, 1896, 1897). By 1918, there were 23 cities, with the incorporation of Fort William, Galt, Kitchener, Niagara Falls, Peterborough, Port Arthur, Sarnia, Sault Ste. Marie, Welland, and Woodstock. Likewise, there were 39 counties in 1882 and 40 in 1895, as the Provisional County of Haliburton in central Ontario was not included separately until 1886 (Legislative Assembly of Ontario 1883, 1887b, 1897). By 1918, there were 48 counties, with the newly included Districts of Kenora, Manitoulin, Nipissing, Rainy River, Sudbury, Thunder Bay, and Timiskaming, reflecting the finalized boundaries of the province. Further, the districts of Muskoka and Parry Sound were now counted separately (Legislative Assembly of Ontario 1919a).

While this time period saw changes to the boundaries and administrative composition of the city, there were also vast internal changes (Table 2.1). In 1881, 70% of the population of Ontario was rural, while only 30% was urban (Statistics Canada 2011).² The largest increase in the urban population occurred between 1901 and 1911: in 1901 43% of the province was living in urban areas, whereas in 1911 this number had jumped to 53% (Dominion of Canada 1912:528). By 1921, the urban population of Ontario was 58% of the total (in comparison, according to the 2011 Canadian census, 86% of the population of Ontario was living in urban areas, Dominion of Canada 1925:32, Statistics Canada 2011). Of those in urban areas in 1921, 31% lived in the city of Toronto (18% of the total population of Ontario) (Dominion of Canada 1925:32,105). As of 1901, the Government of Ontario reported that “less than twenty per cent. of the province has as yet been settled, over eighty per cent. still being in the hands of the crown” (Legislative Assembly of Ontario 1901a). The vast rural exodus during this time meant that much of these lands would remain unsettled. Yet, with over 8,000 miles of steam railways by 1901, people were able to move about the province fairly rapidly (Legislative Assembly of Ontario 1901a:31). Ontario was connected to the surrounding provinces of Manitoba and Quebec, as well as to the American states of Michigan and New York. Efficient

² According to the 1921 census, “in census statistics the population of all cities, towns and incorporated villages, regardless of size, are treated as urban and the remainder of the country as rural” (Dominion of Canada 1925:x). Regarding the definition of rural areas, Statistics Canada states that, “previous to 1981, the definitions differed slightly but consistently referred to populations outside centres of 1,000 population” (Statistics Canada 2011).

railway systems in many parts of the world have been implicated in the rapid spread of both the 1890 and 1918 influenza pandemics (Patterson 1986, Valleron et al. 2010, Le Goff 2011).

Table 2.1 - Population and Demographic Characteristics of Ontario, 1881-1921.

	Year				
	1881	1891	1901	1911	1921
	N				
Population	1,926,922	2,114,321	2,182,947	2,527,292	2,933,662
Toronto	96,196 ^a	181,215 ^a	267,730 ^a	376,538 ^b	521,893
	%				
Urban	30	39	43	53	58
Male	51	51	50	51	51
Canadian Born	78	81	85	80	78
British Born	18		11	14	16
Foreign Born	4	19 ^c	4	6	6

Source: Canadian Censuses. Dominion of Canada 1882, 1902, 1912, 1913, 1924, 1925.

^a. Population of the city municipality

^b. Includes Toronto Centre, Toronto East, Toronto North, Toronto S, Toronto West, and York South.

^c. Includes British-born population

In this analysis, Ontario has been divided into five general regions. This is to account for the different migratory patterns, cultural practices, and economic basis of such a large province. The North region includes the counties and districts of Algoma, Kenora, Manitoulin, Nipissing, Rainy River, Sudbury, Thunder Bay, and Timiskaming. In the early part of the twentieth century much of this region was unsettled and “unorganized” in terms of provincial control. It was an area populated by First Nations individuals, French-Canadians, and British and immigrant settlers (Middleton and Landon 1927). Many of the migratory workers came for positions in forestry and mining and to settle farming lands along the railways (Legislative Assembly of Ontario 1919b:31). Further, many itinerant workers lived in camps run by companies, “the health of whom is often hazarded negligently, carelessly and inadvisedly by these corporations” (1919b:31). The major cities are Fort William, Port Arthur, and Sault Ste Marie, and the major towns are Cobalt, Kenora, North Bay, and Sudbury (based on the classification of the 1918 Report of the Registration of Births Marriages and Deaths, Legislative Assembly of Ontario 1919a:12).

The Central region includes the counties of Dufferin, Haliburton, Hastings, Muskoka, Parry Sound, Peterborough, Prince Edward, and Victoria. The two major cities are Peterborough and Belleville and the towns are Lindsay, Parry Sound, and Trenton. This was a region of farming, lakes, and fishing and a destination of settlement for the United Empire Loyalists and British immigrants, although it was sparsely settled in areas (Weaver 1913).

The East region includes the counties of Carleton, Frontenac, Lanark, Leeds and Grenville, Lennox and Addington, Prescott and Russell, Renfrew, and Stormont, Dundas, and Glengarry. It has the two major cities of Kingston and Ottawa, and the towns of Brockville, Cornwall, Pembroke, Renfrew, and Smiths Falls. According to the District Officer of Health, Paul J. Moloney in 1918, this area was almost “entirely engaged in mixed farming” outside of the urban areas (Legislative Assembly of Ontario 1919b:27), while inside was the domain of manufacturing. Moloney noted that people on farms tended to live shorter lives and had more sickness than those in the cities, which he blamed on the “want of proper sanitary conveniences in the homes and dearth of knowledge of the laws of public health” and municipal water supplies contaminated with sewage (1919b:27). Prescott and the surrounding counties were predominately French-Canadian and there were Polish and German immigrants in the northern part of the region. However, most of the rest of the district was British and United Empire Loyalists (1919b:27). French immigration to Northern North America essentially stopped after 1760 resulting from the loss of French territories to Britain during the Seven-Years War (Charbonneau et al. 2000). Further, the immigration of United Empire Loyalists to Canada resulted directly from the American War of Independence, such that the majority came to Canada in the years around 1784 (McInnis 2000). It would therefore be expected that the East region would be mainly populated by people who were born in Canada, if not Ontario specifically.

The south-central region was a destination for immigration to the large urban centres, but also included areas of settled farming. The counties were Halton, Lincoln, Northumberland and Durham, Ontario, Peel, Simcoe, Welland, Wentworth, and York. It

contains the major cities of Hamilton, Niagara Falls, Toronto, St. Catharines, and Welland as well as the towns of Orillia, Barrie, Midland, and Oshawa.

Finally, the South-West region, with the counties of Brant, Bruce, Elgin, Essex, Grey, Haldimand, Huron, Kent, Lambton, Middlesex, Norfolk, Oxford, Perth, Waterloo, and Wellington is an agricultural area. The population included German-American settlers around the areas of Berlin (becoming Kitchener in 1917) and Waterloo, American immigrants (including a terminus of the underground railway), and English and French farmers (Middleton and Landon 1927). This is the region that contains the most cities: Brantford, Chatham, Galt (now part of Cambridge), Guelph, Berlin (Kitchener), London, Sarnia, Stratford, St Thomas, Windsor and Woodstock. It also has the towns of Collingwood, Ingersoll, Owen Sound, and Walkerville.

Immigration to Ontario kept relatively steady over the 40 year period. In 1881, 78% of the population was Canadian-born, the same percentage as in 1921. However, the percentages did change over the period, with 80% of the population being Canadian-born in 1901 and 85% in 1911. Beaujot and Kerr (2004:97-100) describe three different immigration “phases” during this 40 year time span. The period from 1850-96 was the “period of net out-migration” during which “although large numbers of people immigrated to Canada, even more emigrated” (2004:97). These emigrants consisted both of recent-arrivals and established settlers in Canada, mostly from Quebec. Although there was only an increase of 187,399 between 1881 and 1891, there was a high level of population turn-over, suggesting higher demographic and societal change in Ontario than is implied by the population totals at the time of the censuses.

The next phase of immigration lasted from 1897 to 1913. During this period, “arrivals rose from a low point of 17,000 immigrants in 1896 to 400,000 in 1913” for the entire country and it “contained record numbers which have never since been surpassed” (Beaujot and Kerr 2004:97). During this phase, the population of Ontario grew by 344,345, including both immigration and natural increase. The final phase, from 1914-1945, is dominated by war-time trends. The only part of this phase that is included in the 40-year span of this research is from 1914-1921, during which there was virtually no

immigration to Canada or Ontario, reflective of the restrictive immigration conditions during the First World War (2004:97-100).

For those people residing in the province at each census point, the percentage male and female in Ontario remained steady. Life expectancy at birth for males went from 45.3 years in 1881 to 55.8 years in 1921 and for females from 48.6 years in 1881 to 57.3 years in 1921 (as estimated by McQuillan 1985:41 using the Bourbeau-Légaré method of life expectancy estimation, Bourbeau and Légaré 1982). This is in part reflective of a high infant mortality rate that did not fall below 100 deaths per 1,000 live births until 1917 (Legislative Assembly of Ontario 1919a). In 1901, the middle of the 40 year period, the average age at first marriage in Canada for women was 25 for females and 28 for males (Burke 2007:26). As birth registrations were not complete until the 1930s (Emery 1993) and calculating fertility rates during this period is difficult based on the questions asked on the census, the child-women ratio is used: the number of children under 5 years of age is compared to the number of married women between the age of 15 and 45. Taking into account childhood mortality under the age of 5, Gauvreau and Gossage (2001) estimated this to be 0.86 for Ontario in 1901, meaning that there were 0.86 children under 5 for every married woman of child-bearing years in the province. The ratio was 0.69 for Protestants and 1.05 for Catholic women in Ontario. This is compared to a similar figure of 1.40 for Quebec in 1901 (Gauvreau and Gossage 2001:171). Gauvreau and Gossage (2001:172) estimate a child-woman ratio of 0.81 for women living in rural areas in 1901 as compared to 0.65 in large towns. This contrasts with the statements from the registrar generals who find that “it is in the cities that the greatest increase of population occurs, that in the cities there is the greatest number of births per 1,000, owing to a proportionally large number of persons at child-bearing ages” (Legislative Assembly of Ontario 1896). This could partially be accounted for by the higher infant mortality rates in urban areas (McInnis 1997:266).

Research has shown a relationship between religion and mortality in historical Canadian contexts, especially in terms of infant mortality (Taylor 1986, Mercier 2006). This is not due to the belief system in itself, but with the concomitant behavioural practices, such as family size, crowded living spaces, fertility behaviours, and poverty and its potential

unhealthy sequelae. Of all the possible socio-cultural differences that influenced infant and child mortality in Toronto in 1901, Mercier reports that “the most influential factor was the religious affiliation of the family, with adherence to Judaism associated with low mortality levels, and Catholicism associated with high mortality” (2006:136). Once again, this could be associated with living and working conditions and social class, since in Toronto the city was divided into “two zones; a poorer, and largely industrial Catholic one, and a more prosperous, northern Protestant one” and that “Catholics were, on average, poorer, and hence associated with poor housing and environmental conditions” (2006:139). The behavioural differences he suggests could be partially responsible are “household hygienic practices, breast or artificial feeding of infants, fertility practices, and child care” (2006:142). Religion is an important distinction even between socio-economic zones since Jewish children experienced the lowest mortality and yet one of the highest concentrations of Jewish families was in the poorest area of the city, the Ward (2006:145).³ In her 1910 report on infant mortality in the section entitled “Rachael is a Good Mother,” MacMurchy affirms the lower mortality rates among Jewish families, her reasons for that being that “it is well known that Hebrew mothers almost invariably nurse their children” and that their homes were always clean (MacMurchy 1910:10). Adult mortality correlates of religion might further involve the higher fertility among Catholics leading to more crowded living environments (Gossage and Gauvreau 2007).

The finding of higher Catholic mortality was also observed in the early twentieth century in Ottawa for adults (Taylor 1986) and also for infants (Mercier and Boone 2002). However, Gauvreau (2006) and Gossage and Gauvreau (2007) stress the importance of not merely looking at Catholic-Protestant differences in fertility and mortality, but that it is equally important to examine the differences among the various Protestant denominations in the province, as well as among the non-Christian religions.

³ It is hard to speak to Jewish differences in the WMMIP sample however, since Jewish immigration to Canada was relatively recent (not starting to accelerate until after 1871, Sarna 1976) and if these deaths occurred, they would have occurred to the excluded immigrant population. For example, among the decedents only 2 were identified as Jewish in 1901 (0.07%) and only one in 1911 (0.04%). Unsurprisingly, the one woman found in 1911 is the same person as one of the 2 women in 1901. The sample taken from the Ontario-born selection of the 1901 five-percent sample from the CFP has only one individual (family) who declared himself to be Jewish (Chapter 6).

There were important differences between the Protestant denominations. Although the largest ethnic group in the province was English, the highest percentage of the Protestants was Methodist, not Anglican. Gossage and Gauvreau report that in 1901, Methodism was the denomination of 40% of those who were Protestant (2007:64). United Empire Loyalists brought Methodism to Canada from the United States at the end of the eighteenth century. It was a religion that “emphasized fellowship, free choice, and individual salvation” and where the “clergy generally came from the lower ranks of society” (Baskerville 2005:86-7). It was dedicated to a pious and rigorous following of biblical tenants (Van Die 1989). Regarding fertility, and thus potentially family size, Gossage and Gauvreau found low fertility for Methodist women in Ontario (2007). Creating a scale of child-women ratios centered on Anglicans, they state that “for women aged 25 to 39, Catholics, both English- and French-speaking, had higher fertility, while Methodists and Baptists had significantly lower fertility than Anglicans” (2007:94).

There were some changes in the dominant religions over the forty year period in Ontario (Table 2.2). The number of Catholics increased from 17% of the population in 1881 to 20% in 1921, while the Methodist population decreased from 31% of the population in 1881 to only 24% in 1921. Jewish populations were not significant in Ontario until after 1921 with the entry of Jewish immigrants fleeing pogroms in Europe after the First World War (Kelley and Trebilcock 2010). Catholics were clearly a minority in Ontario, which likely exacerbated the stigmatization resulting from persistent Protestant-Catholic antipathies, leading to possible differences in mortality risk (Chapter 6).

Table 2.2 - Major Religions in Ontario, 1881-1921.

	Year (%)				
	1881	1891	1901	1911	1921
Methodist	30.8	30.9	30.5	26.7	23.5
Presbyterian	21.7	21.4	21.9	20.8	21.0
Anglican	19.1	18.3	16.9	19.5	22.2
Catholic	16.7	16.9	17.9	19.6	20.4
Baptist	4.8	5.0	5.3	5.3	5.1
Lutheran	2.0	2.1	2.2	2.7	2.3
Other	4.2	4.1	4.8	5.2	5.3
Unknown	0.8	1.1	0.5	0.3	0.2

Source: Canadian Censuses. Dominion of Canada 1882, 1902, 1912, 1913, 1924, 1925

Ontario during this time period was mainly British and Protestant, with high levels of discrimination and intolerance toward those who were of other ethnic and religious backgrounds (Masters 1947, Piva 1979, Bothwell 1986). Anti-French, anti-Catholic sentiments were escalating during the first half of the time-period and English-French and Protestant-Catholic tensions persisted over the entire 40 year period (Bothwell 1986). As explained by Middleton and Landon (1927), “Ireland especially provided great numbers of hardy settlers, and whether they came from the North or the South, they contributed greatly to the upbuilding of the Province. *It must be admitted that they brought their prejudices with them*” (1927:889, emphasis added). Anti-French feelings increased in 1917, after the *Military Service Act, 1917* was passed that instated conscription in Canada. A common opinion was that Quebec was not contributing an equal share of men to the war effort, and although the *Military Service Act* did pass, the votes were split, with the majority of English MPs supporting the bill and the majority of French MPs rejecting it (Miller 2002:100, 139-140). Civil unrest and ethnic tensions continued after the war, as there were lingering resentments both to those who did not enlist as well as against Canadians whose ethnic background was from one of the formerly combatant nations (Miller 2002, Crerar 2005).

As was typical both before and after this 40 year period, First Nations people in Ontario experienced high levels of discrimination, loss of lands and resources (Jenness 1937, Baskerville 2005). A government report from Manitoba in 1908 stated that “on the reserve, people sheltered in log cabins during the cold winters; the cabins were purported by Indian Agents to be crowded and poorly ventilated” (Moffat 1992:118). Herring (1993) found that 18 percent of the population of Norway House in Manitoba died from the Spanish flu in the fall of 1918, due in part to the relative isolation of the population and the resulting lack of immunological experience. It is hard to examine the experiences of aboriginal people in Ontario using official statistics, since they are one of the groups who are notoriously underrepresented in these Canadian records (Kuczynski 1930, Emery 1993).

2.1.2 *Social Environment*

Ontario from 1881 to 1921 experienced vast growth in population, commerce, and production, through the combined factors of immigration, natural increase, and territorial expansion (Piva 1979, Careless 1984). This growth centered on Toronto and Hamilton and was not consistent throughout the 40-year time period (Piva 1979, Herring and Korol 2012). There had been a severe economic depression at the end of the 1870s, which lingered on throughout the 1880s and into the 1890s (Masters 1947). Construction stalled and economic concerns caused the amalgamation of several railway companies which led to rampant unemployment (Masters 1947). While conditions did recover in the late 1880s, at the beginning of the 1890s (at the time of the Russian influenza pandemic) there was yet another economic depression, related to a financial crisis in London and New York (Masters 197, Piva 1979:3).

The twentieth century did not bring economic improvements. Rapid immigration into Toronto from 1905 to 1915 led to inadequate and overcrowded housing and slum conditions in parts of the city (Mercier 2006). According to Piva, in 1921, the average annual earnings for blue-collar workers in Toronto were \$1,053.32 and \$1,605.33 for white-collar workers (1979:31, Table 8). However, the Department of Labour in 1921

calculated that “a family of five would require \$31.83 per week and \$1,655.29 per annum to rise above the poverty line” (Piva 1979:38). Poor relief had been allocated by the province to be within the purview of the local municipalities, but the majority of the allocated funds went to private charitable organizations to provide relief (Piva 1979).

All workers were subject to poorly regulated working conditions that had the potential of being highly insalubrious. The length of day and the hours worked per week varied by occupation, with many fields not instituting the 8 hour day until after 1920. Further, while reforms were being enacted through legislation, they were criticized for lack of enforcement, especially in the areas of sanitation, provision of drinking water, adequate ventilation, lighting and the assurance of a comfortable temperature, and in the restrictions against child labour (Piva 1979). Reforms continued during the 1910s with the activities of the labour unions and through the establishment of the Workman’s Compensation Act in 1914, but working conditions were not greatly improved until after 1918 (Piva 1979). Neither was there an extensive social safety net for those who could not work, as it was not created until after the devastation of the Great Depression in the 1930s (Baskerville 2005).

While women had been increasingly entering the work force throughout the First World War, both men and women faced unemployment with the return of soldiers at the end of 1918. Returned soldiers “complained bitterly over poor pensions and the lack of jobs, and they accosted ‘aliens’ who were fully employed” (Baskerville 2005:179). Added to this was the lack of housing, such that “Toronto is short ten thousand dwellings” (The Globe 1918c:6), and that in the spring of 1918, Toronto’s Medical Officer of Health found that “of the 13,000 houses inspected recently, 8,000 were overcrowded . . . most of this doubling-up occurred among working-class families with only one wage earner” (Piva 1979:131).

Outside of Toronto conditions were similar. The Report of the Provincial Board of Health in 1918 has statements from various parts of the province indicating contaminated water and milk (and the resultant typhoid outbreaks and cases of tuberculosis), concerns with city dumps, and industrial waste (Legislative Assembly of Ontario 1919b). The

District Officer of Health (DOH) for Belleville, Dr. George Clayton found shacks outside of the British Chemical Company in Trenton that were “in a dirty, bad condition and crowded” although the main dormitory was in better condition (1919b:26). The DOH for Cornwall spoke of unhealthy conditions in the rural areas. This was in contrast to the conditions in the cities, where citizens were forced by laws and social pressure to keep their homes in more sanitary conditions. In Chatham, seven homes “were placarded as being unfit for human habitation” (1919b:136) while in many towns and cities, the reports speak favourably of attempts to commence garbage collection and of the implementation of sewerage.

2.1.3 *The First World War*

Influenced by its Loyalist, Protestant beginnings, the population of Ontario eagerly participated in the Great War of 1914-1918 (Middleton and Landon 1927). The prevailing sentiment in Ontario was that “its people had always been proud of their British loyalty, of their Imperial idealism, of their readiness to do great things if ever the time for action should come and the life of the Dominion, the honour of the race, the safety of the Mother-land, should be threatened” (Hopkins 1919:1). This meant that not only did large numbers of young men enlist in the armed forces, but those left at home volunteered their time, money, and limited resources to the war effort. Vast government spending on the war meant cut-backs in domestic provisions, but these were accepted without complaint from the people of Ontario, including the implementation of a war-tax to cover the government deficit in 1915 (Hopkins 1919).

Recognizing the unsuitability and general unavailability of housing in Ontario in 1918 and that this problem would increase greatly once the soldiers returned home, the government began a building project that would provide affordable homes to working people (Hopkins 1919:96-97), but conditions were difficult for many people in Ontario. There were constant food, power, and fuel shortages (Wilson 1977:lxii-lxiii) added to rampant inflation (MacDougall 1990). By the end of 1918, “business conditions were most unstable, . . . the cost of living had mounted to an unexampled height, and . . . the

technique of “making ends meet” had become involved and difficult for most classes of every community” (Middleton and Landon 1927:756). The Spanish influenza pandemic of 1918 came right at the end of the war, right after years of public shaming of men who had stayed behind, the constant stress of having loved ones in danger and dying, and the health tolls that these and the food and energy restrictions had on the people of Ontario. Resistance to infectious disease would have been lessened, since it is known that both poor nutrition and stress contribute to increasing the susceptibility to infectious disease (Singer and Baer 1995).

Additionally, the war had removed both money and people from Ontario. In May of 1918, the Canadian Medical Association Journal reported that “Toronto hospitals are undergoing a considerable financial strain owing to the conditions enforced by the war” (CMAJ 1918a:460). Then, in October, during the height of the influenza epidemic, it was reported that “voluntary enlistment of doctors has had the effect of leaving large sections of our population without the aid of a physician within reasonable distance” (CMAJ 1918b:932-3). This necessitated the use of volunteer nurses (the Sisters of Service) and the Victorian Order of Nurses (VON), who were recognized as providing valuable medical assistance during the epidemic (McCullough 1918). It also prompted many Ontarians to seek health care services from the ‘drugless practitioners’ such as osteopaths and chiropractors (Adams 2012).

By the time conscription was implemented on October 31st, 1917, Ontario had already contributed 191,632 out of the total 439,806 Canadian soldiers. By June 17th, 1918, Ontario had sent 232,191 of the total 538,283 Canadian enlistments (Hopkins 1919:15). Many volunteers had been rejected from serving based on physical defects in both this war as well as the prior Boer War from 1899-1902. These rejection rates were evidence to contemporary public health officials of the necessity of the elimination of maternal and child poverty (MacDougall 1990:174). By 1915, the high initial numbers of volunteers were beginning to wane. After running several recruitment drives, the medical standards to enlist were lessened. The minimum height dropped from 5 feet 3 inches to 5 feet 2 inches, and the minimum chest measurement dropped from 33 ½ inches to 32 ½ inches (Miller 2002:69-76). Still, the medical requirements were strict and men could be

rejected for poor eyesight, poor dentition, flat feet, or varicose veins. Even in 1915, “for every ten men who volunteered, three or four failed the medical exam, but it was not unusual for six or seven to be rejected” (Miller 2002: 79-80). Given the numbers that enlisted and the average number of rejections, Miller (2002:80), estimates that over 40% of the eligible men in Toronto had volunteered by 1915. By the end of 1915, the eyesight standards had also been relaxed (Miller 2002:85). In 1918, men who had been rejected, but were still capable, were put into a special non-combatant class, whereby they could still participate in the war in some manner (Dominion of Canada 1919). Although a reserve class of men under 19 were registered by the conscription act, the Class I men were those “who had attained the age of twenty years and were born not earlier than the year 1883, and were unmarried or were widowers without children” (Dominion of Canada 1919:42). Many men who appealed to the tribunals set up to evaluate claims of exemption were denied (Miller 2002). Exemptions were granted for conscientious objectors, those involved in agricultural production or other industries directly related to the war effort (Dominion of Canada 1919:152-153). The result of the enlistment of the young men who were at heightened risk during the pandemic is to change the composition of Ontario immediately prior to the influenza pandemic. For this reason (the unknown population at risk), it becomes difficult to calculate accurate mortality rates by age, since neither the censuses in 1911 or 1921 were representative of the Ontario at the end of four years of war (however, this is attempted in Chapter 4). Further, since enlistment criteria continued to loosen the medical and height restrictions, the result was that those men who were left behind (who had not been exempted on compassionate or needful grounds), were those whose health was insufficient to meet those criteria, or who had been wounded already in the war.⁴ By 1917, the Medical Officer of Health for Toronto stated that “in view of the alteration in the expectation of life of our population due to the removal, by enlistment of Toronto’s best risks, it will be difficult to make comparative mortality statements that are not misleading” (City of Toronto Archives Fonds 200, Series 365, File 20).

⁴ However, there is evidence of the first wave among Canadian soldiers in the spring of 1918 (Rewegan et al. 2015). This could have provided some immunity to those soldiers who were infected and were still in Canada in the fall of 1918.

2.1.4 *Health and Illness*

Our soldiers have already shown us Canadians how to die.
It is up to the health authorities to show the people how to live

Robert Law, Acting Medical Officer of Health,
Ottawa, 1918 (Legislative Assembly of Ontario 1919b:158)

The forty-year study period of this research encompasses two separate systems of disease classification used by the Registrar General of Ontario, reflecting the changing understanding of the concept of disease and illness in society in the late nineteenth century. From 1882 to 1897, a 5 class system was used based on the classification scheme of Dr. Farr and in 1898, the Registrar General switched to the Bertillon System of Classification of the Causes of Death (the precursor to the International Statistical Classification of Diseases, Injuries and Causes of Death (ICD), Legislative Assembly of Ontario 1900, O'Malley et al. 2005, Lilienfeld 2007). The system of Dr. Farr relied heavily on the miasmatic theory of disease causation while the Bertillon system is more reflective of the germ-theory, which was slowly gaining support in biomedical circles around the turn of the century.

The system of disease classification used until 1897 consisted of 5 classes. Influenza was included in the Class I – Zymotic diseases, under “Order 1. Miasmatic Diseases, *Miasmatici*” (Legislative Assembly of Ontario 1883:42). According to Farr, the “Miasmatic diseases (Order 1) are diffusible through the air or water, and are attended by fevers of various forms; the matter by which they are communicated is derived from the human body (as in small-pox) or from the earth (as in ague)” (Farr 1856:9). Therefore, influenza at the time of the Russian pandemic of 1890 was thought to be caused by inorganic emanations from the environment that manifested and reproduced in the blood and were transmissible between individuals (see Figure 2.1, Section 2.2).

Phthisis or consumption (tuberculosis) was frequently the most common cause of death during this period (Legislative Assembly of Ontario 1883-1898, 1919b) and a relationship between co-infection with tuberculosis and influenza has been suggested to

be related to high young adult mortality in 1918 (Noymer and Garenne 2000, Noymer 2009). Under the classification system of Dr. Farr, tuberculosis was in Class II, Constitutional Diseases, being those caused by “bad habit of body” being “sporadic; they are, sometimes discovered to be hereditary; they are rarely confined to one part, but before death ensues they affect several organs, in which new morbid products are often deposited” (Farr 1856:9).

The third class of disease encompassed the Local Diseases, or the “sporadic diseases, in which the functions of particular organs or systems are disturbed or obliterated with or without *inflammation* and its products; some of the diseases are hereditary” (Farr 1856:9). The Registrar-General of Ontario lists among these diseases “Epilepsy, Apoplexy, Convulsions, Paralysis, Insanity, Heart Disease, Congestion of the Lungs, Bronchitis, Pleurisy, Pneumonia, Disease of the Stomach, Liver and Kidneys, etc.” (Legislative Assembly of Ontario 1883:38). Thus, during the 1890 Russian pandemic in Ontario, those diseases which are described in this research as ‘pandemic related’ (influenza, pneumonia, and bronchitis), as well as tuberculosis, which was thought to exacerbate the effects of influenza infection, were not conceptually related diseases, being placed in three separate disease classes.

The implementation of the Bertillon System in 1898 saw causes of death separated into 13 separate groups (Legislative Assembly of Ontario 1900). The main causes of death in this study were still separated with influenza being a communicable disease, tuberculosis and scrofula among the Other General Diseases, and bronchitis and pneumonia being classified as Diseases of Respiration (Legislative Assembly of Ontario 1900). By 1918, the first two groups, Communicable Diseases and Other General Diseases had been collapsed into a single category of “General Diseases,” incorporating both influenza and tuberculosis into the same category (Legislative Assembly of Ontario 1919a). Pneumonia and bronchitis continued to be classified separately as local diseases of the lungs.

Except during the years 1890, 1892 and 1894, influenza, or *la grippe*, was paid very little attention in the Registrar-General’s reports (Legislative Assembly of Ontario 1883-1898).

The department was far more concerned with the diseases that were exacting the greatest toll on the province, such as measles, scarlatina (scarlet fever), typhoid, diphtheria, tuberculosis (phthisis or consumption), smallpox and with the possibility of a cholera outbreak in 1893. When the *Act to Provide for the Registration of Births, Marriages and Deaths, 1869* was amended in 1896 through the *Act Revising and Consolidating the Acts Respecting the Registration of Births, Marriages and Deaths, 1896*, it was now required of each division registrar to make a monthly return (report) of the number of deaths from these specific contagious diseases that occurred in their district (*An Act Revision and Consolidating the Acts Respecting the Registration of Births, Marriages, and Deaths, 1896*, 17 11.4, Legislative Assembly of Ontario 1897:6). Even after the pandemic of 1890, influenza was not considered a serious enough disease to warrant monthly reports of deaths. Cases of influenza among the living were not made “notifiable” until after the 1918 pandemic, in 1923, when the Regulations for the Control of Communicable Diseases were approved by the Lieutenant-Governor in Council (Legislative Assembly of Ontario 1924). After this point, cases of influenza (specified as epidemic influenza) were required to be reported to the Medical Officer of Health or the Secretary of the local Board of Health. Far more people were infected with the virus than killed by it in both the influenza pandemics of 1890 and 1918. But, since influenza morbidity statistics were not kept in Ontario until 1923, all knowledge of the experiences of these epidemics must be gained at the individual level through the analysis of causes of death from the death records (an analysis of the history and completeness of these records is found in Chapter 3). However, since many people were infected in 1890 but few died (Valleron et al. 2010), this creates a natural experiment in which to test influenza-specific hypotheses concerning early life influences on later life health.

As seen through contemporary reports, the Government of Ontario was highly concerned with improving the state of public health in the Province during this period. Efforts to improve public health were abundant in England at the time, following on the establishment of the Office of the Registrar General through the Registration Act of 1837 (Legislative Assembly of Ontario 1889). The Registrar General of England in 1887 gave credit for the increasing interest in public health directly to the collection of the numbers and causes of death. Stating that “it is the *registration of deaths*, and of their causes that

has made sanitation possible” (Legislative Assembly of Ontario 1889:52). Similar emphasis on public health was seen in the United States resulting in the Medical Officer of Health for Toronto frequently interacting with his counterparts in England and the United States (MacDougall 1990).

There was no Canadian Board of Health until 1919. Federal and provincial responsibility was determined in the *British North America Act, 1867* with the responsibility for local institutions being the purview of the provinces while the Federal Department of Agriculture was responsible for marine hospitals and quarantines (Health Canada 2009). After the First World War and the deaths incurred during the Spanish influenza pandemic, several pieces of legislation were passed that allowed for the creation of the national Department of Health (Bryce 1921). Prior to this point, health care in Ontario was overseen by the Provincial and Divisional Boards of Health, while tabulations of births, marriages, and deaths were the responsibility of the Registrar-General.

The Registrar-General was highly concerned with public health in the province, as it had a direct impact on the number of births, marriages, and deaths. His annual reports from 1882 to 1896 provide valuable insight into the state of health of the province during this time. For example, a constant concern was the ever-present cases of diphtheria in the province and the Registrar-General actively sought to reduce the number of deaths from this disease. When there were less diphtheria deaths in 1883 than in 1882, the Sanitary Inspector declared that this “was probably caused by the action of the Provincial Board of Health, lately established in this Province, which encouraged the formation of Local Boards of Health and also disseminated information respecting sanitary measures, and thus caused precautions to be taken to prevent the spread of this and other contagious diseases” (Legislative Assembly of Ontario 1884:43). Based on the miasmatic idea that disease was caused by decay and unsanitary conditions, the purpose of the Boards of Health was to clean up the environment so as to maintain health. The kinds of initiatives undertaken by the boards included milk and water sanitation, food inspection, demolition of the slums, vaccinations, and concerns with high levels of poverty, overcrowding, and infant and maternal mortality (MacDougall 1990:11).

Concerned with high rates of infant mortality in Toronto, Dr. Helen MacMurchy wrote about the issue in 1910. She noted the relationship between poverty and infant mortality in the city, stating that “where the mother works, the baby dies. Nothing can replace maternal care. The destruction of the poor is their poverty” (1910:17). This was thought to be due to the inability of the working mother to nurse her child, since breast-milk replacements at the time were unreliable (1910:17). The Registrar-General kept statistics concerning the numbers and rates of infant mortality and illegitimate births per year (Legislative Assembly of Ontario 1883-1898). The high levels of deaths among illegitimate children are now thought to be caused by poverty, social stigma, lack of access to resources, young age of the mother, and lack of breastfeeding (Kok et al. 1997). Yet, public health officials during this time period were cognizant that illegitimate children had higher mortality than legitimate children (The Lancet 1918:303).

2.2 Influenza

It is difficult to identify historical epidemics without having samples of the virus to analyze genetically. However, past ‘plagues’ have been attributed to influenza through evaluations of their symptoms as described by contemporaries as well as through diagnoses provided by physicians and others. Epidemics have been recognized as early as the twelfth century in Europe (Patterson 1986) and influenza may have been responsible for epidemics in the tenth century as well (Glezen and Couch 1997). However, due to the airborne nature of the disease as well as its zoonotic transmission between pigs, birds, and humans, it is possible that influenza has been endemic in human populations since the inception of agriculture (McNeill 1976, Patterson 1986, Glezen and Couch 1997, Quinn 2008).⁵ According to Patterson (1986), there have been thirteen pandemics (or probable pandemics) since 1700. These occurred in 1729-30, 1732-33, 1781-82, 1788-89, 1830-31, 1833, 1836-37, 1889-90, 1899-1900,⁶ 1918, 1957, 1968, and

⁵ Patterson (1986) notes that since influenza is not found among primates, has a relatively short infectiveness period, and requires a rather large population base, it is unlikely that this disease extends to our early hominid history

⁶ There is debate over whether the 1899-1900 outbreak was pandemic in form or merely epidemic, although it was present in both Europe and North America (Worobey et al. 2014:8108). The 1899 and 1900 Reports of the Registration

1977 (1986:83). Morens and Fauci also include pandemics in 1760-62 and 1847-51 (2007:1025). Adding the recent pandemic of 2009-2010 would make sixteen pandemics in three hundred years. The Provincial Board of Health for Ontario stated in the 1891 report that since the first recorded influenza pandemic in December 1173 in Germany and England, there had been 90 successive pandemics between 1173 and 1874-5. In the nineteenth century, there were reported to be pandemics in 1830-33, 1841-43, and 1860-63 (Legislative Assembly of Ontario 1891b).

Influenza is found in three major types in humans: Types A, B, and C. Types B and C infect only humans while Type A is a zoonotic disease (transmissible between humans and animals) which is hard to treat and identify due to its rapidly changing surface proteins (Glezen and Couch 1997). There are two main surface proteins, hemagglutinin and neuraminidase, and it is the combination of these two proteins that lead to the various

of Births, Marriages, and Deaths for the Province of Ontario show that the disease was present in Ontario in epidemic form. The 1899 Report states that:

The re-appearance of influenza very notably increased the mortality from communicable diseases, which as already mentioned, took place almost wholly in February and March. Indeed if the increase in influenza, not classed as a contagious disease under the Public Health Act, were separated from the other communicable disease therein referred to, the year 1899 would show an actual decrease of 83 deaths. In the report of 1892 is found a reference including an extract from the English Report for 1890, which illustrates very exactly the conditions accompanying the epidemic and extremely rapid character of the spread of this destructive disease of middle and later life. The increase of deaths in February was so sudden and excessive that it almost equaled the births, being 3,152 as compared with 3,581 births, and actually exceeded the births in several cities. [Legislative Assembly of Ontario 1901b:14-15].

Further, the 1900 Report identifies this as an epidemic in that, “the principal cause of increase over 1898 was in both years, influenza – there being in 1899, 990 deaths, as compared with 192 in 1898, and 328 in 1900 . . . every class [of death] shows an increase of 1899, excepting the Respiratory System. The relation of this class to the severe epidemic of influenza is the natural explanation of the excess in 1899 of 225 deaths” (Legislative Assembly of Ontario 1902:25). This is followed by a table that shows the number of deaths in the province from influenza, which identify the years 1892 and 1899 as having strong resurgences of influenza (1900:26):

	1891	1892	1893	1894	1895	1896	1897	1898	1899	1900	Total
Influenza	393	927	483	613	426	502	549	192	990	324	5,399

The Report of the Committee on Contagious Diseases for the Quarter ending March 31st, 1901 from the annual report of the Board of Health of Ontario states higher mortality than the same quarter in 1900 due to the “general prevalence of LaGrippe in January” (Legislative Assembly of Ontario 1901c:45). The numbers of deaths were increased in February and it was projected for March as well. However, both the Reports of the Registrar General and Provincial Board of Health for 1899 and 1900 were more concerned with outbreaks of smallpox, diphtheria, and scarlet fever than with the increase in influenza deaths.

classifications of influenza strains (for example, H1N1, H3N8) (Glezen and Couch 1997, Quinn 2008). The success of the influenza virus in humans is due to its rapid rate of mutation, such that any immunity gained from exposure to an earlier, specific form of the virus can quickly become immunologically irrelevant (Kim et al. 2009). The disease changes in two major ways: through genetic shift and antigenic drift (Webster et al. 1992, Taubenberger 2006, Quinn 2008). Genetic shift occurs when two different strains of influenza co-infect the same cell and, through the process of reproduction, swap part of their RNA, thus changing the make-up of their surface proteins (Glezen and Couch 1997). This process produces an entirely new form of the virus and can lead to sudden and sweeping pandemics, as has been hypothesized for the emergence of H1N1 in 1918 and 2009 (Taubenberger and Morens 2006). New seroarchaeological evidence from Worobey and colleagues (2014), suggests that the H1 protein of the H1N1 virus had appeared in humans by around 1907 and that the N1 protein had been introduced to human populations from birds by about 1913, joining with the H1 protein around 1915. However, the exact timeline behind the appearance of the H1N1 strain in 1918 is still being determined (see the response to Worobey et al. 2014 in Gagnon et al. 2015).

The other major form of change for influenza more generally concerns seasonal flu. Antigenic drift is the slow accumulation of mutations in the surface proteins that can alter the virus just enough that previous infection no longer confers comprehensive immunity (Francis 1953, Glezen and Couch 1997). It may account for the different waves of an influenza pandemic, whereby infection in the first wave provides partial immunity in the second, often more severe wave. Additionally, antigenic drift explains why the ensuing waves after the height of the epidemic decline in virulence until the disease vanishes from the population. As explained by Taubenberger and Morens,

Historical records since the 16th century suggest that new influenza pandemics may appear at any time of the year, not necessarily in the familiar annual winter patterns of interpandemic years, presumably because newly shifted influenza viruses behave differently when they find a universal or highly susceptible population. Thereafter, confronted by the selection pressures of population immunity, these pandemic viruses begin to drift genetically and eventually settle into a pattern of annual epidemic recurrences caused by the drifted virus variants. [2006:16].

Influenza is an airborne infectious disease whereby “most infections are probably acquired by inhalation of small (1- to 5- μ m-diameter) particles” (Glezen and Couch 1997:488). With an incubation period of 1 to 5 days, and

When a large proportion of the population has at least partial immunity, about 20% of infections will be inapparent, and about 30% will be manifested only by signs and symptoms of upper respiratory tract involvement without fever. Febrile upper respiratory illness (URI) or influenzalike illness (sudden onset of fever, chills, sore throat, myalgia, malaise, headache, and hacking, non-productive cough) will occur in about 50% of infected patients . . . About 5% will progress to frank involvement of the lower respiratory tract disease (LRD) with development of tracheobronchitis or pneumonia . . . When there is a predominant bacterial pathogen, it is usually *Streptococcus pneumoniae*, *Staphylococcus aureus*, or *Haemophilus influenzae*. [Glezen and Couch 1997:492].

Additionally, central nervous system involvement may lead to encephalopathy and encephalitis, and manic psychosis (Glezen and Couch 1997, Honigsbaum 2010). The high mortality associated with influenza is often attributed to secondary co-infection with bacterial pathogens (Beveridge 1977).

However, what is scientifically known about influenza today is not the same as what physicians knew about the disease in the late 19th and early 20th centuries. The name *influenza*, derived from the Italian for ‘influence’, speaks to the pre-germ theory notions of the celestial influence on human disease (Parsons 1891, Quinn 2011). Further, the common name for the flu, *la grippe*, is believed to either refer to the grasp that the illness takes on the throat of sufferers or to the speed at which individuals succumb to influenza (Steenhock 2011). While physicians were debating the existence of viral organisms in the late 1800s, Richard Pfeiffer of Germany identified a bacillus he found in the sputum of individuals sick with the flu, which was given the name Pfeiffer’s bacillus (Crosby 1989). Although this bacillus was found in most of those individuals sick with the flu, tests could not satisfy all of Koch’s postulates “required to confirm a specific organism as cause of a specific disease” (Susser and Stein 2009:112, Crosby 1989). Regardless, Pfeiffer’s bacillus as the cause of influenza became known in the public consciousness and was still being advanced as wholly responsible during the Spanish flu pandemic of

1918 (Crosby 1989). The influenza virus as is known today was not identified until 1933 (Crosby 1989, Kolata 1999).

What qualified as the flu also was not initially agreed upon. In 1889, Parsons describes that “some medical men maintained that the, to them, new epidemic was not influenza at all, but some other disease, as dengue, malarious fever, or relapsing fever” (1891:304). Similarly, in 1918, the various causes of the new pandemic were thought to be cholera, or typhus, or simply “pyrexia of unknown origin” (Collier 1974:8). As such, when diseases are not uniformly agreed upon, death is often the result of secondary infections, and it is likely to exacerbate chronic conditions such as tuberculosis, the identification of pandemic influenza using death records must be recognized as being highly conservative.

2.2.1 *Influenza in Ontario*

I am pleased to be able to report that during the year we have been exceptionally free from epidemics and contagious diseases. But, sir, I must not forget to mention “la grippe,” which, about a year ago now, was making us its unwelcome visit. Few persons, indeed, were fortunate enough to be exempted from its attack. Most of the cases were of a mild character, but in not a few households a “vacant chair” stands as an unpleasant reminder of la grippe of 1890. [Angus Ego, Medical Health Officer of Markdale, Ontario, Legislative Assembly of Ontario 1891b:67]

The 1890-1892 influenza pandemic was contemporarily labeled the “Russian Flu” as the first reported cases were in Russia. Although many epidemics are often given ethnic labels erroneously (for example, the “Spanish flu” of 1918 that was named so only because neutral Spain did not experience the war-time media censorship of other nations and was thus the first nation to report the presence of the flu, Crosby 1989), it is probable that the 1889-1892 influenza did have its origins in Russia (Patterson 1986).⁷ Dr. Frank Clemow reported to the British Medical Journal in January of 1890 that there had been cases of epidemic influenza in Siberia as early as October 15, 1889, in the Russian calendar (October 27th, 1889 in the Gregorian calendar) (BMJ 1890). However, there may have been an earlier wave in the spring of 1889 in Russia (Patterson 1986, Taubenberger

⁷ However, Quinn (2008) suggests that it may have started in southern China.

and Morens 2006). It travelled from east to west through Europe, reaching Germany, Austria-Hungary, and France by the end of November, the United Kingdom by mid-December, and had reached the United States and Canada by January, 1890, with epidemics starting within weeks of the first occurrence (Parsons 1891). This pandemic has been referred to as the first flu pandemic that was “truly global in scope” (Patterson 1986), as it spread rapidly along the developing railway system, such that it was able to reach all areas of the world in only a few months (Patterson 1986, Valleron et al. 2010, Le Goff 2011). Additionally, “the near-instantaneous reporting of the outbreaks via the worldwide telegraphic network made the Russian influenza as much a media event as a disease event” (Honigsbaum 2010). Parsons (1891) attributes the penetration of the flu into the British countryside as being a result of the travel of holiday shoppers to and from London and from children returning home from boarding schools at Christmastime. Yet, the apparent simultaneity of the appearance of the Russian Flu in various parts of the world in 1890 often led physicians to affirm their belief in the miasmatic or atmospheric origin of influenza (BMJ 1890, Scholtz 1890, Parsons 1891).

The Russian flu is often overshadowed by the much more virulent Spanish flu of 1918. As a result, research on this flu is often limited to a cursory analysis that merely suggests that those exposed to the flu in 1890 may have had greater immunity in 1918. The literature has been increasing recently (for example, Honigsbaum 2010, Valleron et al. 2010, Le Goff 2011, Valtat et al. 2011, Humphries 2013) while the only study dedicated to the 1890 flu in Canada is focused exclusively on Hamilton, Ontario (Herring and Carraher 2011). Although the epidemic is less fully researched in Canada, it was known to be present for the winter of 1890, mostly in January. Patterson (1986) reports that there were cases in Montreal in December (for the 1890 influenza in Quebec, see Bilodeau Bertrand 2014); however, the Toronto Globe newspaper reported cases in the United States on December 31st 1889, but none yet in Canada (Hallman and Gagnon 2014). The Globe reported many cases in the first two weeks in January, yet there were almost none after mid-month. The pandemic peaked in Canada in mid-January (Maris 2011), yet “In Toronto, the highest numbers of deaths occurred between the end of January and mid-February, consistent with Patterson’s assertion that influenza deaths are normally first noticed to be elevated approximately four weeks after the start of an

epidemic” (Hallman and Gagnon 2014). As in Europe, the Russian flu is hypothesized to have spread throughout Canada and the United States along railway lines, accompanying commercial trade and private travel. Thompson (2011) found that the port of entry of the flu into Canada was either Halifax or the rail lines linking Ontario with New York and Chicago. All of these are major centers of trade and travel.

Influenza epidemics tend to come in waves, with the viruses circulating in less virulent forms in the years following the peak of their epidemic emergence and the Russian Flu was no different. Three smaller waves followed the outbreak in 1889-1890 and the disease generally disappeared after 1892 (Taubenberger and Morens 2006). However, Patterson (1986) reports a severe recurrence in Switzerland in 1893-4 as does Honigsbaum (2010) for the United Kingdom in 1893. Throughout this dissertation, the Russian flu pandemic will be referred to as the pandemic of 1890, since the worst period of the first wave of the pandemic in Ontario was mainly in January-February, 1890 (see Figure 2.1).⁸

The symptoms of the Russian flu were typical of other flu pandemics. Describing the situation in Cape Town, in the Cape Colony, Africa, Dr. William Scholtz reported in March of 1890 that

The character of the symptoms varies somewhat, but generally the muscular pains throughout the body are most marked, headache of a very severe and excruciating nature being the most prominent feature, also pain behind the eyes. Later on catarrhal symptoms set in; prostration in all cases very extreme . . . The symptoms generally are developed very suddenly. [Scholtz 1890:600].

Having had his first case in January of 1890, he emphatically notes that the point of entry was the port, bringing the flu to the Colony via trade with Europe (Scholtz 1890). Clemow describes how the flu took three main forms in Russia “(1) nervous form, with neuralgia and rheumatic pains; (2) with bronchitis, laryngitis, rhinitis, conjunctivitis, and fever – these symptoms last even when the fever falls; (3) gastric form, with indigestion, vomiting, lasts one to three days” (BMJ 1890:47). Scholtz, Clemow, and Parsons note that this epidemic had a high morbidity rate, but a low mortality rate. Looking at the

⁸ Likewise, since the second and most severe wave of the 1918-1920 Influenza pandemic is studied in this research, from September to December 1918, the pandemic will be referred to as the pandemic of 1918.

epidemic globally, Valleron and colleagues estimate that the pandemic had a clinical attack rate of 60% (interquartile range of 45-70%), with the case-fatality rate ranging from only 0.1% to 0.28% (2010:8778). Thus, while about 60% of a population would be expected to have contracted the illness, only 1 out of every 1,000 would have died (Patterson 1986, Morens and Fauci 2007).⁹ This means that many of those exposed in 1890 would have survived, and barring mortality from other causes during the ensuing 28 years, would have faced a similar flu pandemic in 1918. As with other flu pandemics, the 1890 flu had a ‘U’ shaped mortality curve, with the highest number of deaths being among the infants and the elderly (Crosby 1989, Dowdle 1999, Valtat et al. 2011).¹⁰ Patterson estimates that between 270,000 and 360,000 people died in Europe from the 1890 pandemic, with at least the same number dying in other parts of the world and in the following waves (Patterson 1986:72-73).

Although the exact subtype of the 1918 flu has been determined using autopsy tissues preserved since 1918 (A/H1N1, Basler et al. 2001, Taubenberger and Morens 2006), no such evidence remains of the 1890 flu. Seroarcheological evidence suggests that the virus was an influenza A-type virus, likely H3N8 (Dowdle 1999, Taubenberger et al. 2007, Valleron et al. 2010, Worobey et al. 2014), although it cannot be directly ascertained without biological remains.

The evidence that the 1890 Russian pandemic was in Canada can be found in the Reports of the Registrar-General relating to the Registration of Births, Marriages, and Deaths from this time period. The first mention comes from the report for the year 1890 (written in 1892), when it is stated that:

At this moment (Jan. 1892) as in Jan., 1890, as seen in the following remarks from the Registrar-General’s Report for England, “In every country from which returns were received the mortality in 1890 was

⁹ The population of Ontario in 1890 was 2,161,971 (Legislative Assembly of Ontario 1892). Applying these rates to Ontario, a 60% attack rate would mean that 1,297,183 individuals in the province may have contracted the disease in 1890. Of those, 1,297 would have been expected to have died from the pandemic.

¹⁰ However, Martel (2011) reports evidence of a ‘W’ shaped curve for mortality rates in Hamilton, Ontario between the ages of 25 and 54, for the pandemic period January 1 to May 31 1891. Similar results have been found in Montreal (Alain Gagnon, personal communication, December 9, 2013).

higher, and in most cases very considerably higher than it had been in the preceding year. The explanation of this universal increase is doubtless to be found in the very general prevalence throughout Europe of epidemic influenza,” when, through the prevalence of La Grippe with its accompanying pneumonias and bronchitis, the death-toll has doubled the ordinary mortality rates in certain parts of the Province from this class of malady; and when we behold its striking almost invariably at those who, having escaped the diseases of early life, and in so many cases the representative of so much value to the state as actual producers of wealth, we in some degree realize from the economic standpoint what an epidemic of death means. [Legislative Assembly of Ontario 1892:11]

While tuberculosis was usually the most common cause of death in Ontario, in 1890 this was replaced by pneumonia, “owing no doubt to the epidemic of influenza during the year” (Legislative Assembly of Ontario 1892:20).¹¹

The Annual Report of the Provincial Board of Health for the year 1890 sheds more light on the pandemic in Ontario. The Secretary, Peter H. Bryce reported that:

The disease presented most diverse phenomena, and almost every character marking it in other countries was illustrated in the epidemic in Ontario. Superadded to the influenza with its febrile and catarrhal manifestations were pneumonic complications of every variety; neuralgias, general and localized, dominated all classes in adults; while great nervous depression during the acute stage of the disease with extreme subsequent neurasthenia in very many instances were among its most marked characteristics. Gastro-enteric troubles occurred to a notable extent, and in some instances a temporary but marked jaundice was present. During the months which have followed the epidemic numerous instances have occurred where neurasthenias and pneumonic complications ending in phthisis, have had fatal terminations. [Legislative Assembly of Ontario 1891b:lii]

The Board of Health was certain beyond a doubt that the Russian epidemic was in Ontario (Figure 2.1). Their annual report for 1891 reported that the first case of the flu occurred on December 4th, 1889 and that by December 20th, 1889 it had become

¹¹ The most common causes of death for Ontario from 1882 to 1890 were: 1882 – phthisis (11.6% of total deaths); 1883 – phthisis (12.3% of total deaths); 1884 – phthisis (11.1% of total deaths); 1885 – phthisis (10.8% of total deaths); 1886 – phthisis (10.8% of total deaths); 1887 – old age (12.2% of total deaths); 1888 – pneumonia (10.7% of total deaths); 1889 – phthisis (10.3% of total deaths); and 1890 – pneumonia (14.7% of total deaths) (Legislative Assembly of Ontario 1892:38-39).

epidemic. By the close of February 1890, the worst of the epidemic was over in Ontario (Figure 2.1).

Figure 2.1 - The Russian Influenza Epidemic in Ontario.

NAME OF COLONY, CANADA, PROVINCE OF ONTARIO.	
<i>Questions of Local Government Board :</i>	
Has any epidemic "Influenza," particularly if characterized by much nervous depression, severe frontal headache, or various muscular pains, shown itself in your Colony ?	} Yes.
The date of the first occurrence (as far as you know) of such an Influenza.	} December 4th, 1889.
The date of commencement and decline of the epidemic prevalence of such Influenza.	} Probably the most nearly correct date for its epidemic appearance is December 20th, 1889. Its decline as an epidemic would be about the end of February, 1890.
Any evidence as to the mode of origin or introduction of the disease or any opinion on the subject held by the medical adviser of the Government and as to its method of spread.	} It was in the exact sense of the term a pandemic, and was undoubtedly infectious. Its zymotic character is assumed, and would seem to stand in the yet undetermined category in which pneumonia is placed by most physicians.
Has any unusual complaint been observed among domestic animals, and if so, in what animals, and with what symptoms ?	} From various observers it would seem that nothing appeared among animals contemporaneous with La Grippe in man.
Any observations recorded in the Colony as to the behaviour of the Influenza epidemic, especially as to its dissemination among particular communities and its incidence on particular localities.	} The disease was pandemic in Ontario, and prevailed very generally in Manitoba and the North-West Territories during a winter severe even for that climate; the mercury being for days together at from 15°-30° below 0° F.

Source: Report of the Provincial Board of Health, 1890 (Legislative Assembly of Ontario 1891b:lii)

The Annual Reports from the Local Boards of Health in the Various Municipalities of Ontario from 1890 confirm the report from the Provincial Board of Health. Influenza was present in Belleville, where it was the old, the infirm, and the tuberculous who were dying in greatest numbers (Legislative Assembly of Ontario 1891b:4). The same was true from Hamilton, where it was those of "weak constitutions and those predisposed to pulmonary complaints" who were at greatest risk (1891b:10). Further, the Medical Health Officer Isaac Ryall was convinced of the miasmatic nature of the disease, claiming

that “I am not aware that any special microbe has yet been discovered to perpetuate its specie, or that it comes under the head of contagious diseases, but that it is due rather to atmospheric causes producing catarrhal symptoms, accompanying by great debility” (1891b:10). The epidemic was present in London, Chatham, and Goderich, where it killed mainly the elderly (1891b:13, 28, 31) and in Ottawa, where the number of deaths was greatly increased in January and February (1891b:15). Stratford and Collingwood were visited, but had few deaths (1891b:17, 27), while the flu and its related effects were the principal causes of death in Brockville (1891b:26). The report from Galt was typical of many places in the province in that, while there was an epidemic of influenza that increased mortality during January and February, the rest of the year was relatively healthy compared to the previous years (1891b:30). The pandemic was reported throughout the towns and villages of Ontario.¹² The report by S. P Ford, the Medical Health Officer for the township of Norwood is characteristic of the Russian pandemic globally, in that “during the months of January and February, the epidemic of influenza that swept nearly the whole earth, prevailed in our midst, but though scarcely any one escaped an attack, only one case proved fatal” (1891b:67-68). The Provincial and many local Medical Health officers and reported links between infection with influenza and later deaths from pulmonary complications, especially tuberculosis (1891b).

As was typical throughout the world, the Russian Pandemic came to Ontario in subsequent waves in 1892 and 1894. When explaining the decrease in deaths in 1891 over 1890, this is again attributed to the Russian influenza pandemic. The Registrar-General, Richard Harcourt states that “it will be remembered that 1890 showed an increase of some 600 deaths over 1889, due presumably to the fatal epidemic of “*La*

¹² Specifically: Meaford, Orillia, Picton, Paris, Pembroke, Trenton, Wingham, Walkerton, West Toronto Junction, Arnprior, Arkona, Alvinston, Blenheim, Brussels, Bolton, Blythe, Chesley, Port Stanley, Southampton, Stirling, Sidney, Streetsville, Teeswater, Waterdown, Aldborough, Amherst Island, Arran, Barton, Bruce, Bayham, Bosanquet, Brooke, Beverley, Bentinck, Bexley, Clarke, Cardwell, Chaffey, Caledon, Caistor, Dereham, Dysart, Dumfries South, Darlington, Euphrasia, Glanford, Georgina, Hibbert, Hinchinbrooke, Harwich, Moore, Middleton, Mulmer, McKillop, Marysborough, Nepean, Nottawasaga, Oxford North, Osprey, Puslinch, Pilkington, Sarawak, Sullivan, Sundridge, Sherbrooke, Sydenham, Stamford, Sunnidale, Saugeen, Scott, Turnberry, Westminster, Wilmot, Wawanosh West, Woolwich, Williams East, and Wellesley.

grippe.” The lower prevalence of this disease in 1891 may serve to account in part for the decrease of nearly 1 per 1,000 for the whole population” (Legislative Assembly of Ontario 1893:5). With no other reference to influenza in the Registrar-General’s report, or in the report of the Provincial Board of Health (except that the flu was present in the township of Humberstone), it can be assumed that there was no noticeable resurgence of influenza in 1891. The Provincial Board of Health does not mention the flu in 1892, reports only one related death in St Catherines in 1893, and few deaths in 1894. However, the Registrar-General’s reports did show that the flu returned in 1892 and 1894.

Although describing an increase in the incidence of zymotic diseases in 1892, Richard Harcourt attributes much of this to the epidemic of *la grippe* in January and February of 1892. Further, he states that the increases in the local diseases were a result of the increases in pneumonia and bronchitis, seemingly aware that “both these increases are naturally associated with, and a result of the epidemic of *la grippe*” (Legislative Assembly of Ontario 1894:5). During 1892 there was also an increase in developmental diseases, but Harcourt also believes that this is due to deaths at older ages, once again caused by the flu epidemic.¹³

While the pandemic did return to Ontario in 1892, the deaths were not equally distributed across the province and mostly occurred in cities, such that deaths in cities numbered “595 in the total 1,562, or more than 1 in 3, whereas the city population to that of the Province is less than 1 in 5” (Legislative Assembly of Ontario 1894:5). This, again, was attributed to the flu, such that the epidemic of 1892 was mostly a problem of the cities. Comparing the deaths from influenza (not including pneumonia or bronchitis) in 1891 and 1892, the mortality rate in Ontario in 1891 was 0.2 deaths per 1,000 people, while in 1892 this had increased to 0.4 deaths per 1,000 people (an increase from 393 deaths in 1891 to 927 deaths in 1892). The corresponding numbers given for New York State in 1892 were 12,000 deaths at a rate of 2.7 per 1,000 people (Legislative Assembly of

¹³ The developmental diseases, Class IV, encompassed all those diseases which occur through the process or aging or at key points of development. This class was separated into three sub-classes: the Developmental Diseases of Children, the Diseases of Women, and Old Age (Legislative Assembly of Ontario 1883:43).

Ontario 1894:6). Similar data on influenza were not provided from the report for 1890. However in the entire province in 1890 there were 3,474 deaths from pneumonia, giving a ratio of 1.6 deaths per 1,000 people. This is compared to 2,286 deaths in 1889 with a ratio of 1.06 deaths per 1,000 people and 1,432 deaths in 1891 with a ratio of 0.7 deaths per 1,000 (Legislative Assembly of Ontario 1890:43, 1892:40, 1894:28).

The Russian pandemic returned to Ontario in 1894, as the Registrar-General noted that it had throughout the world (Legislative Assembly of Ontario 1896:12). The deaths from influenza in Ontario numbered 927 in 1892, 483 in 1893, and 613 in 1894. Worobey and colleagues (2014) assert that all waves of this pandemic were due to the same H3N8 influenza A strain. As was described in the Spanish Influenza epidemic in 1918, the Russian pandemic was reported to have an impact on the innervation of the heart, being a “disease of the nerve centres” (Legislative Assembly of Ontario 1896:12). It is therefore necessary to consider that deaths at older ages and at later time points can be due to the damage that occurred to the heart from influenza infection. The Registrar-General states that diseases of the nervous system were increasing in 1894, due to “the high pressure rate of living, as seen in our cities” but that some of these deaths would have resulted from earlier damage from influenza (1896:12).

2.2.2 *The Spanish Flu, 1918-1920*

The Spanish flu pandemic occurred in three major waves, Spring 1918, Fall 1918 and Winter/Spring of 1919, but continued to linger until at least 1920 (Crosby 1989). The second wave was the most virulent and accounted for the highest levels of mortality, with the first and third waves being relatively less severe. Although named the Spanish flu, this pandemic is generally thought to have originated either in a military camp in Kansas or on the battlefields of France (Oxford et al. 2002, Barry 2004, Humphries 2005).

The second wave of the global pandemic entered Canada through the Polish Military Camp at Niagara-on-the-Lake, near the border with New York State, in September 1918 (Humphries 2005, Bogaert 2012). From there, the flu spread along transport routes to

Hamilton and Toronto. The Siberian Expeditionary Force (SEF), travelling west to Vancouver, is thought to be responsible for transmitting the disease to the rest of Canada (Humphries 2005). The greatest number of deaths occurred in October, 1918, although deaths from influenza were elevated in November and December as well. The Medical Officer of Health for Toronto, Dr. Charles Hastings reported at the end of December that “the disease is present just as a forest fire smolders after the intensity of the blaze has died down. Every once in a while an area not previously invaded will flare up . . . The cases, however, are of a milder form compared with those of October. The mortality is not nearly so high in proportion” (The Globe 1918b:8, Hallman and Gagnon 2014).

The Spanish flu pandemic is the most virulent and frightening of known flu pandemics. Crosby relates that “nothing else – no infection, no war, no famine – has ever killed so many in so short a time” (1989:311), and the estimated 40 to 100 million deaths worldwide (with a global population of less than 2 billion, Johansen and Sornette 2001) far exceeds the 9 million deaths of the First World War (Patterson 1986, Johnson 2003, Quinn 2008, Storey 2009).¹⁴ It has been estimated that the flu infected up to one sixth of the population of Canada (estimated at 8,148,000, Statistics Canada 2009) and killed between 30,000 and 50,000 people (Dickin McGinnis 1981, Pettigrew 1983). The clinical attack rate of this pandemic ranged from 20% to 60% and had a case-fatality rate greater than 2.5% (Taubenberger and Morens 2006, Morens and Fauci 2007), such that the clinical attack rate in 1890 and 1918 was the same, but the 1918 flu had a much higher case-fatality rate.

Beyond the unprecedentedly high mortality rate, the most noted feature of the Spanish flu pandemic was the people who were targeted. Unlike all other influenza pandemics for which records exist, there was an unexpected increase in deaths among young adults, resulting in a ‘W’ shaped mortality pattern (Crosby 1989).¹⁵ The exact age range of

¹⁴ Hill (2011) believes 100 million global deaths in 1918 to be an overestimate due to miscalculation of excess mortality in India.

¹⁵ However, Francis (1953) reports of the 1782 epidemic in England that “young children and older people were less frequently affected than the middle group. Hamilton says: ‘I think the middle age felt it most . . . I mean from 16 to 45 or so.’ Smyth: ‘Children and old people escaped entirely or were affected in a slighter manner’” (1953:205-6). He also notes for the 1890 pandemic that “there was a definite excess of mortality in the 20 to 40 year age group” and a

‘young adult’ varies by author, but generally the ranges of 20-40, 15-35, 20-29, 30-39, and 25-29 are mentioned as having experienced the highest mortality rates (cf. Pettigrew 1983, Crosby 1989, Noymer and Garenne 2000, Humphries 2005, Taubenberger and Morens 2006, Ma et al. 2011, among others). Previous research tended to use age categories of either 5 years or 10 years in total. This dissertation focuses on the age-specific mortality rates in Canada, since prior explorations (Gagnon et al. 2013, Hallman and Gagnon 2014) show that five-year age groups mask a distinct peak in mortality at the ages of 28 and 30 for selected Canadian cities. However, what is clearly expressed in the category ‘young adult’ is that people who were not normally the victims of influenza were dying in much larger numbers than would be expected during a normal flu epidemic. These were individuals who were responsible for the well-being of their families, were often the primary wage-earners, were of reproductive age and, at a time when the life expectancy at birth was less than 60 (calculated from the mortality rates for Ontario for 1900-1942, McKinnon 1944), were often the most productive members of society. According to both contemporary and modern reports, pregnant women, especially those in their third trimester, were at an even greater risk of death, especially if they contracted pneumonia or if the virus caused a stillbirth or early labour (Oertel 1918, Winternitz et al. 1920, Beveridge 1977, Neuzil et al. 1998, and Reid 2005). This could be due to the normal “suppression of [maternal] immunity that allows retention of the foetus” (Reid 2001:220).

The Ontario-specific experience with the flu has been described for Hamilton by Herring (2005), Herring and Korol (2012), and Rankin (2012), for Toronto by MacDougall (2007), Slonim (2010) and Hallman (2009, 2012), for Kitchener by Johnson (1993), Marion and Scanlon for Kenora (2011) and generally by McCullough (1918), among others. The report of the Registrar-General of Ontario for 1918 explains that in terms of

“proportionately high attack rate among young adults” (1953:216). In Hamilton, Ontario, Martel (2011:55) reports an abnormally elevated death rate among adults aged 25 to 54 during the 1890 pandemic. There is also preliminary evidence that there was excess mortality among young adults throughout Quebec in 1890 (Alain Gagnon, personal communication, December 9, 2013). However, the more common assertion is that the ‘W’ shaped excess mortality pattern was unique to the Spanish flu.

influenza “nothing of the kind ever happened in the history of the province” (1919a:5). and that there were 7,337 deaths from influenza in 1918 and 4,660 deaths from pneumonia. Through calculating that the average deaths per year from pneumonia for the ten previous years was 2,015, he estimates that approximately half of the 4,660 deaths from pneumonia were likely pandemic-related (2,330). Added to the deaths from pneumonia, this gives 9,667 pandemic related deaths or, 3.45 deaths per 1,000 people (1919a:5). Looking only at deaths from influenza, “47.1 per cent., occurred in the cities; 10.5 per cent. in the towns, and 42.4 per cent in the rural municipalities. The cities suffered 47.1 per cent. of the deaths from influenza, whereas they contain collectively but 38.7 per cent. of the total populations” (1919a:5). Likewise, 42.4% of the deaths occurred in the rural areas, but they contained 56% of the population. This is similar to the Ontario experience with the 1890 flu and was explained due to crowding in the cities.

The report of the Provincial Board of Public Health shows that by 1918 the germ theory of infection was more popular than miasmatic theories. In the report for District 3 (including the counties of Norfolk, Haldimand, Welland, Lincoln, Wentworth, Brant, Halton, Peel and York), the District Officer of Health for Hamilton, D. A. McClenahan reports that influenza is “passed from one another by contact, very often by droplet infection” (1919b:25) and that the disease only abated once all those not immune had been infected. A consistent feature from the various district officers was that young people were particularly affected. But, deaths were more numerous in the cities and the death rate varied across the province. It was reported that there were no deaths in the town of Morrisburg, while there were other villages in which 10 per cent of population died. Cornwall and Renfrew were reported to have a death rate of one per cent (1919b:21). Similarly in Guelph Township, the Medical Officer of Health stated that “while the more populous centres were sorely stricken, and a great many deaths recorded, we cannot say that any deaths in the township, outside of St. Joseph’s Hospital, were directly due to it” (1919b:191). The report from Brantford gives a conservative estimate of 35% of the population being infected, and that 50% of the deaths occurred among those aged 21-40 (1919b:133).

Although there was a first wave of the pandemic throughout the world in the spring of 1918, it was not mentioned in the reports of the local boards of health for Ontario. There is evidence of this wave in the province from analysis of death records (Korol 2011, Rewegan et al. 2015), yet it was not of sufficient increase over the expected seasonal influenza to attract the notice of contemporary public health officials. As was similarly reported from many cities in Ontario, the first appearance in London was determined to have occurred on September 22nd, 1918, with the crest of the epidemic being reached around October 18th (Legislative Assembly of 1919b:153). There were 187 deaths reported in the city, with 68% occurring between the ages of 20 and 50 (1919b:154). Niagara Falls reported 3,000 cases of flu, and 70 deaths, for a death rate of 1-3% of the population (1919b:157).

2.3 Social Conditions and Illness

In the epidemiological and the sociology of health literature, the relationship between poverty and illness is well established and is known to have had historical precedents (Blum et al. 1990, Lynch and Kaplan 2000, Susser and Stein 2009). As summarized for HIV by Singer and Baer (1990), “poverty contributes to poor nutrition and susceptibility to infection. Poor nutrition, chronic stress, and prior disease contribute to a compromised immune system, increasing susceptibility to new infection” (1990:213). Link and Phelan (1995) describe social conditions as “fundamental causes” of disease. In this framework, underlying social conditions shape an individual’s level of access to resources, level of stress in their occupation and location of residence, social stigma, and ability to utilize health care opportunities. These distal causes influence the proximate causes of health, or those specific causes that are most often focused on when studying morbidity and mortality, such as diabetes, heart disease, tuberculosis, and cancer. The fundamental causes of disease help to explain why there is a relationship between socioeconomic position and illness, even though the direct cause of disease may be eradicated or alleviated through new biomedical interventions (Link and Phelan 1995). However, there may be fewer links between socioeconomic status and mortality for non-preventable causes of death (such as influenza) and in Canadian as opposed to American contexts

(due to the current sociopolitical climate in Canada [Willson 2009]). However, this may not be the case in the historical Ontario context.

Fundamental cause theory was developed to explain the systematic socioeconomic *gradient* that exists between social conditions and mortality (Link and Phelan 1995, Phelan et al. 2004). While shown to exist in many places in modern society, this phenomenon is exemplified in the Whitehall studies of Michael Marmot (1993, 2004). These studies examined two cohorts of male British civil servants who had a rigid social hierarchy in job restriction, requirements, level of control and freedoms within their jobs, and pay. Except for those in the highest levels and those in the lowest, all of these men were in the same socioeconomic strata in the greater British society and would have been categorized as middle class. Despite this, there was a pronounced socioeconomic gradient in the risk of death from cardiovascular disease, whereby those in the lowest level jobs were at the highest risk (Marmot 1993, 2004). Current research aims to explore the mechanisms behind this relationship, including biological, sociological, and the biosocial and psychosocial ways in which these two mechanisms interact (Barker 1992, O'Rand 1995, Seeman et al. 1997, Ben-Shlomo and Kuh 2002, Dannefer 2003, O'Rand and Hamil-Luker 2005, Ross and Mirowsky 2005, Zarit et al. 2005, Lynch 2008). However, what is still being debated is at what point in time the socioeconomic gradient emerged (Bengtsson and van Poppel 2011).

Historically, differences in health were thought to be the result of varying occupational exposures in diverse social groups. For example, labourers, physicians, artisans, and seamstresses all had differing health risks due to their dissimilar occupational and environmental stresses (Blum et al. 2004). It is thought that those in the highest socioeconomic strata were not protected in the same way as they are today, since medical knowledge was not advanced enough to give effective protective strategies or chemotherapies until well into the twentieth century (Razzell and Spence 2006). However, the relationship between malnutrition, immunodeficiency and infectious disease is unequivocal (Singer and Baer, 1990, Kaslow and Evans 1997, Katona and Katona-Apte 2008), and unequal access to sufficient amounts of nutritional foods has

been a constant distinction between social groups for centuries.¹⁶ The existence of this gradient in 1918 and how much this applies to virulent infectious disease is still being debated, including whether or not the war-induced food restrictions imposed on the Ontario population affected all people equally, or whether they were enough to counteract life-time exposures to inequality. However, it is reasonable to hypothesize that socio-cultural differences may have played a role in the distribution of mortality in Toronto in 1918. This was not a new finding for the province. For 1901, Mercier found that “it did not matter whether the measure of socio-cultural difference was the family’s ancestry, their religious affiliation, or the nativity of the parents – in each case there was a significant association between sociocultural affiliation and infant and child mortality” (2006:136).

There are many pathways through which poverty can lead to increased mortality, such as through population density and poor nutrition. More individuals in a household can lead to a sharing of resources which leads to lower levels of nutrition. Sanitation can also be an issue with overcrowded households. Areas that suffer from the fundamental cause of marginalization, either from income, occupation, religion, or immigration status tend to have many of the qualities that would place them at higher risk: overcrowded living and working conditions (an especially important factor during an airborne infectious disease pandemic), social exclusion leading to less secure or sanitary homes and work environments, and less easy access to social resources and sufficient, if not nutritious, diets (Link and Phelan 1995). For Toronto in 1921, Piva relates the main options that families had to decrease their expenses. These included restricting family size, increasing the number of boarders who paid rent, adding secondary or tertiary wage earners, and also “to rent a house which lacked proper sanitary facilities” (1979:39). Although this could save money, it also meant that “hazards to health would also be increased. And given the financial situation of most families, illness would have a catastrophic effect” (1979:39).

¹⁶ This issue is debated. For example, the paradoxically high infant mortality among nobility who sent their infants to wet nurses (Gadoury 1992, Bengtsson and van Poppel 2011).

The number of individuals living in a household can have an influence on health and mortality through combined biological and social mechanisms. Having more individuals in a household (where only the head is working), can cause resources to be shared to a greater degree than in smaller houses, which can affect the nutritional status of the individuals (although infants appear to be protected from economic stresses, Oris et al. 2004). Nutrition affects the possible immune response when confronted with an infectious disease. Larger households in urban areas may also represent crowded living spaces. Speaking of contemporary reports from Europe in the period after 1850, Taylor claims that “abundant evidence shows that synergism between malnutrition and common infections normally occurred most frequently among the poor” (1983:287). Rotberg and Rabb detail the biological mechanisms whereby:

Nutritional deficiencies are capable of reducing resistance to infection and thereby increasing the prevalence and severity of many infections through a variety of mechanisms including:

1. Reduced production of humoral antibodies
2. Impaired cell-mediated immunity
3. Less effective phagocytosis
4. Weakened epithelial barriers
5. Lower lysozyme production
6. Various other non-specific effects. [1983:307]

They then list numerous infectious diseases by the impact that nutritional status has on the morbidity and mortality risk posed by infection. They categorize nutrition as having an “equivocal or variable” effect on morbidity or mortality from influenza; however, tuberculosis and “most respiratory infections” are categorized as definitely being affected by nutritional status (Rotberg and Rabb 1983:308). Infection with any disease can have the aforementioned detrimental impact on the immune system, such that if an individual was sick with any other disease at the time of the influenza pandemic, would have made them additionally vulnerable. Airborne infectious diseases spread more quickly in crowded environments (Glezen and Couch 1997). The problem of poverty in the form of lowered nutrition and crowded environments is therefore hypothesized to be a means through which socio-economic status may have influenced mortality during the 1918 influenza pandemic (Chapter 6).

There continues to be a debate in the influenza literature about socioeconomic stratification in risk of exposure and risk of death, often resulting from inadequate relevant information on death certificates. The current state of the argument will be explored in the following section

2.3.1 *Social Conditions and Influenza*

It becomes increasingly difficult, in view of accumulated knowledge, to accept the conclusion that war conditions were the deciding influence. Although the disease was marked in military forces, it was also severe in areas little involved in the hostilities. The dislocations and rapid mixing were much greater in World War II than in 1918. Moreover, influenza has not been historically a war disease, like typhus, jaundice and others.” [Francis 1953:213]

Influenza has often been labeled a “democratic” disease, as it has been argued that individuals of all socioeconomic strata were equally likely to contract and die from the disease (Crosby 1989, Tomkins 1992, Barrett and Brown 2008, and as discussed in Mamelund 2006, Hallman 2009, Herring 2009, Herring and Korol 2012, among others). Since pandemics are thought to be created by novel mutations of the virus, all individuals in the population would be at risk due to lack of immunological experience with the new form of influenza (Herring and Korol 2012). This view obscures what is known both about the relationship between socioeconomic groups and infection in general as well as the relationship between influenza and inequality specifically for the 1918 flu. There are many reasons to suspect a relationship between inequality and risk of death from influenza infection and many associations have been noted. It was reported that for morbidity “perhaps no observation during the 1918-1919 was more common than the familiar comment that ‘the flu hit the rich and the poor alike’” (Sydenstricker 1931:154). It had been noted for both the 1890 and 1918 pandemics that death rates were higher in the cities and at both time periods the Registrar-General for Ontario claimed that this was due to the crowding in urban areas. At the height of the epidemic in Toronto, on October 10th, 1918, Dr. Charles Hastings stated that the disease was “spreading most rapidly

among the poor” (The Globe 1918a:6). The Medical Officer of Health for Galt (now Cambridge), J. J. Radford, stated that “the death rate has been greatly increased by the following facts: 1st. – By living in overcrowded and poorly ventilated premises, 2nd. – By being overworked and poorly nourished on account of the high cost of the essentials of life” (Legislative Assembly of Ontario 1919b:146). Through an analysis of surveys of the United States Public Health Service of nine urban centers of over 25,000 people, Sydenstricker found that during the 1918 influenza epidemic in Maryland, USA, “there were marked and consistent differences in its incidence – with respect to both morbidity and to mortality - among persons of different economic status . . . Apparently the lower the economic level the higher was the attack rate. This relationship was found to persist even after allowance had been made for the influence of factors of color, sex, and age, and certain other conditions” (1931:155). Further, there was a clear and progressive gradient in case-fatality rates leading from the ‘very poor’ who were most at risk to the ‘well-to-do’ who were least at risk. The Chief Officer of Health for Ontario claimed that “the mortality is shown to have been the greatest, as might be expected, in crowded and insanitary districts” and that “an underfed people will continue to be more liable to outbreaks of this nature” (McCullough 1920:43-44). Yet, Sydenstricker reports that, while looking at household size as an indicator of crowding conditions, he found that the relationship does not persist when economic status is controlled, such that “household congestion, although a concomitant of poverty, is not per se the determining factor in establishing the association of economic status and influenza in 1918” (1931:166).

Many more modern analyses have come to the same conclusion. Influenza in 1918 is now thought to have affected the poor the hardest, and this has been found in Norway, the United Kingdom, India, and Canada (Smith 1995, Phillips and Killingray 2003, Ramanna 2003, Mamelund 2006, Herring and Korol 2012, and suggested for Toronto by Slonim 2010). Of the 1890 pandemic in the United Kingdom, Smith states that “overcrowding is now recognized as concentrating the virus and multiplying cross-infections” (Smith 1995:56). Phillips and Killingray (2003) agreed that poverty and insanitary conditions would put individuals at a higher risk of both contracting and dying from influenza in 1918, though they remark that this would have varied in terms of degree in different localities (2003:9). They assert that “certainly the poor were rendered more vulnerable

due to low levels of nutrition and poor physical health” (2003:9). Ramanna reported the same in Ahmedabad, India for 1918 where the Health Officer “found high mortality among the low castes, who were both ‘poor and under privileged’” (2003:89). In neutral Norway, apartment size as an indicator of wealth had an impact on mortality during the 1918 epidemic (Mamelund 2006) and the mortality rate in Hamilton, Ontario was higher in the more impoverished and overcrowded areas of the city (Herring and Korol 2012). Herring and Korol argue that while the cases of the flu may have been more democratic (especially if young adults were antigenically primed to produce antibodies to a different strain of the flu), those people who died were disproportionately those who lived in working-class and immigrant areas of the city. Herring and Korol (2012) describe this sentiment more immunologically, in that “everyone is theoretically vulnerable because no one has antigens that confer resistance to the new pathogen” (2012:97). Evidence thus far suggests that there were socioeconomic differences in risk of death in 1918 and Sydenstricker’s (1931) research shows that the gradient was in existence in North America by 1918. Chapter 6 examines differences in socioeconomic groups in Ontario in 1918, through looking at both the declared occupation on the death record as well as at socioeconomic conditions over the lifecourse.

It is known from modern-day studies that poor nutrition and restricted access to resources can result in worse health outcomes in adults (Ben-Shlomo and Kuh 2002). Further, through processes of cumulative-disadvantage, it has been shown that low socioeconomic status in childhood can place children on trajectories that can result in poor educational attainment and poor occupational opportunities and thus low income. This specific trajectory then goes from low socioeconomic status as children directly to low socioeconomic status as adults (O’Rand 1995, Dannefer 2003, O’Rand and Hamil-Luker 2005). Being on the trajectory that exposes an individual to low socioeconomic status throughout the lifecourse can result in a higher risk of both morbidity and mortality in adulthood (Kosteniuk and Dickinson 2003). Fundamental to these theories is that they pertain to the same individuals, or a particular cohort experiencing the same epidemic conditions. Since influenza infection was known to be widespread in January 1890 and that exposure *in utero* can cause lasting effects, date of birth will be used as a proxy to

exposure, both pre- and post-natally and socio-economic status will be evaluated in both 1901 and 1911.

Finally, it was not merely that the poor were most likely to get sick and to die; the surviving family members were also disadvantaged. As Jones explains for Winnipeg, in 1918 the consequences of a woman losing her husband, who was likely the sole or primary wage-earner, were different for those in the poorer classes than for those in the upper classes. Those who were living closer to the poverty line would have suffered disproportionately from the loss of income and savings that could occur following a death. If this death occurred before the end of the pandemic, it would place all the remaining family members at an increased risk of death from the disease through reduced access to resources (in addition to risk of direct infection).

2.4 Specific Influenza Hypotheses

The exact cause of the unusual young adult mortality during the 1918 pandemic remains the ‘missing piece’ in understanding the puzzle of the Spanish Flu (Morens and Taubenberger 2012). Authors have posited theories that could possibly explain it, but no consensus has yet been reached as to the ultimate cause of this phenomenon. There are the somewhat vague comments that it spread quickly among soldiers, cramped together in insanitary conditions and that pregnant women were at greatest risk, but this cannot account for the increased deaths in non-belligerent regions and among non-pregnant women (Glezen 1996). The literature suggests that it was not only the young who were dying, but also the strong (CMAJ 1918c, Crosby 1989), a counterintuitive finding that has challenged researchers. What could possibly occur so that “the severe type affects with predilection young, strong, and plethoric individuals, especially men. In them the prognosis is most unfavourable and the disease rapid” (CMAJ 1918c:1028)?

One popular hypothesis is Burnet’s concept of the overactive immune system (the cytokine storm, Morens and Fauci 2007), first described in the 1960s (Crosby 1989). This argument is based on the common autopsy finding during the pandemic of a

“widespread and edematous process in the lungs” (Crosby 1989:21). In other words, the lungs were often filled with a “thin bloody liquid” (Kolata 1999:4), such that those afflicted would drown in their own bodily fluids. The immune system in these individuals would overreact, causing severe inflammation and an overabundance of immune cells in the lungs, causing “severe tissue damage, disease and death” (Loo and Gale 2007:268). As this is the result of an overreaction of the immune system, it follows that those with the most robust immune system would be most at risk: not only the young, but the strongest of the young. However, some authors have proposed that it is not the strongest who were at greatest risk, but those already suffering from an airborne infectious disease.

For instance, Andrew Noymer and Michel Garenne (Noymer and Garenne 2000, Noymer 2009) suggest that the young adult mortality resulted from co-infection with influenza and tuberculosis. Noting that there were more deaths among men in 1918 in the United States, and that young men were at the greatest risk of contracting tuberculosis, they posit that those who died from the pandemic were those who were already sick. The decline in tuberculosis death rates after the 1918 flu pandemic can thus be explained in that those who would have died post-1918 from tuberculosis had already died from influenza, decreasing the amount of susceptible individuals in the population at risk of dying from tuberculosis. This hypothesis has been questioned by Sawchuk (2009) using data from Gibraltar; however, Noymer (2010) counters that Gibraltar does not have a sufficiently sized population to see the large-scale effects that occurred in the United States. Fundamental to this hypothesis is the finding that more young men died than young women during the 1918 flu, and while this was often reported, it was simply not consistent globally (cf. Johnson 2003, Ramanna 2003, Zylberman 2003).¹⁷ Further,

¹⁷ Using 10-year age categories and a chi-square test, Slonim found for the pandemic for Toronto that “there is no difference in the age-distribution of influenza deaths by sex” (2010:87). Although there were more deaths of men in the city, this was not the case when deaths of soldiers were removed (the deaths of those soldiers who died at Exhibition Camp in Toronto being included in the registry for Toronto, many of whom were not from the city). Comparing the number of deaths of each sex to the population at risk for the city as a whole (as estimated from the 1912 census [sic]), *the death rate for both men and women was 3.7 deaths per 1,000 population* (Slonim 2010:88).

tuberculosis in Ontario had a long-standing history of being a disease that had more deaths among women than men.¹⁸

The final hypothesis that is often proposed is that of the immunological benefits of previous exposure. The focus of this hypothesis is that mortality of those over 40 was less than that of those under 40 (the higher mortality of the elderly resulting from their weakened immune system generally). The explanation is that those over 40 must have been exposed to a similar influenza strain in their youth that gave them protective immunity in 1918, something that those under 40 were too young to have experienced. The most common pandemics cited are 1830, 1847, and 1889 (Morens and Fauci 2007), although, due to the lack of evidence, the incongruous timing of these pandemics, and little to no immunological identification of 1800s influenza strains, this argument is less well supported than others (Taubenberger and Morens, 2006, Morens and Fauci 2007). However, recent seroarchaeological evidence suggests that those born before 1880 and after 1900 may have had exposure early in life to an endemic form of the H1 protein that may have offered immunological protection in 1918 (Worobey et al. 2014, see also Shanks and Brundage 2012 and Gagnon et al. 2015).

Even the type of infection that needs to be explained has not been agreed upon. Since most deaths resulted from secondary bacterial infections, Francis argued in 1953 that:

There is no doubt that bacterial complications were important, but what organisms selectively attack the 20 to 40 year age group? It is not characteristic of pneumococcal infection. *Hemophilus influenzae* pneumonitis may occur at any age but it is most commonly encountered in young children. There is no evidence that hemolytic streptococcal pneumonia concentrated in adults of middle age. The significance of *Staphylococcus aureus* has probably been underestimated but, again, there is no indication that it has a predilection

¹⁸ The 1882 Report of the Registrar-General states that “females are more subject to Phthisis than males in nearly every period of life” (Legislative Assembly of Ontario 1884:41). In 1883, “258 more females succumbed than males succumbed to its effects” (1885:45). Summarily, in 1886, it was reported that “the mortality from Phthisis has always been greater among females, varying in different years” (1887b:45). In 1889, it was reported that “in the returns from the Province a much larger number of females than males were victims of Consumption, especially in the periods of life from 15 to 20 years; 20 to 30 years, and 30 to 40 years. In the cities the mortality from this cause was greater among the males than the females, in the two periods from 15 to 20, and 20 to 30 years” (1889:46). By 1918, female deaths outnumbered male deaths only in the towns, by a ratio of 116 to 100 (1919a)

for persons of 25 to 40 years . . . The conclusion is, then, that influenza virus was the decisive factor of 1918, producing a severe primary damage to the respiratory epithelium and creating a medium in which various bacterial agents could become implanted and produce the multiform pictures which were actually observed. This was predicted from the clinical and pathologic observations by many students of the 1918 epidemic at the time, and I am ready to believe that injury inflicted by the virus itself caused a goodly proportion of the fatal illnesses, to which bacteria were incidental. [Francis 1953:213]

But, neither is a predilection for young adults common for influenza (Glezen 1996). Crosby suggests that in asking what causes pneumonia to attack the young rather than influenza “we haven’t moved one step closer to the Prime Mover Unmoved but one step further away” (1989:218), since it now must be asked what caused the virulence of pneumonia to change so drastically from the forms previously circulating in society (however, the intensification of one illness contingent upon infection with another is similar to theories posited for tuberculosis and influenza, Noymer and Garenne 2001, Noymer 2009, and Shope found similar evidence for influenza and pneumonia in pigs in the 1930s, Crosby 1989). Whether to focus on influenza, pneumonia, or their interactive effects remains a topic for discussion in the study of the 1918 flu and was part of the recent debate in the literature between Shanks and Brundage (2012) and Morens and Taubenberger (2012), with Morens and Taubenberger arguing that “pathogenesis theories of severe or fatal 1918 influenza must account for why the 1918 virus predisposed more persons to secondary bacterial pneumonia, and also look beyond the virus to address bacterial cofactors” (2012:332). However, as of yet, no response adequately explains why young adults may have been at greater risk of contracting influenza and of dying in higher numbers from pneumonia.

Most hypotheses that account for the unusual ‘W’ shaped mortality curve in the 1918 flu attempt to explain why those over age 40 (excluding the elderly) did not appear to have as high excess mortality as younger adults. However, few hypotheses focus specifically on why young adults were at such a higher risk of death, in contrast to previous epidemics. Two theories that do focus on young adult mortality specifically (tuberculosis and influenza comorbidity, Noymer and Garenne 2000 and Noymer 2009, and the overactive immune response, Loo and Gale 2007) can explain why young adult mortality

was higher in general. What they cannot account for is a distinct peak at age 28 or 30 (Hallman and Gagnon 2014). For example, can it be sufficiently argued that, *ceteris paribus*, an individual at age 28 has a stronger immune system than someone at age 27? Similarly, if tuberculosis infection is a requisite precursor to influenza infection, but those aged 28 were at highest risk, what would cause a group of individuals to either all obtain tuberculosis at the same age, or for only those aged 28 to be at higher risk of influenza out of all those currently infected with tuberculosis? Since we examined death records for Ontario for the worst period of the influenza epidemic (September to December, 1918) and demonstrated a distinct peak at the ages of 28 and 30, I will discuss three different hypothetical mechanisms that may account for this phenomenon: 1) gestational growth restrictions (Section 2.4.1); 2) scarring (Section 2.4.2); and 3) antigenic imprinting (original antigenic sin) (Section 2.4.3).

2.4.1 *Fetal Growth Restrictions*

It has been scientifically accepted that the gestational environment to which a fetus is exposed can have direct effects on later, post-natal life. The effects of teratogens on development, such as alcohol or thalidomide, and the lack of folic acid intake implicated in the formation of neural tube defects are part of the public consciousness due in large part to effective public health campaigns (Boyle and Cordero 2005). Barker (1992, 2006) has posited that there is a relationship between low birth weight and coronary heart disease, stroke, diabetes, and hypertension. The mechanism behind this is developmental plasticity during critical periods of development (Barker 2006). Essentially, maternal nutritional stress signals to the developing fetus that it is being born in a period of dearth and consequently, “the baby responds to these signals by adaptations, such as reduced body size and altered metabolism, which help it to survive a shortage of food after birth” (Barker 2006:272). These infants are at higher risk for cardiovascular diseases in adulthood when the environmental conditions of early childhood do not match those for which their phenotype is most well suited. For example, when an infant with an altered metabolism best suited to famine conditions grows up in conditions where calories are amply available. Barker (2006) believes that there are three main processes that are

responsible for the higher risk of disease. Lower birth weight babies have “less functional capacity in key organs” (2006:273), insulin resistance and less adaptive responses to stress, and a higher vulnerability to environmental conditions. Birth weight is a gradient, with no particular ‘low birth weight’ threshold known to exist.¹⁹

Not only can low birth weight lead to a higher susceptibility to degenerative diseases, but it can also increase the risk of dying from an infectious disease later in life. Studying Gambian infants born during periods of food shortages, Moore and colleagues found that low birth weight led to greater risk of death from infectious diseases in young adulthood (between the ages of 14.7 and 47, Moore et al. 1999), such that: “intrauterine growth retardation . . . slows cell division during sensitive periods in the ontogeny of the immune system. This would provide a mechanism by which early insults could be ‘hard-wired’ such that they had a permanent impact” (Moore et al. 1999: 1093). Additionally, maternal malnutrition can result in underdevelopment of the fetal thymus and lymphoid-system, increasing the risk of auto-immune diseases. However, because the flu was of such short duration in Canada (limited mainly to January 1890), and because the clinical attack rate was so high (60%, interquartile range 45% to 70%, Valleron et al. 2010), being *in utero* or alive on January 15th, 1890 will be used as a proxy for exposure. These results have not been reproduced in other regions (Lawlor 2004); however, similar conclusions were reached by McDade and colleagues (2001) studying thymic production of Filipino adolescents who were of low and normal gestational weight for age. Those who had low birth weight had reduced thymic function, the result of which is reduced immunocompetency in adulthood. Thus, individuals who experienced maternal stress while *in utero* were found to be at higher risk for diseases in later life, including those from acute airborne infectious diseases.

Pregnancy, especially further along in development, was a dangerous state for both a mother and a fetus during the 1918 influenza pandemic. In 1920, Winternitz et al. explained that “although in the non-complicated cases of influenza, pregnancy does not influence the course of the disease, if pneumonia supervenes, the mortality for the

¹⁹ Rasmussen (2001) cautions against using birth weight as a proxy for developmental growth restrictions as not all factors resulting in developmental impairments may impact birth weight.

mother, as well as for the child, is definitely increased” and posited that “the hemorrhagic lesion of influenza seems a plausible explanation for the frequency of abortions” (1920:39). A study of English mothers in 1918 found that influenza infection in the first and second trimester could lead to premature birth, a risk to both the mother and the infant (Reid 2005). For those infants who survived gestation, maternal infection with influenza has been known to have deleterious effects on the later-life health of their infants. Brown and colleagues (2004) conducted a case-control study of a cohort born between 1959 and 1966 in California who were diagnosed with schizophrenia in later life. They found that “the risk of schizophrenia was increased 7-fold for influenza exposure during the first trimester. There was no increased risk of schizophrenia with influenza during the second or third trimester” (Brown et al. 2004:774). To explain the mechanism behind this association, they state that since influenza does not usually cross the placenta (although it is possible), there must be indirect mechanisms affecting the fetal brain. These mechanisms could be maternal antibodies or cytokines that can damage the developing brain tissue, hyperthermia, fetal hypoxia, or harmful remedies taken to combat the flu (Brown et al. 2004:778). Studies linking maternal influenza exposure and psychiatric and neural conditions in later life are numerous (for example, Mattock et al. 1988, Adams et al. 1993, Takei et al. 1994). There are also studies linking maternal influenza exposure to the occurrence of childhood neoplasia (Leck and Steward 1972) and to mental retardation (Takei et al. 1995).

Similarly, using census returns from 1960 to 1980, Almond (2006) studied cohorts born in the United States immediately after the pandemic (1919 and 1920), who were thus *in utero* during the worst of the epidemic. He found that “cohorts *in utero* during the pandemic displayed reduced educational attainment, increased rates of physical disability, lower income, lower socioeconomic status, and higher transfer payments compared with other birth cohorts” (2006:672). The mechanism that he believes may be responsible is the fetal origins hypothesis (this has been challenged by Bengtsson and Helgertz (2013) who argue that the socioeconomic gradient resulted from differential enlistment in the First World War). Similarly, using retrospective cohort studies in the United States, Mazumder and colleagues (2010) found an elevated risk of heart disease for cohorts exposed to the 1918 influenza pandemic *in utero*. Using a retrospective cohort

survey, Almond and Mazumder (2005) found more evidence of long-term physiological effects of *in utero* influenza exposure. They found that being *in utero* at the height of the pandemic had a negative effect on the self-reported health of adults over 50, as well as increased the risk of developing cancer, diabetes, heart disease, kidney problems, hypertension, and stomach problems. This was also explained using the fetal origins hypothesis. Additionally, Myrskylä et al. (2013) found that such exposure in the last trimester of pregnancy increased risks for cardiovascular disease, positing a mechanism whereby resources are diverted to the maternal immune response at the detriment of fetal maturation.

However, the findings concerning the long-term sequelae of influenza have been mixed. Cohen et al. (2010) analyzed aggregated data from 24 countries including Canada and found no consistent effect of neonatal or perinatal exposure to the 1918 influenza and later life mortality differentials, although the only birth related data available was at the yearly level. A more fine-tuned, country specific, analysis may show results more consistent with the literature.

In this dissertation, I posit that individuals who were *in utero* during the 1890 influenza pandemic may have experienced fetal growth restrictions that placed them at higher risk of contracting the 1918 Spanish flu. As influenza was not a reportable disease in Canada in either 1890 or 1918 (Zhang et al. 2010), it is not possible to directly assess whether certain individuals' mothers had contracted influenza in 1890. However, because the flu was of such short duration in Canada (limited mainly to January 1890), and because the clinical attack rate was so high (60%, interquartile range 45% to 70%, Valleron et al. 2010), being *in utero* or alive on January 15th, 1890 will be used as a proxy for exposure. As date of birth is known from records linkage to birth records, and it will be assumed that all infants who survived until 1918 were full-term (Toronto, and likely the rest of Ontario, had a high neonatal death rate in 1918, Hallman 2009), the age (or gestational age) of exposure to the 1890 flu can be known with precision at the monthly level.

2.4.2 *Scarring*

The concept of scarring was used in a study by Preston, Hill, and Drevenstedt (1998) that examines the relationship between early life conditions and survival to ages over 85 in a population of African Americans. Arguing that their sample of decedents over age 85 was representative of all those alive over that age, they use records linkage from death records to the 1900 and 1910 US censuses to determine socioeconomic characteristics in childhood. They posit four possible ways through which childhood conditions could be related to adult mortality: positive (childhood infection leads to higher mortality) and negative (childhood infection leads to lower mortality), both with direct and indirect possibilities. Their research supports the direct effects, the scarring (positive) and acquired immunity (negative) hypotheses, but not the argument concerning indirect effects (however, their sample necessarily precludes those who died before the age of 85). Acquired immunity may explain why older adult mortality was low, but does not adequately describe why young adult mortality was so high, only in 1918.

Scarring offers a plausible explanation. Preston and colleagues state that “certain conditions and diseases acquired in childhood may, in a sense, permanently impair the survivors and leave an imprint on death rates at all subsequent ages,” using the examples of tuberculosis, hepatitis B and rheumatic heart disease (1998:1232). With historical data from Sweden, Bengtsson and Lindström (2003) found that “airborne infectious diseases are important for the causal mechanisms linking infant mortality to old-age (55-80 years) mortality” (2003:293) such that “children severely exposed to airborne infectious diseases during their birth year had a much higher risk of dying of airborne infectious diseases in their old age” (2003:286). The mechanism through which the relationship between exposure to severe respiratory illness in childhood and higher mortality in later life actually occurs is less well developed. Bengtsson and Lindström (2003) argue that there is no medical evidence for the suggestion by Fridlitzius (1989) that “diseases during the first years of life would affect survival during adulthood through an irreversible damaging effect on the immunological system” (2003:292) (however, *c.f.* Section 2.4.3). Yet, they report that respiratory infections in the first year of life have been shown to be associated with morbidity and mortality from “cough, phlegm, and impaired ventilator

function” as well as “early bronchitis and pneumonia” providing “evidence of persistent lung damage from respiratory infectious disease during the first year of life” (2003:292). Therefore it is assumed in this dissertation for the scarring mechanism that infection early in life from a severe respiratory infectious disease (the 1890 influenza) causes physiological damage which impairs survival from another severe respiratory infection in later life.

Infection with influenza can lead to lasting physiological impairments. As previously mentioned, the 1890 Russian influenza was thought to impact the innervation of the heart, meaning that the flu left those who had survived it with nerve damage to their essential organs (Legislative Assembly of Ontario 1896, Horder 1918). This nerve damage may also lead to encephalopathy and encephalitis, and manic psychosis (Glezen and Couch 1997, Honigsbaum 2010). Epidemics of encephalitis lethargica and Parkinson’s disease have been proposed as being sequelae of influenza infection (Ravenholt and Foege 1892) and neurological effects have been reported for more recent influenza epidemics (Delorme and Middleton 1979, Fujimoto et al. 1998, Toovey 2008, Baltagi et al. 2010, Chen et al. 2010, Shah et al. 2013). Direct damage to the lungs has also been found (Doblhammer 2004). Laraya-Cuasay and colleagues found that “influenza virus infection may be more serious in infants and young children than has been previously recognized and may contribute to the pathogenesis of unexplained interstitial pneumonitis, pulmonary fibrosis, obliterative bronchiolitis, and bronchiectasis” (1977:617). Further, it was found that “asymptomatic mechanical dysfunction of the lungs is a frequent sequela to acute influenza A virus infection” (Hall et al. 1976:141). Therefore, it is medically plausible that infection with a severe respiratory infection in childhood could lead to later respiratory impairments. These sequelae are not common however (Wang et al. 2010), meaning that it becomes difficult to assert that every individual who contracted the 1890 influenza was subject to this process. Individualized morbidity records and prospective tracking of individuals to the time of death would be a better means to investigate this process. The results from using age at death alone to research this process must therefore be interpreted with caution.

In terms of the relationship between the 1890 and 1918 pandemics, I hypothesize that infection with the Russian pandemic early in life may have damaged lung tissue, placing individuals at risk either for infection with any airborne infectious disease encountered later in life (including the 1918 flu), or specifically to the extreme sequelae resulting from secondary bacterial infection in 1918. There were many airborne infectious diseases circulating in Ontario between 1890 and 1918, including (among others) diphtheria, measles, smallpox, tuberculosis, typhoid fever, and whooping cough. Scarring could have been caused by any of these diseases (both before and after 1890) and would have placed individuals at greater risk from all following infections. For this reason, this mechanism through which early life exposure has an impact on mortality in later life is more speculative and more difficult to assert.

The examination of the scarring hypothesis for these two pandemics must take into account selection biases. It is possible that those individuals who were the most severely scarred in 1890 would have been more susceptible to contracting and dying from an airborne infectious disease between 1890 and 1918. Therefore, the individuals who are of greatest interest would not have survived to be included in the sample. If scarring did affect mortality in 1918, it could be seen in a concentration of deaths among individuals born in 1889, or those who were alive to experience the epidemic for themselves. For those individuals who were breastfeeding at the time and therefore benefiting from their mothers immune system, deaths in 1918 would be expected to be greatest for those exposed to the flu at the time of weaning (or approximately between the ages of 3 and 6 months, when breast milk is no longer sufficient as a sole food source. Timing varies slightly by ethnic and religious origins, Wharton 1989, Hendricks and Badruddin 1992). Infants who were not breastfeeding, and older children, would have been vulnerable to the effects of scarring throughout the period of the epidemic.²⁰ As such, it will likely be

²⁰ It is difficult to get a depiction of common breastfeeding practices in Ontario from 1883-1895. There was a large movement in the 1910s in Ontario focusing on reducing infant mortality, following precedents set in the United States and Britain (Chapin 1921, Piva 1979, MacDougall 1990, Comacchio 1992, Hallman 2009). The imposition of well-baby clinics and milk stations throughout Toronto may indicate that breastfeeding was not widespread during this later period, at least in the cities and among the impoverished. However, at this time there was no reliably sufficient substitute for breast milk, the quality of cow milk was poor, and the water used to clean bottles was often unsafe, such that many bottle-fed babies died (Meckel 1990, Comacchio 1992). MacMurchy notes this in her 1910 report on infant

difficult to separate the exact mechanisms that may have occurred for individuals who were alive during the epidemic (scarring and antigenic imprinting). However, gestational effects would be more likely to have been the main mechanisms for individuals whose exposure occurred *in utero*.

2.4.3 *Antigenic Imprinting (Original Antigenic Sin)*

The hypothesis referred to as ‘the doctrine of original antigenic sin’²¹ for influenza had its origin in the James D. Bruce memorial lecture given by Dr. Thomas Francis Jr. to the American College of Physicians in 1953 (Francis 1953). Involved in the identification of influenza strains and early attempts at vaccine development, Francis was also interested in determining what factors may have contributed to the unusual young adult mortality of the 1918 flu pandemic. Using the work of Collins in the 1930s, Francis determined that the infection rates for influenza were similar across all ages but that young adults (whom he considered to be 25 to 40) were more likely to die from secondary pneumonias. Young adults were more susceptible to bacterial complications due to the more severe damage done to their lungs by the initial influenza infection. He saw that those aged 5 to 9 are generally the most likely to contract influenza and that “they develop a relatively specific immunity against recent strains of virus, reflected in the low point of incidence from 15 to 24 years [when the same, or only moderately changed virus is still in circulation]” (Francis 1953:210). Francis’ hypotheses about the relationship between immunity and age can be seen in Figure 2.2. In 1918, he reasoned that those people over

mortality where she says that “if the baby is nursed by its mother the chances are great that it will live. If the baby is fed in any other way the chances are great that it will die” (1910:5). Infant mortality was blamed on working mothers who were not able to breastfeed, such that “where the mother works, the baby dies . . . she cannot nurse the baby if she works” (1910:17). Since the subject period of birth for individuals in this study was before the 1900, and due to the high levels of mortality for bottle-fed infants, for the purposes of this research it will be assumed that all individuals who survived to age 23 were breast-fed in some regard and thus were under the protection of the maternal immune response to a certain extent.

²¹ First called so in Francis (1955) and Davenport and Hennessy (1956). It is now more useful to refer to this theory as ‘antigenic imprinting’ (Ma et al. 2011) or ‘antigenic seniority’ (Lessler et al. 2012, Miller et al. 2013, Gagnon et al. 2015). Antigenic imprinting implies that the virus an individual is first exposed to may be *similar* (but not identical) to the one encountered later in life.

the age of 40 had a more successful immunity to the 1918 strain and those under 40 had a less successful immunity. Importantly, “the more virulent a strain, the greater, presumably, is its ability to override the lesser degrees of resistance [immunity]” (Francis 1953:214).²² Francis explains the elevated immunity of young adults as a byproduct of the immunity to influenza of those over the age of 40 (resulting in less likelihood of dying from pneumonia), creating the ‘W’ shaped mortality curve which otherwise would have been a linear increase from the age of around 20 (1953:214). He explains that,

The combined effect is that the middle age group possessed a lower margin of the physiologic tolerance which protected the five to 24 year group from pneumonia and a less complete resistance to the virus than that which protected the older ages against the essential virus injury (Figure 6). It shows, instead, the results of a high incidence of influenza and the increased liability of its age to pneumonia and fatality following influenza virus infection. [Francis 1953:215].

Francis’ Figure 6 is reproduced as Figure 2.3. Francis states that “the data support the conclusion that the primary determining factor in the development of fatal infection is the viral constitution and the degree of injury it creates.” (1953:217).

²² Francis was writing only twenty years after the influenza virus had first been identified and the isolation of various strains was only in its initial phases. He is working under the assumption that there was a similar viral strain circulating before 1918 while more recent research claims that “the 1918 virus appears to be an avianlike influenza virus derived *in toto* from an unknown source” (Taubenberger and Morens 2006:18). Contrastingly, Worobey and colleagues suggest that both the H1 and N1 strains had been circulating for at least 5 years and that they had joined together by 1915 (2014).

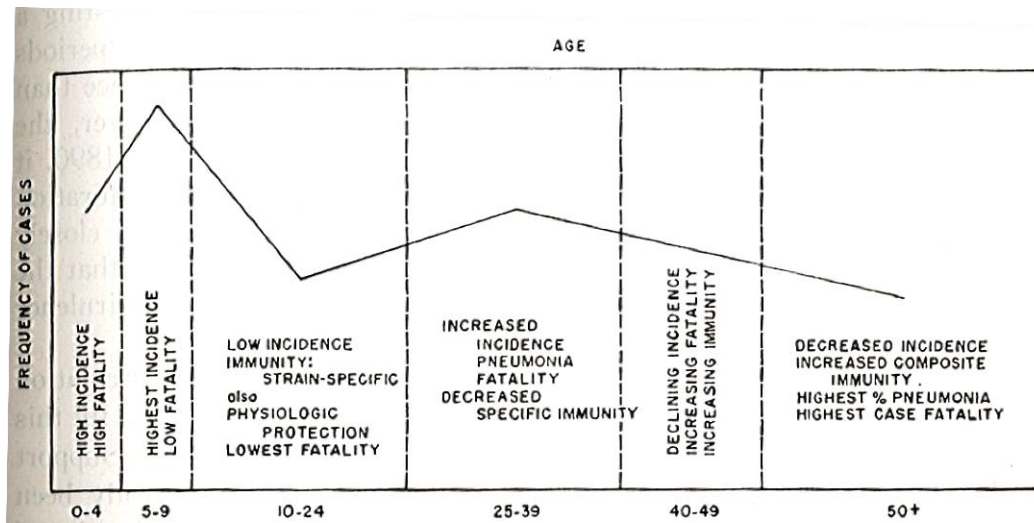
Figure 2.2 - Relation of Age to Features of Incidence and Immunity in Influenza.

Age	Incidence of Illness	% Pneumonic Complications	Case Fatality	Immunity to Virus
0-4	High	High	High	Low
5-9	Highest at all times	Lowest (5-20)	Lowest (5-20)	Lowest
10-24	Usually lowest			High-specific
25-39	Increases	Increases [•]	Increases	Waning of specific
40-49	Declines	Declines	Increases	Increased composite
50+	Declines [•]	Highest	Highest	Increased composite

[•] Degree varies with severity of epidemic

Source: Francis (1953:210, Figure 3).

Figure 2.3 - Relation of Age to Feature of Incidence and Immunity in Influenza.



Source: Francis (1953:215, Figure 6).

Davenport, Hennessy and Francis expanded this in an article later in 1953. Experimenting with blood samples from individuals of different ages and using different strains of influenza, they were able to hypothesize about the strains that were circulating in the years prior to 1953. Additionally, by focusing on the ages of the individuals and the types and amount of antibodies to certain strains produced, they came to three important conclusions. First, the antibodies that an individual obtains from their first exposure to influenza in early life (influenza infections being ubiquitous among the very young) are specific to that strain encountered; however, the long-term immunity gained from this experience is of limited strength. Second, exposure to various different types of influenza over the lifecourse will result in a broader range of antibodies to help protect individuals against antigenic shift.²³ Finally, however,

The antibody-forming mechanisms appear to be oriented by the initial infections of childhood so that exposures later in life to antigenically related strains result in a progressive reinforcement of the primary antibody. The highest cumulative antibody levels detectable in a particular age group tend, therefore, to reflect the dominant antigens of the virus responsible for the childhood infections of that group. [Davenport et al. 1953]

The finding that upon vaccination, individuals will produce antibodies to the virus first encountered in childhood was reinforced by Davenport and Hennessy using experimental data in 1956 (Davenport and Hennessy 1956). Similarly, from experiments with both humans and ferrets, Jensen and colleagues reported that “whatever the mechanism may be, it [is] clear that each antibody response to the various strains is dependent on preexisting immunologic factors and that antigenic similarities are emphasized in human hosts rather than slight antigenic differences which can be demonstrated among influenza viruses” (Jensen et al. 1956:208).

In response to authors who found little evidence for original antigenic sin in experimental studies (Gulati et al. 2005 and Wrammert et al. 2008) and to a lack of consensus over the existence of original antigenic sin among researchers, Kim and colleagues (2009)

²³ However, recent longitudinal analyses have shown that exposure to variant strains later in life that share similar proteins may actually reinforce the immunological response to the strain the individual first encountered in life (Miller et al. 2013).

conducted experiments in mice finding that “original antigenic sin could be a potential strategy by which variant influenza viruses subvert the immune system” (2009:3294). Using live rather than inactivated viruses, they found a profound effect of original antigenic sin but note that the original strain and the more recent strain must be antigenically similar for the phenomenon to occur (2009:3300). They admit that the mechanisms that cause original antigenic sin are still unknown.

In 2012, Shanks and Brundage (2012) attempted to explain the mortality of young adults using immunological theories and previous exposure to the 1890 influenza. As with other theorists, they try to explain elevated young adult mortality using a range of 18-43. They use 43 as the upper bound, since they believe that the 1890 pandemic strain was circulating in a less virulent form as early as 1875. Likewise, 18 is the lower age bound, as they attribute the 1899-1900 epidemic to the same strain as was circulating in 1890. They argue that “mortality rates during the lethal second wave were highest among persons with prior exposures to heterosubtypic influenza strains that enhanced immunopathogenic effects when a person was infected with the 1918 pandemic strain and had limited exposures to other respiratory infectious agents” (2012:205). They explain their finding that medical personnel and those who had been in the army for longer periods of time were as likely as non-medical personnel and new recruits to contract influenza, but they were less likely to die during the second wave. They argue that both previous exposure to a strain similar to pandemic influenza as well as limited exposure to the bacteria that cause pneumonia resulted in higher death rates in 1918 (for example, in those recruits raised in rural areas who had less exposure to the bacteria that cause pneumonia throughout their lives). Once these individuals were exposed to pandemic influenza, it resulted in “high viral loads, dysregulated and pathogenic cell mediated immune responses, and transient increases in susceptibility to invasive bacterial infections” (2012:205). If those individuals also contracted a strain of bacteria to which they had no immunity, they were more likely to die.

This argument is not accepted by Morens and Taubenberger (2012) who state that the knowledge of the 1890 virus and its circulation in the 1890s is speculative at best, and it is hard to base immunological arguments on it. They conclude that the ‘W’ shape of

mortality is as of yet unexplained and that there are still missing pieces to understand the puzzle of the 1918 influenza pandemic. This dissertation, focusing on the young adult age category specifically onto the ages of 28 and 30, adds a piece to that puzzle (Chapter 5).

Chapter 3

3 Materials and Methods

3.1 Materials

In order to answer the question of whether exposure to the 1890 Russian influenza pandemic influenced mortality during the 1918 Spanish influenza pandemic, it is necessary to utilize historical data sources that have not been previously compiled into a usable database. This is typical of historical demographic studies, where databases are typically not available to answer the questions of interest. The information that is most suitable to the answer these questions needs to be assembled (typically through archival research), even though the best data available may not be considered adequate if this research were to be conducted on modern populations (Section 3.2). Since it is not possible with historical samples to ask further questions of the individuals, to add questions to the census, or to recover data that have been lost, historical demographers utilize the data that is available to the best of their abilities. Therefore, to investigate the research questions of this study, a new database was created, compiled from various civil and governmental records available through archival sources. Throughout this dissertation, this database will be referred to as the Western, McMaster, Montreal Influenza Pandemic (WMMIP) Database.

This research is based on the death records of those who died in Ontario between September and December, 1918. It includes the pre-epidemic month of September, 1918; October, 1918, which was the worst month of the second wave of the Spanish flu; and November and December, 1918, when the epidemic was waning in Ontario. As the age-range of interest is young adults and previous research has indicated clustering at the ages of 28 and 30 (Gagnon et al. 2013, Hallman and Gagnon 2014), the age-range of 23-35 was selected. Age 23 represents five years younger than the peak at age 28 and age 35 is five years older than the secondary peak at age 30.

In order to establish whether or not the decedent had been exposed to the 1890 Russian pandemic (and if this exposure occurred pre- or post-natally), the exact birth date for each individual in the database is required. Since the majority of the death records from this time period only recorded age at death in full years (i.e., 28 years, 30 years, etc.), linkage to the birth records is important to analyze exact date of birth. As the birth records are not readily available for those born outside of Ontario, the sample is limited to individuals who died in Ontario and were born in Ontario. Of the original 23,183 individuals whose deaths were registered in Ontario from September to December, 1918, the elimination of those under 23, over 35, and those not born in Ontario left an initial sample of 3,878 individuals on which to link records (this included 577 individuals who were missing either a declared age at birth, a declared place of birth, or who were declared to have been born in “Canada” or “unknown,” Section 3.2).

During the process of creating the WMMIP database, I discovered that some individuals were included in the sample who did not meet the inclusion criteria. This is because: a) their place of birth was in error (either they were recorded as being born in “Canada” and upon investigation it was found that they were not born in Ontario, or their place of birth was incorrect, for example, if they immigrated at an early age, this may have not have been known at the time of death); or b) their age was incorrect. As will be discussed in Chapter 5, if age is not known, it tends to be rounded to either a number that ends in -5 or -0, or to a more socially desirable number, a process known as age-heaping (Hobbs 2004). For example, an individual who does not know their age will say they are 25 or 30, rather than 27. Further, if there is social stigma against being 30, an individual might say they are 29. Excluding those individuals who did not meet the criteria, the final sample size was 3,316.²⁴

²⁴ As individuals had to have been born in Ontario to be included in the study, immigrants were by definition excluded. However, some individuals in the database were born and died in Ontario, but spent the majority of their lives in the United States (as evidenced by border crossing records and their presence in either the 1900 or 1910 United States Censuses). The American censuses were not transcribed for these individuals and their linkages are missing for the censuses in this study, but further research could examine the effects of international return migration on death during the 1918 influenza pandemic.

The 3,316 individuals included in the study were linked to their birth records, their marriage records, their records in the 1891 census (if applicable and required), the 1901 and 1911 census, and to their Attestation papers (if applicable) (Section 3.2). Each of these sources are discussed, including their completeness and potential biases.

3.1.1 *Civil Registration*

Following upon the successes of the British *Birth and Deaths Registration Act, 1837*, civil registration began in Ontario in 1859 (then the province of Upper Canada) when clergymen performing marriages were required to submit a return to the County Registrar (Legislative Assembly of Ontario 1894). Formal registration of births, marriages and deaths began in Ontario on July 1st, 1869, when the *Act to Provide for the Registration of Births, Marriages and Deaths, 1869*, came into force (Statutes of the Province of Ontario 1869, Cap 30, *An Act to Provide for the Registration of Births, Marriages and Deaths*). This act established the roles of District Registrars (clerks of the peace in counties, cities, and towns) and Division Registrars (clerks in townships, villages, and city wards). The Registrar-General for Ontario (the former Provincial Secretary) was required to distribute the necessary registration books to the District Registrars, who would then distribute them to the Division Registrars of their district.

The Division Registrars were required to record every birth, marriage, and death that occurred in their region throughout the year and return the completed books to the District Registrar no later than January 15th of the following year. The District Registrars were to forward these books to the Registrar-General by February 1st. The Registrar-General would again transcribe these reports into one book; however, the originals would also be bound together into one volume. By June 1st of each year, the Registrar General was to make a full annual report of the records received by his office to the Lieutenant Governor in Council. These annual reports were published in the Sessional Papers of the Legislative Assembly of Ontario. The reports from 1882 to 1919 are used heavily in the research for this dissertation.

Errors were to be corrected in the margins of the books within a one-year period after the registration, following an inquiry to verify the error. Monetary fines were to be charged to any registrar or informant who neglected their duties or who knowingly submitted an inaccurate return; however, it proved to be “almost as much as his position is worth for a Division Registrar to prosecute residents of their municipalities for non-registration” (Legislative Assembly of Ontario 1895:32). Also, at any point, for a fee, individuals could request to have certified copies of these civil records. These requests for birth and marriage documents overtaxed the office of the Registrar-General during the First World War, when proof of age and marriage were required (Legislative Assembly of Ontario 1919a).

The *Act to Provide for the Registration of Births, Marriages, and Deaths, 1869* (Statutes of the Province of Ontario 1869, Cap 30, *An Act to Provide for the Registration of Births, Marriages and Deaths*), was amended at two different points (1875 and 1896) prior to the inception of the *Vital Statistics Act, 1908* which replaced it (Emery 1993). The 1875 amendment eliminated the position of the District Registrars such that “the Division Registrars were brought into direct relations with the Registrar-General” (Legislative Assembly of Ontario 1894:10). The revision in 1896, *An Act Revising and Consolidating the Acts Respecting the Registration of Births, Marriages, and Deaths, 1896*, had the greatest effect on civil registration in the province (Statutes of the Province of Ontario 1896, Chapter 17, *An Act Revising and Consolidating the Acts respecting the Registration of Births, Marriages and Deaths*). In this revision, the role of inspector was created who would “inspect the different registration offices throughout the Province, and carefully examine the different schedules, to see that the entries and registrations are made and completed in a proper manner and in legible handwriting” (c.17 s.3). This act also changed how the returns were reported to the Registrar-General, adding a semiannual return in July of each year. Concerned with common infectious diseases in Ontario (and reflecting the concerns of the registrars), Division registrars were required to send in monthly postcards with the total numbers of deaths from contagious diseases within the

past month. This represents the start of the system of notifiable diseases in the province.²⁵

The 1896 amendment to the act deemed that “all territory within the limits of the Province of Ontario, shall, for the purpose of this Act, be a part of some registration district” (c.17 s.9). This is important for the purposes of the records linkage in this project, since the final borders of Ontario were not extended to their present position until 1912 (see Section 2.1, Zaslow 1967, Baskerville 2005). While the death records used in this study hypothetically represent all the deaths that occurred within the province,²⁶ the birth and marriage records for those individuals in the far north of the province would not have been included (the former Keewatin Land and in the region contested with Manitoba, Zaslow 1967, Dean 1967, Baskerville 2005). Added to the lack of completeness of the vital registration system itself (Emery 1993), it is understood that it is not possible to locate all records of all individuals in this study.

The second *Vital Statistics Act, 1919* came into force in 1920 and is the format that the Registration Acts are known as today. The Registrar-General explains in the 1918 report that in this year “the Dominion Bureau of Statistics at Ottawa expressed a desire to compile vital statistics for the whole of the Dominion” and hosted a conference in Ottawa to do so (Legislative Assembly of Ontario 1919a:10). The provinces then passed separate acts that would facilitate the creation of a national registration area (Emery 1993). The governments of Ontario and Canada recognized the various problems in the vital registration system prior to 1918 and made changes that enabled the completeness of modern day records. In doing historical research on these records, understanding the process through which they were created aids in recognizing that they are socially constructed, socially negotiated, and contain errors. Once it is known where the errors

²⁵ Specifically, smallpox, scarlatina (scarlet fever), diphtheria, measles, whooping cough, typhoid or malaria, and tuberculosis/consumption (1897:6). Influenza was not a notifiable disease until after the 1918 pandemic (Legislative Assembly of Ontario 1924).

²⁶ Through an extensive analysis of the deaths in Ingersoll, Ontario from 1880-1972, Emery and McQuillan argue that “death registrations are shown to be seriously incomplete for the 1870s, but their coverage sharply improves during the 1880s and is nearly complete by the 1890s”. They further note that the death records appear to be unbiased after 1890 (1993: 61). For the province as a whole, Emery (1993) believes that mortality registrations were complete by 1911.

occur and the time point at which they were amended, the limitations of their use can be mitigated.

In the *Act to Provide for the Registration of Births, Marriages, and Deaths, 1869* (Statutes of the Province of Ontario 1869, Cap 30, *An Act to Provide for the Registration of Births, Marriages and Deaths*) a birth of a child was to be reported (with all the necessary details of the birth) to the Division Registrar within one month of the birth taking place. This was the job of the father of the child first, then the mother if the father was deceased or absent, next a guardian if the parents were absent, and finally, if no-one else was available, this job fell to the owner of the house in which the birth occurred or the nurse present at the birth. The person reporting the birth was required to pay the division registrar the sum of ten cents. It was illegal to state the name of the father of the child when the birth was illegitimate, “unless at the joint request of the mother and of the person acknowledging himself to be the father” (C.30 s.9). However, in all cases, it was required that the word ‘illegitimate’ be clearly stated on the birth registration, since it was known that the mortality rate of illegitimate children was greater than for legitimate children but also so as to not burden men with the legal and financial responsibility of paternity which may not be theirs. This represents the “distrust of women” that existed in Ontario legislation that persisted from their origins in the English poor laws (Chambers 2007:17). Only 10 individuals who were linked to a birth record in the study were noted as having been illegitimate.²⁷ However, 1,237 individuals in the database were not linked to a birth (or baptismal) record (including both the 2,965 linked and the 351 unlinked individuals). As was similarly expressed many times in his annual reports, the Registrar-General of Ontario stated in 1891 that “for evident reasons, there is a greater likelihood of these births not being registered than of others” and illegitimate births were often spoken of as a form of immorality (Legislative Assembly of Ontario 1893:9). It is possible that

²⁷ The father’s name of one individual is listed as “mother won’t tell”. He was born on Garden Island, Frontenac in 1883. There is no mention of a father in the 1901 census, at which point his mother was single and they were living in his uncle’s house in Toronto. He was married in 1904, at which point his father’s name is listed as “Joseph” after his mother had died in 1903, still single. The only mention of a Joseph in the history of the mother was the head of the household she was living in in Kingston in 1881. He was married, of African descent, born in the United States.

some of those 1,237 individuals without a birth record may not have been registered due to illegitimacy.

In the early part of this time period, there was a problem that “many of the counties in Ontario have been settled very recently, and it is found difficult to get returns from sparsely settled districts” (Legislative Assembly of Ontario 1883:7). Regardless, the Registrar still found that the birth rate for Ontario was close to that of Massachusetts, which suggested to him that the recording process for births in Ontario was progressing well. The faith in the completeness of the Ontario returns did not last long, however. By the 1890 annual report, lack of completeness among birth records was attributed to a) physicians and midwives not being present at all births; b) instances where the name of the infant was not finalized for weeks or months after the birth; and c) the aforementioned lack of willingness to register illegitimate births (1892:4).

The 1896 Amendment to the Registration Act required that medical practitioners also send in reports of the births that they attended, based mainly on the finding that physicians had been negligent in registering all births. Births that were to be recorded more than one year after they had occurred were required to obtain the permission of the Registrar-General and to fill out a separate form. These forms asked further questions of the informant, such as the marital status and place of marriage of the parents, and required that the informant swear that the information was true. Emery notes a distinct increase in the number of delayed registrations of births that occurred after the inception of the *Military Service Act, 1918*, which instituted conscription for men over the age of 18 (Legislative Assembly of Ontario 1919a, Emery 1993). Some of the birth records linked in this study were late returns; additionally, sometimes it was possible to find the late returns of the individual’s siblings, dated at some point after 1918 (for example, at the time of retirement or qualification for old age benefits). This may have indicated that all the births in the family were not registered and that, had the individual lived long enough, the birth might have been registered late.

The birth records that are used in this study, for the period 1883-1895, remained the same. The information provided includes: date of birth, name of the infant, sex, the name

and surname of the father, the name and maiden surname of the mother, the rank or profession of the father, the signature, description (relationship), and residence of the informant, the date of registration, name of the accoucheur (midwife or physician), the signature of the Division Registrar, and any remarks necessary.²⁸ They were often almost completely filled out, and if information was missing, it was usually the name and the address of the informant (sometimes useful in records linkage) and the name of the accoucheur (never useful for records linkage).

The death records for 1,237 individuals in the study could not be linked to birth records, meaning that the returns of the births that were recorded were filled out rather fully, but not all births were registered (for example, only 61 records of the 2,079 linked were missing the maiden name of the mother). These problems are addressed in Chapter 4. When the birth record was found and there were discrepancies between the declared year of birth on the various linked records, the date on the birth record is taken as correct, since it is the record that was created nearest to the actual event.²⁹

The registration of marriages had begun in Ontario ten years prior to the 1869 Registration Act (Legislative Assembly of Ontario 1894). From 1859-1869, clergymen were to keep their own book in which they would record the marriages that they solemnized throughout the year. The clergyman was then to send a “certified list” of these marriages to the County Registrar by February 1st of the following year (Legislative Assembly of Ontario 1894:9). The County Registrar would then record each list that he received into a book kept by his office.

With the passage of *Act to Provide for the Registration of Births, Marriages, and Deaths, 1869* (Statutes of the Province of Ontario 1869, Cap 30, *An Act to Provide for the Registration of Births, Marriages and Deaths*), it became required for clergymen to report marriage ceremonies to the Division Registrar within ninety days. He was obliged

²⁸ These were often “illegitimate”, “twins/triplets” or “delayed registration”. However, someone found it necessary to write on the birth record of one individual born in 1889, “wish it had been a boy.”

²⁹ Unless the birth record is the only discrepancy, whereas the other records, including the death record and attestation papers, gave a different year of birth. This happens very infrequently, however (see Appendix A for the linkage procedure).

to pay 10 cents to the Division Registrar, but was able to charge this fee to the couple being married. It was clearly stated that clergymen were only required to report on marriage ceremonies personally solemnized and not on any other births, baptisms, marriages, or death (except where they fulfill the requirements of other parts of the act, for example, when they were present at a death and there was no-one else able to report it to the Division Registrar).

The earliest marriage recorded in the WMMIP database is from 1890, and the latest is from 1918. The records change over this time period, with more information being given in the latter years. However, most records provide the names of the spouses and their place of residence, the occupations of the spouses (usually only the groom), the names of the parents of both spouses, and the location of the marriage. These records are particularly useful to link married women whose parents' names are not listed on their death records. Some of the marriage records come from church records, both from Ontario and Quebec, and the majority of individuals who are either listed as married, divorced, or widowed on their death record (2,045) were linked to a marriage record (1,761, 86.1%). This percentage rises to 94.3% (1,761 of 1,867) when considering only those who were linked to at least one other record.

With the commencement of the *Act to Provide for the Registration of Births, Marriages, and Deaths, 1869* (Statutes of the Province of Ontario 1869, Cap 30, *An Act to Provide for the Registration of Births, Marriages and Deaths*), deaths were to be reported to the Division Registrar “before the interment of the body or within ten days after” (c.30 s.11). This was to be done by the owner or occupant of the house in which the death occurred, by any person present at the death or by any person having knowledge of the circumstance of the death, or by the coroner in the case of an inquest. Again, the informant of the death was required to pay 10 cents to the Division Registrar.

The 1869 Act mandated that the physician or medical attendant at the death report it to the Division Registrar within ten days, stating the particulars of the cause of death. However, the 1896 Amendments to the Act stated that the reporting of the death must occur before interment of the body (c. 30 s.21). It was explained that “no removal for

burial of the dead body of any person shall take place, and no undertaker, clergyman, sexton, householder or other person shall engage in the burial of the dead body of any person unless a certificate of registration has been previously obtained and shown to the person so removing or engaging in the burial of the dead body” (c. 30 s. 23). Further, “the caretaker or owner of any cemetery or burial ground, whether public or private, or any clergyman having charge of a church to which a burial ground is attached shall not permit the interment of the dead body of any person . . . unless he has received a certificate under the hand of the division registrar . . . that the particulars of the death have been duly registered” (c. 30 s.24). It was also required for the person in charge of the burials to send a bi-annual return of the interments in the cemetery to the Division Registrar. Both fees and jail terms were threatened for anyone who neglected their duties or who willfully supplied incorrect information. It was these changes, so that a death registration was required before burial could take place, that were likely responsible for death registrations being complete earlier than birth records (Emery 1993).

The death records that form the basis of this research come from a very limited period (September to December, 1918) and the forms used did not change during this time. The death record for each person is divided into two sections, one filled out by the informant of the death and the other filled out by the attending physician. The informant’s report contains the following information: Christian name and surname, sex, age, date of death, place of birth, place of death, occupation, marital status (single, widowed, or divorced), name of father, maiden name of mother, cause of death (if known), name of attending physician, name and address of the informant, and the date of the return. The physician’s portion of the return includes the full name of the deceased, the date of death, cause of death, the physician’s name and address, the date of the return, and any remarks. The cause of death on the physician’s return is divided into two sections, the “disease causing death” and the “immediate cause of death,” and the duration of each cause is stated on the following line (Appendix B).³⁰ When determining whether a death is pandemic related³¹,

³⁰ For example, the physician’s return of George G. Stewart, aged 26, who died in Toronto on October 9th, 1918 is fairly typical of returns during the worst of the epidemic in October, 1918. The cause of death section states:

Disease causing death: Spanish Influenza

Duration: 10 days

or tuberculosis-related,³² if the disease is indicated in any *one* of the three cause of death listings on the death records (the one from the informant and the two from the physician), it is included as being either pandemic or tuberculosis related. A death is considered to have been pregnancy-related if there is any mention of pregnancy on the death record, including in the remarks section, or if it was noticed that the death record of a mother was immediately beside the death record of an infant and their deaths were within days of each other.³³

3.1.1.1 Sources of Error

If, then, it is now conceded that for economic, sanitary, and legal purposes, vital statistics are of inestimable value, it must be granted that necessity demands that they shall be as perfect as circumstances make possible.

Peter H. Bryce, M.D.
Deputy Registrar General of Ontario
Legislative Assembly of Ontario 1892:2

The *Registration Act, 1869*, and the subsequent amendments were concerned with the issue of payment to the various Registrars for the collection of information regarding

Immediate cause of death: Pneumonia

Duration: 3 days

³¹ Deaths during the Spanish influenza pandemic were the result of influenza infection complicated by secondary bacterial infections of the lungs (further, the course of the disease was hastened by co-infection with tuberculosis or pregnancy; CMAJb 1918, Oertel 1919, Young 1919). Therefore, pandemic related deaths as defined for the purposes of this study include the following: influenza (whether specified as acute, epidemic, virulent, or Spanish), la grippe, toxæmia of influenza, pneumonia (whether broncho, lobar, double, pleuro, septic, or influenzal), bronchitis, edema (oedema) of lungs, or congestion of lungs.

³² Including: tuberculosis (including pulmonary and miliary), consumption, anything with the adjective tubercular (for example, tubercular kidney, tubercular arthritis, tubercular laryngitis), Pott's disease, and phthisis.

³³ Not all maternal mortality during the pandemic would have been caused by the flu. However, it has been noted that the flu increased maternal mortality, especially for those women in their third trimesters (Chapter 2, Hallman 2009).

births, marriages, and deaths. This was because of the concerns voiced by the Registrar-General of the quality of the collection and recording of these data.

This was a longstanding issue for the Registrar-Generals. The 1884 annual report exclaims that “in numerous instances Division Registrars exhibit great carelessness in making their entries in the schedules. The writing is almost illegible, and many items of information required by the Act omitted” (Legislative Assembly of Ontario 1885:48). It called on the Division Registrars to remember the instructions from their office, that:

Division Registrars are requested to remember that the original forms will be bound into books to be kept in the office of the Registrar General, and care, therefore, must be taken that they are neatly filled in with writing as small as is consistent with clearness and legibility, and that they are in no way mutilated or disfigured; and it is further absolutely necessary that they in every case, see that the whole of the information required by the Act is obtained. It should also be borne in mind that there is no context to assist the eye, and that the legibility of the name depends solely on the actual formation of each letter composing it. What is called running hand, or one decorated with unnecessary flourishes, is fatal to the accurate indexing or transcription of proper names. [Legislative Assembly of Ontario 1885:48]

Yet, even with this rejoinder in 1884, the problem was not resolved. The birth registrations in Ottawa in 1891 were so incomplete (with there being 1,106 fewer births registered than in 1890), that they were said to “indicate so grave a state of affairs in the municipal office of that city” such that “said returns have to be wholly removed from any table, if correctness is to be even approximated” (Legislative Assembly of Ontario 1893:2). In comparing the rate of natural increase in selected cities to those cities in which the census indicated population growth, the Registrar-General concluded that “a neglect to register births exists to an extent which is simply lamentable” (Legislative Assembly of Ontario 1893:2) and that “until a radical improvement in the returns takes place it is apparent that conclusions based upon them are, except in the most general way, wholly fallacious” (1893:3). In regards to the Division Registrars themselves, the Inspector R. B. Hamilton found that “as a rule [they] are active and intelligent men; but though painstaking and energetic in their capacity as municipal clerks, I regret to say that they look upon the registration of vital statistics as of minor importance, and consequently their returns are not at all reliable” (1893:12). The most common reasons

given to the Inspector were 1) that “people are careless and will not register births and deaths of their own free will”; that 2) “physicians are negligent and will not send in their reports on the causes of death”; while 3) clergymen fail to send in their reports of marriages, and few of them ever think of making a report of a death where no certificate is provided at the time of burial;”; 4) that many burial grounds have no caretaker to make a report; and finally 5) that in regions dominated by religions with no permanent clergy (examples were given of the Quakers and the Tunkers) “many of these people object to furnishing the required information to any Government official” (1893:12). While the inspector praises those Division Registrars who go out of their way to obtain the required data, he suggests that they are not sufficiently compensated for their work and that the duties they must perform are not explicitly specified in law. In the course of his annual work in 1891, the inspector notes a town in Eastern Ontario in which 11 deaths were returned. He asked two local undertakers to see their books and found that together they had buried 44 individuals. The errant Division Registrar responded with “well, what have I got to do with that; if the parties interested do not bring in the returns, I am not going to hunt them up, my time is too valuable for that” (1893:13). The inspector then gives an analysis by township of some of the counties that he visited and the state of the returns in each township varied from “very incomplete” to “seem very complete” (1893:16-28).³⁴

In the annual report for 1892, the Registrar-General bemoaned the current system whereby registrars were paid per registration or on an annual salary. He complained that, “the result in practice is, as many of the answers made to the Inspector show, that the Division Registrars will not make any serious attempt to get complete returns, as they say they are paid no more for all their pains whether the number be 100 or only 10” (Legislative Assembly of Ontario 1894:12). He believed that there would be no improvements in the quality of the returns until the issue of payment to the Registrars was resolved (as it was in the 1896 amendment). In comparing the birth and death rates for various cities in Ontario, as well as an analysis of late returns submitted in 1892 and

³⁴ The counties visited were Algoma, Carleton, Essex, Lanark, Leeds and Grenville, Ontario, Prince Edward, Simcoe, Waterloo, and Welland.

1893 (359 births, 16 marriages, and 8 deaths), he concluded that there were great deficiencies in the recording of births in the province (Legislative Assembly of Ontario 1894:13). This was especially the case for illegitimate births, since “there is a likelihood of this class of births not being in all cases registered” (Legislative Assembly of Ontario 1894:3). By 1893, the Inspector stated that the Registration Act “has been practically a dead letter” (Legislative Assembly of Ontario 1895:28), with individuals flaunting the act and violations going unpunished. It remained the case that “. . . in some municipalities the birth returns seem to be fairly complete, while the death returns show very poorly; in other instances the reverse is the case. Very seldom indeed does it appear that births and deaths are both nearly reported in full” (1895:32).

The 1896 Amendment to the act had the “distinct object of remedying evils, which past years have made abundantly apparent” (Legislative Assembly of Ontario 1897:3). It now required the Division Registrars to keep their own copies of the registry which residents could access. It also reinforced the monthly return of deaths from contagious diseases and “the Division Registrar is now required under the Act to make diligent enquiry into the facts regarding any birth, marriage, or death coming to his knowledge, which has not been registered” (1893:4). It became explicitly illegal to bury a body without a death certificate. Moreover, the Division Registrar would not issue either a death certificate or a burial permit until he had received the return of death from the attending physician. Finally, the Division Registrars were now paid twenty cents for every *complete* registration and the registrations were sent to the Registrar General through the post office at no charge (1893:4).

Six months after the inception of the Amendments to the Act, the Registrar General found that it had “proved itself a notable advance toward the attainment of that completeness of returns so much sought for” (1897:12). These changes resulted in increases in the number of vital events returned from 1895 to 1896, being 16% more birth registrations, 6% more marriage registrations, and 20% more death registrations (in total numbers, not accounting for actual increase in number of events) (1897:14). There were also 2,400 delayed registrations of births, marriages, and deaths in 1896 (for those events that occurred more than one year prior to the date of registration) (1898:8). The marriage

records were the earliest that can be considered complete (1890), followed by the death records (1911) and the birth records (1920) (Emery 1993:41-43). However, even after 1920, Canadian birth statistics were “still rather deficient for the Indian population in general” (Kuyczynski 1930:27) and “in the 15 years from 1876 to 1890 perhaps two-thirds of the births may have been registered” (1930:108).

Further to the issue of the legibility of the records is the issue of the physical state of the records. Some portions of some of the books kept by the Registrar-Generals have been destroyed, either through ink, water, or damage that looks as though it might have been fire-related. Some parts of some pages are ripped or missing. Unfortunately, this data cannot be recovered and may add to the reasons why particular individuals could not be linked, or their birth record could not be found (a form of random error, except that the damage usually applies to a particular part of a single county, since the registrations are organized by county). The marriage records for the years surrounding 1900 span two pages and in some instances the second page is not available in the digitized records (for more on the process of obtaining the records, see Section 3.2). The books were microfilmed and kept by the Archives of Ontario and these microfilmed records were later digitized by Ancestry.ca. The microfilming process involves taking a picture of the open book, both verso and recto sides exposed. The resulting crease of the book is inconsistently imaged, with some microfilmed pages having a dark line down the middle. This, at times, results in the records closest to the spine being fully obscured and these data being lost. This is most extreme for the birth records, on which there are six records to a page, three on top and three on bottom. Since this data loss affects both sides of the page, up to four records may be partially or fully illegible. Both the issue of the completeness of the recording and legibility their current state may contribute to the reasons why some individuals could not be linked, beyond that analyzed in Chapter 4.

A source of error unique to the marriage records (and also to the attestation papers) is intentional age-misreporting. The age of either spouse may be erroneously stated in direct response to the minimal legal age of marriage (Acheson 1967). In Ontario, the *Marriage Act, 1897*, states that “no license or certificate shall be issued to any person under the age of fourteen years, except where a marriage is shown to be necessary to

prevent the illegitimacy of offspring” (RSO 1897 C. 162, s. 16 1 Geo. V. Cap 32). However, since the marriage records are only used as a determinate of age as a last resort (they only included age in whole years), this source of error does not affect the linkages in any great manner.

The Spanish influenza pandemic itself may also have had an impact on the completeness of the death records. The underreporting of deaths during epidemics is commonly hypothesized and is a process by which deaths occur in too great a number, too quickly, such that physicians simply do not have the time to register a death at all, or they are not able to completely fill out a death record (Johnson and Mueller 2002). For the Spanish influenza pandemic, it was contemporarily recounted that “the recent epidemic has been such a rush for those who have to deal with the victims that any detailed account of a number of cases is likely to be left until till the pressure is over, and so notes of the earlier cases may be lost for lack of leisure” (Wirgman 1918:324). The Medical Officer of Health for Toronto did not believe this was happening during this pandemic, citing the legislation that required a death certificate to be registered before a grave was dug (The Globe 1918d). The extent to which underreporting during the epidemic occurred in Ontario amid the Spanish influenza pandemic is hard to assess. There are many forms that are missing information on the physician’s return (some missing this section altogether) and likewise, some records are missing either part or all of the informant’s return (see Chapter 4 for further discussion concerning missing information on the death records and non-linkage of records). Underreporting may also be evidenced by comments on the death record such as “buried before burial permit issued or Doctors report received,” “I presume the cause of death was influenza, I was not present at the time,” and “saw her after.” What is more difficult to assess is underreporting that resulted in individuals not being included in the death registries at all.

Based on those individuals for whom information is missing, I propose that if the deaths of individuals were not registered, it would be for those individuals on the margins of society. They might include the impoverished (more likely outside of cities than within), immigrants (who are excluded from this study), aboriginal Ontarians – especially those living in remote areas of the north, individuals living in rural areas, and those involved in

itinerant labour. It is therefore necessary to view the results of this study as potentially biased towards those living in cities and those of higher socio-economic status. These issues are address in Chapter 4.

3.1.2 *Census Records*

The Canadian censuses have their own developmental history and sources of error. As described by Sager and Baskerville,

While acknowledging the uniquely comprehensive scope of censuses, we remember that they are constructions of the national population created by individuals and groups within the state. Routinely generated information in official sources is never a transparent window into a past social reality. Censuses are surveys, the results of personal interviews with large numbers of people who spoke on behalf of their families, answering questions framed by census officers working (in the case of the 1901 census) within the Canadian Department of Agriculture. The enumerators' forms are the record of a multitude of dialogues, a long series of questions and answers in which class, race, gender, language, and other influences guided the conversations. It is a mistake even to see the questions as determined by the state or state officials alone, because the questions had multiple origins. We may use the census to draw tentative conclusions about people and society in the past, but we do so most convincingly when we put the census dialogues in the context of their creations. The information has the great advantage that it is self-reporting by individuals speaking on behalf of their families or households; they spoke, however, in conditions that they did not alone create. [Sager and Baskerville 2007:4-5]

The modern day census has its beginnings in the *British North America Act, 1867* (later the *Confederation Act, 1867*), which established a decennial census that would begin in 1871 (Trovato 2009). The census returns that are used in this study (1891, 1901, and 1911) therefore represent the third, fourth, and fifth Canadian censuses, respectively. The specifics of the census are formalized in *The Census Act, 1870* which provided that the details of the census were to be determined during the census year through a Proclamation by the Governor in Council, "provided always that such period for taking the Census shall not be later than the first of May" (*The Census Act, 1870* 33 Vict c.2). The specific census divisions and sub-divisions would likewise be created through

proclamation “into so many and such . . . as may be deemed convenient” (*The Census Act, 1870* 33 Vict c.4, 5).

The census was divided into families, households, and institutions, where “as defined in the Census Act, the term ‘house’ includes whips, vessels, dwellings, or places of abode of any kind.” Further, “any structure which provides shelter for a human being is a house, and if it has only one entrance it counts only as one dwelling, no matter how many families it may shelter” (Dominion of Canada 1902:xvii). Any house that had two doors was counted as two separate houses. In defining a family, the enumerators in 1901 were told that “a family consists of parents and sons and daughters united in a living and housekeeping community: but in the larger sense it may include other relatives and servants.” Further, “a household may include all persons in a housekeeping community, whether related by ties of blood or not, but usually with one of their number occupying the position of head” (Dominion of Canada 1902:xvi).

The censuses of 1891, 1901, and 1911 differ in the information they collected and are therefore of different uses in this study. They each provide a picture of the socioeconomic environment of the individual at a certain point of time throughout his or her lifecourse (the census of 1891 was only used in this research if it was necessary to determine year of birth. As the sample population was born from 1883-1895, many individuals included in this research were not born at the time of this census). The digitized census records were obtained either from Ancestry.ca (www.ancestry.ca) or through Automated Genealogy (<http://automatedgenealogy.com>).

The 1891 census consists of nine schedules with 216 questions (Dominion of Canada 1902). This research utilizes Schedule I, “Nominal Return of the Living” which asks 25 questions and was begun on April 6th, 1891 with 4,324 enumerators (1,887 of which were employed in Ontario) (Dominion of Canada 1902). The questions asked by enumerators included the type of dwelling, the names of individuals (grouped into families), age, sex, marital status, relationship to the head of the family, the country or province of birth, whether the individual was a French Canadian, the birth place of an individual’s father and mother, religion, field of employment, whether a wage earner, whether employed in

the week following the census, number of hands employed, whether can read and write, and whether deaf and dumb, blind, or unsound of mind. Age was recorded in full years only, such that this census was not sufficient to establish exact date of birth.

The 1901 census expanded from nine to eleven schedules and asks 561 questions, increasing from the 216 asked in 1891 (Dominion of Canada 1902). This research once again utilized Schedule I “Populations”, which asks 34 questions to respondents for a one month period starting on March 31, 1901 and employing 8,800 enumerators (3,759 in Ontario) (Dominion of Canada 1902). According to the proclamation for 1901, “the decisive hour of reckoning is made 12 o’clock or midnight on the night of 31st March to 1st April, so that every one born before that hour and every one dying after it are to be counted in the population” (Dominion of Canada 1902). Differing from 1891, this census no longer asks the place of birth of the father and mother, if French Canadian, whether unemployed the week before the census and the number of hands employed (Dominion of Canada 1902). However, it now asks about colour, the month and date of birth as well as the year of birth and age at last birthday, year of immigration and naturalization, racial or tribal origin, nationality, whether living on own account, whether working in a factory or home and how many months in each type of employment, wages from occupation, extra earnings not from occupation, months at school, whether can speak English, whether can speak French, and the mother tongue spoken. As these returns provide the exact date of birth, they are more useful in ascertaining this information than either the 1891 or 1911 censuses (however, see Chapters 4 and 5 for a discussion of the correspondence date of birth between the censuses and the civil registrations).

The fifth Canadian census began on June 1st, 1911 and employed 9,703 enumerators (the number in Ontario was not specified, Dominion of Canada 1912). This census has thirteen schedules with 549 questions in total and Schedule I, “Population,” is used in this research. Where the 1901 census asks for date of birth, the 1911 census only asks for year and month of birth and age at last birthday. As with the 1891 census, the 1911 is less useful in ascertaining age of exposure to the 1890 Russian Flu pandemic than the 1901 census. In 1911, questions are also asked as to the amount of insurance held per individual, and whether a person could read or write. The question of the mother tongue

remains, but specific questions on the ability to speak either English or French are dropped. Also new to the 1911 census is the cost of education for individuals over the age of 16 if they are at a college, convent, or university. Useful to this project, the 1911 census also asks for the place of habitation, which is often provided as a street address for people living in large urban areas. If an individual remained in the same residence from 1911 to 1918 and died at their home (and this is listed on the death record), it is another means to link the records of individuals directly.

3.1.2.1 *Sources of Error*

The census records contain similar sources of error as do the civil registrations. As was an issue throughout the records linkage process, it could be difficult at times to read the handwriting of the enumerators. The instructions to the enumerators clearly state that “every name, word, figure or mark should be clear and legible. If a schedule cannot be read . . . the work of the enumerator may be wholly wasted” since “the census is intended to be a permanent record, and its schedules will be stored in the Archives of the Dominion” (Dominion of Canada 1902:xvii). However, this is clearly not always the case. The census records also have some unique sources of error, and these are addressed by the Department of Trade and Commerce as early as 1925 (the ministry responsible for the administration of the sixth Canadian census in 1921).

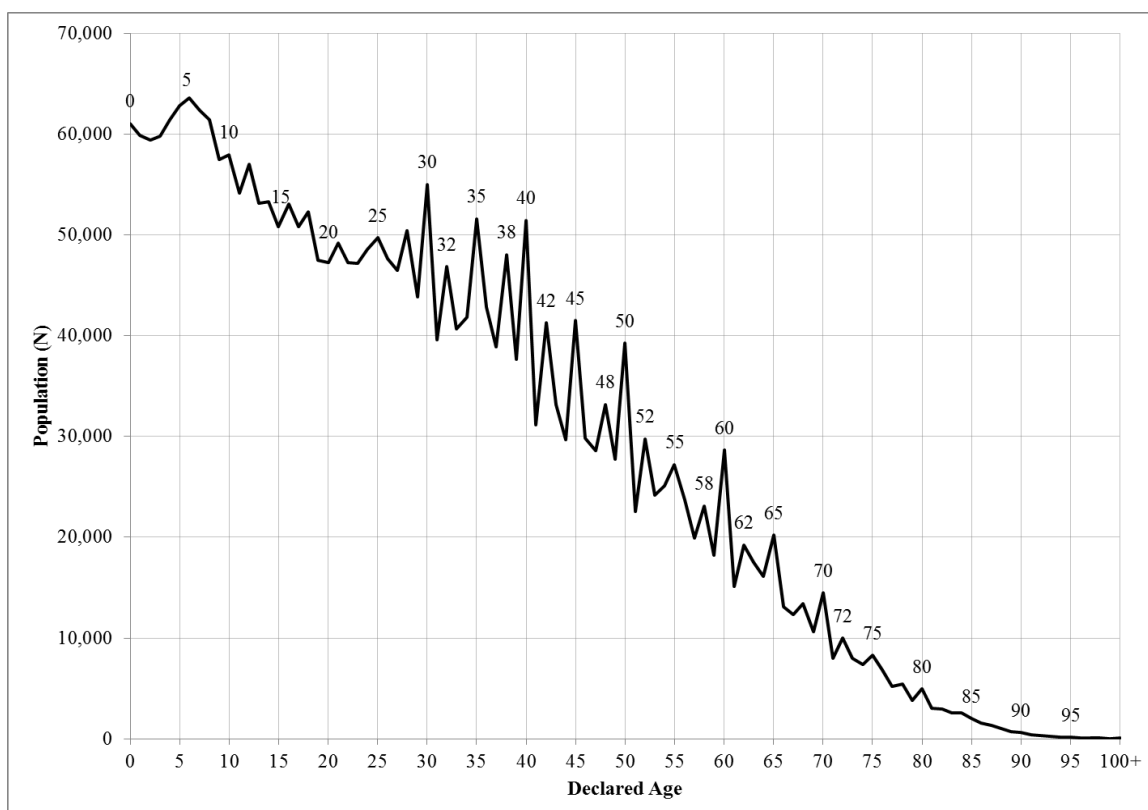
In regards to errors in the declared age of individuals, they state that:

It is impossible to claim absolute accuracy for census statistics of ages because (1) a considerable number of persons do not know their true age, (2) in a number of instances the enumerator obtained information relating to the person enumerated, from a third party, either some member of the family found at home, or the proprietor of a hotel or boarding house, who could give the age only approximately, and (3) the age, in certain cases, is intentionally misstated. When the age is not accurately known there is a tendency to state it in years ending with 5 or multiples thereof. For example, the number of persons reported at the ages of 30, 40, 50, or 60 is much greater than the numbers reported for either the years immediately preceding or following. The number of persons reported at the age following the decennial or quinquennial group when compared with the number at the age immediately preceding would seem to indicate that the

round age method of reporting was not due to a desire to misrepresent, but rather because of carelessness, illiteracy, or ignorance of the correct age. It is probable that the degree of inaccuracy is greater for adults than for children or young people. [Dominion of Canada 1925:vii-viii].

Age heaping in the 1921 census can be seen in Figure 3.1. Note that the heaping begins at the age of 20 and appears to be extreme between the ages of 30 and 60. This may be a result of the necessity to prove age resulting from the *Military Service Act, 1918* (Legislative Assembly of Ontario 1919a). With the age of conscription set at 18 in 1918, by the time of the 1921 census, those individuals would have been 21. If those individuals close in age to 18 in 1918 also needed to prove their age, it would follow that heaping would be more evident in people starting around the age of 25.

Figure 3.1 - Total Population by Age for Ontario in the 1921 Census.



Source: Dominion of Canada (1925), N= 2,929,103.

Age heaping can be seen in the differences between the ages and year of birth as given in the 1901 census as compared to the 1911 census, but also in the date of birth given on the birth certificate and the reconstructed date of birth for this project (for discussion, see Chapters 4 and 5). While it would be ideal to be able to use the birthdate provided in the 1901 census for this project (and reduce the amount of records linkage necessary), the discrepancies between the declared and reconstructed dates of birth are simply too large. Since potential exposure to the 1890 influenza at its more virulent phase could only have occurred in the first few months of 1890, it is not feasible to allow the amount of error that exists when the year of birth is off by one year (of the individuals who were linked and had a reconstructed age at death, 1,097 of 2,945 (37.2%) individuals had a different reconstructed age at death than the declared age at death listed on the death record, Chapter 5).

Another source of error relates to the problem of where people live. The Canadian census to this day is a *de jure* census, meaning that individuals are enumerated at their usual place of residence, whether or not they happen to be away on the date of the visit of the enumerator (as opposed to a *de facto* census, in which individuals are counted in their present location, no matter if it is their permanent residence or not, Trovato 2009). However, it was described in the 1901 Census Report that “in providing that the census of the people is to be taken by the *de jure* system, the proclamation does not give a meaning to that term. Neither does the Census Act, nor any other statute. It must be determined largely by usage, and therefore the practice of former censuses in Canada should be followed with reasonable closeness. In the great majority of the people, their home is to the place where they should be counted” (Dominion of Canada 1902:xiv). This ambiguity towards the term led to the warning that,

There is a probability that some persons may be counted in two places at once, and that others may not be counted at all. A domestic servant, for example, may be reported at the home of her parents as a member of the family *de jure*, and she may also be reported as *de jure* of the household where she is employed. Or, if absent from home for a comparatively long time and in her present place of service for only a short time, she may be left out of the enumeration altogether. The same thing may occur in the case of clerks, salesmen, artisans, labourers, railway employees, etc. [Dominion of Canada 1902]

While 82.5% of the individuals in the sample of death records were found in the 1901 census and 73.1% were found in the 1911 census (with 69.1% being found in both), it was not possible to locate every individual, for various reasons (as discussed in Chapter 4). One of these reasons may have been that individuals were not enumerated due to their transient employment and living arrangements. Commonly, when an individual was found in the 1901 census, but not in 1911, it is because in 1901 they were living in their natal home, while in 1911 they had not yet married and were living somewhere else as borders, potentially in a distant town. The result of this was that there typically is less information to be sure of a link in 1911. The effect on this research is that it is likely the more vulnerable individuals in the sample were not located, skewing the results in favour of the wealthier or more well-established (likely older individuals and those who were not indigenous). As was reported in the 1911 Census Report regarding men in mines, for those who worked on the railway, and for whom marital status was unknown, “it was difficult for the enumerators to get personal access to them or to procure information on them excepting from employers or the men in charge of their boarding or lodging places, and a like remark applies in the case of women who were not found at their homes” (Dominion of Canada 1912:vii). This is a common element to these records and is discussed in Chapter 4.

3.1.3 *Other Sources*

The attestation papers of individuals who either enlisted or were conscripted into the Canadian Expeditionary Forces (CEF) were also collected in the course of this research. These records relate only to those members of the CEF and not to other branches of the Canadian forces. Individuals who were members of the air force in the First World War were members of the British Royal Flying Corps prior to April 1st, 1918 or the Royal Air Force (RAF) after April 1st, 1918 (The National Archives n.d.). These records are kept

by the British National Archives in London, England, and are not available online. Likewise, the records for the Royal Canadian Navy are not available.³⁵

The Attestation Papers of the more than 600,000 members of the CEF from 1914-1918 are kept by Libraries and Archives Canada (www.collectionscanada.gc.ca) and also through Ancestry.ca. The digitized records are available through a searchable database. There are various forms of these records throughout the years, but generally they provide information on name, place and date of birth, name and address of next of kin, occupation, marital status, previous experience in the military, place of attestation and medical examination, age, height, chest measurements (girth when fully expanded and range of expansion), complexion, eye and hair colour, religion, and “distinctive marks, and marks indicating congenital peculiarities or previous disease” which would be useful if it became necessary to identify a deceased soldier who had lost his identification. While these records are mostly used in this project as a source of verification of linkages and of the birth date, they provide valuable biometric data that could be analyzed at a later date.

The attestation papers are subject to similar types of error as are the other sources used in this research. Some of the records are not available online, suggesting that they may either be missing from Library and Archives Canada, or that they have not been reached in the process of digitization. Some individuals in this study were identified through the search engine on the Library and Archives Canada website, however their relevant file was not attached. It is possible that the declared date of birth may have been inaccurate, since it was known that individuals may misrepresent their age in order to participate in the war.³⁶ However, since the youngest people in this sample were aged 23 at the time of death (18 or 19 at the start of the war), they were legally able to enlist.³⁷ Also, as discussed previously, it was necessary for men to prove their age in order to enlist in the war through presenting their birth certificate. As was stated in the Report Relating to the

³⁵ The Royal Canadian Navy Ledger Sheets are available for search through the Library and Archives Canada website (<http://www.collectionscanada.gc.ca>). However, the information provided by these online records is limited.

³⁶ The Canadian Great War Project (Leroux 2010) reports that of 2.71% of enlistments were under the age of 18.

³⁷ As established in the *Militia Act, 1906*, men were eligible to participate in military service if they were between the ages of 18 and 60 (*The Military Service Act, 1917*, 7-8 geo v: c 19).

Registration of Births, Marriages and Deaths in the Province of Ontario for 1918, “during the early part of the year, the Military Service Act became operative in Canada, which made it necessary for practically all men from sixteen or seventeen years of age and upwards to carry certificates of birth and, in some cases, certificates of marriage” (Legislative Assembly of Ontario 1919a:10). This led to many delayed registrations of birth and, barring any illegal alterations to these documents, it is likely that the declared age on the attestation papers is correct. However, these papers were found for only 188 (5.7%) individuals in this sample (multiple records were found for some individuals. Only one record belonged to a woman – a graduate nurse).

3.2 Methods

In its most basic definition, “demography is the scientific study of human population, including its size, distribution, composition, and the factors that determine changes in its size, distribution, and composition” (Swanson and Siegel 2004:1). Contemporary demographers draw on many sources for their data, including the modern-day successors of the vital registration and census data described above. A distinct advantage of working with living populations is the ability to collect the data that are required for the desired statistical analyses. Further, demographers working in Canada today are able to utilize the resources of Statistics Canada, which produces data of high quality, complexity, and coverage. However, numerous indirect estimation techniques have been developed to compensate for deficient data, such as in areas with either poor or non-existent national registration systems (Preston et al. 2001). Even in places with satisfactory data collection, often the data that are used to create population estimates are obtained from the decennial censuses, an issue which can be problematic if the population experiences rapid and age-specific changes.

Historical demography, by extension, is the scientific study of historical human populations. What can be known about the past must be gleaned from the extant evidence, which can range from tombstones, to vital registrations, to personal accounts, to parish registers. Creativity is required in order to extract the required data from

records which may never have been created for such purposes. As such, “historical demography is really a science not unlike archaeology” (Hollingsworth 1969:13). The challenge of historical demography is that the data gathered from these sources may not be sufficient for the typical analyses of contemporary demography, and thus may entail greater use of indirect methods. Although the kinds of analyses which may be conducted can be limited, it is nonetheless a worthwhile endeavor to examine that which has not been previously explored. By compiling and analyzing previously disaggregated data, it is possible to illuminate salient aspects of human history which may otherwise have been lost. As was conveyed by Laslett, historical demography is able to “answer questions which have hitherto been regarded as unanswerable, beyond the pale of knowable fact” (1966:12).

Records linkage, family reconstitution, and a multitude of techniques and sources are an integral part of this process. Hollingsworth asserts that “corroboration from independent sources by independent methods is the very kernel of historical demography, for two fairly weak but quite different arguments that reach the same conclusion are very much better than one” with the caveat that “all historical arguments are more or less weak, for no one can ever be totally certain or totally uncertain of anything” (1969:12). While not technically a family reconstitution (I am reconstructing the course of individuals’ lives rather than whole family units), this research owes much to the work of the founders of academic family reconstitution (Henry and Fleury 1956, Laslett 1966, Wrigley 1966, Hollingsworth 1969, among others). The techniques that they developed help to form the basis of the records usage protocol used in this study (Appendix A).

It is useful to conceptualize the individuals in this research as belonging to a particular ‘death cohort’ (c.f. Oeppen and Wilson 2006). This research focuses on *every* individual who was born in Ontario between 1883 and 1895 and who also died in Ontario from September to December, 1918. The benefits of a cohort approach to historical demography are detailed by Eversley (1966) for a marriage cohort. Through records linkage, it becomes possible to “describe a particular set of people in an entirely unambiguous way, and we [can] distinguish them from other people who lived before or after them, or in other areas” (1966:35). If the 1918 Spanish influenza pandemic did not

preferentially strike certain young adults, there should be no difference between this particular death cohort and any other. For example, there would be no reason to expect clustering of births in particular years or for individuals to be different from the rest of the population in 1901 and 1911. Detailed examination of the micro-level data also gives greater depth to the wide breadth of data gathered at the aggregate level. Not only can we determine occupation at death (if given), but also the occupation of that individual in 1901 and 1911 (or, the occupation of the head of household if they were not yet employed).³⁸ Likewise, where it is possible to study the impact of urban environments on mortality by comparing place of death, records linkage allows us to study this in comparison to location of birth. In this research, I am using both micro-level and macro-level data to develop a more complete understanding of young adult mortality in Ontario in 1918.

3.2.1 *Transcription*

The transcribed death records for the second wave of the 1918 flu pandemic in Ontario were obtained from Dr. David Earn, Department of Mathematics, and Dr. D. Ann Herring, Department of Anthropology, both at McMaster University. The microfilmed reels were acquired from the Archives of Ontario (Registrations of Deaths, Series RG 80-8, Appendix A7, Microfilm MS 935, Reels 239–261, Archives of Ontario) and transcribed by research assistants into an Excel database. The data for deaths in Ontario from September to December 1918 were made available by Drs. Earn and Herring through the International Infectious Disease Data Archive (IIDDA) at McMaster University (<http://iidda.mcmaster.ca/>). The microfilmed death records for Ontario from 1869-1938 have also been digitized by Ancestry.com. These records were used if clarification of the IIDDA transcriptions were needed.

As detailed in Section 3.1, the deaths were recorded by both the doctor and the informant on the death certificate. This was forwarded to the Registrar General for Ontario, where

³⁸ The census returns were subject to revision after the forms had been completed. This applies to such categories as ethnicity, nationality, and occupations (Adams 2011). This project transcribed the corrected entries.

it was transcribed into one large Registration Book (Statutes of the Province of Ontario 1869, Cap 30, *An Act to Provide for the Registration of Births, Marriages and Deaths*). These records were then microfilmed and made available to the public by the Archives of Ontario.³⁹ The microfilmed records were interpreted and transcribed by research assistants at McMaster University. There are many individual areas where error may have crept into these records through the process of transcription at each point at which this occurred. The sometimes illegible handwriting of the Division Registrars needed to be learned and deciphered in order for transcription to take place. In describing the quality of historical demographic data, Hollingsworth (1969) notes that

The most common errors are probably mistakes in copying. People who have never tried to work with figures for long may not appreciate that 2 or 3 per cent of any figures copied in a hurry will commonly be wrong, and that if the copier has no real interest in the figures he will scarcely realize it when he has made a mistake. Such mistakes must have been very common in the past, since many sets of records were copied and recopied several times before they reached their extant form. As Hume pointed out 200 years ago, a copying mistake in a word usually spoils the grammar and is easily detected, whereas a copying mistake in a figure is often impossible to spot. [1969:299]

In addition to the various forms of error previously described, there were inevitably mistakes made in the transcription process of this research, even though care was taken in the course of this project.

The death records obtained from the IIDDA were filtered to select those individuals who were both born and died in Ontario and who were between the ages of 23 and 35 at the time of death. The death records of these individuals were transferred into a Microsoft Access database (as well as any individual with a missing age at death). The records that were linked through this research were transcribed directly into the same Access database and are subject to the same errors and concerns through the transcription process as were the other records.

³⁹ The release of vital statistics records from the Registrar General to the Archives of Ontario is regulated by the *Vital Statistics Act, 1990* (R.R.1990, Regulation 1094, Appendix). Births are to be sealed for privacy reasons for 104 years, Deaths for 69 years, and Marriages for 79 years. After this point, the records are transferred to the Archives of Ontario and digitized for public use.

3.2.2 *Records Linkage*

Nominal Records are those in which individuals are distinguished by name, and by that token are potentially linkable to other nominal records. There is point in doing this only when two conditions are met. First, that it can be shown that it is possible to distinguish satisfactorily between true and false links. Second, that the complex of information which can be assembled by linking records that concern one man reveals something about him which would otherwise remain obscure. [Wrigley 1973:1].

The records linkage component of this project which created the WMMIP database was completed by Stacey Hallman (SH) and research assistants at Western University,⁴⁰ McMaster University,⁴¹ and the Université de Montréal.⁴² Of the 2,965 records that were linked, SH linked 1,257 records (42.4%) and the Research Assistants linked 1,708 records (57.6%). The records linkage completed by each research assistant was completed in separate Access databases. When complete, they were transferred to the master database and reviewed for completeness. At this point, 730 (24.6%) individuals had additional records linked to them by SH and, in total, 351 individuals could not be linked (each of these records were reviewed before determining that they could not be linked. For an analysis of these records, see Chapter 4). Linkages were transcribed using the digitized primary source and the transcription provided by Ancestry.ca was only used if clarification was needed.

The records linkage was completed using a protocol developed by SH (Appendix A). As per Winchester, “almost any kind of information item can function as an identifying item for records linkage purposes. The only condition is that it be present on more than one record relating to a particular historical individual. Naturally, other than proper names, such descriptive items as age, sex, place of birth, address, occupation, and the like are the most frequently employed for identifying historical persons” (1973a:130). The informant’s name, place of death, and parents’ names were frequently used. A record linkage score was tabulated at the end of each link to account for the number of correspondences and dissimilarities between each of the records (as per Wrigley and

⁴⁰ Jennifer Brooks.

⁴¹ Kandace Bogaert, Mikaela Colville, Alex Rewegan, Saryah Wahoush, and Melissa Yan.

⁴² Enrique Acosta, Marilyn Amorevieta-Gentil, Astrid Flénon, Julie Lacroix, Vincent Lortie, and Sophie-Andrée Piché.

Schofield 1973, Winchester 1973a. For more information on how the score was calculated in this research, see Appendix A). The information being linked did not have to be exact; to account for these discrepancies, a score of 5 was deducted for each piece of information that was discordant. Further, the individual doing the linkage was asked to state whether the link was a “positive”, “likely”, or “maybe” link. All the records could be used to make a link, even though the score ties directly to the death record. For example, as per Winchester (1973b), if a census record could be positively linked to a death record, and that same census record could be positively linked to a marriage record, then the death record and the marriage record are also linked, even if there might not be sufficient evidence on each record alone to link them to each other.

3.2.3 *Mortality Rates*

In the absence of age-specific population totals created for some other purpose, the best way to estimate the total population at each age from 23 to 35 who were alive in September, 1918 and at risk from dying of the flu is to create an intercensal estimate (necessary for the denominator in the calculation of mortality rates).⁴³ By this process, the population would be estimated using the 1911 census and the 1921 census, the two closest points at which the population of Ontario was enumerated. This can create problems, since “populations change quite rapidly, making census statistics for every tenth year, even every fifth year inadequate for most purposes” (Bryan 2004:523). This is the case for the 1918 Spanish influenza pandemic. It occurred in the midst of vast population change, including the First World War, high immigration up to 1913 and

⁴³ As defined by Preston et al. (2001:7):

$$\text{crude death rate } [0,T] = \frac{\text{number of deaths in the population between times 0 and T}}{\text{number of person-years lived in the population between times 0 and T}}$$

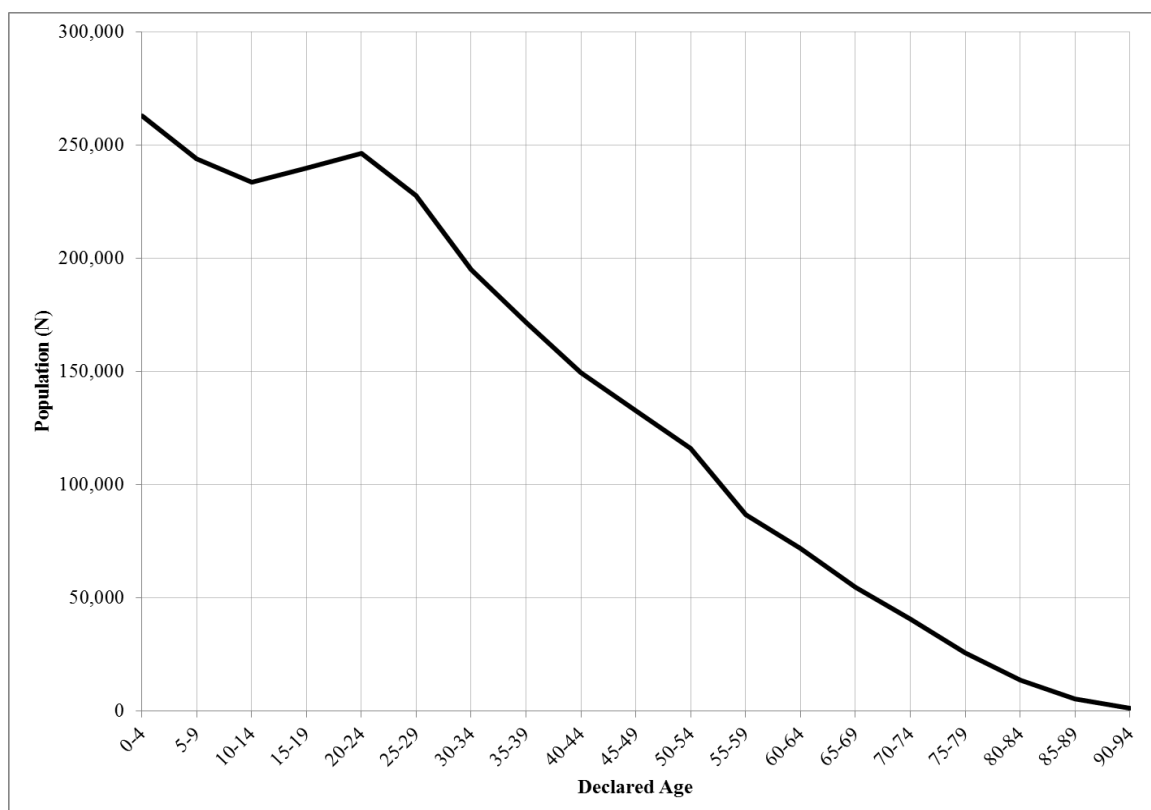
soldiers returning from the war,⁴⁴ and the pandemic itself had an unequal effect upon the age structure of the society. This was described in the 1921 census report:

Three factors, more or less, closely related to the War and resulting consequences (a) fluctuations in the birth rate, (b) cessation of immigration during the war period, and (c) losses occasioned by the war, have seriously influenced the variations in age constitution during the census period, 1911-1921. . . In the groups 20-24 and 25-29 there were absolute decreases in the number of males of 34,871 and 22,849 respectively from 1911 to 1921. The per cent proportion at these age groups also showed a large falling off for which the war and the influenza epidemic of 1918 were largely responsible. [Dominion of Canada 1925:viii]

The use of the 1911 and 1921 Canadian censuses to estimate population totals in 1918 poses two additional problems. The interest of this research is in age-specific mortality. The 1911 census only provides total population counts by area and does not break this down into age-specific population totals (Dominion of Canada 1912). The 1921 census does provide this information for 1911, but only in 5-year age categories and at no lower level of aggregation than at the province level (Figure 3.2. These population counts are not separated by place of birth, so it is not possible to isolate the Canadian-born).

⁴⁴ This includes not just soldiers themselves but also their dependents. This is an important aspect to consider, since “Canadian soldiers serving overseas during the First World War brought home nearly thirty-five thousand British and European war brides” (Jarratt 2008:127).

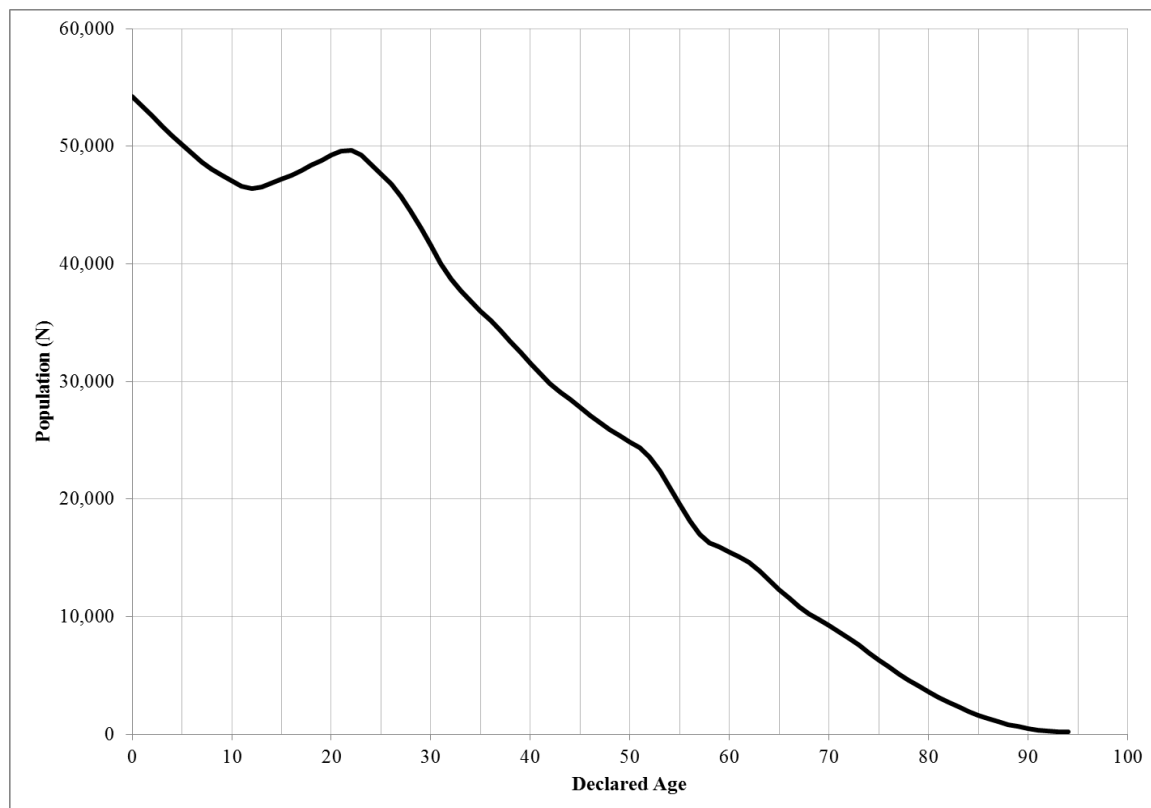
Figure 3.2 - Population of Ontario from the 1911 Census by Declared Age in 5 Year Age Categories.



Source: Dominion of Canada 1925:11, N=2,519,922.

In order to be able to use the 1911 census population totals to create an intercensal estimate for 1918, it is first necessary to interpolate the age-specific population totals in 1911. This was done using Sprague's Fifth-Difference Equation whereby a series of multipliers are applied to the total of each category (Judson and Popoff 2004). This method is currently used by Statistics Canada in order to estimate population (Statistics Canada 2012). Once the Sprague's multipliers were applied to the population totals from 1911, the estimated age-specific population totals in 1911 were created (Figure 3.3).

Figure 3.3 - Age-Specific Population of Ontario from the 1911 Census



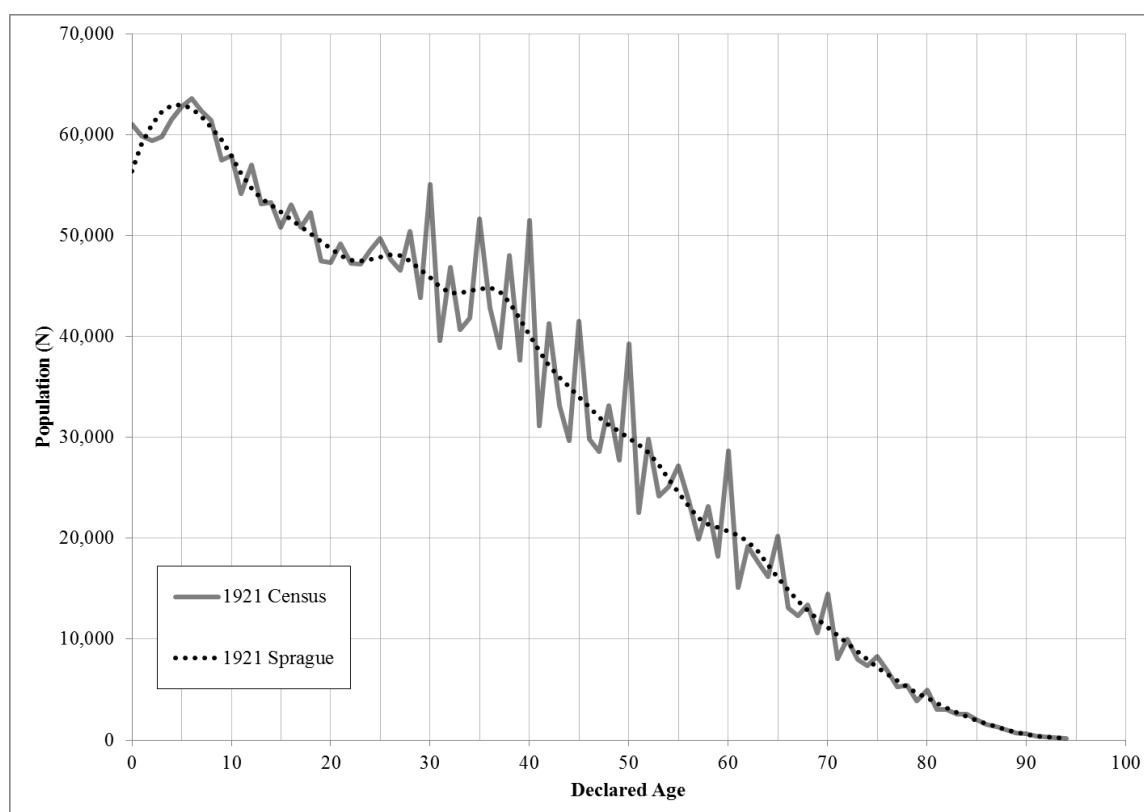
Note: Created using Sprague's multipliers

Source: Dominion of Canada (1925) N=2,519,922, Judson and Popoff (2004).

As shown in Figures 3.1 and 3.4, the age-specific population totals in the 1921 census are subject to noticeable age-heaping throughout the ages that are pertinent to this study. In order to attempt to create accurate mortality rates through interpolation, it was necessary to smooth these data in some manner. Since Sprague's multipliers were applied to the 1911 data, I applied them to the 5-year age-totals for Ontario in 1921 as well, in order to maintain a consistent methodology. The interpolated age-specific population totals are shown in Figure 3.4 along with the declared age-specific population totals from the 1921 census report (Dominion of Canada 1925). The process of interpolating the age-specific population totals has the effect of smoothing the heaped data. However, the smoothing is quite extreme and may be masking legitimate age-specific variation. There is no means to account for this other than by using different smoothing techniques; yet, in using any

smoothing technique there is no way to ensure that actual population variation is being maintained while the variation that is due to heaping is corrected. However, variation in the numerator is more important than heaping in the denominator due to the large population totals.

Figure 3.4 - Age Specific Population Totals of Ontario from the 1921 Census and Interpolated Age-Specific Population Totals.

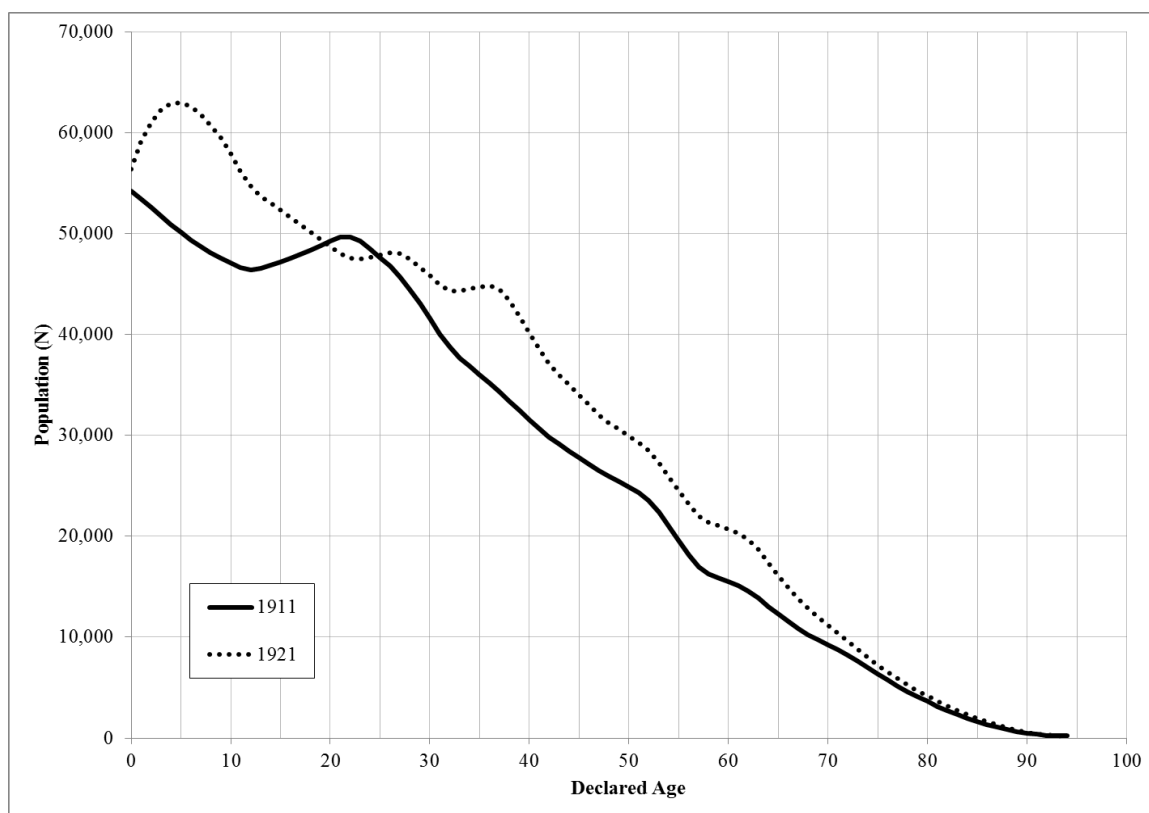


Note: Created using Sprague's Multipliers,

Source: Dominion of Canada (1925), N=2,929,103.

The 1911 and 1921 interpolated age-specific population totals for Ontario are shown in Figure 3.5. The unequal effects of population changes during this period can be seen in the unusually low numbers in the young adult population around the age of 22 in 1921.

Figure 3.5 - 1911 and 1921 Age-Specific Populations of Ontario.



Note: Interpolated from the 1911 and 1921 5-Year Population Totals using Sprague's Multipliers

Source: Dominion of Canada 1925, 1911 N=2,519,922, 1921 N=2,929,103.

With the establishment of age-specific population totals in both 1911 and 1921, the final step in creating mortality rates is the interpolation of the 1918 age-specific population. This was done using Waring's two-point formula (Judson and Popoff 2004, Appendix C), where $f(x)$ is the population in 1918, $f(a)$ is the population in 1911, and $f(b)$ is the population in 1921 and x is 1918, a is 1911, and b is 1921:

$$f(x) = \frac{f(a)(b-x) - f(b)(a-x)}{(b-x) - (a-x)}$$

The age-specific mortality rates, as compared to the frequencies of deaths at each age between 23 and 35, are shown in Figure 3.6. The frequencies of deaths and the mortality rates follow the same pattern and the peak at age 28 is maintained.

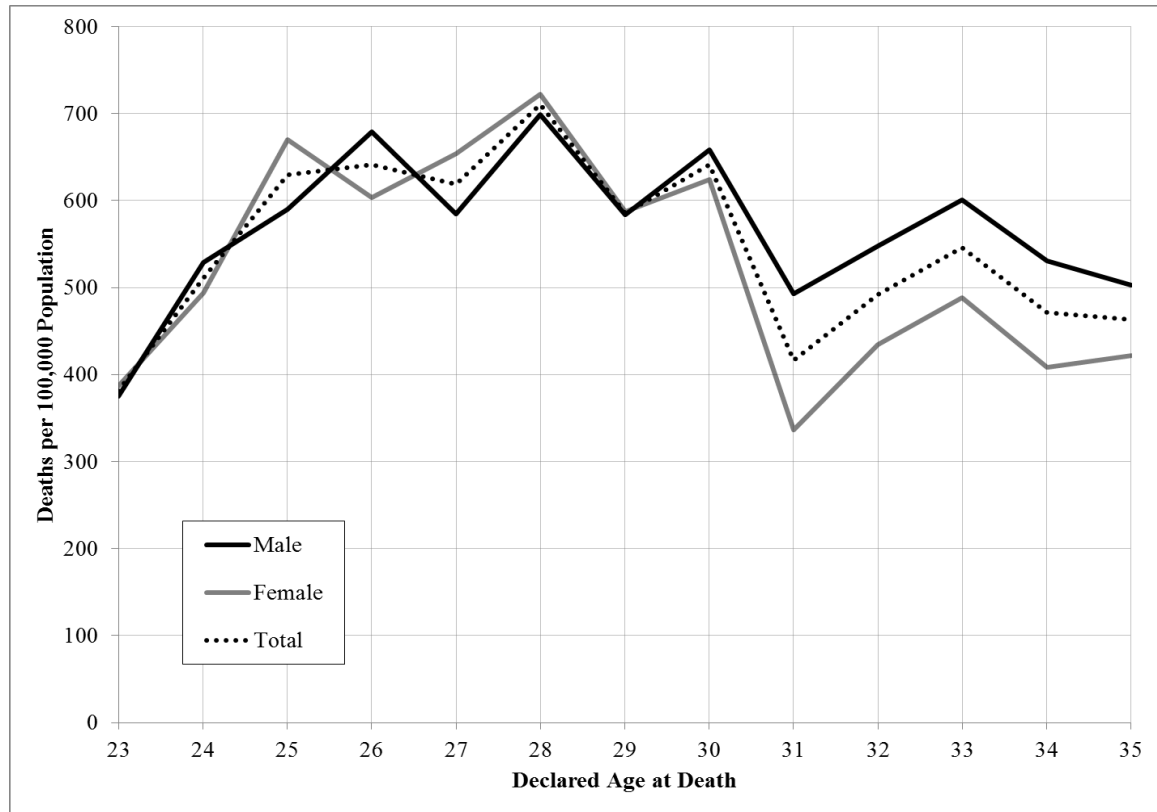
Figure 3.6 - Declared Age at Death from the Death Records in the WMMIP Database.



Note: For those with a declared age at death, N=3,230. Frequencies as Compared to Mortality Rates. Calculated using the 1918 Population Age-Specific Population Interpolated from the 1911 and 1921 Canada Censuses using Sprague's Multipliers and Waring's 2-Point Formula.

Mortality rates by sex are presented in Figure 3.7. While there is variation from ages 23 to 27, the rates are similar at the age of 28 and older, except for lower rates among women in ages above 30. As with the total mortality rate, the highest peak at age 28 remains the same when comparing mortality rates with frequencies of deaths. Due to the inaccuracies introduced by calculating the mortality rates, and due to the similar results from the death numbers to the death rates, the death numbers will be used in this dissertation.

Figure 3.7 - Mortality Rates by Sex for the individuals in the WMMIP Database.



Note: Using Declared Age at Death and the Interpolated 1918 Population Totals. Male (N=1,693) and Female (N=1,539).

3.3 Conclusion

This chapter discussed the materials used in the creation of the WMMIP database and the methods utilized in its creation. While there are biases with each record source and areas in which error can occur, this database compiles archival evidence that had previously been underutilized. With these cautions understood, the next three chapters analyze what can be learned from the WMMIP database. Chapter 4 addresses the database itself, including who was linked and who was not. Chapter 5 analyzes the relationship between the 1890 and 1918 influenza pandemics through declared age at death and the reconstructed age at death. Lastly, Chapter 6 uses the linked records to provide an understanding of the environmental circumstances of the decedents at various time points as compared to the rest of Ontario, to reveal if those who died were similar, or distinct from, the rest of the population.

Chapter 4

4 Assessing Data Quality: Using Ontario Historical Records to Study the 1918 Influenza Pandemic

This chapter addresses the following methodological research question:

1. *Through a historical demographic lens, are the extant historical records in Ontario suitable for demographic analyses of past infectious disease?*

The legislative history, coverage, and completion problems of the historical records used in this study were outlined in Chapter 3 and are highlighted where they appear to be problematic in Chapters 5 and 6. This chapter systematically addresses the quality of each source of extant records in terms of their suitability for use in historical-demographic analyses of past infectious diseases in general, as well as their applicability for studies that require exact age determinants.

It will specifically address the issues in relying on any one particular record to analyze declared age, outlining the benefits and deficits of each type of record. Most of these issues can be addressed by the kind of records linkage done for this study; however, this process requires more time than most researchers have available. This chapter begins with an analysis of the final Western, McMaster, Montreal Influenza Pandemic (WMMIP) database as created through the records linkage process. This is followed by analyses of each type of record as a source of historical data through comparison to each other and to the results of the records linkage process.

4.1 Final WMMIP Database

There are 3,316 decedents in the final WMMIP database, 1,743 male (52.6%) and 1,573 female (47.4%).⁴⁵ The declared ages at death range from 22 to 37 with 89 people of

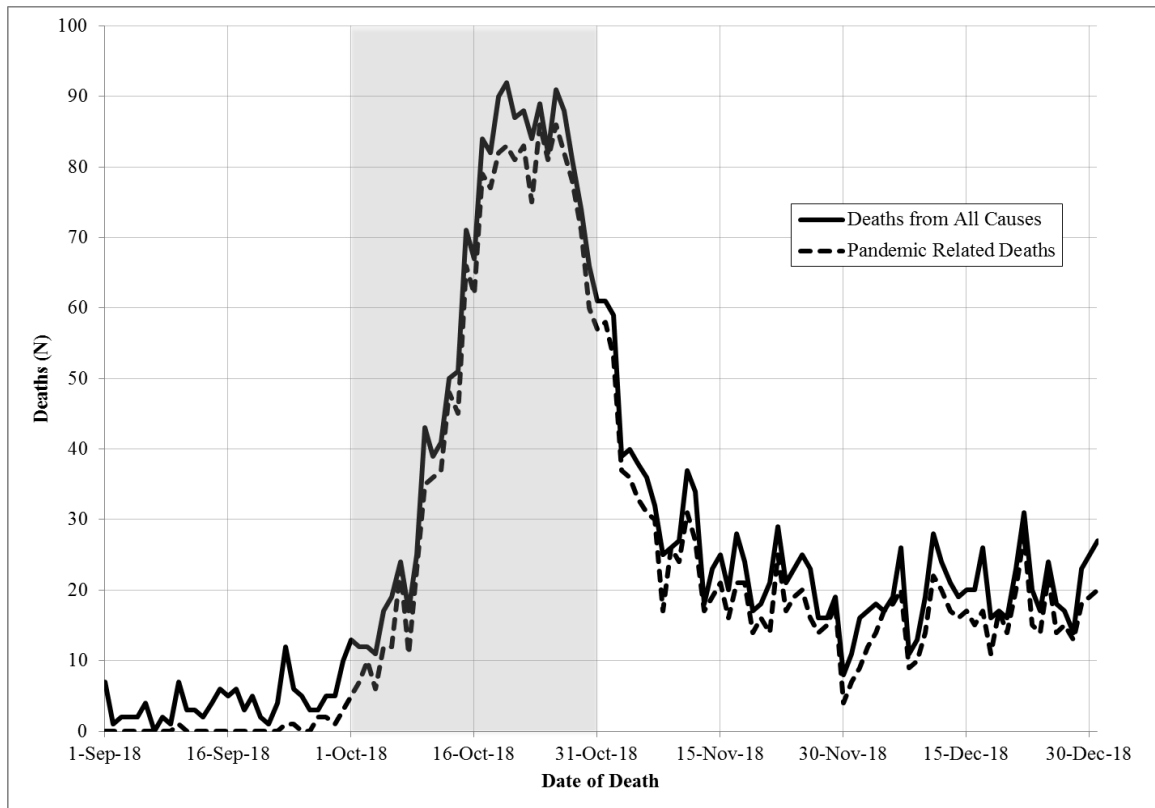
⁴⁵ Twenty individuals did not have a sex declared on the death record and these were imputed based on the likely gender of the given name. Ten were classified as Female and ten as Male. Only one name, Francis, was possibly unisex. However, of the 33 individuals who had either the name Francis or Frances as their first or second name on the

unknown age (the reconstructed ages at death range from 23 to 35 – see Section 5.1). Of the 3,316, 2,816 died of a pandemic related cause (84.9%; 1,318 female, 1,498 male), 200 had some form of tuberculosis (6.0%; 98 female, 102 male), and 71 died of an obvious pregnancy-related cause (4.5% of the women).

The distribution follows the expected epidemic curve (Figure 4.1) whereby the majority of deaths occurred in the month of October. There were 121 deaths in September (3.6%), 1,753 in October (52.9%), 828 (25.0%) in November, and 613 (18.5%) in December (one individual was found floating in Mohawk Lake, near Brantford, and his exact date of death was not known. He was included in the study because his death certificate was issued on October 1st, 1918). Figure 4.1 shows the number of deaths from all causes as well as deaths from epidemic related causes only with the majority of deaths during this time period being pandemic related. To show the similarities between the mortality rates and the frequencies of deaths, the epidemic curve is shown in Figure 4.2 using mortality rates per day.

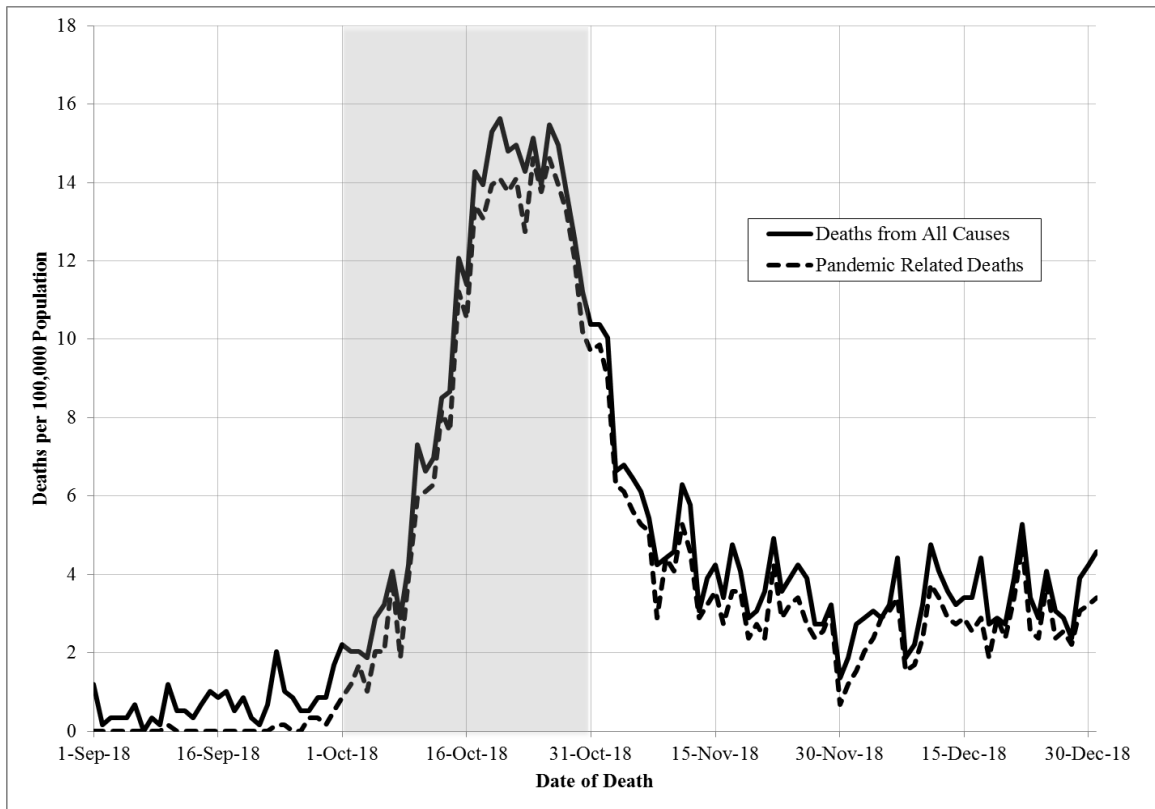
informant's return of death, 10 were female and 23 were male. Of the 10 females, 9 had the spelling Frances, while only one was named Francis. Every male with this name used the spelling Francis and therefore this individual was classified as male.

Figure 4.1 – Deaths from All Causes and Pandemic Related Deaths, Ontario, September to December 1918.



Note: For those in the final WMMIP database (N=3,313) and pandemic related deaths (N=2,814) for those with both a month and date of death. Highlighted section is October 1918.

Figure 4.2 – Death Rates from All Causes and Pandemic Related Deaths, Ontario, September to December 1918



Note: For those in the final WMMIP database (N=3,313) and pandemic related deaths (N=2,814) for those with both a month and date of death. Rates calculated by the total number of people in the interpolated population between the ages of 23 and 35 (N= 588,191.22). Highlighted section is October 1918.

Table 4.1 shows the distribution of the deaths in the final database by county, categorized by region in Ontario. The largest percentage of the deaths (17.3%) occurred in York County, which contained Toronto, the largest city at the time. Carleton County, which included Ottawa, accounted for 5.8% of the deaths and Wentworth, including Hamilton, had 5.1% of the deaths. No other individual county had more than 5% of the total deaths. Whether a death occurred in an urban or rural area was determined from the 1918 Report of the Registration of Births, Marriages, and Deaths (Legislative Assembly of Ontario 1919a:12). Any death that occurred in places designated as a City or a Town (population greater than 5,000) was considered to be an urban death. Deaths in other areas were

designated to have occurred in rural areas.⁴⁶ For the province as a whole, the deaths were fairly evenly split between rural (48.7%) and urban (51.3%) locations. There was much greater variation among the counties, however, with 17 counties having no deaths in urban areas while 84.8% of all deaths in York County were urban. While 48.7% of the total deaths from September to December, 1918, occurred in rural areas, 56.1% of the population was rural at the time. Of all the deaths, 51.3% occurred in urban areas, while only 43.9% of the population at the time lived in urban centres ($\chi^2=72.81$, $p<0.001$).⁴⁷

⁴⁶ Cities: Belleville, Brantford, Chatham, Fort William, Galt, Guelph, Hamilton, Kingston, Kitchener, London, Niagara Falls, Ottawa, Peterborough, Port Arthur, St Catherines, St Thomas, Sarnia, Sault Ste Marie, Stratford, Toronto, Welland, Windsor, and Woodstock. Towns: Barrie, Brockville, Cobalt, Collingwood, Cornwall, Ingersoll, Kenora, Lindsay, Midland, North Bay, Orillia, Oshawa, Owen Sound, Parry Sound, Pembroke, Renfrew, Smith's Falls, Sudbury, Trenton, and Walkerville.

⁴⁷ Unless otherwise specified, all statistical tests used in this dissertation are chi-square tests for independence.

Table 4.1 - Distribution of Death Records and Population by County.

Region	County/District	Death Records			Population ^a			
		N	%	% Urban	N	%	% Urban	
North	Algoma	35	1.1	65.7	51,680	1.8	37.9	
	Kenora	23	0.7	13.0	18,370	0.7	32.0	
	Manitoulin	20	0.6	0.0	11,465	0.4	0.0	
	Nipissing	48	1.4	45.8	40,700	1.5	21.0	
	Rainy River	7	0.2	0.0	11,875	0.4	0.0	
	Sudbury	61	1.8	41.0	45,560	1.6	15.2	
	Thunder Bay	38	1.1	71.1	40,900	1.5	84.6	
	Timiskaming	64	1.9	3.1	31,460	1.1	19.0	
Total		296	8.9	34.5	252,010	9.0	32.3	
Central	Dufferin	17	0.5	0.0	15,380	0.5	0.0	
	Haliburton	8	0.2	0.0	5,280	0.2	0.0	
	Hastings	75	2.3	45.3	58,610	2.1	32.7	
	Muskoka	32	1.0	0.0	18,090	0.6	0.0	
	Parry Sound	48	1.4	29.2	32,000	1.1	17.9	
	Peterborough	38	1.1	60.5	41,700	1.5	47.7	
	Prince Edward	13	0.4	0.0	15,610	0.6	0.0	
	Victoria	21	0.6	57.1	30,080	1.1	24.2	
Total		252	7.6	32.9	216,750	7.7	24.0	
East	Carleton	192	5.8	80.2	133,940	4.8	74.7	
	Frontenac	76	2.3	72.4	46,340	1.7	51.2	
	Lanark	37	1.1	35.1	32,880	1.2	20.0	
	Leeds and Grenville	79	2.4	39.2	51,090	1.8	18.5	
	Lennox and Addington	20	0.6	0.0	18,870	0.7	0.0	
	Prescott and Russell	89	2.7	0.0	52,990	1.9	0.0	
	Renfrew	86	2.6	47.7	56,350	2.0	26.4	
	Stormont, Dundas, and Glengarry	111	3.3	29.7	60,720	2.2	11.9	
	Total		690	20.8	47.4	453,180	16.2	35.7

*The estimated population in 1918 for the individual counties was taken from the 1918 Report of the Registration of Births Marriages and Deaths (Legislative Assembly of Ontario 1919a). The populations were combined from Tables 1 and 2 (1919a:1-12).

Table 4.1 cont'd - Distribution of Death Records and Population by County.

Region	County/District	Death Records			Population ^a		
		N	%	% Urban	N	%	% Urban
South-Central	Halton	19	0.6	0.0	22 120	0.8	0.0
	Lincoln	51	1.5	47.1	44,470	1.6	40.2
	Northumberland and Durham	42	1.3	0.0	54,460	1.9	0.0
	Ontario	48	1.4	43.8	42,480	1.5	23.0
	Peel	21	0.6	4.8	21,850	0.8	0.0
	Simcoe	127	3.8	54.3	77,780	2.8	28.7
	Welland	76	2.3	46.1	59,010	2.1	39.0
	Wentworth	170	5.1	79.4	147,020	5.3	74.2
	York	573	17.3	84.8	582,240	20.8	84.2
	Total		1127	34.0	68.4	1,051,430	37.6
South-West	Brant	86	2.6	58.1	53,860	1.9	52.8
	Bruce	46	1.4	0.0	45,470	1.6	0.0
	Elgin	55	1.7	54.5	44,930	1.6	35.2
	Essex	88	2.7	54.5	84,880	3.0	40.7
	Grey	42	1.3	33.3	69,260	2.5	28.4
	Haldimand	16	0.5	0.0	21,280	0.8	0.0
	Huron	43	1.3	0.0	47,880	1.7	0.0
	Kent	74	2.2	31.1	59,680	2.1	25.4
	Lambton	43	1.3	25.6	51,560	1.8	24.8
	Middlesex	138	4.2	76.1	102,290	3.7	55.0
	Norfolk	30	0.9	0	25,970	0.9	0
	Oxford	44	1.3	25.0	46,230	1.7	31.9
	Perth	49	1.5	57.1	50,040	1.8	30.9
	Waterloo	133	4.0	54.9	70,020	2.5	45.8
	Wellington	64	1.9	37.5	52,250	1.9	32.5
Total		951	28.7	43.8	825,600	29.5	31.7
	Province	3,316	100.0	51.3	2,798,970	100.0	43.9

*The estimated population in 1918 for the individual counties was taken from the 1918 Report of the Registration of Births Marriages and Deaths (Legislative Assembly of Ontario 1919a). The populations were combined from Tables 1 and 2 (1919a:1-12).

Related to the issue of rural and urban mortality are deaths that occurred in institutions (Table 4.2).⁴⁸ For the entire province, only 864 (26.1%) died in institutions and the remaining 2,452 (73.9%) died in some other place, often at home. In rural areas, only 9.3% of deaths occurred in institutions, while in urban areas institutions accounted for 41.9% of all deaths. Of the 487 individuals who died in Toronto, 194 (39.8%) died in institutions while 293 (60.2%) did not. Deaths in institutions are more subject to underreporting during the epidemic and contain less information for linkage (Section 3.1).

Table 4.2 - Deaths in Institutions by Location in Ontario.

	Death Occurred in Institution		Death Did Not Occur in Institution		Total
	N	%	N	%	
Rural	151	9.3	1,465	90.7	1,616
Urban	713	41.9	987	58.1	1,700
Toronto	194	39.8	293	60.2	487
Province	864	26.1	2,452	73.9	3,316

Of the 3,316 individuals in the WMMIP database, 812 (24.5%) did not have an occupation declared. The occupations of a further 675 (20.4%) were classified as a variation of ‘wife’ (for example, housewife, baker’s wife, farmer’s wife, or simply ‘wife’). Of the remaining 1,829 (55.2%), 634 (19.1) were unlikely to have been itinerant labourers and 1,214 (36.6) were possibly in itinerant occupations.⁴⁹

⁴⁸ Including hospitals, sanitarium, asylums, jails, and religious institutions.

⁴⁹ As classified by Stacey Hallman. Jobs not likely to be itinerant: At Home (various forms), Civil Servants, Farmers (also Farmers’ Sons, Farmers’ Daughters), Foremen, Managers, Masters, Merchants, and Professionals.

Jobs possibly itinerant: Agents; Artisans and Craftsmen; Assistants, Clerks, Hired Hands, or Employees; Auctioneers; Bookkeepers; Bushmen, Fishermen, Indians, Lumbermen, and Trappers; Cashiers and Salesmen; Clergy (any form), Collectors; Cooks; Dealers; Drivers; Electricians; Farm Hands; Foundrymen; Housekeepers, Housemaids, Domestic and Servants; Inspectors; Labourers; Mail Carriers; Masseuses; Mechanics; Miners; Nurses; Operators; Photographers; Rail-Road Employees (any form); Secretaries; Soldiers and Sailors (any form); Students; Superintendents; Teachers; Teamsters; Telegraphers; Tradesmen (various forms); Travelers; Waiters; and Watchmen.

As explained in Appendix A, the Access Database contains a different table for each of the various records that were linked. At times, there were multiple records for a single individual (such as for multiple marriages or multiple enlistments). Further, since every member of the household in which the decedent was living was transcribed for each of the three censuses in the study, the largest number of records was transcribed from the censuses. In total, there are 2,081 birth records, 2,879 entries from the 1891 census, 19,175 from the 1901 census, 14,174 entries from the 1911 census, 1,748 marriage records, and 193 attestation papers (there were also three entries from the 1916 prairie census).

Of the 3,316 death records in the final database, 2,965 (89.4%) were linked to *at least one* other record, meaning that 351 individuals could not be linked (10.6%, Table 4.3. See Section 4.2 for an analysis of these individuals).⁵⁰ There were 2,079 (62.7%) people who were linked to a birth (or baptismal) record, 2,737 (82.5%) linked to the 1901 census, and 2,424 (73.1%) linked to the 1911 census. Notably, 2,292 (69.1%) individuals were linked to both the 1901 and 1911 censuses, while 1,667 (50.3%) individuals could be linked to a birth record, and both the 1901 and 1911 censuses. The 1891 census records were transcribed for 444 individuals (13.4%) only. Since this census began on April 6th, 1891, it was only possible to be enumerated in this census if an individual was born before April 1891 (Dominion of Canada 1902). This applies to only 1,873 of the 2,965 linked individuals (63.2%). Their records were only transcribed when the 1891 census was necessary to establish a reconstructed age at death (due to either a lack of other information or conflicting data on date of birth). Therefore, the 444 individuals (13.4%) who were linked in 1891 do not represent the total number of individuals who could possibly be linked in future analyses.

⁵⁰ Including “positive”, “likely” and “maybe” links, cf. Section 3.2 and Appendix A.

Table 4.3 - Linkage Success by Type of Record (N=3,316)

Linkage	Linked		Unlinked	
	N	%	N	%
To <i>at least</i> one other record	2,965	89.4	351	10.6
Birth	2,079	62.7	1,237	37.3
1901	2,737	82.5	579	17.5
1911	2,424	73.1	892	26.9
Marriage	1,762	53.1	1,554	46.9
Attestation	188	5.7	3,128	94.3
1901 and 1911	2,292	69.1	1,024	30.9
Birth, 1901, 1911	1,667	50.3	1,449	43.7

Of the individuals declared to be ‘married’ (1,972), the marriage records were found for 1,665 (84.4%) individuals and they could not be located for 307 (15.6%) people. The marriage records for both of the people who were declared to be ‘divorced’ were found. For the individuals who were ‘widowed’ (71), the marriage records could be found for 55 (77.5%), while the remaining 16 (22.5%) could not be found. Of the 1,142 individuals who were declared to be ‘single’, 7 (0.6%) could be linked to a marriage record, another indication of the possible error in these sources, or in the records linkage process. Finally, of the 129 people who did not have a marital status declared on their death record (who may or may not have been married), marriage records were found for 33 (25.6%) people and were not found for 96 (74.4%).

Of the individuals whose death records in some way indicated that they were a soldier (115, 3.5%),⁵¹ 84 (73.0%) were linked to an attestation paper and 31 (27.0%) could not

⁵¹ Indications that an individual was a soldier include occupations of: Cadet, Cadet RAF, Captain Militia, Farmer and Soldier, Labourer and Soldier, Lieut. RAF, Retired Soldier, Returned Soldier, Sailor (although included, this occupation might not necessarily have referred to individuals in the navy), Soldier, and Munition Worker. Also

be linked. As per Section 3.1, the only records available to be linked were from the Canadian Expeditionary Forces (CEF). Some of these individuals' records indicated that they were in the Royal Air Force (RAF), so their military records would not be expected to be found. The remaining 3,201 death records did not provide any indication that the decedent was in the CEF; but, it was possible to link 104 (3.3%) of those people to attestation papers.⁵²

With 89.4% of the death records being linked to *at least one* other record, the linkage rate of this study compares favourably to other historical records linkage projects. With the completeness issues addressed in Chapter 3, all records are not expected to be found and linked for all individuals. As explained by Hollingsworth, “the proportion of families that have been reconstituted in any parish using the Fleury-Henry method [of family reconstitution] is usually disappointingly small. It rarely reaches 10 per cent, and the main reason for this seems to be migration from parish to parish [where the adjoining parishes have not yet been analyzed]” (1969:181). When looking at more closed communities, “in Gautier and Henry’s pioneer study of Crulai, migration could largely be discounted by keeping only to those married couples both of whom had been baptized in the parish.” When this was done, “Only 14 per cent of men and 22.5 per cent of women were then left whose age at death was unknown, and only 3 per cent of men and 5 per cent of women had no indication at all of their subsequent careers after marriage” (Hollingsworth 1969:186). This is summarized by Wrigley, who states that “perfect accuracy is beyond attainment in historical record linkage” (1973:14). Knodel (1988) and Smith (1973) had family reconstitution rates of closer to fifty percent. Yet, record linkage outside of family reconstitution studies has been generally more successful.

When Emery and McQuillan (1993) linked death records from Ingersoll, Ontario, to “newspaper death notices, community directories, and manuscript censuses” (1993:63) in order to classify individuals by occupation, they were able to classify 71% of individuals

included were individuals who died in a Military, Base, or Camp Hospital, and one individual whose given name was Major John.

⁵² The individuals may have been returned soldiers, may have died in some place other than a military base, or be similar to one individual who deserted in 1916.

in their sample from the 1871-74 period and 85% of the individuals in their sample from 1881-1884. Through a family reconstitution project on Anglican parishes in England, Wrigley et al. (1997:54) were able to link 75% of burials in their selected parishes to their Family Reconstitution Forms, which required that an individual already be linked to either a preceding marriage or baptismal form (towards the end of the study period, 1580-1837). Norton successfully linked 71.3% of male-headed census families to families reconstructed from vital records in three Massachusetts towns from 1790-1850 (1980:13). Using the death records of children in Connecticut in the 18th and 19th centuries, Swedlund and colleagues were only unable to link “slightly over 10% for Shelburne and almost 20% for Deerfield” (1980:143): to censuses, family genealogies, and taxation lists. Therefore the linkage rate of this study is comparable to other studies in historical demography and represents a successful research project. The following section analyzes those 351 death records of individuals who could not be linked.

4.2 Linkage Analysis

The process of tracing persons will almost always involve a process of selection; simply put, in almost every society, some persons are more stable and more visible than others . . . The investigator must consider the chance that records linkage, the very successes of her research, have introduced biases into her data . . . At least she must determine where in her conclusions she should be cautious. [Herlihy 1973: 41, pronouns changed to feminine]

It was not possible to link 351 individuals in the WMMIP database. Of these 351 individuals, 68 (20.4%) did not have a declared age at death on the death record. Individuals without a declared age a death were originally included in the sample unless there was some obvious indication of age somewhere else on the record (for example, an occupation of “infant”, or a cause of death such as “premature” or “old age”). If it could be verified from another record that the individual was younger than 23 or older than 35, that individual was removed from the database. However, the records of the 68 individuals without a declared age at death and who could not be linked were not excluded because it was not possible to ascertain their age from another record.

Although they may have died outside of the ages of 23 to 35, this was not possible to determine, so they remain in the database as unlinked individuals.

In addition to age, the two other inclusion criteria for this sample were that individuals were both born and died in Ontario. Information on place of death was not declared for 23 of the 351 individuals (in total, 41 individuals in the entire sample did not have a declared place of death). However, all of these deaths were registered in Ontario and there was nothing to indicate on the records themselves that the deaths occurred in another location.

A birth having occurred in Ontario was more difficult to establish than was death in Ontario. Of the 351 unlinked individuals, the declared place of birth for 158 was indicated as being somewhere in Ontario.⁵³ However, the declared place of birth for a further 193 individuals was not somewhere in Ontario (once again, these individuals were originally included since they potentially could have been born in Ontario. Individuals were only eliminated from the database when it could be proven that they were *not* born in Ontario). Of these 193 individuals, 70 had a declared place of birth of “Canada,” 109 did not have this information filled in, and 14 were either “do not know,” “na,” “not declared,” “not known,” or “unknown”. Fifty-seven individuals were missing both age and place of birth information entirely, while there were 8 individuals whose ages were approximate (for example, “about 26” or “about 35”) and who were also missing information on place of birth.

The physician’s return of death was left blank for 28 individuals (the bottom of the form) and the informant’s return of death was blank for 14 individuals (the top of the form). Seven women did not have a declared given name and were only referred to as the wife of their husband, for example, Mrs. Andrew Carrier or Mrs. Francis Mejaki. One individual was adopted and it could not be ascertained whether the names of her parents referred to her birth parents or her adoptive parents. At least 4 individuals were members

⁵³ The place of birth for 17 individuals was simply “Ontario.” For one individual, the best interpretation of what was written for place of birth was “Rosmonky,” which was hypothesized as possibly being a misspelling of Rimouski; however, it was not possible to find a source which definitively located his birth in Quebec and thus he was not able to be excluded.

of the clergy, which presents difficulties in determining name at birth. One individual had no identifying information other than sex, since he was found in Mohawk Lake. The contemporary authorities were unable to identify him; therefore, he was impossible to link to any record. The notes by the research assistants conducting the linkages suggest that some of the individuals were likely not born in Ontario or were outside of the required age range, yet this could not be proven. Of the 285 individuals with a declared place of burial, two were buried in Quebec, possibly indicating that they had been born in that province. One married woman died in Toronto's Western Hospital and was declared to have been born in "Canada". She was buried in Prince Edward Island however, suggesting she also may not have been born in Ontario. These individuals represent problems with establishing the inclusion criteria rather than problems with the records linkage process.

Table 4.4 indicates the county in which the death was registered for all the 351 individuals who were not linked, separated by region of Ontario. Most notably, 39.9% of individuals in Northern Ontario were not able to be linked (33.6% of the unlinked records). This may be due to the more frequent reorganization of the districts in the North, that the highest percentage of itinerant workers was in the North,⁵⁴ and that the largest number of aboriginal Canadians who died was from Northern Ontario (38, 46.3%). Successful linkages were fewer for the death records of aboriginal Canadians than for the records of non-aboriginal Canadians (see Table 4.4).

⁵⁴ The percentage of all workers in each region with jobs that were possibly itinerant are: North = 43.9%, Central = 34.1%, East = 33.9%, South-Central = 38.2%, and South-West = 35.0%.

Table 4.4 - Records that could not be Linked by County of Death Registration.

County/ District	Records (N)	County/ District	Records (N)	County/ District	Records (N)
North		East		South-West	
<i>Algoma</i>	12	Carleton	18	Brant	6
<i>Kenora</i>	10	Frontenac	7	Elgin	4
<i>Manitoulin</i>	5	Leeds and Grenville	5	Essex	5
Nipissing	8	Prescott and Russell	10	Grey	2
<i>Rainy River</i>	4	Renfrew	9	Haldimand	1
<i>Sudbury</i>	23	Stormont, Dundas, and Glengarry	8	Lambton	1
Thunder Bay	18			Middlesex	5
<i>Timiskaming</i>	38			Oxford	3
				Waterloo	3
				Wellington	6
Total (%)	118 (33.6)		57 (16.2)		36 (10.3)
County/ District		Records (N)			
Central		South-Central			
Hastings	6	Lincoln	3		
<i>Muskoka</i>	5	Peel	1		
Parry Sound	8	Simcoe	7		
Peterborough	3	Welland	3		
Prince Edward	1	Wentworth	11		
Victoria	1	York	87		
Total (%)	24 (6.8)		116 (33.0)		
Province (%)	351 (100)				

Note: Counties in italics did not exist throughout the entire study period, 1883-1918. Kenora District was created in 1909 out of parts of Rainy River District, Rainy River District was created in 1885 from parts of Thunder Bay District, Sudbury District was created in 1907 out of Algoma District, Manitoulin District was created in 1888 out of Algoma District, Muskoka was created in 1888 from the Unorganized Territories, and Timiskaming District was created in 1912 from parts of Algoma, Nipissing, and Sudbury (Archives of Ontario 2004).

The differences between the unlinked individuals (N=351) and the linked individuals (N=2,965) is shown in Table 4.5, cross-classified by the demographic characteristics of

the decedents, the circumstances surrounding the death, and the condition of the records themselves. The four important demographic characteristics are hypothesized to be: sex, declared years of age, marital status, and indigenous status. Of the 351 unlinked individuals, 141 were declared to be female, 192 (54.7%) were declared to be male, and 18 individuals were missing information on sex. Of those, 10 were identifiable as female through their given name and 8 were identifiable as male, giving a total of 151 female deaths (43.0%) and 200 (57.0%) male deaths. Among the linked individuals, 1,422 (48.0%) were female and 1,543 (52.0%) were male, a difference which is statistically significant ($\chi^2=3.07$, $df=1$, $p=.079$) at $\alpha=.1$. The average declared age of the individuals was slightly higher among the unlinked individuals (two-tailed t-test for means, $t=-1.97$, $df=432.07$, $p=.05$), which may represent the improving coverage of the birth records over time and that older individuals were more likely to have left their homes of birth by the time of the censuses and to have potentially changed their names through marriage (Section 3.1).

Table 4.5 – Descriptive Statistics of Linked and Unlinked Records.

		Linked		Unlinked		χ^2	df	p
Demographic Features		N	%	N	%			
Sex	M	1,543	52.0	200	57.0	3.07	1	.08**
	F	1,422	48.0	151	43.0			
Declared Age	Mode	28		28		2.02 ^a	3314	.05*
	Median	28		29				
	Mean	28.7		29.1				
	SD	3.5		3.6				
Marital Status	Single	1,038	35.7	104	36.9	0.15	1	.70
	Ever-Married	1,867	64.3	178	63.1			
Indigenous	Y	53	1.8	29	8.3	54.55	1	<.001*
	N	2,912	98.2	322	91.7			
Soldier	Y	103	3.5	12	3.4	0.003	1	.96
	N	2,862	96.5	339	96.6			
Itinerant Occupation	Y	1,071	46.3	143	69.1	39.63	1	<.001*
	N ^b	1,244	53.7	64	30.9			
Conditions of Death								
Flu	Y	2,531	85.4	285	81.2	4.25	1	.04*
	N	434	14.6	66	18.8			
Tuberculosis	Y	176	5.9	24	6.8	0.45	1	.50
	N	2,789	94.1	327	93.2			
Urban	Y	1,510	50.9	190	54.1	1.29	1	.26
	N	1,455	49.1	161	45.9			
Toronto	Y	415	14.0	72	20.5	10.64	1	.001*
	N	2,550	86.0	279	79.5			
Institution	Y	724	24.4	140	39.9	38.97	1	<.001*
	N	2,241	75.6	211	60.1			
Region	North	178	6.0	118	33.6	294.40	1	<.001*
	Not-North	2,787	94.0	233	66.4			
State of Death Record								
Occupation	Y	1,662	56.1	167	47.6	9.12	1	.003*
	N	1,303	44.1	184	52.4			
Father's Name	Y	2,689	90.7	132	37.6	696.43	1	<.001*
	N	276	9.3	219	62.4			
Mother's Name	Y	2,379	80.2	75	21.4	565.37	1	<.001*
	N	586	19.8	276	78.6			

* p<.05

**p<.10

^a. t-value calculated from a two-tailed t-test for means.^b. Includes the category of 'wife'.

Among the unlinked individuals 166 (47.3%) were married, 104 (29.6%) were single, 12 (3.4%) were widowed, one individual was “married but husband overseas”, and there were 69 (19.7%) who were “not-known” or left blank. Collapsing these categories into ‘single’ and ‘ever-married’ gives a 104 (36.9%) of the unlinked who were single and 178 (63.1%) who had ever been married. Similar statistics for the linked records are 1,038 (35.7%) single and 1,867 (64.23%) ever-married, while 60 (2.0%) were unknown. These differences are not statistically significant ($\chi^2=0.15$, $df=1$, $p=.701$)⁵⁵.

Of the 351 unlinked individuals, 29 (8.3%) were identifiable as aboriginal either through location of death or location of the death registration, location of burial, or from an occupation declared as “Indian”. However, only 53 (1.8%) of the linked individuals were aboriginal. These differences are statistically significant ($\chi^2=54.55$, $df=1$, $p<.001$). Aboriginal Ontarians were more difficult to link for various reasons. They were less likely to have a birth certificate (birth or baptismal records were found for only 2, or 2.4% of the total 82 aboriginal decedents in the database), the spelling of names was inconsistent from record to record, the date of birth was inconsistently recorded in the censuses (perhaps because it was an estimation) and the death record itself was often incomplete. These issues are addressed in Section 4.3.

Concerning the conditions surrounding the death itself, 285 of the 351 unlinked individuals (81.2%) died of a pandemic related cause, while 2,531 of the 2,965 linked individuals deaths were related to the pandemic (85.4%, $\chi^2=4.25$, $df=1$, $p=.039$). Twenty-four (6.8%) of the unlinked had tuberculosis while 176 (5.9%) of the linked were declared to have had the disease ($\chi^2=0.45$, $df=1$, $p=.50$). The urban or rural location of the deaths were not statistically significantly different among the linked and unlinked individuals ($\chi^2=1.29$, $df=1$, $p=.256$). However, there was a significant difference among the linked and the unlinked for those who died in Toronto ($\chi^2=10.64$ $df=1$, $p<.001$). Of the 87 unlinked individuals who died in York County (Table 4.3), 72 (20.5%) died in the city of Toronto, while only 415 (14.0%) of the linked individuals died in the city. The unlinked represent 14.8% of all the individuals who died in Toronto. This is important

⁵⁵ Does not include the records of unknown marital status. When this is included, the chi-square test is statistically significant ($\chi^2=261.16$, $df=2$, $p<.001$). Missing information made the records-linkage process more difficult.

because it was more difficult to link residents who lived in cities, as there were more possible matches. Further, of those 72, 41 (56.9%) died in one of the cities hospitals or institutions, higher than the city total of 670 (23.7%) who died in institutions. For the province as a whole, there were more individuals among the unlinked who died in institutions, which is statistically significant ($\chi^2=38.97$ df=1, $p<.001$), again suggesting underreporting caused by the epidemic

The records of the individuals who died in hospital were more likely to be incomplete or inaccurate and therefore more difficult to link. For example, it was noted in the remarks section for two individuals who died in the Emergency Hospital in Timmins that “the above named were taken to the emergency hospital and we did not get the required information. They were attended to by Dr. Moor, McInnis, or Minthorn.” Another individual at the same hospital had the remark on her death record that “the above answers not given are not known. The patients were attended by whichever ones of the doctors who visited the emergency hospital each day.” This may be an example of underreporting during epidemics discussed in Section 3.1. Of the 17 individuals who died in jails, it was not possible to link 10 (58.8%) of them. This was also a result of the incompleteness of the records.

Among the unlinked records, there were 143 individuals who had occupations which could involve travelling (69.1%, and thus a fewer number of potential individuals to report accurate information following their death). The higher percentage of individuals with possibly itinerant occupations among the unlinked is statistically significant ($\chi^2=39.63$ df=1, $p<.001$). As the majority of these individuals were single or widowed (56.0%, 18 of these individuals did not have a declared marital status), it would be more likely that they were travelling for work. This may also explain why many of the unlinked deaths occurred in the major city of Toronto and in Northern Ontario (Table 4.3 and Section 6.2).

The conditions of the death records themselves were also important in determining whether a death record could be linked to another record or not. More of the unlinked individuals had records that were not complete. For those who were linked, 1,662

(56.1%) had an occupation declared while this was true for only 167 (47.6%) of the unlinked (importantly, there was a *smaller* percentage of women among the unlinked). This difference is statistically significant ($\chi^2=9.12$ df=1, $p=0.03$). Similarly, a far smaller percentage of the unlinked had the names of their parents declared on their death record, which provided the research assistants with fewer data-points on which to conduct the linkage (both are statistically significant using chi-square tests. For father's name declared: $\chi^2=696.43$, df=1, $p<.001$, and for mother's name declared $\chi^2=565.37$, df=1, $p<.001$).

Table 4.6 presents the results of five logistic regression models including those characteristics from Table 4.5 that were statistically significant at $\alpha=0.1$. These are sex (0=male, 1=female), age (continuous: min=22, max=37), indigenous status (0=no, 1=yes), if the individual had a possibly itinerant occupation (0=no, 1=yes), whether a death was pandemic related (0=no, 1=yes), occurred in Toronto or an institution (0=no, 1=yes), whether the death occurred in Northern Ontario (0=no, 1=yes), and whether the occupation, father's name, or mother's name was declared on the death record (all: 0=no, 1=yes). The dependent variable is whether or not the death record was linked to at least one other record (0=no, 1=yes), with odds ratios greater than 1.00 indicating a higher likelihood of being linked and odds ratios less than 1.00 indicating a lower likelihood of being linked (or thus, a higher likelihood of being unlinked). Models were estimated in a sequential manner in order to determine the various impacts of demographic factors, the environment surrounding the death, and missing information on the death record.

Table 4.6 - Logistic Regression of Records Linked to at Least One Other Record, by Demographic Features, Conditions Surrounding Death, and Conditions of the Death Record.

	Model 1	Model 2	Model 3	Model 4	Model 5
	OR	OR	OR	OR	OR
Female	1.15	1.08	1.07	1.60	0.70
Age	0.96**	0.96**	0.99	0.98	0.97
Indigenous	0.19***	0.24***	0.24***	1.21	1.02
Flu		1.19	1.17	1.13	1.22
Toronto		0.38***	0.99	1.03	1.50
Institution		0.36***	0.53***	0.53***	0.67**
North Region		0.15***	0.20***	0.25***	0.26***
Occupation			1.21	1.19	0.83
Father's Name			4.11***	3.87***	3.74***
Mother's Name			4.50***	3.29	2.07
North				0.08***	0.08***
Region*Indigenous					
Mother*Father				1.51	2.45
Itinerant					0.59**
Model χ^2	38.73***	222.94***	532.75***	544.18***	382.79***
Pseudo R Square	.020	.116	.278	.284	.292
AIC	1,887.26	1,711.05	1,407.24	1,399.81	956.56
BIC	1,911.58	1,759.69	1,474.11	1,478.84	1037.99
N	3,227	3,227	3,227	3,227	2,481

*** p<0.01

**p<0.05

*p<0.1

Model 1 in Table 4.5 presents the demographic features that were significant in Table 4.4 (having an possibly itinerant occupation was not included until Model 5 because it reduces the total N from 3,227 to 2,481). While controlling for sex, having a lower age and being indigenous were both associated with lower odds of being linked. For every

one year increase in age, the odds of being linked decreased by 4% and indigenous status decreased the likelihood of being linked by a factor of five (OR=0.19). Model 2 adds in the conditions surrounding the death into the demographic features model. Age and indigenous status remain significant, but indigenous status now divides the odds of being linked by four (OR=0.24). While controlling for demographic features, dying in Toronto decreases the odds of being linked by 62%, dying in an institution decreases the odds of being linked by 64%, and dying in Northern Ontario decreases the odds of being linked by 85%.

Model 3 adds in the conditions of the death records themselves to the demographic features and environment in which the death occurred. Taking into account the condition of the records themselves, neither age at death nor death in Toronto remain significant, which means that parents' names were less likely mentioned for older individuals and for Toronto residents, contributing to why record linkage was less likely for these individuals. On the other hand, indigenous status, death in an institution, and death in Northern Ontario continue to decrease the odds of being linked. Whether an occupation was declared on the death record did not significantly affect the odds of being linked. However, having the parents' name declared on the death record has a very strong impact on the odds of being linked. Father's and mother's name respectively multiplied these odds by 4.1 and 4.5. This may indicate that much more of the record was incomplete than just the parent's name, or be the result of fewer data-points on which to link to other records.

Model 4 introduces interaction terms between indigenous status and a death that occurred in the North, since the highest percentage of deaths among aboriginal Canadians occurred in Northern Ontario. Also, there is an interaction term between father's name declared and mother's name declared, since having one of these pieces of information missing may increase the odds of having the other missing as well. Since this interaction term is not significant, having either the mother's or the father's name missing decreases the odds of being linked, but having both data missing does not further decrease the odds of being linked.

In model 4, the interaction term between indigenous status and region is significant. The odds of being linked are lower if an individual is both indigenous and died in Northern Ontario than for an indigenous person who died outside of Northern Ontario. This suggests that place of death may be more important in terms of linkage than indigenous status itself and that it is easier to link indigenous people if they did not reside in the north.

The final model, Model 5, takes into account all the previous elements, but adds in whether or not an individual possibly had an itinerant occupation. This was added last, because of the high level of missing data from this variable, meaning that the total sample size reduces from 3,227 (smaller than 3,316 because of the records that do not include age at death) to 2,481. However, this model had the smallest AIC and BIC suggesting that it is a better model (Tabachnick and Fidell 2007) and a higher pseudo R^2 , meaning that it explains a higher percentage of the variation in whether or not an individual record was linked (29%). Having both the mother's and father's name missing were not important and being both indigenous and from the north decreases the odds of being linked. Last, having a job that is possibly itinerant decreases the odds of being linked by 41%. This means that it was not just deaths among aboriginal Canadians that increased the risk of being unlinked in the North, but was probably also influenced by the influx of young individuals into the region for jobs (Chapter 6) and the reorganization of the North, such that some early records were either not kept, or not the responsibility of the Government of Ontario (Chapter 2).

In these models, at most, only 29.2% of the variation in whether or not a record could be linked is explained. The rest is unexplained heterogeneity which can be the result of many factors. For example, some of the pages in the registration books were destroyed by ink or water, at times data were transcribed incorrectly on one of the many iterations of a records, individuals may have had differing personal beliefs about government registration, and death records may have been included that should not have been, such as for immigrants for whom there are no other records to which linkage could be possible. Finally, late registrations may have been issued for these individuals had they lived long enough to need to prove their age (Chapter 3). While many of these factors cannot be

accounted for, the 29.2% of the variation that can be explained does appear to have some systematic biases, in directions expected by other research (Kuczynski 1930, Emery 1993).

Only 351 (10.6%) of the death records in the study could not be linked to at least one other record. The logistic regression indicates that these records represented individuals who were systematically different from those who could be linked in several important ways. They were more likely to have died in Northern Ontario, more likely to be aboriginal, and more likely to have had missing information on their death records. Those who died in institutions and who had jobs that were possibly itinerant were also more likely to be unlinked. It is known that certain groups are underrepresented in historical Ontario vital and census records, such as Aboriginal Canadians, Immigrants, and the impoverished (Kuczynski 1930, Emery 1993). While this study necessarily excludes immigrants, it confirms the lack of completeness among the records of Aboriginal Canadians. Social class is harder to study, but this task is undertaken in Chapter 6. While it is difficult to account for these biases, it is important to recognize that they do exist and that it is necessary to be cautious with the conclusions drawn from this study. However, there were no rural/urban biases in the records, nor any biases by sex. The linkage of deaths caused by the influenza epidemic was no different from deaths caused by other diseases, suggesting no bias in linkage by cause of death, which would be indicative of sampling error. Further, underreporting of the deaths in hospitals is likely due to doctors and hospitals being overwhelmed during the epidemic and may be corrected in future study by the examination of extant hospital records or military records (such as ongoing research by Bogaert). Underreporting of deaths of Aboriginal Canadians and those in the north could be addressed by detailed archival research, such as was done by Herring (1993) in Norway House, Manitoba, for the 1918 influenza pandemic and by Moffat (1992) in Fisher River, Manitoba, for the period 1907-1939. Future analyses may focus solely on those 351 records that could not be linked. However, this study examines the trends that were found among the 2,965 (89.4%) of the records that could be linked while recognizing that there may be important differences between these individuals and the 351 unlinked decedents.

4.3 Analysis of the Extant Records Sources

4.3.1 *Age*

The following section addresses the accuracy of the birth records, 1901 and 1911 censuses, and the death records, especially in comparison to the reconstructed age at death. Since many historical analyses of infectious disease depend on these records, including studies of the 1918 influenza pandemic, it is important to have an understanding of their utility for this type of research.

Chapter 5 examines the age at death as compared to the reconstructed age at death as determined by this research process. This section analyzes the reconstructed age at death and the date of birth on the birth record and the declared age on the 1901 and 1911 censuses. It also compares the vital and census records to each other in order to detail both the accuracy of each type of record, as well as their utility as an objective measure of declared age.

Once all the records that could possibly be found for an individual were transcribed and given a record linkage score, the research assistant determined the exact date of birth based on those records (see Appendix A for a description of this process). If all dates and ages were consistent, that date of birth was entered for the reconstructed date of birth; however, if there were some discrepancies between the dates or ages, the birth record was taken as correct, unless there was an obvious reason not to do so (Chapter 3). After the reconstructed date of birth was established, the research assistant identified whether all of the ages across all of the records for that individual were the same or if any differed. Table 4.7 shows the results of this process. All ages across all linked records for an individual were the same in only 1,209 (40.8%) of the 2,965 linked records. This means that there was at least one age that was different among the linked records for 1,756 (59.2%) of the decedents. This could indicate a problem for research that depends on age at death, if the declared age for a particular individual is not consistent on records taken throughout their life (see Chapter 3 for a discussion of age heaping).

Table 4.7 – Age Consistency across Linked Records.

	Ages the Same		Ages Different		Total Linked Records	
	N	%	N	%	N	%
Records	1,209	40.8	1,756	59.2	2,965	100

Of the 2,060 individuals whose birth records were “positive” or “likely” links (PL links, see Chapter 5, Appendix A), the declared date of birth was different from the reconstructed date of birth for only 106 records (5.1%, Table 4.7). The high correspondence between the declared date of birth on the birth record and the reconstructed date of birth is an artifact of the calculation of the reconstructed age at death (Appendix A). If there was a disagreement between the date of birth on any of the records, the age or date on the birth record was almost always taken to be correct since, in almost all cases, the birth record was the document that was created closest in time to the event itself. However, some birth records contained errors. There were also instances when the birth record was the only linked record that was different and there was overwhelming evidence that the birth record was incorrect. This explains the 106 (5.1%) individuals with both a declared date of birth and a reconstructed age at birth where there is some discordance between the two.

Table 4.8 - Correspondences among Dates of Birth.

Correspondences with Birth Records (PL Links N=2,060)													
	Year, Month, Day		Year, Month only		Year Only		Month Only		None		Not Linked to Birth Record		
	N	%	N	%	N	%	N	%	N	%	N	%	
1901 Census PL Links (N=2,703) ^a	1,042	53.7	332	17.1	66	3.4	78	4.0	74	3.8	1,219	37.1	
1911 Census PL Links (N=2,382) ^b	N/A	N/A	1,015	59.7	92	5.4	454	26.7	95	5.6	1,217	37.2	
Reconstructed Date of Birth (N=2,965) ^c	1,954	66.3	34	1.2	8	0.3	3	0.1	7	0.2	807	38.8	

Note: Rows do not sum to 100% because this analysis does not include those records where there were inconsistencies in the day of birth. Records that were off by less than a month would have a very limited effect on the error surrounding birth during the 1890 epidemic. Those records where only the day match include so much error that the consistency in the day is not useful for analysis.

^a. Both Birth Record and 1901 Census Record Linked, both PL Links N=1,939

^b. Both Birth Record and 1911 Census Record Linked, both PL Links N=1,700

^c. For Birth Records with a Reconstructed Age at Death N=2,946

When only considering the year and month of birth,⁵⁶ there are 11 records (0.5%) where either the year or the month is incorrect and 7 (0.3%) records where there is no correspondence between the reconstructed date of birth and the date of birth declared on the birth record. The most variation for the month and the year is by only one year, or one month.

It is difficult to draw conclusions about the accuracy of the birthdate from the birth record as compared to the reconstructed age at birth, since it was usually based on the birth record. However, it can be compared to the date of birth as given in the 1901 census, since this was the only other record which asked for the complete date of birth. Table 4.8 shows the comparison between the date of birth on the birth record and the date of birth

⁵⁶ Errors in the date of birth would have little effect on the error in calculating exposure to the 1890 pandemic. The day can only be off by a maximum of 30 days and the epidemic was in Ontario for months. There is some error when the date is incorrect, but it does not compare in magnitude to errors in the month or year of birth.

as declared on the 1901 census using only PL links for both the birth records (N=2,060) and for the 1901 census (N=1,703).⁵⁷ Additionally, the birth record had to have been linked to the 1901 census records. This gives a total sample of 1,939. Of those 1,939, 1,042 (53.7%) of the records declared the same date of birth on the birth record and the 1901 census, while another 332 (17.1%) varied only by the day. This gives 1,374 (70.9%) matches that can be considered correct within an acceptable level of error. The year was the only matching datum for 66 (3.4%) and the month was the only correspondence for 78 (4.0%) of the records. Finally, there were 74 linkages (3.8%) for which there were no correspondences between the day, the month, or the year, even though they were both considered to be “positive” or “likely” links.

There were 166 PL linkages (8.6%) where the month of birth did not correspond with the declared month of birth from the 1901 census. The average number of months difference was 3.2 months, with a minimum of 1 month, a maximum of 11 months, and a median of 2 months. Further, there were 454 (23.4%) PL links where the year did not correspond. The average difference was 1.6 years, with a minimum of 1 year, a maximum of 10 years, and a median of 1 year. Throughout this records linkage research, we have assumed that (in almost all cases), the date of birth as declared on the birth record was the most likely to be accurate. Further, the birth record was to be completed by someone associated with the birth, while the census declaration could be made by someone in the household, even when they were not certain of a lodger’s or employee’s date of birth (Chapter 3). This assumption could be contested in future research; however, if choosing to use only one record as evidence of date of birth, it is important to realize that there is an exact correspondence between the declared date of birth on the birth record and the declared date of birth in the 1901 census for only 70.9% of PL links.

The 1911 census does not provide the day of birth, but comparing the month and year to the birth records (Table 4.8) shows that 1,015 (59.7%) of the dates are the same between the PL birth records and the PL 1911 census records (N=1,700). This is less accurate

⁵⁷ The linkage rating is always between the death record and the record in question, so does not specifically refer to the linkage between the census records and the birth records (Chapter 3, Appendix A).

than the 1901 census and thus of lesser value in determining exact date of birth, if both data sources are available.

Table 4.9 shows the comparison between the declared age on the 1901 census (given as year, month, day) and the 1911 census (year, month). The month and year as declared on the 1901 and 1911 censuses are in accordance for 53.0% of the records that refer to the same individual. In contrast, when comparing the month and year from the 1911 census to the birth record, 59.7% of the records match. Relating the 1901 census records to the birth records, where there is a match between the year and month (it does not matter whether the day matches or not), 70.8% of dates match. It appears as though the 1901 census is a closer match to the birth records (and thus the reconstructed date of birth) than the 1911 census. This is likely because the 1911 census was taken farther away in time from the birth, was not guaranteed to be declared by a parent or relative, and the lack of information on the precise date may have made the overall declaration less precise.

Table 4.9 - Declared Age on the 1901 and 1911 Censuses.

	Correspondences with the 1901 Census (PL Links N=2,703)									
	Year, Month		Year Only		Month Only		None		Not Linked to 1901 Census	
	N	%	N	%	N	%	N	%	N	%
1911 Census PL Links (N=2,382) ^a	1,190	53.0	90	4.0	718	32.0	162	7.2	595	25.0

^a. Both 1901 Census and 1911 Census Record Linked, both PL Links N=2,245

Taking only those records where the year of birth on the 1901 census was different from the 1911, the average years of difference was 1.9, with a minimum of 1 year, maximum of 20 years, and a median of 1 year. When specifying those records where there was a difference between the months, the average number of months difference was 3.7, with a minimum of 1, a maximum of 11 and a median of 3.

Table 4.10 shows the age as declared on the 1901 and 1911 censuses. The reconstructed age at birth from the project was used to calculate the reconstructed age at the census (as

taken by the date that each census was taken: March 31st, 1901 and June 1st, 1911, Dominion of Canada 1902, 1912). The declared age was given in whole years.

Table 4.10 - Declared Age on the 1901 and 1911 Census as Compared to the Reconstructed Age at the Census.

	Reconstructed Age at the Census							
	Same		+/- 1 Unit		+/- 2 Units		+/- >3 Units	
	N	%	N	%	N	%	N	%
Declared Age At the 1901 Census, PL Links N=2,701 ^a	183	8.3	324	14.6	295	13.3	1,415	63.8
Declared Age At the 1911 Census, PL Links N=2,380 ^b	160	8.1	287	14.6	269	13.7	1,254	63.7

^a. With both declared age at the 1901 census and a reconstructed age at the census, PL links, N=2,239.

^b. With both a declared age at the 1901 census and a reconstructed age at the census, PL linkage N=1,970.

The 1901 census and the 1911 census are similar in the differences they show to the reconstructed age at the census. Both have just over 8% for which the age on the censuses is the same as the reconstructed age at the census. The greatest discrepancy is by more than three years, which is the case in 63% of both censuses. The age as declared on the census did not necessarily match to the declared date of birth, and it appeared in some cases that the enumerator may have either guessed at the age based on the year of birth, or guessed at the year of birth based on the age. If there is a choice between using the date of birth (either the day, month, and year as given in 1901 or the month and year in 1911) and the declared age, it would be more prudent to use the declared date of birth, since it is more accurate than the declared age.

When trying to determine age at exposure to the 1890 influenza pandemic, it is important to have an accurate date of birth, something which is not provided on the death records. It therefore becomes necessary to link individuals to other records in order to ascertain these data. From this analysis, the most accurate information on date of birth is the birth record. However, while the birth record is almost always entirely filled out, for the time

period 1883-1895, birth registration was not complete for the province as a whole. Therefore, while the data provided are accurate, they are not robust enough to provide information for every decedent. The 1901 census provides the day, month, and year of birth and the 1911 census provides the month and the year. The 1901 census is more consistent with the birth records and the reconstructed date of birth from this project than is the 1911 census. It is also the type of record for which we were able to link the greatest number of individuals. Therefore, while there are more inaccuracies in this source, they may be acceptable if it is more important to have information on a greater number of the decedents, accepting a certain level of error. On both the 1901 and 1911 censuses there is a declared age at the last birthday. This age is highly inaccurate and should be avoided if possible in historical demographic analyses that require an exact date of birth or age.

4.3.2 *Demographic Characteristics*

The characteristics of the individuals who could not be linked to a birth record were similar to those who could not be found in any record (Appendix D, Table 4.5). As compared to those who could be linked to a birth record, there was a higher percentage of individuals among the unlinked who were indigenous and a higher percentage with possibly itinerant occupations. A higher percentage of the unlinked died in institutions and in the North than among the unlinked and a far smaller percentage of individuals' birth records could be found if either their father's or their mother's name was missing from the death record. Due to the similarities to those who could not be linked overall, a separate logistic regression for those who could not be linked to a birth record was not computed.

Those who could not be found in the 1901 census were similar to those who could not be found overall and those for whom no birth record could be found (a higher percentage were indigenous, had possibly itinerant occupations, died in institutions or in the north, and were without a father's or a mother's name on the death record, Appendix D). As with those who could not be found at all, those who could not be found in the 1901

census were statistically significantly older than those who could be found. Those who could not be found in the 1901 census are different from those who could not be found overall and in the birth records in that a higher percentage died in urban locations and died in Toronto. Further, fewer individuals who did not have an occupation declared on their death records were found in the 1901 census than among those who did.

There is a difference between group 1: those who could not be found overall, those who could not be found in the birth records, and those who could not be found in the 1901 census and; group 2: those who could not be found in the 1911 census, and those who could not be found in all three types of records combined. In group one, sex, marital status, and death by influenza or tuberculosis were not significantly different among those who could be linked and those who could not be linked (the significant difference for deaths by the flu in the overall linked records does not remain when other factors are controlled for in the logistic regression). If any of these factors were important, it could influence the results of this research (for example, since there are anomalous findings of sex differences among those who died by the flu globally (Chapter 2), it is important that the sex differences for those who could not be linked are small). There would be a bias problem if those who died from the flu could not be found in the same amount as those who died from other causes. There are many more significant differences between the linked and the unlinked individuals in group 2. Records appear to be more systematically biased among those who could not be found in 1911 and for those who could not be found in all three of the birth record, the 1901 census, and the 1911 census. This is partially accounted for by there being fewer individuals linked to the 1911 census, and even fewer to all three.⁵⁸ In both cases in group 2, each factor on the descriptive statistics tables are significant except for death from influenza, death from tuberculosis, and the presence of a declared occupation (Table 4.11, Appendix D). Since these 2 cases are similar and there were more individuals found in the 1911 census, a separate logistic regression will only be run for those who cannot be found in the 1911 census (Table 4.11).

⁵⁸ Linked to the birth record, N=2,079. Linked to the 1901 census, N=2,737. Linked to the 1911 census, N=2,424. Linked to the birth record, 1901 census, and 1911 census, N=1,667.

Table 4.11 - Descriptive Statistics of Records Linked and Not Linked to the 1911 Census.

		1911 Record		No 1911 Record		χ^2	df	p
Demographic Features		N	%	N	%			
Sex	M	1,242	51.2	501	56.2	6.35	1	.01*
	F	1,182	48.8	391	43.8			
Declared Age	Mode	25		30		5.88	3314	<0.01*
	Median	28		29				
	Mean	28.5		29.3				
	SD	3.5		3.4				
Marital Status	Single	877	36.9	265	32.7	4.59	1	.03*
	Ever-Married	1,500	63.1	545	67.3			
Indigenous	Y	40	1.7	42	4.7	25.29	1	<.001*
	N	2,384	98.4	850	95.3			
Soldier	Y	70	2.9	45	5.0	9.06	1	.003*
	N	2,354	97.1	847	95.0			
Itinerant Occupation	Y	827	43.5	387	62.3	51.65	1	<.001*
	N ^b	1,074	56.5	234	37.7			
Conditions of Death								
Flu	Y	2,067	85.3	749	84.0	0.87	1	.35
	N	357	14.7	143	16.0			
Tuberculosis	Y	140	5.8	60	6.7	1.04	1	.31
	N	2,284	94.2	832	93.3			
Urban	Y	1,191	49.1	509	57.1	16.41	1	<.001*
	N	1,233	50.9	383	42.9			
Toronto	Y	308	12.7	179	20.1	28.20	1	<.001*
	N	2,116	87.3	79.9	79.9			
Institution	Y	561	23.1	303	34.0	39.66	1	<.001*
	N	1,863	76.9	589	66.1			
Region	North	135	5.6	161	18.1	124.9	1	<.001*
	Not-North	2,289	94.4	731	82.0			
State of Death Record								
Occupation	Y	1,345	55.5	484	54.3	.40	1	.53
	N	1,079	44.5	408	45.7			
Father's Name	Y	2,212	91.3	609	68.3	271.16	1	<.001*
	N	212	8.8	283	31.8			
Mother's Name	Y	1,967	81.2	487	54.6	238.93	1	<.001*
	N	457	18.9	405	45.4			

*P<0.05

There are many statistically significant differences among those who could not be linked in the 1911 census and those who could be linked (Table 4.11). More of the unlinked were women, were older, had ever been married, had possibly itinerant occupations, while fewer were soldiers. Greater percentages of the unlinked died in urban areas, in Toronto, in institutions, and in the north. More of the linked individuals had their parents' names declared on their death records. The results of a logistic regression of these significant factors are found in Table 4.12. As with the overall records, the models are run in a sequential manner in order to separate the demographic characteristics of the individuals, the environment of their death, and the conditions of their death records.

Table 4.12 - Logistic Regression of Records Linked to the 1911 Census, by Demographic Features, Conditions Surrounding Death, and Conditions of the Death Record.

	Model 1	Model 2	Model 3	Model 4	Model 5
	OR	OR	OR	OR	OR
Female	1.13	1.16*	1.13	1.14	0.95
Age	0.94***	0.94***	0.94***	0.94***	0.92***
Married	0.83**	0.81**	0.83*	0.83*	0.69***
Indigenous	0.33***	0.35***	0.38***	0.81	0.83
Soldier	0.47***	0.60**	0.63**	0.63**	0.60**
Urban		0.76***	0.79**	0.79**	0.94
Toronto		0.54***	0.74**	0.75**	0.84
Institution		0.63***	0.71***	0.71***	0.77**
North Region		0.33***	0.39***	0.45***	0.43***
Father's Name			1.95***	1.85***	1.72***
Mother's Name			1.58***	1.08	0.89
North Region*Indigenous				0.17***	0.12***
Mother*Father				1.53	1.85
Itinerant					0.52***
Model χ^2	66.40***	201.50***	283.76***	294.31***	262.52***
Pseudo R Square	.019	.057	.081	.084	.098
AIC	3,470.53	3,343.44	3,265.17	3,258.63	2,451.53
BIC	3,506.85	3,403.97	3,337.81	3,343.37	2,538.59
N	3,144	3,144	3,144	3,144	2,450

***p<0.01

**p<0.05

*p<0.1

Examining those who could be linked to the 1911 census as compared to those who could not, Model 1 uses logistic regression to test the demographic characteristics identified in Table 4.11. Sex is not significant, but age, being married, being indigenous, and being a soldier decrease the odds of being linked. In Model 2, these factors still decrease the odds of being linked, but now death in an urban area, death in Toronto, death in an institution,

and death in the North also decrease the odds. This is the only model in which being female increases the odds of being found, but it is significant only at $\alpha=0.1$. Model 3 adds in the conditions of the death record, and both having a father's name declared and having a mother's name declared increase the odds of being linked, such that records with missing information are harder to link. The interaction terms for Northern Ontario and indigenous status and mothers' and fathers' names present are added in the fourth model. The important factor in the missing data when linking to the 1911 census is whether the fathers' name is missing, since the mother's name is less important for records linkage, although it is useful to have the mother's maiden name to link to birth records. When the father's name is missing it suggests that there is further information missing, while it was more common for the mother's name to be missing alone. As with those who could not be linked to any record at all, it was more difficult to link an indigenous individual who also lived in Northern Ontario than to link an indigenous person who lived elsewhere in the province. The final model, Model 5, adds in the effect of possibly having an itinerant occupation. Once again, this was added last because it decreases the sample size from 3,114 to 2,450. However, as with the logistic regression of whether a record could be linked at all, the final model is better, since the AIC and BIC are smaller and the pseudo R square is larger. In this model, both death in an urban location and death in Toronto are no longer significant, but having a possibly itinerant occupation is significant. All of these factors suggest that death records that were filled out incompletely, by individuals who did not know the decedent, or by someone reporting on the death of an indigenous person were systematically harder to link. Interestingly, the interaction between indigenous status and region changes direction once itinerant status is added in Model 5. However, again, this model explains only a small percent of the variation in whether or not a record could be linked to the 1911 census (9.8%). Other factors that are likely important are the size of the household (smaller households, such as those for the newly married) are harder to link because of the fewer number of possible data points. Also, at the time of the 1911 census, the individuals in this study ranged in age from 15 to 28. This is the time in their lives when they would be most likely to be living in another household as a border or a servant, to be travelling to remote locations for work, to join the military, or to be recently married. As shown in

Chapter 6, the average size of the household among the decedents was larger in 1911 than it was for the rest of the population of Ontario. This is likely partially explained by individuals in smaller households in 1911 being harder to link than individuals in larger households.

Based on the analysis of age and the systematic biases that are found between the linked and unlinked individuals for each type of record, future analyses that require exact age at death should first consult the 1901 census. The birth records are more accurate, but have more coverage problems and the individuals who could not be linked to the 1901 census and to the birth records are similar. Further archival research should be conducted to determine the ages of those who are missing systematically, such as indigenous individuals and those from the North. While acknowledging these deficits, the 89.4% success rate of linkage to *at least one* other record is encouraging and conclusions can be drawn about the majority of Ontarians, especially those who are non-indigenous and live in areas other than Northern Ontario.

4.4 Conclusion

This chapter addressed the research question:

1. *Through a historical demographic lens, are the extant historical records in Ontario suitable for demographic analyses of past infectious disease?*

Historical demographers must use the data available in order to explain previously unexplored events in history. The historical nature of these data means that follow-up interviews to account for missing information are not possible. However, there are many extant archival sources that are underutilized and that can be drawn upon to expand and augment (or create) databases. Due to time constraints, it is likely not feasible to conduct the type of records linkage research presented in this dissertation for every question of historical interest concerning exact date of birth. Yet, the records that are available are suitable for research of this kind, although it is important to accept their limitations. For completeness, it would be best to use the 1901 census to establish age. However, for

accuracy, the birth records are more appropriate. The errors in the 1911 census mean that it should not be the first source used to ascertain date of birth, but it is useful for an understanding of the socioeconomic environment of individuals' as they approach adulthood (Chapter 6).

Other sources could be used for this type of information, but any contemporaneous secondary source will be subject to the same types of errors. For example, the death rates by age published by the Medical Officer of Health will not include the late registrations of birth or death that had not yet been received at the time of publication. Tax assessment rolls are not good sources to obtain information about those with insufficient housing. It may be possible to find these missing individuals with different types of data sources, such as obituaries and family genealogies in future research. Despite these limitations, the extant historical records in Ontario, such as those used to create the WMMIP database, are suitable for demographic analyses of past infectious disease.

Chapter 5

5 Young Adult Mortality and Records Linkage

This chapter presents the literature illustrating the high young adult mortality during the 1918 Spanish Influenza pandemic, the lack of age-specific mortality data and the necessity of this information, and the age-specific mortality totals and rates for native-born Ontarians found from this research. It explores the implications of the reconstructed ages at death in terms of current hypotheses to explain excess young adult mortality in the 1918 influenza pandemic. In so doing, it addresses the following research questions:

2. *Were all ages among young adults in Ontario at equal risk of death from the 1918 flu pandemic?*
3. *Since young adults have already been shown to be at an unusually high risk of death, could this be due to:*
 - a. *Previous exposure to influenza, resulting in physiological impairments or immunological conditioning?*
 - b. *Co-morbidity with tuberculosis?*

5.1 Were All Young Adults in Ontario at Equal Risk of Death from the 1918 Flu Pandemic?

This section examines previous literature as well as the linked WMMIP database in order to answer the question:

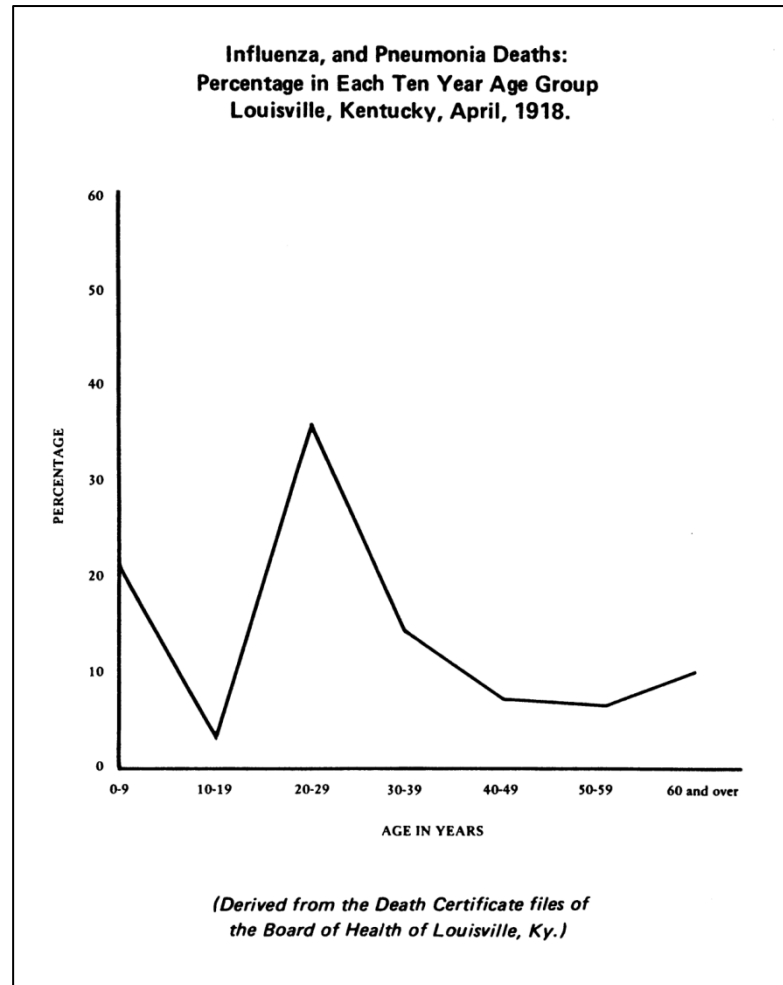
2. *Were all ages among young adults in Ontario at equal risk of death from the 1918 flu pandemic?*

The 1918 Spanish influenza pandemic is thought of as a disease of young adults. They did not die at the highest rate, those were as always the very young and the very old, but their deaths were the most unexpected. This is distinctive because all other influenza pandemics that have been recorded show low excess mortality in adulthood (Patterson 1986; Crosby 1989; Reid, Taubenberger and Fanning 2001; Taubenberger and Morens

2006, see discussion in Chapter 2). While it is understood that young adults were dying in unprecedented numbers, it is less clear who is being referred to as “young adults”.

Mortality from the 1918 flu is rarely presented in either age-specific death totals or as age-specific rates. For example, in his seminal book, Crosby (1989) graphs deaths in Louisville, Kentucky in April 1918 (the first wave of the pandemic) from influenza and pneumonia as a percentage of total deaths (Figure 5.1, 1989:24). Since this graph shows the percentage of total deaths, high young adult mortality from the flu does not indicate that these individuals were dying in higher numbers, but that among those who did die, the pandemic caused more deaths among young adults than among other ages. There is a clear increase in deaths caused by influenza and pneumonia among those aged 20-29 and pandemic deaths are increasing among ages 10-19 and decreasing for those aged 30-39.

Figure 5.1 - Influenza, and Pneumonia Deaths: Percentage in Each Ten Year Age Group, Louisville, Kentucky, April, 1918.

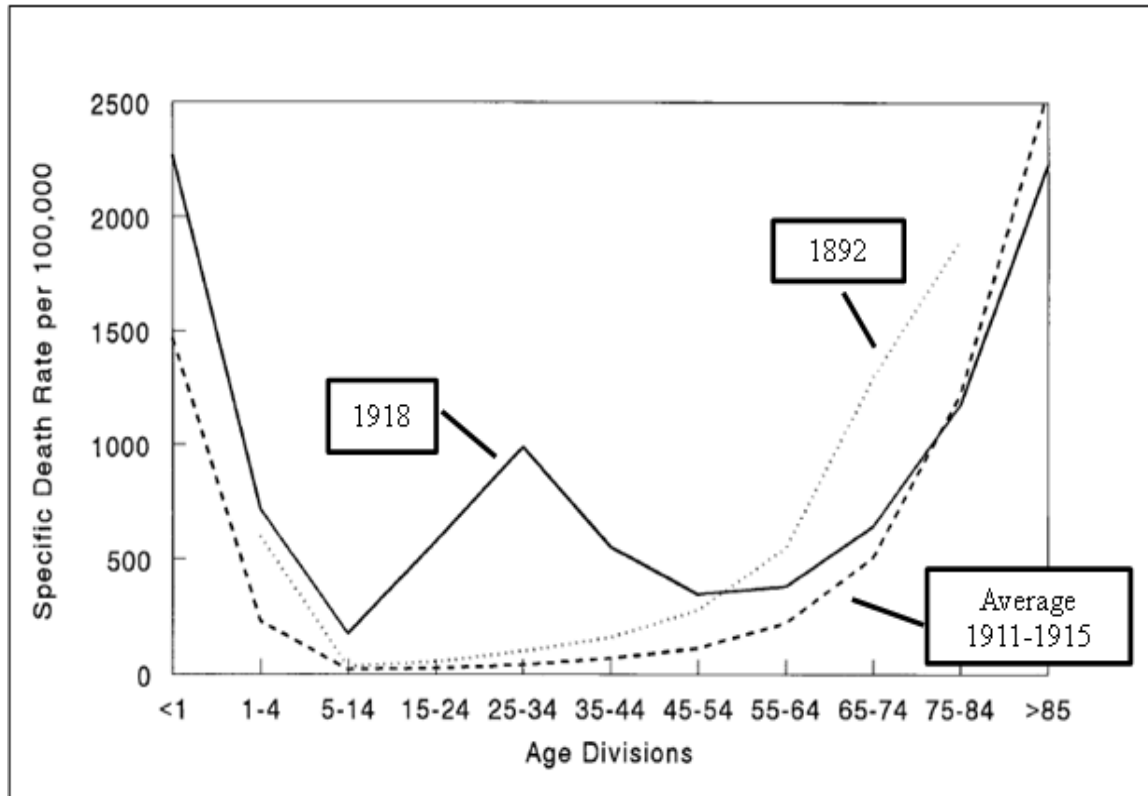


Source: Crosby (1989:24).

Similarly, Reid, Taubenberger, and Fanning (2001) graph the mortality rate from influenza and pneumonia in 1918 as compared to the mortality rate from the 1892 pandemic (a successive wave of the 1890 pandemic), as well as the average mortality rates from the interpandemic years of 1911-1915 in the United States (Figure 5.2). This graph highlights how the 1918 pandemic was different from the outbreaks that came before it, specifically in terms of the elevated young adult mortality; the ‘W’ shaped mortality curve of 1918 stands in contrast to the more typical ‘U’ shaped curve of previous influenza epidemics (Crosby 1989). Once again, mortality is presented in 10

year categories, with a clear peak among young adults. However, the category with the highest mortality is now 25-34 and mortality is increasing in the age categories of 5-14 and 15-24, and decreasing in the age categories 35-44 and 45-54.

Figure 5.2 – Influenza and Pneumonia Mortality by Age, USA, 1892-1918.



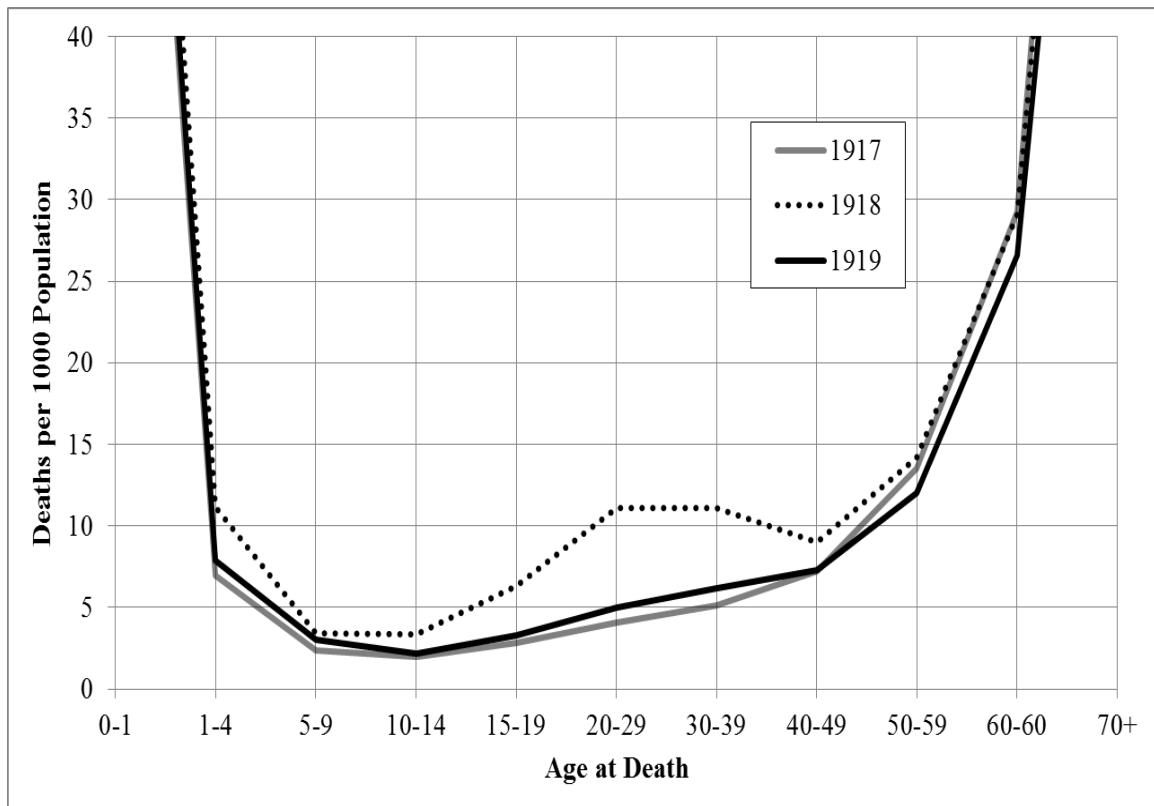
Source: "Figure 1. Influenza and pneumonia mortality by age, United States. Influenza and pneumonia specific mortality by age, including the pandemic years 1892 and 1918, and the average of the inter-pandemic years 1911–1915 is shown. Specific death rate is per 100,000 of the population for each age division." Reid, Taubenberger, and Fanning (2001:83).

These variations in the presentation of mortality from the 1918 influenza pandemic are common in the literature. Not only do the 10-year age categories differ as to what ages they cover, some authors present mortality in 5-year age categories (Taubenberger and Morens 2006, Morens and Fauci 2007) or as a mixture of 1-year, 3-year, and 5-year categories (Department of Commerce, Bureau of the Census 1920). Additionally, the age category with the highest mortality fluctuates as certain authors indicate a peak in the 20s (Crosby 1989), some show highest mortality in the 30s (Morens and Fauci 2007), while

yet others report the highest mortality between the ages of 25 and 34 (Noymer and Garenne 2000, Taubenberger and Morens 2006). These studies give a global indication of higher risk of death among young adults, but they are less precise in indicating whether all young adults were at similar risk of death, or whether that risk varied by single-year increments.

In 1944, McKinnon published a table of mortality-rates for Ontario from 1900-1942 for both sexes combined, and in both 5-year and 10-year age groupings. He does not specify where the data on the age-distribution of the population was obtained from (the denominator in the calculation of the mortality rates), but the 'W' shaped mortality pattern is evident in his data from 1918 in Ontario in comparison to the more typical mortality patterns in 1917 and 1919 (Figure 5.3). According to McKinnon (1944), the death rate in Ontario was elevated throughout the entire 20-39 age group, when looking at mortality for the whole year of 1918.

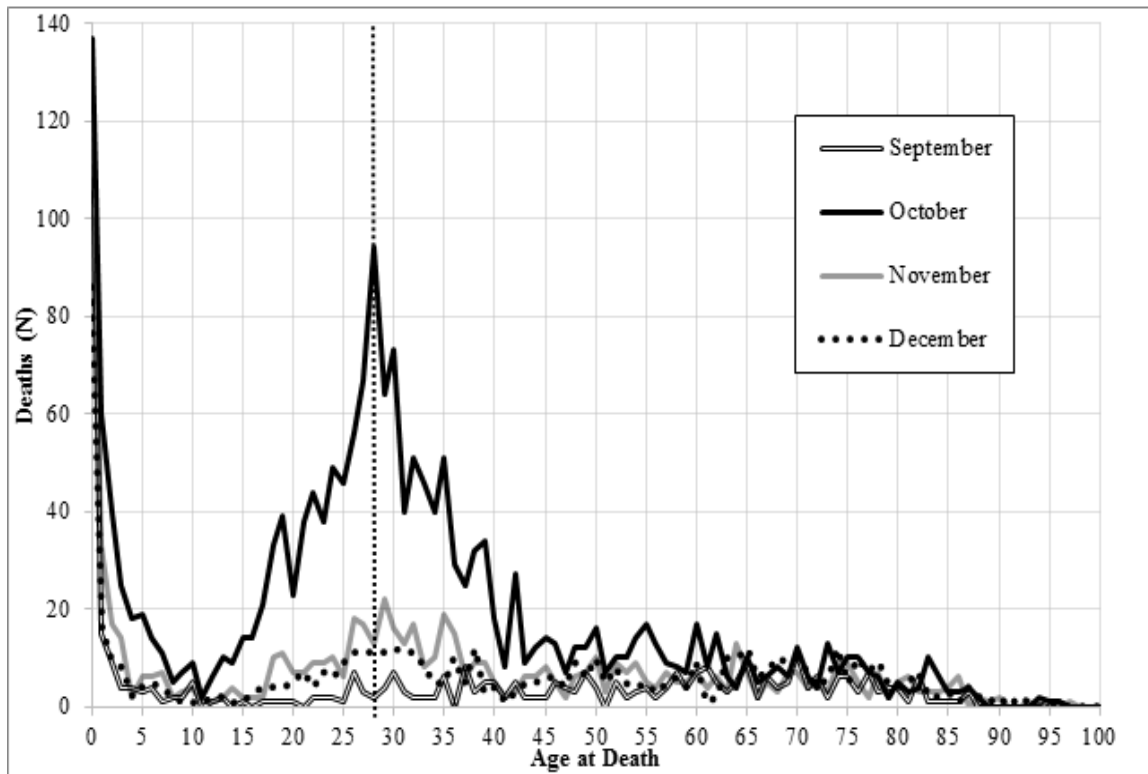
Figure 5.3 - Death rate in Ontario, 1918.



Source: McKinnon (1944).

My past work with Alain Gagnon begins to bring consistency to the question of what ages experienced the highest mortality, or at which age exactly the centre of the “W” shaped curve fell (the following results are published in Hallman and Gagnon 2014). Looking at the declared age at death on the death records for Toronto separated by month, we are able to separate the pre-pandemic month of September from the pandemic month of October, followed by November and December 1918, when the pandemic was waning in intensity (Figure 5.4). In accordance with the findings of other researchers, overall young adult mortality was elevated in Toronto during the worst month of the second wave of the pandemic (October 1918). This is in stark contrast to 1) the pre-pandemic month of September where young adult mortality is not increased, 2) to November, where the elevated mortality of young adults has abated but is still present, and 3) to December, where young adult mortality is elevated, but the pattern is muted. The important revelation of this analysis of young adult mortality by age is that there is an evident peak in mortality at the age of 28, with a secondary peak at age 30. It is now possible to see that young adult mortality was not elevated equally over the ages 20-40, but that increase in mortality is centered on the age of 28.

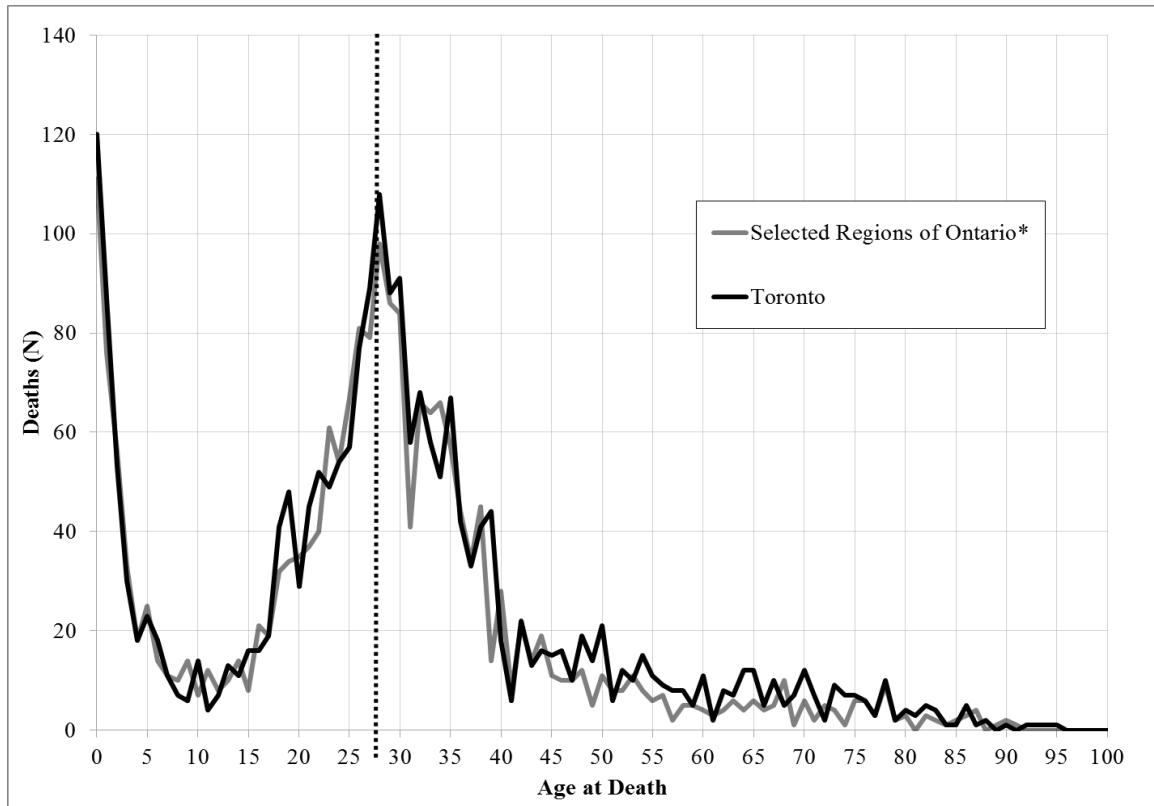
Figure 5.4 – Deaths from All Causes, Toronto, September to December, 1918.



Source: "Figure 1. Number of deaths by age from all causes in the City of Toronto, September to December, 1918 (September, n=441; October, n=1,885, November n=731, December n=618. Total n=3,675). The vertical line indicates age 28." From Hallman and Gagnon (2014).

In order to determine whether this pattern was localized in Toronto or more widespread, we compared other cities and regions in Canada. The patterns in the declared ages at death were almost identical when comparing Toronto to the pooled deaths from London, Ottawa, Hamilton, and the counties of Lincoln and Welland (Figure 5.5). The deaths were pooled since the number of deaths at each age for the smaller cities was very small, diluting the overall pattern. Since the pattern was specifically caused by the influenza pandemic, we restricted the analysis to those deaths caused by influenza, pneumonia, and bronchitis. The height of mortality at age 28 was spread throughout Ontario and was not merely a Toronto-specific phenomenon.

Figure 5.5 – Pandemic Related Deaths in Ontario, September to December, 1918.



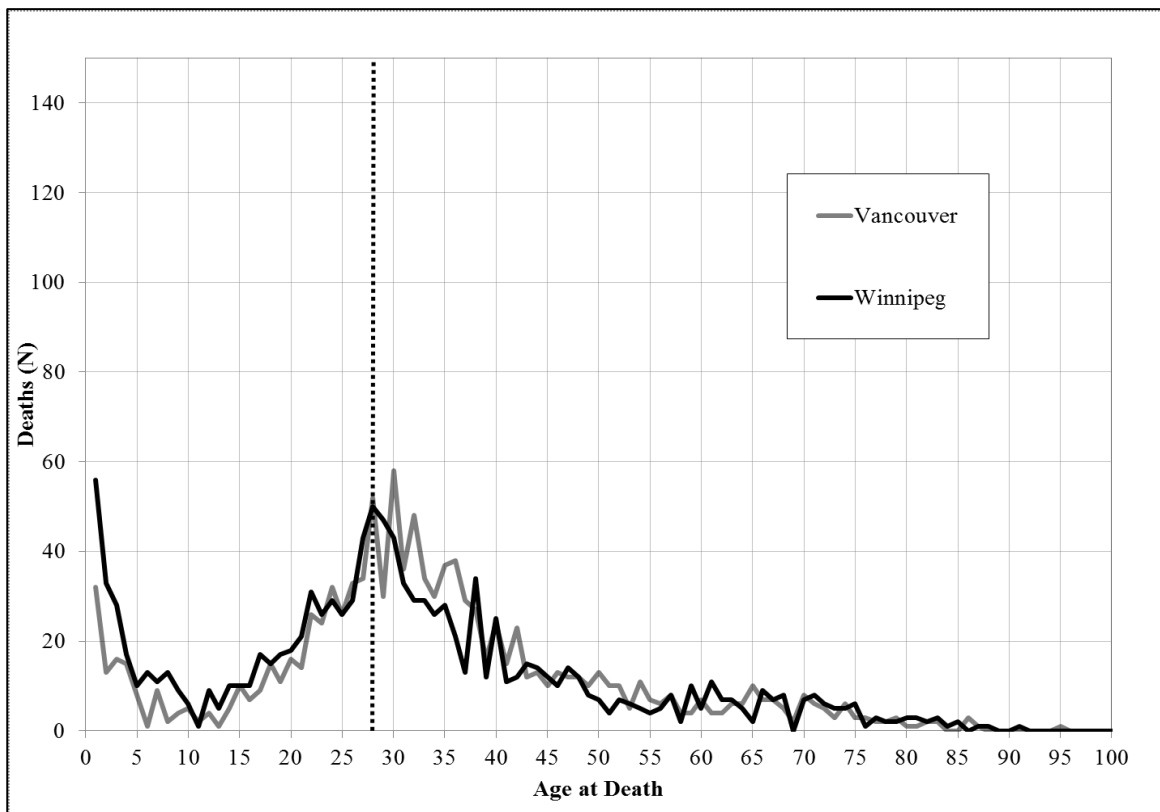
Source: "Figure 2 - Deaths from influenza, pneumonia, and bronchitis, September to December, 1918, in selected Ontario cities. Toronto n=2,195. The vertical line indicates age 28.

*Includes London (n=290), Hamilton (n=536), Ottawa (n=640), and Lincoln and Welland Counties (n=550)." From Hallman and Gagnon (2014).

Registered death records are readily available online for the province of Ontario (www.Ancestry.ca). Yet, for other provinces the records are only available at the local provincial archives and are not amenable to rapid analysis. The index for the death records (including sex, date of death, and age of death) for the cities of Vancouver and Winnipeg are available online through the Vital Statistics Agency of Manitoba (<http://vitalstats.gov.mb.ca/index.html>) and the British Columbia Archives (www.bcarchives.gov.bc.ca/index.htm). The age distribution of death from all causes is presented in Figure 5.6. The indexes do not provide information about cause of death, so we are not able to restrict the analyses to pandemic related causes. However, the pattern of increased young adult mortality is still present. In Winnipeg, the pattern is similar to

those cities and regions in Ontario, with the peak of mortality at age 28. Vancouver differs from the other cities in that there is a peak at age 28, but it is smaller than the elevated mortality at age 30. The focus of this dissertation is young adult mortality in Ontario, but further British Columbia specific research in the manner of this dissertation would be needed to determine the exact cause the excess mortality at age 30 in Vancouver.

Figure 5.6 – Deaths from all Causes, Winnipeg and Vancouver, September to December, 1918.



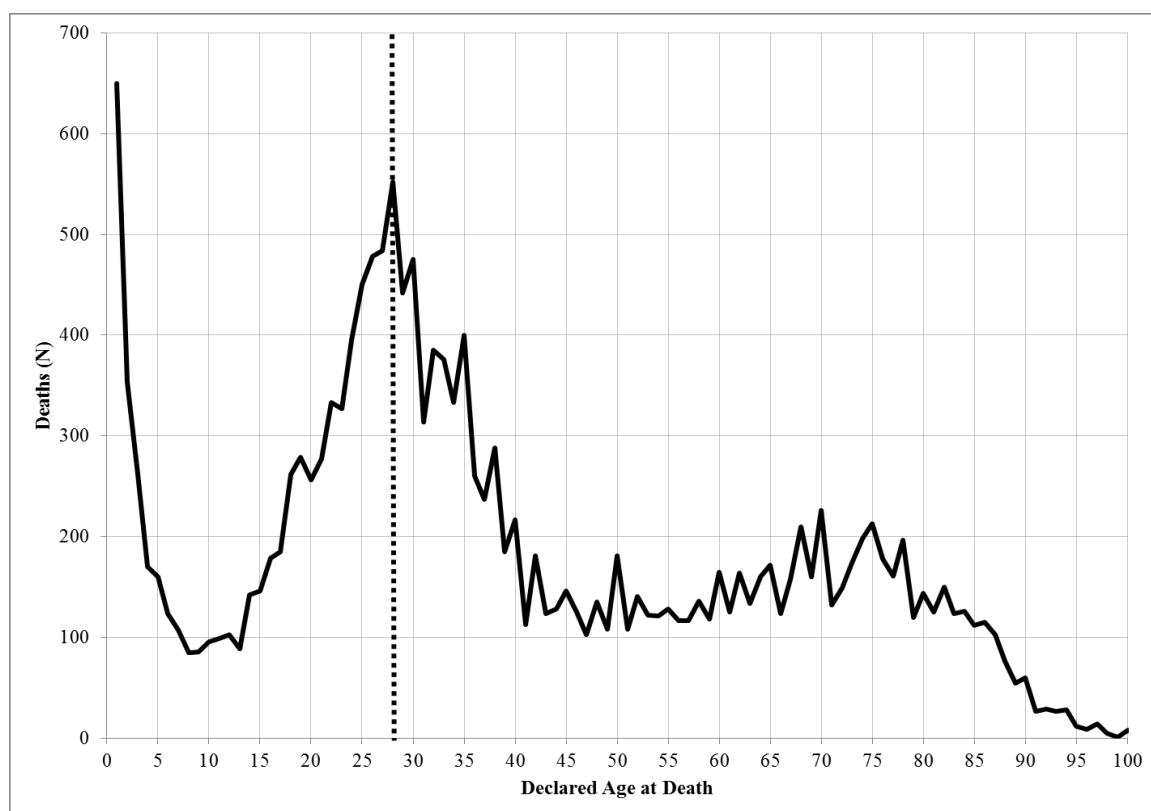
Source: “Figure 3 - Deaths from all causes, Winnipeg (n=1,193) and Vancouver (n=1,141), September to December, 1918 (excluding infants). The vertical line indicates age 28.” From Hallman and Gagnon (2014).

We furthered this research (with the collaborators Matthew Miller, Robert Bourbeau, D. Ann Herring, David Earn, and Joaquín Madrenas) with the addition of data from Montréal, Philadelphia, Kansas, and Indiana (Gagnon et al. 2013). Similar results were

found from these areas as was found in the Canadian specific analyses (Hallman and Gagnon 2014).⁵⁹

As discussed in Chapter 3, the death records from this study came from Drs. Earn and Herring through the International Infectious Disease Data Archive at McMaster University. These death records represent the 21,712 deaths that occurred in Ontario from September to December 1918. The distribution of deaths can be seen in Figure 5.7. As with the previous findings, among young adults, the greatest number of deaths occur at age 28, with a secondary peak at age 30.

Figure 5.7 – Declared Age at Death in Ontario, September to December, 1918.



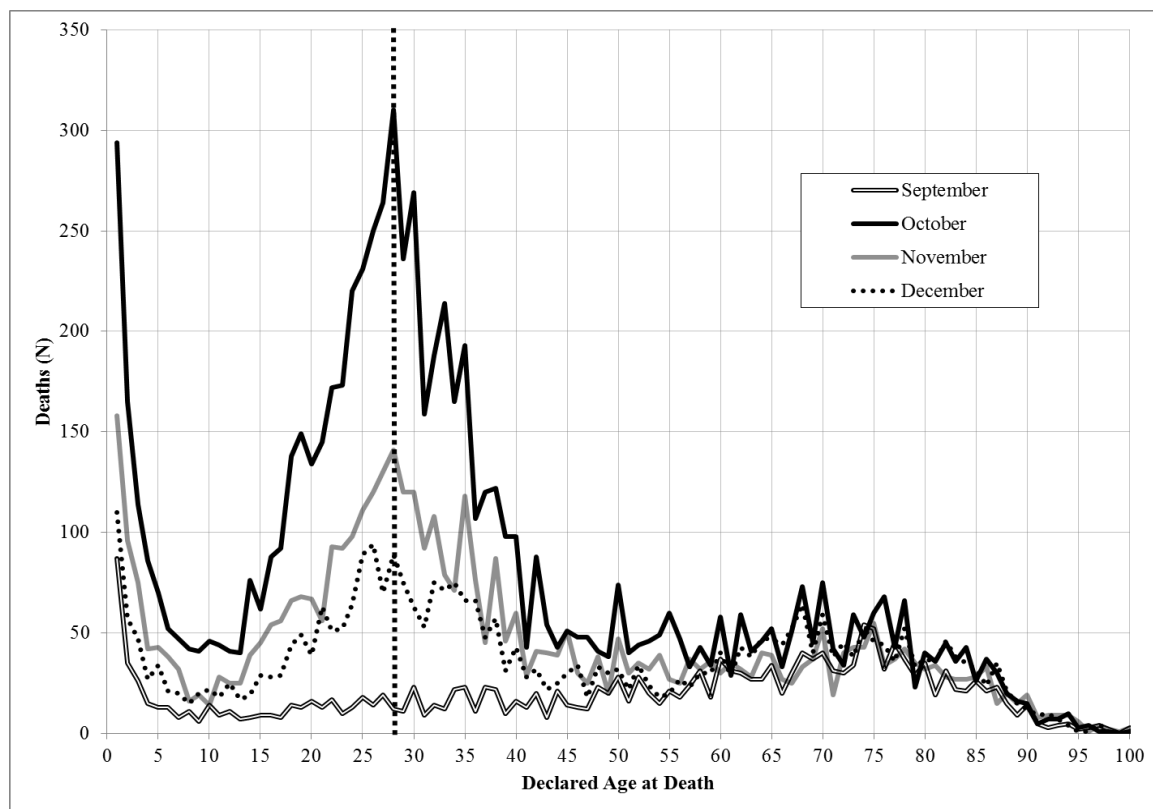
Note: Deaths in Ontario from September to December, 1918, using Declared Age at Death (N=18,173). The 3,284 infant deaths and stillbirths are excluded and 255 individuals did not have a declared age at death. The dotted line represents age 28.

Source: International Infectious Disease Data Archive at McMaster University.

⁵⁹ For similar research by other authors, see Viboud et al. 2013, Yang et al. 2013, Wilson et al. 2014.

Figure 5.8 shows the total deaths for the province of Ontario separated by month. Once again, October, 1918 has the most deaths. Young adult deaths at age 28 are highest for October and November, while in December the peak is at age 26. There is no increase in young adult deaths for the pre-epidemic month of September.

Figure 5.8 - Total Deaths in Ontario from September to December, 1918, by Month.



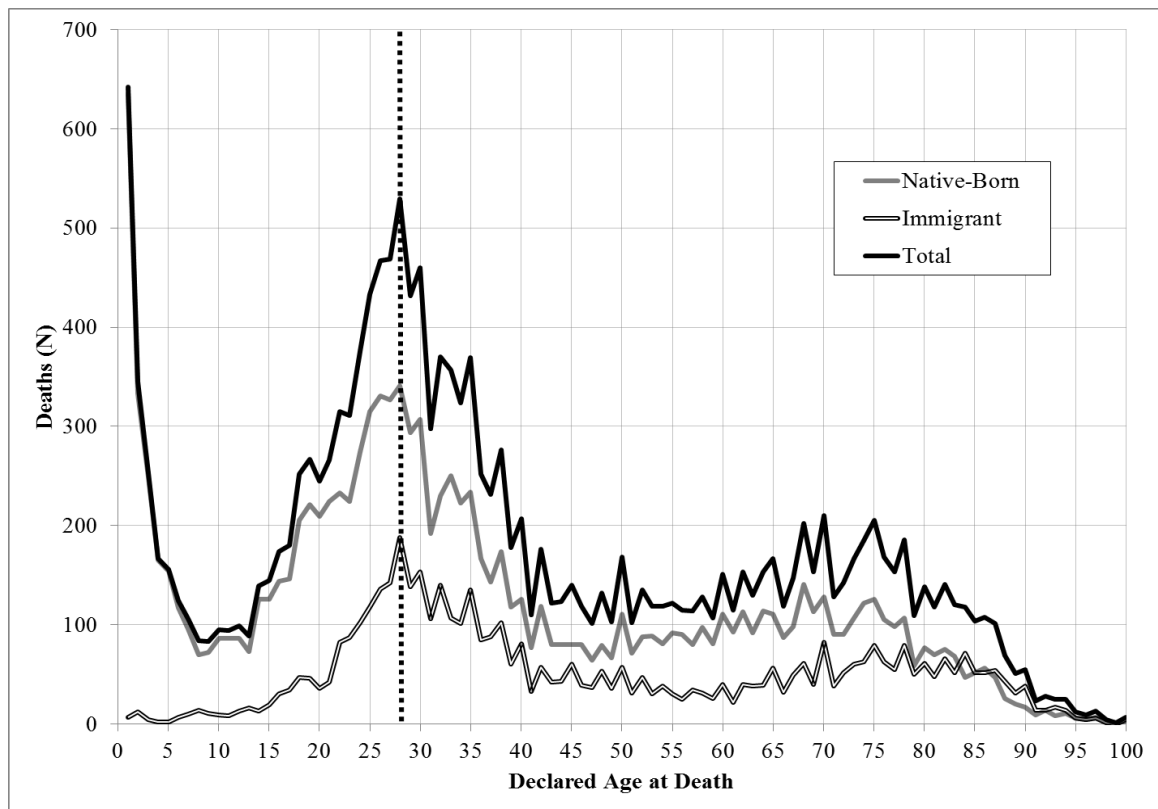
Note: Graph only shows those who had both a declared age at death as well as a declared month of death, excluding infants and stillbirths (N=18,159). September (N=1,972), October (N=7,871), November (N=4,579), December (N=3,737). The dotted line represents age 28.

Of the 21,712 individuals who died in Ontario from September to December, 1918, 15,747 (72.5%) were native-born Canadians and 5,061 (23.3%) were foreign-born (including Newfoundland). Place of birth was missing from 904 (4.2%) records. The distribution of deaths by place of birth can be seen in Figure 5.9. While the peak of deaths at age 28 is still present for Native-born Canadians, it is more prominent among immigrants; excess deaths among the Canadian-born were more numerous than the

deaths of immigrants at ages 25-27. As with the native born, immigrant deaths are subject to age heaping over the age of 30. Very few of the infants and children who died in Canada were born elsewhere.

As seen in Figure 5.9, the peak at age 28 in Ontario is more pronounced for the immigrant population than for native-born Canadians. However, it was necessary to only include the native-born Canadians (specifically those born in Ontario) in order to be able to link an individual to a birth certificate. This also provides a bounded area within which to study the effects of the 1918 Russian pandemic during a limited time frame. It may be that the effects of the spread of the 1890 influenza differed based on the location of exposure, the socio-economic environment of that location, the selection effects through which certain individuals immigrate while others do not, and the length of time spent in Canada. The inclusion of the immigrant population would give a greater understanding of the circumstances in Ontario in 1918, while their exclusion provides a better insight into the experience of the very young in Ontario in the 1880s and 1890s.

Figure 5.9 - Total Deaths in Ontario from September to December, 1918, by Place of Birth.



Note: Total Deaths (N=20,643), Native-Born Canadians (N=15,650), and Immigrants (N=4,993). Excludes infant births, stillbirths, and individuals without a declared place of birth or age.

5.1.1 *Using the WMMIP*

Consistently, among those who died during the pandemic in Ontario, those aged 28 were dying in greater numbers than any other young adults, according to their declared age at death. The age-specific distributions of mortality presented above were created using the declared age at death as found on the death record. This depends on either a statement of age by the individual prior to death, the report of age by a knowledgeable informant, or a determination of age by the attending physician or an unacquainted informant. In order to provide a more accurate distribution of age at death during the pandemic it is necessary to confirm the date of birth of the decedents through comparison with either birth records or the declared date of birth in the 1901 census. Then, by using the date of death as

specified on the death record, an accurate age at death can be determined. The results of this analysis for the native-born Ontario population who died in Ontario between September and December 1918 can be found in the following section. Specification of the date of birth is also necessary to determine whether exposure to the 1890 Russian influenza pandemic, whether *in utero* or in very early life, had an influence on risk of death during the 1918 Spanish influenza pandemic. This is presented in Section 5.3.

Because we only have information on those individuals who died between September and December, 1918, it is only possible to draw conclusions about this particular death cohort. In order to calculate risk of death based on date of birth, it would be necessary to have a control group of individuals born around the same time who did not die. Further, we do not have information on those who died before September 1st, 1918. If we hypothesize that those who were born in the time surrounding the 1890 Russian influenza pandemic were weaker due to gestational impairments or physiological scarring, it is likely that those individuals would have been at greater risk of death from the many dangers that existed throughout the lifecourse. Infant mortality was high in Ontario during this time, ranging from a low of 94 deaths per 1,000 live births in 1891 to a high of 125.2 deaths per 1,000 live births in 1888 (the Registrar General of Ontario reported a severe lack of completeness of the registrations in the 1891 returns, suggesting that these rates are likely an underestimation. The rates are calculated from reports of total births per year and deaths from all causes under 1 year of age in the Reports Relating to the Registration of Births, Marriages and Deaths in the Province of Ontario, Legislative Assembly of Ontario 1883-1898. See also Chapter 3).⁶⁰ In addition, there were many lethal diseases circulating in Ontario at this time which would have place children and

⁶⁰ Infant mortality rates for each year are as follows (rate per 1000 live births, Legislative Assembly of Ontario 1883 – 1898):

Year	IMR	Year	IMR	Year	IMR
1883	113.6	1888	125.2	1893	109.0
1884	117.8	1889	118.6	1894	117.7
1885	107.3	1890	118.9	1895	123.4
1886	113.9	1891	94.0		
1887	116.4	1892	104.4		

In comparison, the infant mortality rate in Ontario in 2011 was 4.6 deaths per 1000 live births (Statistics Canada 2013).

youths at risk of death (Legislative Assembly of Ontario 1883-1898). Then, those who survived to 1914 and participated in the war were subject to a higher mortality risk than normal. The death rate among Canadian soldiers was 9.5% of all enlistments, while 24.1% returned from the war wounded, so even those men who were not weakened by the timing of their birth may have been weakened through injury (Leroux 2010).

In order to establish a risk of death in the 1918 Spanish influenza pandemic by date of birth, it would be necessary to follow a cohort of individuals born around 1890 until their deaths (however, death records after 1941 (or after 1946 for those who died overseas) are not yet publically available, Archives of Ontario 2014). In the absence of this type of large-scale longitudinal database, we are analyzing that cohort of individuals who died between September and December, 1918. This enables us to test the hypothesis of no difference among the decedents, since there is no reason to expect a clustering of deaths at a certain age among young adult individuals who died at the same time (see Figure 5.2 for other influenza pandemics and Figure 5.8 for pre-epidemic mortality patterns in Ontario).

The ages of death as declared on the registered death records for the 3,316 people included in this study are presented in Table 5.1. Individuals whose declared age was “about X” were considered to be that age (N=20). For example, the 22 year old woman in the sample had a declared age of “about 22.” Upon research, her reconstructed age was determined to be 24 (see Table 5.4) and she was left in the database. Any individual who had an “about X” age and who was shown to be outside of the age range of 23-35 was deleted from the sample (see Chapter 3). The 37 year old man was originally deleted from the database for falling outside the prescribed age range. However, while researching his wife, it was found that his declared age at death was in error and his reconstructed age at death was 26. He was then re-added to the final database. The differences by age are statistically significant (all the statistical tests for the declared age at death do not include ages 22 and 37, chi-square test: $\chi^2=130.88$, $df=12$, $p<.001$). Further, the differences in total deaths by age in the pre-epidemic month of September, 1918 are not statistically significant using a chi-square test ($\chi^2=13.96$, $df=12$, $p=.303$) while the differences by age are statistically significant during the height of the epidemic

in October, 1918 ($\chi^2=114.01$, $df=12$, $p<.001$), although this may be affected by sample size. Neither the differences by sex at each declared age at death are significant at $\alpha=0.05$ ($\chi^2=19.17$, $df=12$, $p=.085$) nor are the differences by urban or rural location of death and declared age at death ($\chi^2=15.98$ $df=12$, $p=.192$).

Table 5.1 - Declared Age at Death.

Declared Age At Death	Total		Male		Female		Urban		Rural	
	N	%	N	%	N	%	N	%	N	%
22	1	100.0	0	0.0	1	100.0	1	100.0	0	0.0
23	183	100.0	90	49.2	93	50.8	100	54.6	83	45.4
24	245	100.0	125	51.0	120	49.0	119	48.6	126	51.4
25	300	100.0	141	47.0	159	53.0	143	47.7	157	52.3
26	306	100.0	163	53.3	143	46.7	152	49.7	154	50.3
27	292	100.0	139	47.6	153	52.4	145	49.7	147	50.3
<u>28</u>	<u>331</u>	<u>100.0</u>	<u>165</u>	<u>49.8</u>	<u>166</u>	<u>50.2</u>	<u>186</u>	<u>56.2</u>	<u>145</u>	<u>43.8</u>
29	267	100.0	135	50.6	132	49.4	147	55.1	120	44.9
30	286	100.0	149	52.1	137	47.9	169	59.1	117	40.9
31	181	100.0	109	60.2	72	39.8	86	47.5	95	52.5
32	210	100.0	119	56.7	91	43.3	109	51.9	101	48.1
33	231	100.0	130	56.3	101	43.7	124	53.7	107	46.3
34	199	100.0	115	57.8	84	42.2	105	52.8	94	47.2
35	194	100.0	108	55.7	86	44.3	101	52.1	93	47.9
37	1	100.0	1	100	0	0.0	1	100.0	0	0.0
Unknown	89	100.0	54	60.7	35	39.3	13	14.6	76	85.4
Total	3,316	100.0	1,743	52.6	1,573	47.4	1,700	51.3	1,616	48.7
χ^2					19.17			15.98		
df					12			12		

Note: For the 3,316 people who were born in Ontario and who died in Ontario from September to December 1918. Individual's whose age was listed as "about ___" were included in the age that they were estimated as (for example "about 30" is included in age 30). For any individual who was missing a declared sex, a value for sex was imputed based on the likely gender of the given name.

The ages of death of the same population in Table 5.1 as reconstructed from the records linkage analysis is presented in Table 5.2 (see Section 4.3 and Appendix A for the calculation of the reconstructed age at death). Since it was not possible to successfully

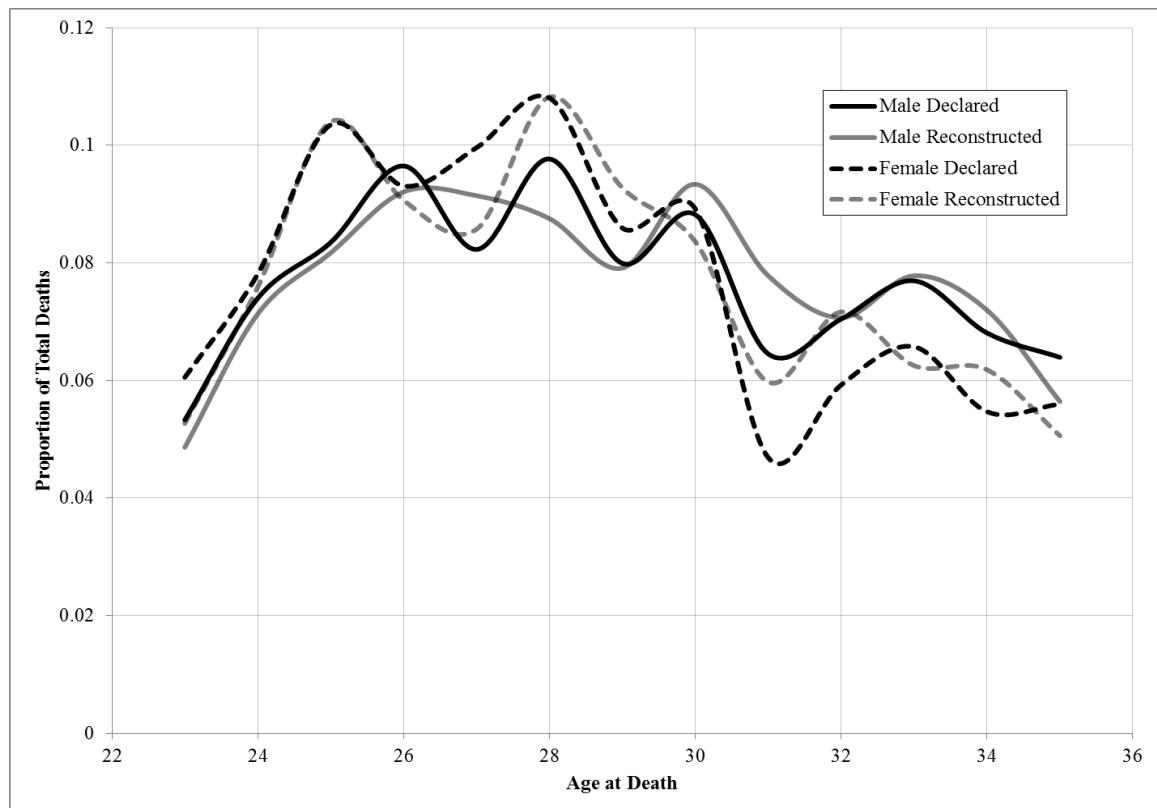
link every individual in the database and thus determine a reconstructed age at death, the number of individuals of unknown age increased from 89 to 350 (total population with declared age at death, N=3,227; total population with reconstructed age at death, N=2,966). As with the declared age at death, the differences by age are statistically significant (chi-square test: $\chi^2=104.01$, df=12, p<.001) and this result is not found in the pre-epidemic month of September (chi-square test: $\chi^2=15.22$, df=12, p=.230) while it is found during the epidemic in October (chi-square test: $\chi^2=74.71$, df=12, p<.001). Again, neither the differences by sex at each reconstructed age at death are significant (chi-square test: $\chi^2=18.17$, df=12, p=.111) nor are the differences by urban or rural location of death and declared age at death (chi-square test: $\chi^2=11.30$, df=12, p=.504). A major difference between the declared age at death and the reconstructed age at death is in the peak age for men. It is at age 28 for the declared age at death, but occurs at age 30 in the reconstructed age at death (using the proportion of total deaths at each age, the peaks at age 26 and 28 are almost the same, Figure 5.10). However, the peak at age 28 remains for women. Although this may be an accurate depiction, that it happens for men and not for women is important. It may have been influenced by greater male deaths during the First World War, male migration related to the war, and the greater number of unknown individuals in the reconstructed sample which reduced the sample size. The reconstructed ages for women are more representative of a non-combatant and less migratory population.

Table 5.2 - Reconstructed Ages at Death.

Reconstructed Age At Death	Total		Male		Female		Urban		Rural	
	N	%	N	%	N	%	N	%	N	%
23	150	100.0	75	50.0	75	50.0	69	46.0	81	54.0
24	218	100.0	110	50.5	108	49.5	104	47.7	114	52.5
25	274	100.0	126	46.0	148	54.0	136	49.6	138	50.4
26	271	100.0	142	52.4	129	47.6	131	48.3	140	51.7
27	263	100.0	141	53.6	122	46.4	122	46.4	141	53.6
<u>28</u>	<u>289</u>	<u>100.0</u>	<u>135</u>	<u>46.7</u>	<u>154</u>	<u>53.3</u>	<u>151</u>	<u>52.2</u>	<u>138</u>	<u>47.8</u>
29	254	100.0	122	48.0	132	52.0	140	55.1	114	44.9
30	263	100.0	144	54.8	119	45.2	138	52.5	125	47.5
31	206	100.0	120	58.5	85	41.5	108	52.4	98	47.6
32	211	100.0	109	51.7	102	48.3	117	55.5	94	44.5
33	209	100.0	120	57.4	89	42.6	114	54.5	95	45.5
34	199	100.0	111	55.8	88	44.2	98	49.2	101	50.8
35	159	100.0	87	54.7	72	45.3	84	52.8	75	47.2
Unknown	350	100.0	200	57.1	150	42.9	189	54.0	161	46.0
Total	3,316	100.0	1,743	52.6	1,573	47.4	1,700	51.3	1,616	48.7
χ^2					18.17				11.30	
df					12				12	

Note: For the 3,316 people who were born in Ontario and who died in Ontario from September to December, 1918, and who were successfully linked to at least one record (one date of birth was obtained from a tombstone, but no other records of her were found).

Figure 5.10 - Proportion of Deaths by Age and Sex.



Note: Male declared N=1,689, male reconstructed N=1,542, female declared N=1,536, female reconstructed N=1,423.

The differences between Tables 5.1, 5.2, and Figure 5.10 can be seen in Table 5.3. In total, there were 3,227 individuals in Table 5.1 who had a declared age at death, while we could reconstruct age at death for only 2,966 individuals in the sample. This means that there are 261 fewer individuals in Table 5.2 than in Table 5.1. Age 28 lost the most individuals (42), and more than two thirds of this loss came from men (30). The only ages that gained individuals were 31 (24) and 32 (2). The gain was relatively equal between the sexes at age 31, while at age 32, there were 9 less men in Table 5.2 but 11 more women at this age. As seen in Figure 3.1 for the 1921 census, age heaping in Ontario increased after age 30. The relatively fewer individuals at age 31 in the declared age at death and the increase at this age from the reconstruction process support the idea of age heaping over age 30. This can be seen in Figure 5.11. The curves until age 30 are similar, but after age 31 the reconstructed age at death line is flatter, suggesting that the

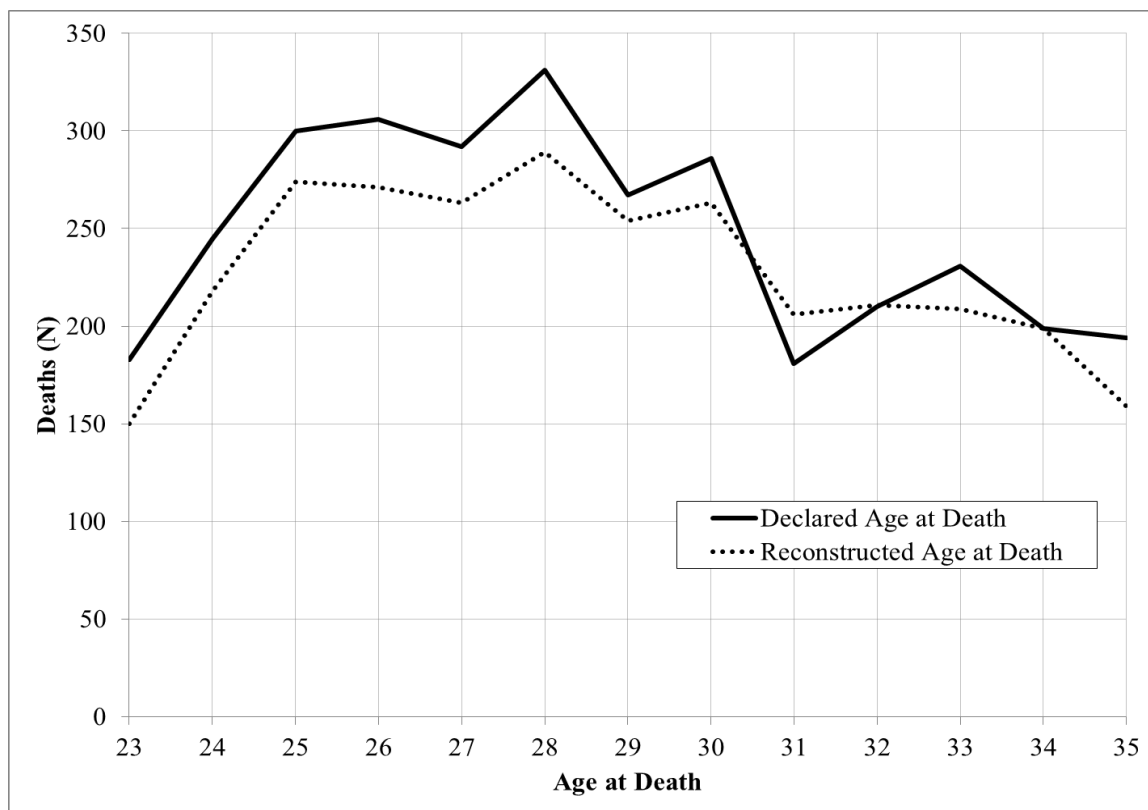
reconstruction process is able to compensate for age heaping in the death records. This allows for a more accurate determination of age of exposure to the 1890 pandemic.

Table 5.3 - Changes in Number of Individuals from the Declared Age at Death to the Reconstructed Age at Death by Age and Location.

	Total	Male	Female	Urban	Rural
Age at Death			N		
22	-1	0	-1	-1	0
23	-33	-15	-18	-31	-2
24	-27	-15	-12	-15	-12
25	-26	-15	-11	-7	-19
26	-35	-21	-14	-21	-14
27	-29	+2	-31	-23	-5
28	-42	-30	-12	-35	-7
29	-13	-13	0	-7	-6
30	-23	-4	-18	-31	+8
31	+25	+11	+13	+22	+3
32	+1	-10	+11	+8	-7
33	-22	-10	-12	-10	-12
34	0	-4	+4	-7	+7
35	-35	-21	-12	-17	-18
37	-1	-1	0	-1	0
Unknown	+261	+146	+115	+176	+85

Note: Declared Age at Death (Table 5.1, N=3,227), Reconstructed Age at Death (Table 5.2, N=2,966).

Figure 5.11 – Age at Death from All Causes, September to December, 1918.



Note: Declared age at death, N=3,227 and Reconstructed Age at Death, N=2,966. The two declared ages of death at age 22 and 37 are not included.

While there is relatively little difference between the aggregate declared age at death and the reconstructed age at death tables ($r^2=.88$), the differences become more acute when the correspondences between these two ages are compared for each individual (Table 5.4). Of the 2,945 individuals who had both a declared age at death and a reconstructed age at death, only 1,848 (62.8%) had the same declared and reconstructed age, while 1,097 (37.2%) differed in some manner. If age at possible exposure to the 1890 influenza pandemic had an influence on mortality in 1918, and that pandemic was primarily in Ontario in January of 1890, it is imperative to use a correct date of birth in the analyses (Section 5.2).

Table 5.4 – Correspondences between the Declared Ages at Death and the Reconstructed Ages at Death.

Reconstructed Age at Death	Declared Age at Death															Total
	22	23	24	25	26	27	28	29	30	31	32	33	34	35	37	
23		<u>112</u>	24	8	1	2		1			1					149
24	1	37	<u>141</u>	28	6	2	1	1								217
25		9	37	<u>179</u>	37	3	3		1			1		2		272
26		2	14	50	<u>167</u>	26	8	1							1	269
27		1	4	8	49	<u>165</u>	32	2	1		1					263
28		2	3	4	13	<u>53</u>	<u>170</u>	33	2	1	2	2				285
29			1	3	5	12	58	<u>148</u>	21	2	1		1			252
30			1		2	4	16	43	<u>169</u>	17	6	1		2		261
31							3	10	45	<u>111</u>	23	5	4	3		204
32		1					4	4	10	27	<u>123</u>	28	10	3		210
33		1	1				3		5	11	22	<u>131</u>	22	11		207
34			1	1	1				3	1	6	35	<u>119</u>	30		197
35		1			1	1			1		6	6	30	<u>113</u>		159
Total	1	166	227	281	282	268	298	243	258	170	191	209	186	164	1	2,945

Note: For those individuals who were linked and who had a declared age at death (N=2,945).

Table 5.5 - Classifications and Misclassifications of the Declared Age at Death as Compared to the Reconstructed Age at Death.

Reconstructed Age at Death	2+ Years Younger	1 Year Younger	Properly Classified	1 Year Older	2+ Years Older
			%		
23 ^a			<u>75.2</u>	16.1	8.7
24	0.5	17.1	<u>65.0</u>	12.9	4.6
25	3.3	13.6	<u>65.8</u>	13.6	3.8
26	5.9	18.6	<u>62.1</u>	9.7	3.7
27	4.9	18.6	<u>62.7</u>	12.2	1.5
28	7.7	18.6	<u>59.6</u>	11.6	2.5
29	8.3	23.0	<u>58.7</u>	8.3	1.6
30	8.8	16.5	<u>64.8</u>	6.5	3.4
31	6.4	22.1	<u>54.4</u>	11.3	5.9
32	9.0	12.9	<u>58.6</u>	13.3	6.2
33	10.1	10.6	<u>63.3</u>	10.3	5.3
34	6.6	17.8	<u>60.4</u>	15.2	
35 ^a	10.1	18.9	<u>71.1</u>		
Total	6.3	16.5	<u>62.8</u>	10.9	3.5

Note: N=2,945

^a. Age 23 exhibits left truncation while age 35 exhibits right truncation (inclusion years 1883-1895 and ages at death 23-35).

Table 5.5 simplifies the correspondence between the declared age at death and the reconstructed age at death at each individual age between 23 and 35. The WMMIP database is limited to individuals who were born between January 1st, 1883 and December 31st, 1895, provided that they died after having turned 23 (for example, an individual who died October 15th, 1918 and was born on October 15th, 1895 would have been included, whereas an individual who died on the same date, but who was born on October 16th, 1895, would not have been included). Therefore, age 35 exhibits left truncation since it does not include those individuals who died at age 35 but whose birth occurred in 1882 (for those born between September to December 1882 and who died before their birthday in 1918). Age 23 exhibits right truncation because it does not include those individuals who were born in 1895 and who would have turned 23 in 1918 had they survived until their birthday.

The majority of each age was classified correctly on the death records, ranging from a low of 54.4% at age 31 to a high of 75.2% at age 23. Age 31 was the age least likely to be classified correctly, which can also be seen in Table 5.3 and is again indicative of age heaping (age 28 is among the lower percent of ages classified correctly, at only 59.6%). Those individuals whose reconstructed age at death did not match their declared age at death were most likely to be misclassified by one year only, and more likely to be declared to be one year younger than one year older (t-test: $t=4.562$, $df=19$, $p<.001$). There is no statistically significant difference between those misclassified as two or more years younger and those misclassified as two or more years older than their actual age. Chapter 4 analyzes these differences between the records used in this study in terms of their reliability and usefulness for historical demographic research.

5.1.2 *Conclusion*

This section addressed the research question:

2. *Were all young adults in Ontario at equal risk of death from the 1918 flu pandemic?*

While it is not possible to determine epidemiological risk of death without knowledge of those who did not die, it is possible to examine trends among the decedents. The results are similar using both the declared age at death from the death records as well as the reconstructed ages from this research. In the pre-epidemic month of September, 1918, young adult mortality was not elevated, as would be expected from a population experiencing normal adult mortality patterns (see Figure 5.3 for mortality patterns in Ontario in 1917 and 1919). Further, there was no statistically significant difference between the numbers of deaths at each age from 23 to 35 in September. This is to be expected, as there is no reason to hypothesize that there would be a larger difference between the number of deaths by age for these ages in a population not experiencing an epidemic. During the worst month of the second wave of the Spanish influenza pandemic, October, 1918, deaths among young adults are elevated and there is a

statistically significant difference between the death totals at each age. Graphically, this peak is centered at age 28. This is unexpected and requires a theoretical explanation. Since the 1890 Russian influenza pandemic occurred 28 years prior to the Spanish influenza pandemic, possible early life physiological and immunological conditions that may have impacted mortality later in life are explored in Section 5.2.

5.2 Early Life Influences on Mortality in 1918

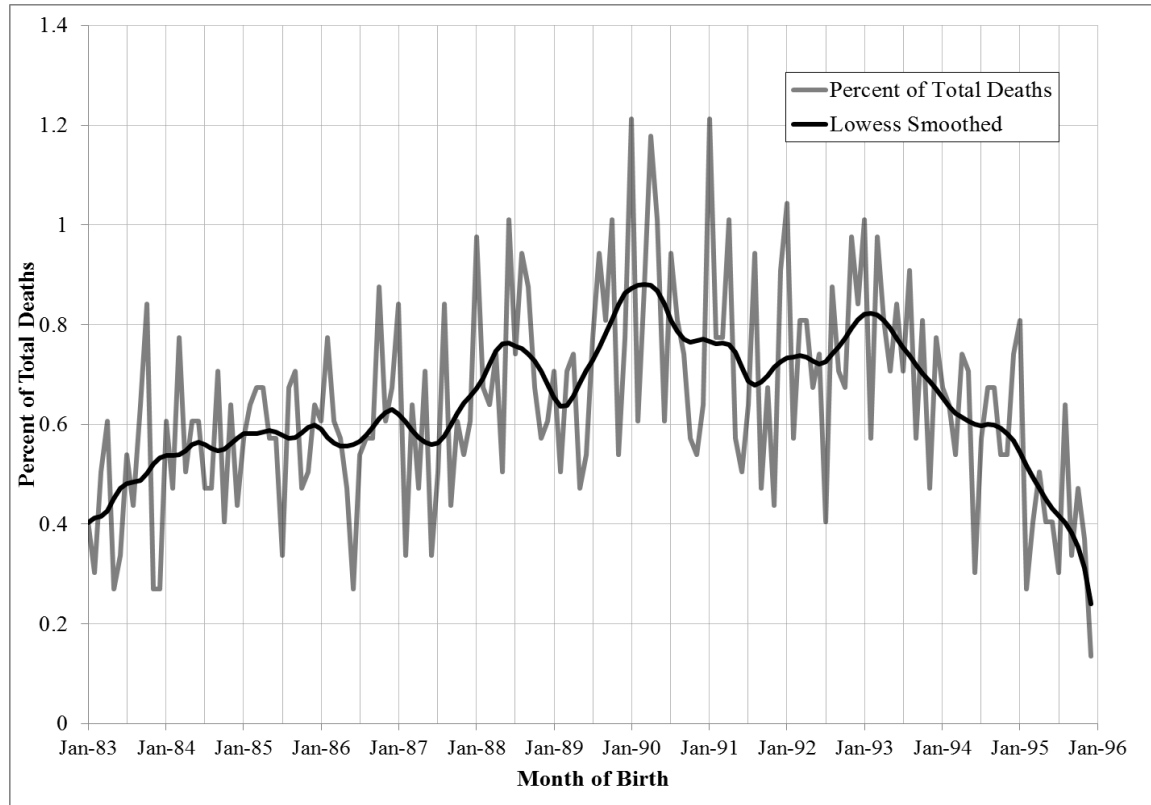
It has been ascertained that young adults died disproportionately in Ontario from the ages of 23 to 35 between September and December, 1918. This pattern is apparent among the native-born Ontarians, using both the declared and reconstructed ages at death. This section addresses the third research question, using the reconstructed date of birth:

3. *Since young adults have already been shown to be at an unusually high risk of death, could this be due to:*
 - a. *Previous exposure to influenza, resulting in physiological impairments or immunological conditioning?*

Figure 5.12 shows the number of deaths by month of birth (using the reconstructed month of birth) as a percentage of the total births among those who died from September to December 1918. For the unsmoothed numbers, the highest percentages of the deaths occurred among those born in January 1890, April 1890, and January 1891 (the differences in the numbers of death by month are significant with a chi-square test: $\chi^2=297.86$, $df=156$, $p<.001$). Using the smoothed line, the highest number of deaths occurs for those born in the first quarter of 1890, with the highest percentage of deaths occurring for those born in March, 1890. The numbers of deaths per *day* of birth ranged from zero to five, with only three days of birth having five people die during the time period: June 27, 1888; January 30, 1891; and, March 6, 1893 (average 0.6 deaths per day of birth). If, *ceteris paribus*, this group of decedents was experiencing the mortality patterns seen in Figure 5.3, the greatest number of deaths should be seen amongst those born in 1883, with a steady decline to those born in 1895. However, it is necessary to

take account for the fact that there might have been unequal numbers of individuals at each age group at the time of the pandemic (for example, if more individuals were alive at age 28, it would be expected to see more deaths at this age).

Figure 5.12 - Total Deaths by Month of Birth as a Percentage of Total Deaths, September to December, 1918.



Note: Figure includes only those individuals with a reconstructed month of birth (N=2,965). Lowess smoother applied using Stata with bandwidth 0.1.

The percent of total deaths by month of birth for each year are shown in Table 5.6 (using the reconstructed month of birth), along with the percent of total births per month for each year that were recorded by the Registrar General of Ontario for that month (Legislative Assembly of Ontario 1883–1898). Mortality in general was high during this time period, especially for infants and during the First World War. As discussed in Chapter 2, many other diseases were circulating in epidemic form at this time, including smallpox, diphtheria, and scarlet fever. As a result, it is difficult to know how many

individuals who were born in a particular month in Ontario survived to face the Spanish influenza pandemic in 1918. For example, if neo-natal mortality was significantly higher in one month, more infants born in that month would have died than in other months,

leaving fewer individuals alive from that cohort in 1918 (for use as a denominator in the calculation of rates. Infant mortality by month of birth was not collected by the registrar general at this time). Keeping in mind this limitation, it is possible to see trends among the decedents in 1918 by month of birth.

In most years (except 1889, 1892, and 1895), March was the month with the highest percent of total births. The month of birth in each year with the highest percentage of deaths among the decedents in 1918, can be divided into two separate groups. From 1883-1889, the highest percentage of deaths was usually in the autumn, in the months of August to October (the exception being 1888, with the greatest percentage of deaths among the decedents for those born in June of that year). However, this abruptly changes in 1890. From 1890-1893 and 1895, the month with the highest percentage of deaths among the decedents each year is January, with the highest percentage of deaths in 1894 being for those born in April of that year.

Table 5.6 - Births per Month in Ontario, 1883-1895, and Number of Deaths for Those Born in Those Months.

	1883		1884		1885		1886		1887		1888		1889	
	%													
	B	D	B	D	B	D	B	D	B	D	B	D	B	D
Jan	8.3	7.5	8.2	9.0	8.2	8.1	8.4	8.5	8.4	<u>12.3</u>	8.4	10.9	7.7	8.3
Feb	8.0	5.6	8.3	7.0	8.4	9.1	8.0	10.8	8.1	4.9	8.5	7.5	7.6	5.9
Mar	<u>9.3</u>	9.3	<u>9.4</u>	<u>11.6</u>	<u>9.0</u>	9.6	<u>9.2</u>	8.5	<u>9.5</u>	9.3	<u>8.9</u>	7.1	8.7	8.3
Apr	8.7	11.2	8.4	7.5	8.6	9.6	8.7	8.0	8.7	6.9	8.2	8.3	8.5	8.7
May	8.3	5.0	8.3	9.0	8.8	8.1	8.3	6.6	8.5	10.3	7.9	5.6	8.2	5.5
June	7.7	6.2	7.8	9.0	7.7	8.1	8.0	3.8	7.9	4.9	8.1	<u>11.3</u>	8.2	6.3
July	8.2	9.9	8.3	7.0	8.3	4.8	8.3	7.5	8.2	7.4	8.6	8.3	8.7	9.1
Aug	8.5	8.1	8.8	7.0	8.5	9.6	8.7	8.0	8.5	<u>12.3</u>	8.8	10.5	9.0	11.1
Sept	8.9	11.8	8.8	10.6	8.7	<u>10.0</u>	8.5	8.0	8.5	6.4	8.7	9.8	<u>9.2</u>	9.5
Oct	8.4	<u>15.5</u>	8.2	6.0	8.2	6.7	8.1	<u>12.3</u>	8.3	8.8	8.5	7.5	8.6	<u>11.9</u>
Nov	7.8	5.0	7.8	9.5	7.5	7.2	7.6	8.5	8.0	7.8	7.6	6.4	7.8	6.3
Dec	7.9	5.0	7.7	6.5	8.0	9.1	8.0	9.4	7.5	8.8	8.0	6.8	7.7	9.1
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

Source: For births, Legislative Assembly of Ontario (1883-1898).

Note: Deaths utilizes the Reconstructed Age at Death, for those who died between September – December 1918. Births, N=581,842, Deaths=2,965. Underlined cells represent the highest percent in the year. When there are two cells that are the same, the cell that had the highest raw number is underlined.

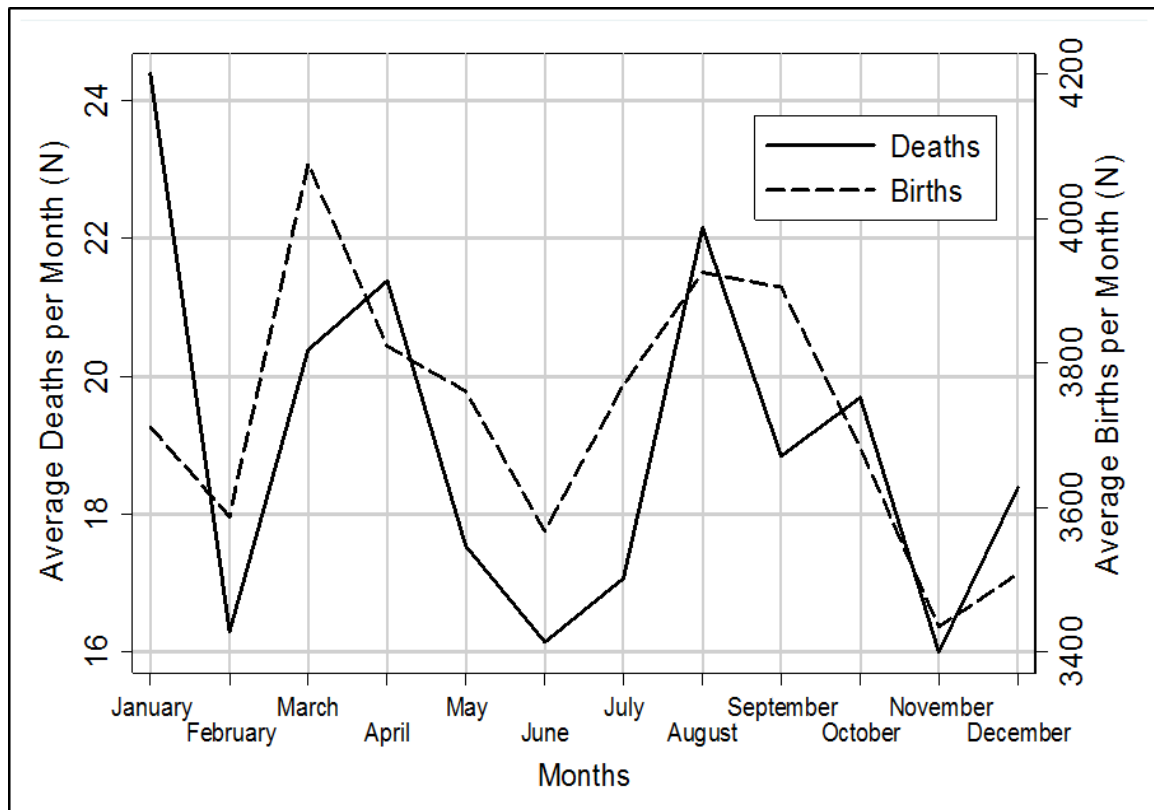
Figure 5.13 shows the average number of births per month from January, 1883, to December, 1895, plotted against the average number of deaths by month of birth for the same period, using the reconstructed month of birth. Since it is difficult to get an estimate of the Ontario-born population who were alive in Ontario at the start of September 1918, using the number of births that occurred each month gives a place (albeit far from ideal) to begin looking for divergences.⁶¹ Births in Ontario in the late 19th century followed a cyclical pattern, which was recognized at the time. Marriages were more likely to occur during the fall and winter months (December registered the most marriages in 12 of the 13 years, Legislative Assembly of Ontario 1883-1898). This had to do with the cycle of farming, with the fewest marriages being in August which was the peak harvest period (Legislative Assembly of Ontario 1884). The pattern of births thus followed the pattern of marriages quite closely, as was noted by the Registrar General in 1891 when he said:

This difference of births may be in a minor degree explained on the assumption made by statisticians that nature is more fertile at some periods of the year than others, but as far as Ontario is concerned, the explanation is better explained by a reference to the Table of Marriages. It is there seen that from December to August, with variations to some extent in other months, the monthly number of marriages declines. [Legislative Assembly of Ontario 1893:3]

Similar to many other years, in 1890 the Registrar General proclaimed that “March, as usual, returned the highest number of births . . . and November the lowest” (Legislative Assembly of Ontario 1892:2, Table 5.6).

⁶¹ Using number of births per month may be an advantage in this case. As seen in Figure 5.9, immigration into Ontario did not begin to increase until between the ages of 10-15. If population totals were used to calculate mortality rates of adults from this period, it would be difficult to separate the immigrants from the native born. The number of births per month in the province exclusively refers to those born in Ontario, while the infant mortality rate almost entirely relates to the Canadian-born (as in fn. 54, the infant mortality rate was relatively constant through this period, with a few exceptions, but these measures were notoriously unreliable due to the deficiencies of the records used in the calculations).

Figure 5.13 – Average Number of Birth per Month and Deaths by Month of Birth in Ontario, 1883 to 1895.



Note: Births, N=581,842, Deaths (for those with a reconstructed month of birth), N=2,965.

The average number of deaths per month of birth that occurred from September to December, 1918, for each month of birth from January, 1883, to December, 1895, follows the pattern of births per month closely, with a few differences. The month with the highest average number of births per year from 1883-1895 was March, and there were increases in the average number of births in both August and September. The fewest births each year took place in February, June, and November. While March had the highest average number of births per year, the deaths to those born in April were more numerous than for those who were born in March. The second highest average number of births in August and September accords with the high number of deaths during the epidemic to those born in August (however, there is a decline in the average number of deaths during the pandemic for those born in September). The only major divergence between the two curves occurs in January. For the period between January, 1883, and

December, 1895, the month of January was average in terms of number of births (the two modal months being January and May). However, by far the highest average numbers of deaths from September to December, 1918, occurred among those who were born in January. Granting full-term pregnancies, these individuals were in their first and second months of gestation in the less plentiful spring and summer months. Their third trimester and births then occurred in the more plentiful autumn and winter, with the birth occurring in the midst of influenza season (and during the 1890 influenza pandemic). Season of birth and resources available per trimester may have an influence on later mortality through fetal conditioning (Barker 1995, Gagnon 2012 and also environmental conditions at time of birth, Doblhammer 2004). Using the exact numbers of deaths by month of birth for each month during the study period, an Edwards test for seasonality was significant ($\chi^2=20.90$, $df=2$, $p<.001$). However, the Walter and Elwood test which includes the population at risk (births in that month) was not significant ($\chi^2=4.04$, $df=2$, $p=.1328$) (Walter 1977, Rau 2007).^{62, 63}

In order to determine if this increase in the number of deaths to those born in January was in some way related to the age of exposure to the 1890 influenza, it is necessary to calculate a hypothesized potential age of exposure to the pandemic, based on the reconstructed date at birth. The 1890 influenza was only present in epidemic form in Ontario in the first wave for a very short time - from December, 1889, through the end of February, 1890. As this disease had an estimated 60% clinical attack rate (Valleron et al. 2010) and a low mortality rate (such that many people were infected but few died) age in January, 1890, is used as a proxy for age of exposure to the Russian influenza pandemic. In order to more accurately specify exact age at exposure, I used age on January 15, 1890, as the day of potential exposure.

⁶² The Edwards test looks for seasonal differences in disease occurrence or mortality. In this test, “the only data required . . . are the numbers of cases of the disorder in question, grouped into appropriate time intervals such as months” (Walter 1977:137). The Walter and Elwood test extends the Edwards test to include differences in the population at risk for each month.

⁶³ I am using the births per month as the population at risk, instead of the actual population alive in Ontario between September and December, 1918, born in January 1890 who were at risk of dying. Taking intervening mortality and migration into account would change the result of the Walter and Elwood test for seasonality.

Based on this hypothetical exposure date of January 15, 1890, every individual in the database with a reconstructed age at death was given an ‘exposure age.’ Using this date, I categorized each individual into one of 6 different age categories (Table 5.7), assuming an average length of gestation of 283 days (Smith 2001). In Category 1 (N=1,258) are those who were older than one year at the time of exposure to the epidemic influenza in 1890. They were born between January 1, 1883, and January 14, 1889 (2,206 days), and the first influenza epidemic that they were exposed to was a likely a form of H1N8 (Worobey et al. (2014) posit that this strain was circulating between 1847-1889). If they somehow did not contract influenza before 1890, they would have had a better chance at mounting an immune response to influenza than for those under one year of age: Infants are usually protected from influenza through maternal antibodies acquired in breastfeeding, which begin to taper off at the introduction of solid foods around six months of age (Kreijtz et al. 2011). Category 2 contains those less than one year of age at the time of potential infection (N=256). These individuals were born between January 15, 1889, and January 14, 1890, (366 days) and their first exposure to influenza would have been either the (hypothesized) H1N8 or H3N8 forms. Infants at the time of exposure are separated in order to determine whether this age group was at increased risk in 1918.

Table 5.7 - Age at Potential Exposure to the 1890 Influenza Pandemic.

Age at Exposure ^b	Age at Death	Deaths		Days in Time Period		Expected Deaths ^a	Deaths per Day
		N	%	N	%	N	N
1. > 1 year	29-35	1,258	42.5	2,206	46.46	1,375.2	0.57
2. 1 to 365 days	28-29	256	8.7	366	7.71	228.2	0.70
3. 3 rd Trimester ^c	28	82	2.8	95	2.00	59.2	0.86
4. 2 nd Trimester ^c	28	84	2.8	94	1.98	58.6	0.89
5. 1 st Trimester ^c	27-28	67	2.3	94	1.98	58.6	0.71
6. Not exposed	23-27	1,213	41.0	1,893	39.87	1,180.2	0.64
Total		2,960	100.0	4,748	100.1	2,960	0.62

Note: Age on January 15th, 1890, using the Reconstructed Date of Birth for Individuals born between January 1883 and December 1895, for those who had a reconstructed date at birth.

^a. Calculated as the number of deaths multiplied by the proportion of days in the time period.

^b. Age at Potential Exposure to Epidemic Influenza

^c. Refers to maternal exposure while the decedent was *in utero*.

Categories 3, 4, and 5 represent different stages of gestation. Maternal infection in the different trimesters of fetal development pose different risks to the developing infant (Chapter 2). Those who were in their third trimester of gestation at the time of exposure to the 1890 pandemic were born between January 15, 1890, and April 20, 1890 (N=82, 95 days). Those in the second trimester of gestation were born between April 21, 1890, and July 23, 1890 (N=84, 94 days). Finally, those who were in the first trimester of gestation at the time of exposure to the pandemic in 1890 were born between July 24, 1890, and October 25, 1890 (N=67, 94 days). The final age group, Category 6, includes those born between October 25, 1890, and December 31, 1895 (N=1,213, 1893 days). The youngest person in the WMMIP database was actually born on December 27, 1895). These individuals had not yet been conceived when Ontario was exposed to the worst of the pandemic influenza strain in 1890. The first influenza strain that they would have been exposed to was the H3N8 that was declining in virulence from the peak in 1890 (Worobey et al. 2014).

These age categories can be seen in Table 5.7 along with the number of days in each time period, the expected number of deaths in each period if the number of deaths were distributed equally, and the average number of deaths per day to those individuals in each time period. For example, among those who died between September and December, 1918, 1,258 were older than one year at the time of their exposure to the 1890 pandemic strain. There were 2,206 days between January 1, 1883 and January 14, 1889, giving an average number of deaths per day for those born in that period of 0.57.

The difference between the number of deaths from those born in each time period that would have been expected by the number of days in that time period as seen in Table 5.7, is statistically significant (chi-square test: $\chi^2=35.28$, $df=5$, $p<.001$). There are fewer deaths than would be expected for those who experienced the Russian pandemic at the age of one year or older (Category 1). This could be the influence of either previous exposure to a similar influenza strain or due to the selection effect of intervening mortality. Because those who were older at the time of exposure to the 1890 influenza had necessarily lived longer than those born in later years, they had been at risk of death for a longer period of time. Therefore, there may have simply been fewer individuals present in 1918 who were at risk of dying. Those who were not yet conceived at the time of the 1890 pandemic (conception took place after January 16, 1890 and birth took place on or after October 26, 1890, Category 6) had slightly more deaths than were expected. Infants (those between 1 day and 365 days old) and those who were in the 2nd and 3rd trimesters of gestation on January 15th, 1890 died in greater numbers than expected (Categories 2, 3, and 4).

There is also a significant sex difference by age at exposure (Table 5.8, chi-square test: $\chi^2=12.16$, $df=5$, $p=.033$). As with general flu deaths and tuberculosis deaths, the sex distribution of death is relatively equal for most of the categories of potential exposure to the 1890 influenza pandemic. The only notable exceptions are in the categories of older than one year, where more males who were exposed at this age died in 1918 than females, and in the maternal exposure in the first trimester of pregnancy, where more females died than males in 1918. This may have to do with the differential robustness of

male and female fetuses, the effect of intervening mortality, and potential scarring, which is explored in the following section.

Table 5.8 – Age at Potential Exposure to the 1890 Influenza Pandemic by Sex.

Age at Exposure ^a	Male		Female		Total	
	N	%	N	%	N	%
1. > 1 year	696	55.3	562	44.7	1,258	100.0
2. 1 to 365 days	123	48.0	133	52.0	256	100.0
3. 3 rd Trimester ^b	41	50.0	41	50.0	82	100.0
4. 2 nd Trimester ^b	43	51.2	41	48.8	84	100.0
5. 1 st Trimester ^b	27	40.3	40	59.7	67	100.0
6. Not exposed	612	50.5	602	49.6	1,213	100.0
Total	1,418	47.9	1,542	52.1	2,960	100.0
χ^2				12.16*		
df				5		

Note: Age on January 15th, 1890 using the Reconstructed Date of Birth for Individuals born between January 1883 and December 1895, for those who had a reconstructed date at birth.

*p<.05

^a. Age at Potential Exposure to Epidemic Influenza

^b. Refers to maternal exposure while the decedent was *in utero*.

5.2.1 Fetal Growth Restrictions

Using the fetal growth restrictions hypothesis in order to explain the excess mortality among young adults in 1918 by definition must focus on those individuals who were *in utero* at the time of their exposure to the 1890 Russian influenza pandemic. Therefore, it applies only to those who are in Category 3 (exposure in the third trimester), Category 4 (exposure in the second trimester), and Category 5 (exposure in the first trimester). From Table 5.7, it can be seen that there were slightly more deaths from September to December, 1918, for those who were *in utero* at the time of exposure to the 1890 Russian influenza pandemic (necessarily referring to maternal exposure which through some mechanism may impact fetal development, see Chapter 2). Deaths were elevated in Categories 3 and 4, which lends support to the hypothesis that maternal exposure during the second and third trimesters of gestation is detrimental to fetal development. Maternal deprivation of resources (which can also occur through maternal illness) can lead to higher risk of death from airborne infectious disease in adulthood, through a deleterious

effect on the fetal immune system (Moore et al. 1999). As with the scarring hypothesis, it is harder to argue that these individuals are at greater risk from influenza specifically, since the physiological impacts are global rather than specific. Further, as discussed in Chapter 2, there were many diseases circulating in epidemic form at the time in Ontario and fetal growth restrictions would have elevated the risk of death from all of these causes. However, the severity of the 1918 influenza pandemic may have been such that it affected even those who were only mildly impaired from their maternal infection in 1890. Those individuals who had only been weakened slightly may have been able to survive other insults until the 1918 virus proved to be too strong. Without a prospective cohort study starting from birth records, this is only speculative; however, an increase in deaths among those *in utero* at the time of exposure to the 1890 does provide some support for the fetal growth restrictions hypothesis. This is stated with the caveat that it is not possible with these data to separate the effects of the fetal growth restrictions (maternal exposure *in utero*) from antigenic imprinting (neo-natal exposure), since these individuals were born into an environment with circulating H3N8, in either epidemic or endemic form. If they were exposed to this form of influenza, it may have primed them to this particular strain, placing them at greater risk in 1918. However, they might also have been doubly impacted by a compromised immune system from maternal exposure which compounded the pre-conditioning to the H3N8 strain of influenza.

More support for the fetal growth restrictions hypothesis is found by looking at those individuals in Category 5, or those who were in their first trimester of gestation at the time of the 1890 influenza pandemic. While deaths among these individuals in 1918 were elevated, it was not as great as for the other two categories of gestation and contributes very little to the chi-square (Table 5.7). What separates this from the other two categories is the differences by sex of those individuals who died in 1918 (Table 5.8). The average male-to-female sex ratio at birth falls between 1.04:1 to 1.07:1 which means that more male infants are born than female infants (Hobbs 2004:133). The Registrar General for Ontario reported the male-to-female sex ratio at birth (based on birth records) at 1.06:1 in 1890 (Legislative Assembly of Ontario 1892). This advantage soon disappears however, since neonatal deaths target males more than females, at a ratio of around 1.28:1 (Naeye et al. 1971). The lowest male-to-female sex-ratio at birth in

1890 (of registered, not late return, births) occurred in June, approximately 6 months after maternal exposure in the first/second trimester of pregnancy (1.03:1, calculated from Legislative Assembly of Ontario 1883-1898). However, this was not anomalously low for the time period, and the lowest male-to-female sex-ratio at birth was found in March 1889, (0.98, the only time between 1883 and 1895 where the sex-ratio fell below 1.00. See figure in Appendix E).

It has been noted that the male-to-female sex ratio at birth drops when populations are under stress, such as with increasing unemployment as found by Catalano and colleagues (2005), but also from “natural disasters, pollution events and economic collapse” and also the population stress following the World Trade Tower bombings in the United States in 2001 (Bruckner et al. 2010:273). The mechanisms put forth as potentially being responsible are 1) physical stresses to which males are more susceptible, such as hormonal imbalances, 2) that stressed men are less likely to make sperm that produces male offspring, and 3) that stressed individuals are less likely to have sex, and that conception later in the menstrual cycle is more likely to produce female offspring (2005:944). The evidence linking reduced unemployment and the population stress following the bombings to a lowered sex ratio at birth both supported the first mechanism (2005:947), which would also be consistent with the acute physical stress of maternal influenza infection. This is supported by a study by Byrne and Warburton (1987) on sex-typed embryonic and fetal loss. They found that “the male excess was confined to normally formed specimens (1.30) and was present at all gestational ages up to 23 weeks and all sizes over 5 cm” but that “among malformed specimens the sex ratio was close to unity (0.92)” (1987:605). This means that both male and female fetuses have an equal risk of suffering from a congenital malformation and being subject to a spontaneous abortion. However, among those fetuses that are not malformed, males are more sensitive to extraneous stresses which would cause the pregnancy to end. That there were fewer deaths than expected in 1918 among those who were in the first trimester of gestation at the time of maternal exposure (compared to the other trimesters), but that there were more female deaths, suggests the possibility that the weaker male fetuses were miscarried after their mothers contracted the 1890 Russian influenza. This would suggest that there were fewer Ontario-born men who were at risk of dying in 1918 who had been

born between July 24, 1890 and October 25, 1890. This could also be ascertained through a prospective records linkage project that starts with the birth records. However, the male-to-female sex-ratios at death for 1890 as provided by the Registrar General also suggest that this mechanism may have been in operation. Based on the variability of the date of exposure (January 15, 1890 is arbitrary), the lowest sex-ratio of the year occurring in June might offer support for the loss of weaker male fetuses in the first trimester of gestation. The highest ratio in October may indicate that this process is not at work in the earliest weeks of conception and pregnancy. Although the evidence suggests support that the fetal growth restrictions hypothesis might have been a factor in the higher excess mortality among young adults in Ontario in 1918, the evidence provides better support for the antigenic imprinting hypothesis.

5.2.2 *Scarring*

In terms of this research, the scarring hypothesis posits that some of individuals who contracted the 1890 influenza would have had physiological damage to their lungs that would make them less able to survive a later, severe respiratory infection. This presumes that individuals were alive at the time of the epidemic (or January 15, 1890) and therefore only those in Category 1 (born between January 1, 1883 and January 14, 1889) and those in Category 2 (born between January 15, 1889 and January 14, 1890) would have been subject to this process. Therefore, if scarring was at least partially responsible for the increase in young adult mortality in 1890, there should be an elevated number of deaths for individuals who died in 1918 who were born in those two categories.

As seen in Table 5.7, this was not the case for individuals in Category 1, who were between the ages of 1 and 7 at the time of their first exposure to the H3N8 influenza virus in 1890. Mortality among this age group was less highly elevated than for the other categories, meaning that mortality in 1918 was not concentrated among those who were older at the time of their first infection. However, mortality for those in Category 2 who were infants (less than one year) at the time of potential infection to the 1890 strain was higher than expected, which may provide support for the scarring hypothesis. Nursing

infants are able to mount an immune response to influenza using the maternal antibodies in breast milk, but this protection decreases with the introduction of solid foods which has usually occurred by the age of six months (Wharton 1989, Jackson and Nazar 2006). The scarring hypothesis would predict that those infants who had either begun or completed weaning at the time of exposure to the 1890 Russian influenza pandemic would be at higher risk of scarring since they were dependent upon their own, immature, immune system (Hanson et al. 1985, Holsapple et al. 2003). Younger infants would have been at less risk of scarring as they would have been able to utilize their mothers' immunological memory.

The difference by age (in months) at exposure for Category 2 is not significant (chi-square test: $\chi^2=17.62$, $df=11$, $p=.09$). However, the ages of exposure that had the highest numbers of deaths in 1918 were all under 6 months (less than one month (neo-natal exposure), $N=27$; 2 months, $N=28$; and 5 months, $N=28$). There is a much larger difference when the infants are separated in to those under six months ($N=142$, 55.9%) and those six months and over ($N=113$, 44.1%), which runs directly counter to the scarring hypothesis (assuming an age at weaning of 6 months). The evidence garnered from the deaths of those individuals who died in Ontario between September to December 1918 and who were born in the province between 1883 and 1895 does not support the scarring hypothesis as the predominant cause of elevated young adult mortality during the 1918 influenza pandemic. As mentioned in Chapter 2 and for the fetal growth restrictions hypothesis, the scarring process is not influenza specific; it may have been caused by any severe respiratory infectious disease in childhood and would have placed an individual at risk from a variety of other airborne infectious diseases, which were abundant in this time period. Individuals may have been scarred from the 1890 flu, which had a high morbidity rate and low mortality rate (Valleron et al. 2010). If so, they were at an increased risk of death at all stages of life and may not have survived until 1918. The scarring process did not have an impact in causing the unexpectedly high surplus mortality of young adults in 1918, but it may have severely increased the mortality risk at the individual level at any point after the scarring occurred.

5.2.3 *Antigenic Imprinting (Original Antigenic Sin)*

The antigenic imprinting hypothesis posits that those individuals who were at greatest risk during the 1918 pandemic were those who were immunologically primed to produce antibodies to a different strain of influenza (Ma et al. 2011). The 1918 strain was H1N1 (Taubenberger and Morens 2006), while the 1890 strain was potentially H3N8 (Worobey 2014). According to this hypothesis, the first strain that an individual is exposed to in their lives is the one that they will at first produce antibodies for whenever they next become exposed to influenza. Further, this process is stronger the more virulent the first strain is: this reaction is stronger the more experience an individual has with the particular strain (Davenport and Hennessey 1956). Therefore, it would be the older individuals in the sample who would have been at greatest risk as they would have had longer potential exposure to the strain. This would be Categories 1 and 2. Category 1 contains those older than one year of age at the time of the epidemic (born between January 1, 1883 and January 14, 1889 and aged 29-35 at the time of death). All of these people would have been reliant upon their own immune systems to fight the influenza. As influenza is widespread among infants and children (Glezen and Couch 1997), these individuals would likely have first been exposed to a circulating form of H1N8 (Worobey 1918). Individuals in Category 2 were those who were one year and under at the time of the 1890 pandemic (born between January 15, 1889 and January 14, 1890 and aged 28-29 at the time of death). Those individuals over six months of age, and those younger who were not breastfeeding, would have first been exposed to the same H1N8. However, those who were being protected from the H1N8 strain by their maternal antibodies through breast milk likely would have first faced the H3N8 as their first influenza experience.

As in Table 5.7, those in Category 1 died in lower numbers than expected. Again, this could be due to the issue of intervening mortality, in that it may have been that this group of individuals was smaller among the decedents in 1918 because they had lived longer and were subject to mortality risks for a longer period of time. As there is no way to determine this with this study (it would take a prospective records linkage study starting with the birth records of an entire cohort), I will analyze this data with the understanding

that the populations at risk were different at each age, but that with the large denominators at the province level these differences are of less importance (for example, 100 deaths at age 23 would give a rate of 2.08 deaths per 1,000 population, while 100 deaths at age 37 would give a rate of 2.38 deaths per 1,000 population).⁶⁴

The findings in this database for deaths in Ontario from September to December, 1918, are consistent with the antigenic imprinting hypothesis. The fewer deaths than expected in the older ages (Category 1) are in accordance with the idea that these individuals were first conditioned to a form of H1N8. Being pre-conditioned to this strain had a less deleterious impact than being pre-conditioned to the epidemic form of H3N8, perhaps because the H1N8 strain pre-1889 and the H1N1 strain in 1918 shared the H1 antigen.

Higher deaths among infants (those under one year of age, Category 2) also provide support for the antigenic imprinting hypothesis. Those who were older than six months of age in this Category and past the age at which the introduction of solid foods is necessary (Wharton 1989, Hendricks and Badruddin 1992) and thus no longer fully under the protection of their maternal antibodies, would have been first exposed to the H1N8 strain, similar to those in Category 1, with some variation due to breastfeeding practices. However, if exposed in this first year, it would be to the H3N8 epidemic variant while their immune systems were still immature (Hanson et al. 1985, Holsapple et al. 2003). Those younger than 6 months would have had maternal protection from the H1N8 strain before the epidemic and also the H3N8 strain during the epidemic in 1890 (if exposed at all, cf. Bodewes et al. 2011). The first strain that they would have encountered while reliant on their own immune response would be the H3N8 Russian influenza strain, after

⁶⁴ Using the interpolation and Sprague's multipliers techniques from Chapter 3, I am able to have a rough estimation of the population at risk in Ontario at each age. However, this includes all the residents in Ontario, and not only those who were born in the province. The populations at risk at each age, rounded to the nearest whole number, are:

Age	Population in 1918	Age	Population in 1918	Age	Population in 1918
23	48,029	28	46,579	33	42,307
24	47,910	29	45,572	34	42,235
25	47,777	30	44,571	35	42,069
26	47,687	31	43,471		
27	47,336	32	42,651		

it had declined from its epidemic form. Accordingly, if antigenic imprinting was in effect, we would expect to see higher deaths in 1918 among those who were infants at the time of the 1890 pandemic, but that among those infants there would be higher deaths among those who were younger at the time of the pandemic in 1890.

As mentioned above for the scarring hypothesis, the difference by age (in months) at exposure for Category 2 is not significant, but there is a larger difference when the infants are separated in to those under six months (N=142, 55.9%) and those six months and over (N=113, 44.1%). Opposite to the scarring process, the increased number of deaths among the younger infants supports the antigenic imprinting hypothesis.

Categories 3 to 6 all include individuals who died in 1918 who had not yet been born at the time of the 1890 pandemic. Barring seasonal flu strains, each individual in these categories would have first been exposed to the H3N8 form after it had become endemic and lessened in virulence. While there are more deaths than expected in these categories, the difference is greatest for those who were born closest to January 15, 1890 and declines thereafter. Again, consistent with the antigenic imprinting hypothesis, those who are first exposed to more virulent forms of a different strain of influenza (and thus more encounters with the virus) would suffer the worst consequences from the H1N1 in 1918 (Davenport and Hennessey 1956) Therefore, the data from this study does offer support for the antigenic imprinting hypothesis.

5.2.4 *Conclusion*

In this section, I addressed the research question:

3. *Since young adults have already been shown to be at an unusually high risk of death, could this be due to:*
 - a. *Previous exposure to influenza, resulting in physiological impairments or conditioning?*

I evaluated the deaths among young adults who were born in Ontario and who died in the province between September and December, 1918 to address three main hypotheses: fetal growth restrictions, scarring, and antigenic imprinting (original antigenic sin). These data provide cautious support for fetal growth restrictions hypothesis and stronger support for the antigenic imprinting hypothesis, while the scarring hypothesis is not evidenced by the data. We know that young adults had higher than expected excess mortality in 1918 and we know that this was worse for those aged 28. In Chapter 2, I asked the question “can it sufficiently be argued that, *ceteris paribus*, an individual at age 28 has a stronger immune system than someone at age 27?” since it has been thought that those with stronger immune systems were at greater risk of dying from the cytokine storm. The answer to this question is no – there is no reason to expect that a difference of one year of age would make substantial alterations to the quality of the immune system. Yet, the antigenic imprinting hypothesis allows us to say that, in 1918, an individual at age 28 had a sufficiently different *immune response* to the H1N1 influenza strain than someone at age 27 and that it was this difference that in some instances proved lethal.

5.3 Tuberculosis and Influenza Co-Morbidity

The last section of this chapter analyzes the reconstructed data set to answer the second part of the third research question:

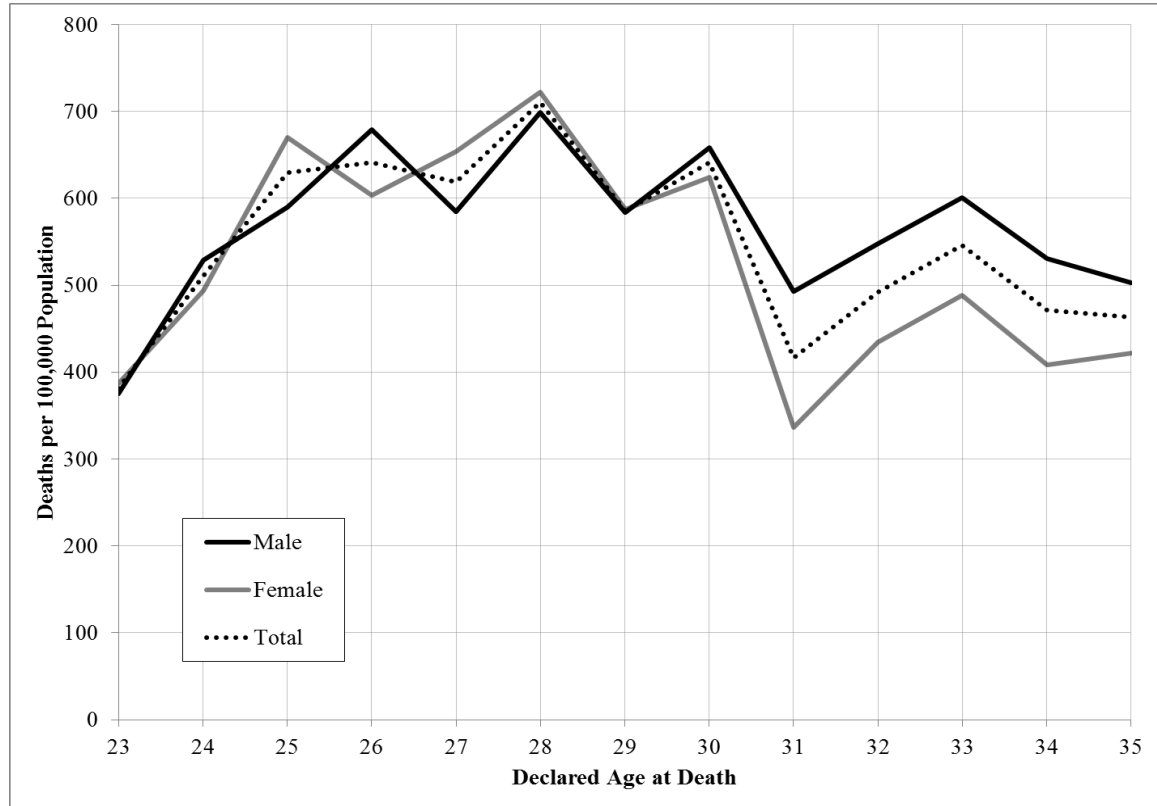
3. *Since young adults have already been shown to be at an unusually high risk of death, could this be due to:*
 - b. *Co-morbidity with tuberculosis?*

In their earliest article, Noymer and Garenne report that the 1918 Spanish influenza pandemic was known for its “maleness” since there was “a difference between male and female age-standardized death rates of 174 per 100,000” (Noymer and Garenne 2000:565). This high number contrasts with a male minus female age-standardized death rate of 38 per 100,000 in 1917 and of 13 per 100,000 in 1919 (2000:579). The cause of this was that “those with tuberculosis (TB) in 1918 were more likely than others to die of

influenza. This outcome affected males more than females because TB morbidity was disproportionately male” (2000:565). They were trying to explain the reduction in tuberculosis mortality after 1918, by arguing that there were fewer young men at risk to die from tuberculosis because they had already died during the influenza pandemic. This was later renamed by Noymer (2009, 2010) as active selection, whereby those with tuberculosis were more likely to die from influenza. He argues with more recent evidence that passive selection is more likely, which occurs at a population level, such that there were fewer tuberculosis deaths in the years following 1918 because the pool of susceptibles was greatly reduced by the influenza pandemic in 1918. However, contingent on both of these processes is that males died in greater numbers than females.

Of the 3,316 individuals in the database who died in Ontario between September and December 1918 and who were also born in the province, 1,573 (47.4%) were female and 1,745 (52.6%) were male. As seen in Figure 5.14 (also Figure 3.7), the mortality rate by age and sex for Ontario did show some discrepancies. Using the declared age at death, both male and female rates of death were highest at age 28 and low at age 31. After age 30, male death rate was systematically higher than female, while they were more similar between the ages of 23 and 29. These differences by sex are not statistically significant using a chi-square test (does not include ages 22 or 37, $\chi^2=19.17$, $df=12$, $p=.085$).

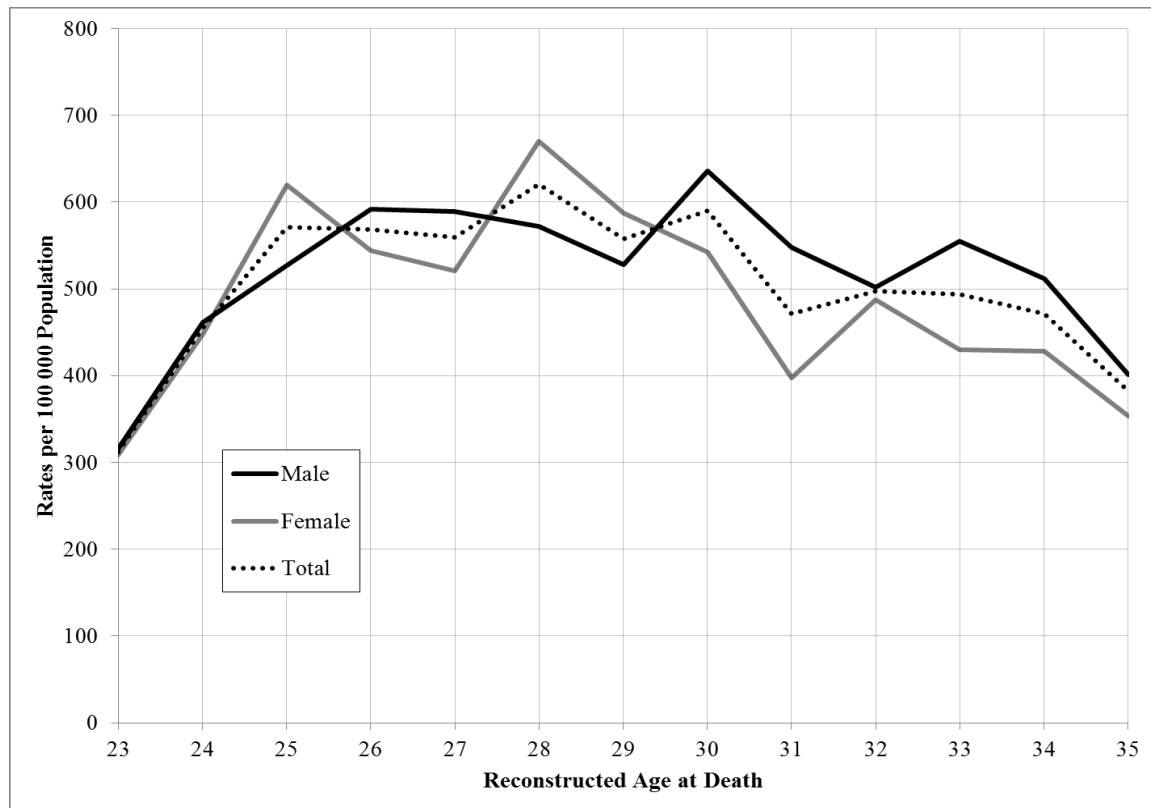
Figure 5.14 - Mortality Rates by Sex for the individuals in the WMMIP Database.



Note: Using Declared Age at Death and the Interpolated 1918 Population Totals. Male (N=1,693) and Female (N=1,539).

There appears to be more discrepancies between the age-specific death rates for males and females using the reconstructed ages at death (Figure 5.15). Most notably, while the peak at age 28 remains for females, it has shifted to age 30 for men. Male mortality remains higher than female mortality at ages 30 and above. These differences are also not statistically significant (chi-square test: $\chi^2=18.17$, $df=12$, $p=.111$) which does not support the active selection hypothesis that influenza and tuberculosis co-morbidity had an influence on mortality during the second wave of the Spanish influenza pandemic in Ontario. This finding is reinforced by an analysis of those individuals whose deaths were caused by tuberculosis.

Figure 5.15 - Age Specific Death Rates using the Reconstructed Age at Death in Ontario, September to December, 1918.



Note: For those who had a reconstructed age at death, Male (N=1,543), Female (1,423), Total (N=2,966).

Of all the causes of death among the 3,316 individuals in the database, 200 had some indication on their death record that death was either caused by, or related to, tuberculosis (see Section 3.1, fn. 30). Of those 200 deaths, 102 were male and 98 were female ($\chi^2=0.21$, $df=1$, $p=.645$). Separated by month of death, 25 of the deaths occurred in September (14 male, 11 female), 75 occurred in October (37 male, 38 female), 43 occurred in November (22 male, 21 female), and 55 occurred in December (28 male, 27 female). The differences between the month of death and death caused by tuberculosis is significant ($\chi^2=68.19$, $df=3$, $p<.001$). However, this is because there are more tuberculosis deaths than would be expected in September, November and December, and fewer deaths than expected in October. As discussed in Section 3.1, this may be a result of underreporting during the epidemic. Deaths may have occurred so quickly and in such

great numbers, that the informant and the physician may have reported the immediate cause of death as due to pandemic influenza without recording the prior infection with tuberculosis. This can be seen in records of 3 individuals who died in tuberculosis hospitals but whose death records did not indicate infection with tuberculosis (these 3 men died at three different hospitals, but all died in the epidemic months. They died at 1) the Mowat Sanatorium in Kingston on October 23, 1918; 2) the Queen Alexandra Sanatorium in Byron on October 29, 1918 and; 3) the Mountain Sanatorium in Hamilton on November 29, 1918). If it was not caused by underreporting, the finding of more deaths than expected in September and fewer than expected in October would also not support the active selection hypothesis.

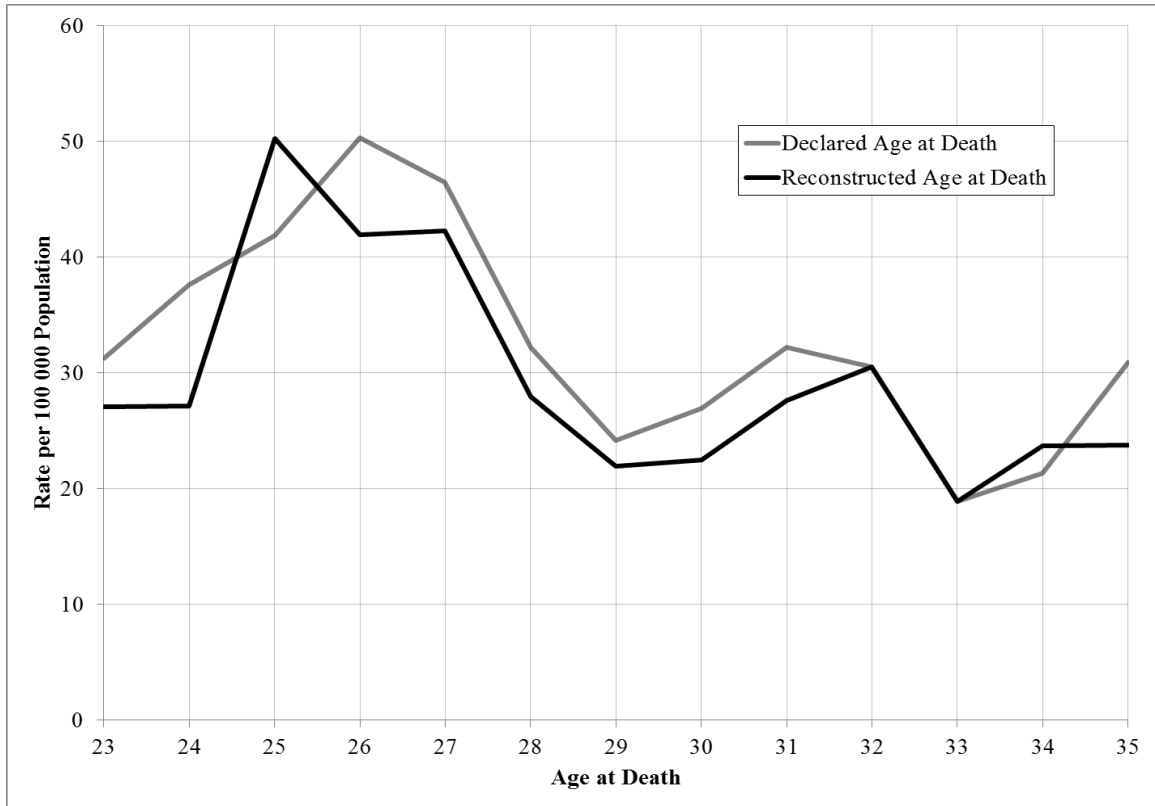
Only 25 individuals in the WMMIP database had both influenza and tuberculosis mentioned on their death records (12.5% of the individuals who died of tuberculosis, 0.89% of those who died of influenza, and 0.75% of all individuals in the sample). Yet, since tuberculosis can have a long latency period during which the individual has been infected but does not yet have the disease, and a large percentage of the population may be infected at any one time, this does not preclude a relationship between tuberculosis infection and influenza mortality (currently, the World Health Organization estimates that up to one-third of the global population has been infected with tuberculosis, but that only about 10% of those infected will become ill, World Health Organization 2014, Noymer and Garenne 2000). However, our data do not provide evidence of a relationship between tuberculosis infection and influenza mortality.

Among those who died from tuberculosis, 68 (34.0%) died in urban areas, while 132 (66.0%) died in rural areas. This contrasts with the entire database, where 1,700 (51.3%) died in urban areas and 1,616 (48.7%) died in rural areas ($\chi^2=22.50$, $df=1$, $p<.001$). This is not accounted for by death in tuberculosis hospitals either, which tended to be in more rural areas due to Ontario's support of the "Open Air Treatment of Consumption" in which the disease could be cured or at least mitigated through, *inter alia*, access to fresh air, sunlight, verandahs, solaria, and "walks through the grounds with graduate inclines" (Legislative Assembly of Ontario 1901c:72-3, Adams and Burke 2006). The death records show that of the 200 individuals who died from tuberculosis, 58 died in

institutions, while only 15 of those were specifically tuberculosis sanitarium. Further complicating the issue is that tuberculosis was “a disease historically associated with the density of population in cities, with the insanitary conditions especially incident to the accumulations of organic matters due to the existence of life on close areas, to the ill ventilated abodes of the poor, and to the factories wherein the millions of city dwellers spend their existence” (Legislative Assembly of Ontario 1901c:16). Deaths would therefore be predicted to be more numerous in urban areas.

Using both the declared age at death and the reconstructed age at death, the majority of tuberculosis deaths occurred for the younger individuals in the sample. As seen in Figure 5.16, the highest rate of deaths was at age 26 using the declared age at death and at age 25 using the reconstructed age at death (of the 200 individuals who died of a tuberculosis related cause, 176 were able to be linked through the records linkage process). However, these differences were not significant at $\alpha=0.05$ using declared age at death with a chi-square test ($\chi^2=20.48$, $df=12$, $p=.059$) or the reconstructed age at death ($\chi^2=18.57$, $df=12$, $p=.099$); however, they are significant at $\alpha=0.1$ which may be a result of the small numbers of deaths at each age (range 8-24).

Figure 5.16 - Age-Specific Death Rates for Deaths from Tuberculosis, September to December, 1918.



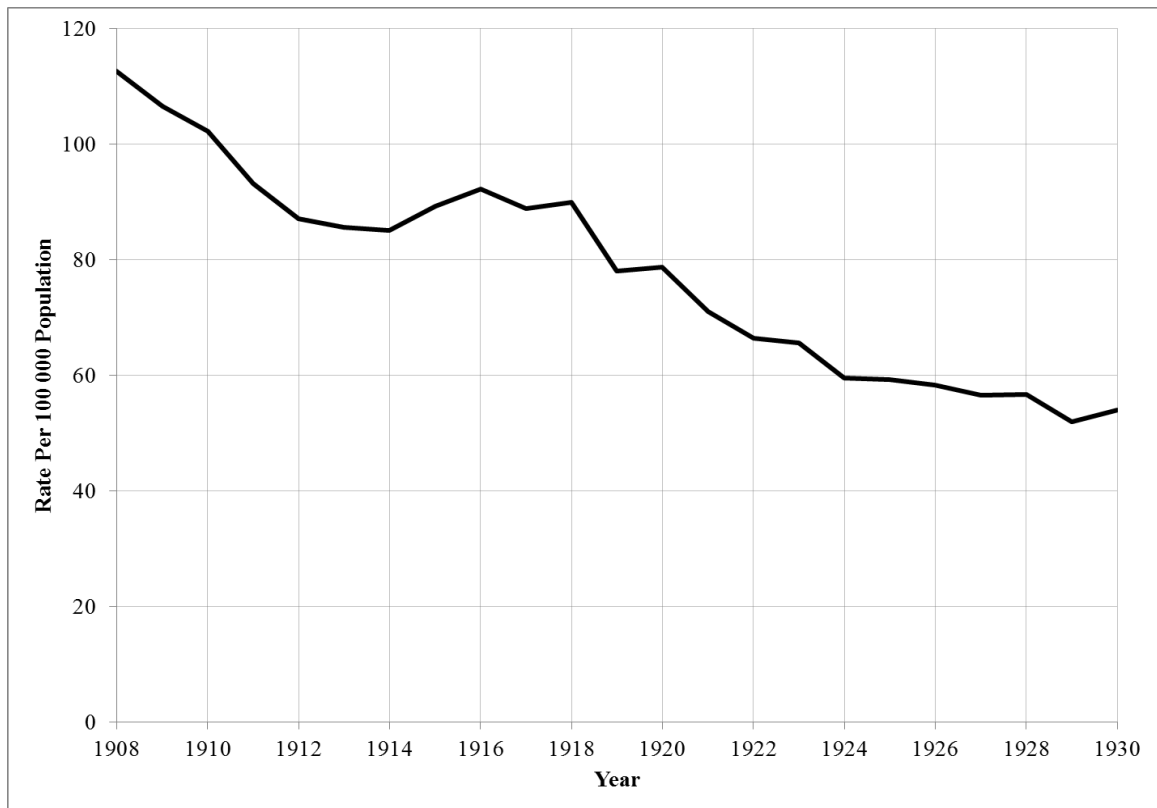
Note: Rates created using Sprague's Multipliers (Chapter 3). Declared age at death (N=200), reconstructed age at death (N=176).

Both the tendency towards younger ages as well as the large number of rural deaths may be due to this pandemic having occurred so close to the First World War, as there appears to be a relationship between tuberculosis and service in the military. While only 6.0% of the total population in the sample died of tuberculosis, 12.1% of those who were identified as soldiers died of tuberculosis ($\chi^2=7.75$, $df=1$, $p=.005$). Of those who died of tuberculosis, 7.0% were soldiers, while only 3.5% of the people in the database were soldiers. Tuberculosis is an airborne infectious disease, often noted to be associated with crowded conditions, lack of ventilation, and poverty (Sherman 2006). Its association with service in the army is not surprising, considering the amount of individuals who were brought together, housed in close barracks, and who endured the conditions of trench warfare, as was reported by the Registrar General in 1920 (Legislative Assembly of

Ontario 1920). Since the average age at enlistment in the First World War was 26 (Leroux 2010) and men were being brought together from all over the country, this could explain the preponderance of tuberculosis among those of younger ages and those from rural locations (especially if those from rural locations had not previously been exposed to the virus). Further, 52.0% of the tuberculosis deaths occurred among unmarried individuals while only 33.3% of the total sample was single. Most of those who enlisted in the Canadian Expeditionary Force were unmarried (79.7%, Leroux 2010). However, an association with the war would also suggest an increased number of tuberculosis deaths among men, which was not found in this data.

Noymer's (2009, 2010) passive selection argument shows that the large decline in tuberculosis rates after 1918 was because those who normally would have died from tuberculosis in the years following 1918 had already died from the pandemic. In order for deaths in Ontario to support this hypothesis, there would have to be a noticeable decline in the rate of deaths from tuberculosis in the years following 1918. The Registrar General in 1917 already gave evidence of a declining rate of tuberculosis in Ontario since 1908, with a slight increase during the war years (Legislative Assembly of Ontario 1918). This was affirmed by the report in 1920, which stated that "while army experience seems to have shown that the stress of war conditions greatly excited latent tuberculosis among soldiers, it is gratifying to note that the reduction in the mortality of this disease among our people during the period is a substantial one" (Legislative Assembly of Ontario 1922:11). However, when the tuberculosis rates for Ontario are graphed, the reduction is not centered on 1918. There is a drop in 1919, but the trend is an overall slow decline that started before 1908 (Figure 5.17).

Figure 5.17 - Tuberculosis Mortality Rates in Ontario from 1908 to 1930.



Source: The Reports Relating to the Registration of Births, Marriages and Deaths for the Province of Ontario for the years 1917, 1920 and 1930 (Legislative Assembly of Ontario 1918, 1922, 1932).

Since influenza deaths during the Spanish influenza pandemic from September to December, 1918, did not disproportionately target individuals by sex and deaths caused by tuberculosis did not increase during the worst of the pandemic, there is no evidence to support the active selection hypothesis put forward by Noymer and Garenne (2000) and Noymer (2009, 2010) and countered by Sawchuk (2009). Also, since mortality rates from tuberculosis had been declining since at least 1908, and the increase during the war was due to the activation of latent cases and spread among men in close quarters (Legislative Assembly of Ontario 1922), there is also no support for the passive selection hypothesis. In agreement with Noymer (2009), it may very well be that “that those who died in the middle of the W in 1918 influenza were unhealthy to begin with”

(2009:1600). However, the evidence from Ontario does not support the hypothesis that “unhealthy” refers to the “*disproportionally tuberculous*” (2009:1600).

5.4 Conclusions

This chapter has answered the following research questions:

2. *Were all ages among young adults in Ontario at equal risk of death from the 1918 flu pandemic?*
3. *Since young adults have already been shown to be at an unusually high risk of death, could this be due to:*
 - a. *Previous exposure to influenza, resulting in physiological impairments or immunological conditioning?*
 - b. *Co-morbidity with tuberculosis?*

In terms of the first research question it was found that in Ontario from September to December, 1918, mortality among those who had been born in the province between 1883 and 1895 was more severe for those individuals who were born in and around the time of the Russian influenza pandemic of 1890. Of the hypotheses that have been proposed to account for this process in the third research question, the data best support the antigenic imprinting hypothesis and there is cautious support for the fetal growth restrictions hypothesis. Using this data, there is no evidence that the scarring process had any impact in shaping the pattern of mortality in Ontario during the 1918 Spanish influenza pandemic. Additionally, I found no evidence of a relationship between tuberculosis and influenza, such that those with tuberculosis do not appear to have been at an elevated risk of death. Chapter 6 will explore whether there was socioeconomic differences among those individuals who died in the pandemic of 1918.

Chapter 6

6 Mortality Implications of Socioeconomic Differentials

This chapter addresses the following research question:

4. *Among young adults, were there unequal mortality experiences resulting from socio-cultural or demographic differences among individuals or groups?*

This chapter explores whether there is a relationship between different socio-cultural groups and mortality during the 1918 influenza pandemic and, if so, the nature of that relationship. This is done by analyzing all the linked records in the WMMIP database for indication of group membership and, where possible, how this changed over time. This provides previously unknown information about the lives of the decedents, moving away from their particular circumstances at the time of death, to provide a more nuanced understanding about how people's living conditions may have influenced the way they died. Consistent with Link and Phelan's (1995) theory of fundamental causes, it is hypothesized that those individuals experiencing any form of socioeconomic disadvantage would be at greater risk of dying from influenza, even if rates of infection rates were equal among groups. The mechanism is that social inequality leads to insufficient response to infection and thus higher mortality rates during the 1918 pandemic. While a specific socioeconomic *gradient* may not have been established by 1918 (Bengtsson and van Poppel 2011), I posit that there were mortality differentials among social groups in Ontario consistent with previous findings for both the 1918 influenza pandemic and for historical Ontario in general (Chapter 2).

In order to determine whether certain socio-cultural groups were overrepresented among those who died Ontario in 1918, it is logical to start with the information provided on the death records. Additional information on socio-cultural groups will come from birth and census records.

6.1 Occupation in 1918

On the death records, occupation was declared for 2,580 (77.8%) of the individuals in the study. However, when excluding any individual whose occupation was listed as “wife”, “housewife”, “married woman”, or any version of “not given” or “not known” (as this does not provide any useful socioeconomic information), this number drops to 1,829 (55.2%). This results in an over-representation of men, as 1,512 (82.7%) were men and 317 (17.3%) were women; however, mortality in the Western, McMaster, Montreal Influenza Pandemic (WMMIP) database is relatively equally split between the sexes (52.6% male and 47.4% female).⁶⁵

To maintain contemporaneous conceptions of occupations (as well as what were seen as important distinctions), I classified each individual according to the occupational categories used in the 1911 census (Dominion of Canada 1915).⁶⁶ These categories are: *Agriculture; Construction; Domestic and Personal Service; Civil and Municipal Government; Fishing, Hunting and Forestry;*⁶⁷ *Manufacturing; Mining; Professional; Trade; and Transportation.* I added the category of *Gentleman or Lady*, as well as the category of *Student*. The distribution of these occupational categories among the decedents can be seen in Table 6.1. Using a chi-square test, the differences by sex are statistically significant (the statistical tests in this chapter do not include the categories of *Gentleman or Lady*, or *Student*; $\chi^2=499.87$, $df=9$, $p<.001$). This is to be expected by the “sexual discrimination practiced against women workers” found in Toronto and the gendered nature of work at this time in the entire province (Piva 1979:17, Acton et al. 1974, Cohen 1988). The differences between the occupations by urban and rural status are also statistically significant using a chi-square test, as would be expected since most

⁶⁵ However, this is more in accordance with the distribution of workers by sex. The 1911 census reports that for all of Ontario aged 10 years and older in 1911, of those “employed in gainful occupations,” 84.4% were male and 15.6% were female. (Dominion of Canada 1915:xv, Table 5). Analogous numbers from 1891 show that 87.1% were male and 12.9% female, and from 1911, 85.6% of the workers were male and 14.4% were female. This provides evidence of the slow increase of women in the workforce seen since the end of the nineteenth century (Ramkhalawansingh 1974).

⁶⁶ As detailed by Katz (1972), this classification scheme only measures social structure and not mobility.

⁶⁷ *Fishing and Hunting* is a separate category from *Forestry* in the 1911 Census classification system. I combined the two categories due to the low numbers of individuals in these occupations in the WMMIP database.

farming would have taken place outside of the cities and towns ($\chi^2=375.52$, $df=9$, $p<.001$). It is important to note that the occupational category does not necessarily correspond to economic status since, as Piva explains, “unemployment was an everpresent fact of life for most workers” especially those in the *Construction* trades (1979:30). Further, there was a hierarchy within each of the categories, ranging from labourers to management and owners (Katz 1972, Sager 2007). The question of interest is how closely does the occupational distribution of decedents in 1918 reflect the occupational distribution of all Ontario-born individuals living in Ontario in 1918 (as a proxy for those who did not die).

Table 6.1 –Occupational Category of the Decedents.

Occupational Category	Total WMMIP		Male		Female		Urban		Rural	
	N	%	N	%	N	%	N	%	N	%
Agriculture	413	22.6	359	23.7	54	17.0	39	4.5	374	38.7
Construction	84	4.6	84	5.6	0	0.0	62	7.2	22	2.3
Service ^a	121	6.6	37	2.4	84	26.5	55	6.4	66	6.8
Government	98	5.4	97	6.4	1	0.3	69	8.0	29	3.0
Forestry	24	1.3	23	1.5	1	0.3	3	0.3	21	2.2
Manufacturing	507	27.7	472	31.2	35	11.0	260	30.1	247	25.6
Mining	12	0.7	11	0.7	1	0.3	3	0.3	9	0.9
Professional	185	10.1	88	5.8	97	30.6	119	13.8	66	6.8
Trade	196	10.7	160	10.6	36	11.4	120	13.9	76	7.9
Transportation	180	9.8	175	11.6	5	1.6	127	14.7	53	5.5
Gentleman or Lady	1	0.1	0	0.0	1	0.3	1	0.1	0	0.0
Student	8	0.4	6	0.4	2	0.6	5	0.6	3	0.3
Total	1,829	100.0	1,512	100.0	317	100.0	863	100.0	966	100.0
Missing	1,487		231		1,256		837		650	
χ^2				499.87*				375.52*		
df				9				9		

Note: For those who died from September to December 1918. Included are only those with a declared (meaningful) occupation (N=1,829).

a. Service = Domestic and Personal Service. Forestry = Fishing, Hunting, and Forestry

*p<0.05

As seen in Table 5.1, the majority of those with a declared occupation were in the fields of *Agriculture* and *Manufacturing*. As would be expected, more agricultural jobs occurred in rural areas and there were more manufacturing jobs in urban centres. In terms of the percent in each category, males outnumbered females in jobs in all trades but

Domestic and Personal Service (mostly maids and laundresses), *Professional* (mostly nurses, bookkeepers, and teachers), and in *Trade* (mostly clerks, although the difference by sex is slight). The distribution according to urban or rural location of death is in accordance with what would be expected resulting from the nature of the occupation.

As seen in Table 6.2, the type of industry also varies by region in Ontario (chi-square test, $\chi^2=401.51$, $df=36$, $p<.001$).⁶⁸ This is also to be expected, with most of the *Agriculture* jobs coming from outside of south-central Ontario and most of the *Mining* and *Fishing, Hunting and Forestry* jobs in the North. *Domestic and Personal Service* jobs are mostly equal throughout the province, as are *Professional* jobs. *Transportation* occupations are found more in the North and South-Central regions.

⁶⁸ Including the 10 occupational categories (excluding *Gentlemen or Lady*, and *Student*) creates a chi-square test where 14% of the expected frequencies are less than 5 and 1 is less than 1 (0.76). However, excluding the category of *Mining* (which includes 10% of the expected frequencies that are less than 5 and the frequency that is less than 1), the chi-Square test remains statistically significant (chi-square test: $\chi^2=313.61$, $df=32$, $p<.001$).

Table 6.2 - Occupational Category by Region of Ontario.

Occupational Category	Total		North		Central		East		South-Central		South-West	
	N	%	N	%	N	%	N	%	N	%	N	%
Agriculture	413	22.6	25	14.9	43	36.8	126	31.0	78	13.0	141	26.3
Construction	84	4.6	3	1.8	4	3.4	13	3.2	40	6.6	24	4.5
Service ^a	121	6.6	10	6.0	6	5.1	36	8.9	31	5.1	38	7.1
Government	98	5.4	5	3.0	6	5.1	19	4.7	49	8.1	19	3.5
Forestry	24	1.3	20	11.9	0	0.0	1	0.2	1	0.2	2	0.4
Manufacturing	507	27.7	38	22.6	30	25.6	108	26.6	167	27.7	164	30.6
Mining	12	0.7	11	6.5	0	0.0	1	0.2	0	0.0	0	0.0
Professional	185	10.1	12	7.1	11	9.4	30	7.4	72	12.0	60	11.2
Trade	196	10.7	10	6.0	6	5.1	39	9.6	88	14.6	53	9.9
Transportation	180	9.8	34	20.2	10	8.5	31	7.6	74	12.3	31	5.8
Gentleman or Lady	1	0.1	0	0.0	1	0.9	0	0.0	0	0.0	0	0.0
Student	8	0.4	0	0.0	0	0.0	2	0.5	2	0.3	4	0.7
Total	1,829	100	168	100	117	100	406	100	602	100	536	100
Missing	1,487		128		87		284		573		415	
χ^2							401.51*					
df							36					

Note: Included are only those with a declared (meaningful) occupation (N=1,829).

a. Service = Domestic and Personal Service. Forestry = Fishing, Hunting, and Forestry

*p<0.05

Although the distribution of occupations of the decedents is interesting, it is hard to draw definitive conclusions without a direct comparison group in 1918. Society in Ontario was changing rapidly at this time, with more women working (especially during the last year of the First World War), and with the slow process of urbanization (Ramkhalawansingh 1974, Cohen 1988, Sylvester 2007). Without a census of 1918 to directly compare, it is difficult to determine how representative these individuals are of the population from which they came. Through the process of records linkage, it is possible to get an understanding of how these individuals were living at various points in their lives, and

then compare that to the rest of Ontario, or even to the rest of the Ontario-born population in Ontario at the same time point. In addition to the problem of a lack of comparative sample in 1918 is that there are systematic gaps in the completeness of the records, especially in term of sex.

Attempting to get a complete understanding of the socioeconomic characteristics of individuals who died in Ontario during the 1918 influenza using the occupation as listed on the death record is problematic in many ways. First, meaningful information is only available for 55.2% of the sample and, of that, 82.7% are male. This is necessarily an unrepresentative sample (of the social status of all individuals, however, it may be a representative sample of those who were working). Further to this issue is the question of *who* is not being included. This analysis is missing information on 79.8% of the women in the database (1,256 of 1,573). For those women for whom occupational information is declared, the largest categories are *Professional* and *Domestic and Personal Service*. For those in the *Professional* category, it is those who would be expected to contract influenza due to their daily interactions: 43 (44.0%) are nurses, 31 (32.0%) are bookkeepers or stenographers, and 17 (17.5%) are teachers. Those in the *Domestic and Personal Service* are almost exclusively some type of maid or laundress. These are women who are either in close contact with the major transmitters of the disease (those who are already sick and children – Glezen and Couch 1997) and those who are in other people's homes. Those in the *Domestic and Personal Service* category are also of low social standing. The demand for domestic servants, both in urban homes and on farms was extremely high in the early twentieth century. However it was an undesirable job that left women open to abuse, with few freedoms or social supports, and with an increased risk of participation in sex work (Leslie 1974, Rotenberg 1974, Darroch 2007). It was “quite possibly, the most despised form of nineteenth-century wage work” (Darroch 2007:214). What is missing is information on those who would not be expected to become ill due to their occupational exposures: who are the ‘married women’? What is the living condition of those who were ‘living at home’ at the time of death? As Johnson notes for Ontario in the nineteenth century, “while class relations among men were structured economically, class relations among women were structured socially – that is, defined not by what women did, but where they were placed socially” (1974:14). It

would be inappropriate to consider a woman's occupation (or lack thereof) as representative of her standing outside of the occupational category of her husband or father.

Further, this evaluation is missing information on 45.7% of the individuals who died in institutions (395 of 864). It is missing information on 40.2% of individuals who died in rural areas (650 of 1,616) and 49.2% of individuals who died in urban areas (837 of 1,700). Of those who died in Toronto, occupational data is missing for 53.8% (262 of 487). Without engaging in mapping procedures to plot locations of death in order to determine if more deaths occurred in areas known to be impoverished (as per Herring and Korol, see also Mercier and Boone 2002, and Mercier 2006), little further information can be garnered from the death records alone. However, since the question of socio-cultural stratification in relation to mortality in the 1918 influenza pandemic is worth exploring, it is necessary to go beyond the death records to get this information. Although it is almost impossible to reconstruct this information for the missing individuals at the time of their deaths using extant records, it is possible to use other sources to get an understanding of their socio-cultural status relative to the rest of society at certain points over the life-course. As argued by proponents of the life-course approach, socioeconomic status and exposure to health risks are not static phenomena: the ways in which they change can have an important impact on the lives and deaths of individuals (Elder et al. 2003, McDonough and Berglund 2003). This is especially important, since the socio-economic position of an individual at any one given time point may only represent a very short phase in the lives of individuals and not be representative of their usual environment (Berkner 1975). By using the records linkage procedure, we can more accurately see changes in social position, as it would vary throughout an individual's life, especially since we have information at birth, in youth (1901 Census), and in adolescence/adulthood (1911 census). Although not ideal (the ideal would be to have more complete information on the death records), this analysis is able to provide a broader picture of those who died in Ontario in 1918 than has yet been conducted.

Using the linked birth records, 1901 and 1911 census records, marriage records and attestation papers, we have information on socioeconomic status at some point during the

lifecourse for all but 12 of the linked individuals (2,954 of 2,965, 99.6%). Of those individuals for whom occupational information could be found at some point during their lives, 1,490 (50.3%) have information on parental occupation on the birth record and either their parents' or their own occupation in both the 1901 and 1911 censuses.

The first socioeconomic indication we have for the decedents concerns the environments into which they were born.⁶⁹ The birth records from 1883-1895 ask for information on the father's occupation. We were able to find birth records for 62.7% of the individuals in the sample (2,079 of 3,316) and 2,060 of those records (62.1%) were "Positive" or "Likely" links (hereafter referred to as PL links).⁷⁰ Data on father's occupation is missing from only 97 (4.7%) of the 2079 birth records (94, 4.6%, of the 2,060 PL links). Although this does not present information for everyone about their father's occupation at the time of their birth, it does provide data on 57.2% of the individuals who did not have information about occupation listed on their death record (851 of 1,487, 728 being female). This then gives some socio-economic information for at least one point over the lifecourse. The distribution of father's occupation at birth is seen in Table 6.3. The occupational classification follows that used in the 1911 Census and urban and rural status as identified in 1918 (making this analysis conservative – Chapter 3). These births occurred between 1883 and 1895.⁷¹

⁶⁹ It is important to consider issues of selectivity during this analysis. This is only a description of those who died in Ontario in 1918 (with an accurate registered death record), were born in Ontario, and were successfully linked. It does not provide evidence on those who were born in Ontario but who had left before 1918, those born during the time period in Ontario but who had died prior to September 1918, or immigrants who died in Ontario during the pandemic. It is therefore difficult to speak of how early life conditions increased the *risk* of dying during the pandemic, but can provide an idea of how these particular individuals may or may not have differed from the rest of Ontario at certain points during their lives. This could suggest important areas of difference that can be analyzed in terms of mortality risk in further research.

⁷⁰ All analyses using the linked records in this chapter will only include those linkages which were rated by the researcher as either a "Positive" link or a "Likely" link (see Chapter 3 and Appendix A). Links which were classified as "Maybe" are not included. For each type of record, the number of "Maybe" links are: Birth, 19 of 2,079 (0.9%); 1901 Census, 34 of 2,737 (1.2%); 1911 Census, 42 of 2,423 (1.7%); Marriage 12 of 1,777 (0.7%); and Attestation, 1 of 186 (0.5%).

⁷¹ There were slight variations in the distribution of occupations by year. These seem to follow general economic trends (Piva 1979, Harris 1992) rather than systematic changes in occupational classes of the natal home over time. For example, the largest percentage in the *Construction* trades occurs in 1888 and 1889 (both years 10.4%), right before the

Table 6.3 - Occupational Category of Father from Birth Records.

Occupational Category	Death Records		Birth Records		Urban		Rural	
	N	%	N	%	N	%	N	%
Agriculture	413	22.6	892	45.4	21	4.5	871	58.1
Construction	84	4.6	142	7.3	64	13.7	77	5.1
Service ^a	121	6.6	36	1.8	13	2.8	23	1.5
Government	98	5.4	13	0.7	8	1.7	5	0.3
Forestry	24	1.3	14	0.7	3	0.6	11	0.7
Manufacturing	507	27.7	576	29.4	201	43.1	375	25.0
Mining	12	0.7	0	0.0	0	0.0	0	0.0
Professional	185	10.1	55	2.8	26	5.6	29	1.9
Trade	196	10.7	102	5.2	47	10.1	55	3.7
Transportation	180	9.8	130	6.6	80	17.2	50	3.3
Gentleman or Lady	1	0.1	6	0.3	3	0.6	3	0.2
Student	8	0.4	0	0.0	0	0.0	0	0.0
Total	1,829	100.0	1,966	100.0	466	100.0	1,499	100.0
<i>Missing and Maybe</i>	<i>1,487</i>		<i>1,350</i>		<i>47</i>		<i>61</i>	
χ^2			424.37*				463.72*	
df			9				8 ^b	

Note: For those linked to a birth record where the linkage was either "Positive" or "Likely" (N=1,966). One individual is missing an exact place of birth beyond "York County".

*p<0.05

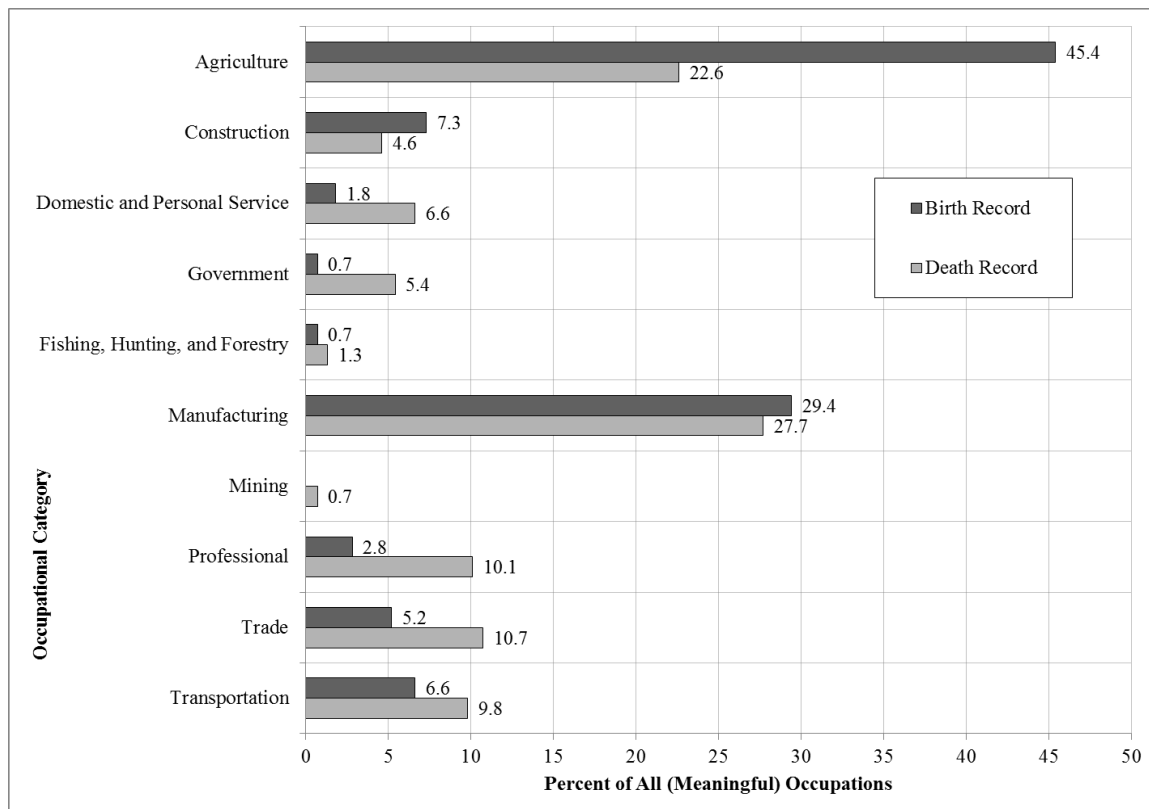
^a. Service = Domestic and Personal Service. Forestry = Fishing, Hunting, and Forestry

^b. Does not include "mining"

recession of the early 1890s. 1889 has the second highest percentage of *Manufacturing* (31.9%) jobs and the fewest *Agriculture* jobs (36.8% - the highest percent was at 52.8% in 1883).

The difference between the distribution of occupational environments at birth and the distribution of occupations at death is statistically significant (chi-square test: $\chi^2=424.37$, $df=9$, $p<.001$). This means that there were some important changes that occurred between the time of birth and the time of death and potentially reveals the bias from the limited information on the death records. The most noticeable of these changes is the drastic decrease in the percentage of total occupations in *Agriculture*. At the time of birth, 892 (45.4%) of the decedents entered into a household where the main occupation was *Agriculture*. However, by the time of death, only 413 (22.6%) of the decedents were still engaged in occupations relating to farming (Figure 5.1). The percentage in the *Construction* and *Manufacturing* occupations declined slightly, while the increases in the *Domestic and Personal Service* (1.8% to 6.6%), *Government* (0.7% to 5.4%), *Professional* (2.8% to 10.1%), *Trade* (5.2% to 10.7%) and *Transportation* (6.6% to 9.8%) occupations were more noticeable (these are, generally, the more urban occupations). Among those who were born into *Agriculture* households and who had an occupation on their death record (N=530, 59.4%), less than half were in *Agriculture* at the time of their death (N=236, 44.5%), with the greatest amount of movement being into the *Manufacturing* (N=105, 19.8%), *Professional* (N=49, 9.3%), and *Trade* occupations (N=49, 9.3%). This does not appear to be only a function of the dearth of declared occupations on the death record, since only 231 males were missing an occupation and 23.7% of men were in *Agriculture*. While 1,256 women were missing an occupation at death, 17% of the women with a declared occupation were in *Agriculture*. This suggests that at some point, individuals who had been born into a farming home moved into another type of occupation. As will be discussed in Section 5.2, of the 1,548 individuals who were born into a rural environment, by the time of death 917 (59.2%) were still living in rural areas and 631 (40.8%) had moved into an urban environment. Although this could be related to individuals coming into urban centres for medical treatment and hospital stays during an epidemic, the fewer than expected occupations in *Agriculture* at the time of death suggests that migration into cities was also an important element.

Figure 6.1 - Occupational Environment at Birth and Occupation at Death.



Note: Occupation at Birth, N=2,060, and the Occupation at Death, N=1,829. Birth records are for those with a “Positive” or “Likely” link and show only “meaningful” occupations.

Table 6.4 shows the occupational environment into which the decedents were born according to the region of their birth. In all regions except for South-Central, most families were engaged in *Agriculture*, with the largest percentage of families participating in *Agriculture* occurring in Central and Eastern Ontario. In South-Central Ontario, most of the families were engaged in *Manufacturing*, while *Agriculture* occupations were almost as numerous. As compared to the distribution of occupational categories at death (Table 6.2), the decrease in *Agriculture* occurred in all regions of Ontario.

Table 6.4 - Occupational Category of Father from Birth Records by Region of Birth.

Occupational Category	WMMIP Total		North		Central		East		South-Central		South-West	
	N	%	N	%	N	%	N	%	N	%	N	%
Agriculture	892	45.4	14	46.7	94	55.6	209	54.7	190	31.9	385	48.7
Construction	142	7.3	3	10.0	8	4.7	16	4.2	63	10.6	52	6.6
Service ^a	36	1.8	1	3.3	2	1.2	9	2.4	14	2.4	10	1.3
Government	13	0.7	1	3.3	2	1.2	2	0.5	6	1.0	2	0.3
Forestry	14	0.7	0	0.0	2	1.2	2	0.5	7	10.2	3	0.4
Manufacturing	576	29.4	6	20.0	46	27.2	94	24.6	201	33.8	229	29.0
Mining	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0
Professional	55	2.8	0	0.0	2	1.2	8	2.1	23	3.9	22	2.8
Trade	102	5.2	2	6.7	7	4.1	17	4.5	40	6.7	36	4.6
Transportation	130	6.6	3	10.0	6	3.6	24	6.3	48	8.1	49	6.2
Gentleman or Lady	6	0.3	0	0.0	0	0.0	1	0.3	3	0.5	2	0.3
Student	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0
Total	1,966	100	30	100	169	100	382	100	595	100	790	100
<i>Missing and Maybe</i>	<i>1,350</i>		<i>8</i>		<i>5</i>		<i>60</i>		<i>24</i>		<i>17</i>	

Note: For those linked to a birth record where the linkage was either "Positive" or "Likely" (N=1,966), χ^2 not calculated due to expected frequencies less than zero.

a. Service = Domestic and Personal Service. Forestry = Fishing, Hunting, and Forestry

Although occupational information was missing on only 94 (4.6%) of the birth records that were PL links, the more important issue is the 1,255 individuals (37.8% of the total database) for whom no birth record could be located (or, 904 of the 2,965 individuals (30.5%) who were linked to at least one other record).

Among the 2,060 PL linked birth records, 6 (0.3%) gave some indication that the birth was illegitimate (either the word illegitimate was somewhere on the birth record, or there was no father listed and the infant had the surname of the mother). Illegitimate births were a concern to the Registrar General of Ontario at the time, and the numbers of these

births were recorded in his annual reports. Between 1883 and 1895, the percentage of illegitimate births per year ranged from 0.9 to 2.3 (Legislative Assembly of Ontario 1892:28, 1895:14, 1896:28, 1897:36). He stated in 1890 that “illegitimate births are for manifest reasons very commonly not registered in Ontario” (1892:4). Although the infant mortality rate for children born outside of marriage was higher than for children born to a married couple (MacMurchy 1910), it is unlikely that this accounts for all of the missing illegitimate children. Rather, lack of linked birth records among the decedents in 1918 is likely a result of the lack of registration of their births. The coverage of birth records was also known to be lower for those in the north and more newly settled parts of Ontario as well as for aboriginal individuals (Chapters 3 and 4). However, information on the familial environment can be found for almost all individuals in the 1901 census, and further information can be gathered from the 1911 census.

Of the 2,965 linked individuals, 2,737 have entries in the 1901 census (92.3% of the linked individuals, 82.5% of the total database) and 2,703 of those are PL links (91.2% of the linked individuals, 81.5% of the total database). Once individuals were located in the census records, the records for the entire household in which they lived were transcribed. In so doing, I am able to get an understanding of the socioeconomic environment in which they lived, through analysis of the occupation of the head of the household, their own occupation (if applicable), and whether anyone else in the household was working.^{72,73} The age range for these individuals (using the reconstructed date of birth, Chapter 4) is between 6 and 18.

Of the 2,703 PL links in the 1901 census, 2,608 had information about the occupation of the head of household (96.5%). A further 219 individuals were themselves working (8.4% - does not include the 53 individuals who had “Student” listed as their occupation), yet only 20 were working where the head of the household was not (0.07%). Of those 20 individuals, 14 were living in a household that did not include their father (or step- or

⁷² In this analysis, a family is a nuclear family, including step- or adopted-children. Any other type of relationship (including grandchild, sibling of the head, niece or nephew) is considered as a lodger (used in the general sense of someone else living in the household. I do not distinguish between boarders, extended family, or roomers, see Harris 1992).

⁷³ The occupation refers to the original entry, or the corrected entry if applicable.

adoptive-father). For those who were working, 173 of the 219 were male (79%). In families where the head of the household was not working, but the decedent was working, the occupation of the decedent is treated as the occupation of the head of the household. When this is done, there is occupational information for 2,614 individuals, treating the occupation of the head of the household as indicative of the family environment. Another person (beside the head or the individual) was working in 37.7% of the families while 62.3% did not have another individual who was working (or whose occupation was recorded).

The occupational categories in the 1901 census (published in 1910 as Bulletin XI, Dominion of Canada 1910) are not directly congruous with the 1911 categories used in this analysis and cannot be separated by Canadian- or foreign-born. As seen in Table 6.5, the categories of *Trade* and *Transportation* are combined, and the occupations I have classified as *Construction* are combined with *Manufacturing*. The category of *Domestic and Personal Service* (in the Bulletin is *Domestic and Personal Class*) is more broadly defined than in 1911, including many of the individuals in the *Government* category (such as police officers) and many of those in the *Professional* category (such as nurses). It is included for the sake of completeness, but the more useful comparison is between the 1901 occupational environments of the individuals in the WMMIP database and the database created by the Canadian Families Project (CFP).

The CFP is a five-percent sample of the entire 1901 census of Canada that was created through a SSHRC funded multi-university collaborative project based at the University of Victoria and headed by Dr. Eric Sager and Dr. Peter Baskerville. The sampling procedure is outlined in Ornstein (2000). The sample used in this study included all those who were living in Ontario (from the entry on the top of the census schedule 1), who were born in Ontario (as determined from the declared birth place), and who were listed as the head of the household (either the head or, when absent in a household, the individual most likely to fill that role, for example “Mother” or “Husband”). The comparison to the WMMIP sample in 1901 is for the entire family unit, based most often on the occupation, ethnicity, and religion of the head of the household. Selecting these individuals from the CFP sample is to obtain an analogous sample of Ontario-born

individuals living in Ontario in 1901.⁷⁴ The total sample size of the selected CFP dataset is 15,178.

⁷⁴ Not all of the heads of households in the WMMIP database were born in Ontario since the requirement is that decedent was born in Ontario. The latest that this Ontario birth may have occurred was in 1895. This means that, by 1901, although the head of the household may have been an immigrant, in general, they had been living in Ontario for at least 6 years. Eliminating immigrants from the CFP sample means that recent immigrants (with potentially different cultural and occupational environments) are not included.

Table 6.5 - Occupational Category of Head of Household from the 1901 Census.

Occupational Category	Total		1901 Census ^a		CFP ^b		Urban		Rural	
	N	%	N	%	N	%	N	%	N	%
Agriculture	1,200	46.2	306,431	40.8	7,201	51.8	52	7.2	1,148	61.3
Construction	167	6.4			783	5.6	91	12.6	76	4.1
Service ^c	94	3.6	112,232	14.9	342	2.5	42	5.8	52	2.8
Government	25	1.0			98	0.7	15	2.1	10	0.5
Forestry	29	1.1	7,680	1.0	135	1.0	5	0.7	24	1.3
Manufacturing	650	25.0	178,939	23.8	2,827	20.4	273	37.7	377	20.1
Mining	7	0.3	3,81	0.5	54	0.4	1	0.1	6	0.3
Professional	7	2.7	38,311	5.1	608	4.4	44	6.1	26	1.4
Trade	164	6.3	104,465	13.9	1,070	7.7	92	12.7	72	3.8
Transportation	187	7.2			726	5.2	107	14.8	80	4.3
Gentleman or Lady	4	0.2			45	0.3	1	0.1	3	0.2
Student	1	0.04			21	0.01	1	0.1	0	0.0
Total	2,598	100	751,875	100	13,890	100	724	100	1,874	100
Missing and Maybe	718		2,441 ^c		1,288		22		66	
χ^2					89.00*				670.58*	
df					9				9	

Note: Refers to the Decedent if Head of Household is Not Working. For those linked to a 1901 Census record where the linkage was either "Positive" or "Likely" and who was living in Ontario at the time of the 1901 census (N=2,598).

Source: Ontario-born Ontario population from the Canadian Families Project (N= 13,890) and Census data from Dominion of Canada (1910).

*p<0.05

^a. For all of Ontario

^b. The Ontario-born only

^c. Service = Domestic and Personal Service. Forestry = Fishing, Hunting, and Forestry

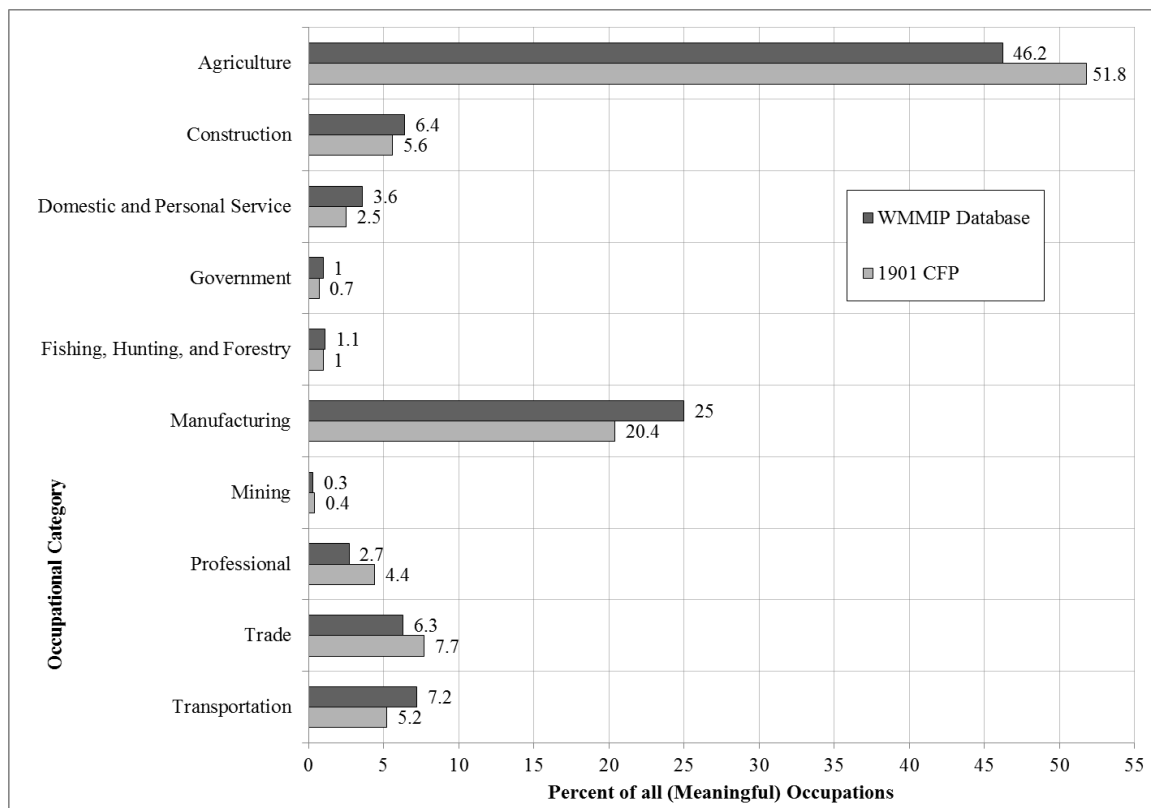
^d. Includes those in the *Miscellaneous* and *Non-Productive* classes.

The comparison between the WMMIP individuals and the CFP is more fruitful than looking at the 1901 census publication. The difference between the occupational categories of the decedents in 1901 and at the time of their deaths is statistically

significant ($\chi^2=409.62$, $df=9$, $p<.001$). Similarly, the differences between the 1901 census and the occupational environment at birth are also significant ($\chi^2=32.86$, $df=9$, $p<.001$), which could either mean that the changes that occurred between birth and death were already beginning by 1901, or it could represent the higher linkage rates to the 1901 census than to the birth records.

Further, the WMMIP population of decedents was significantly different than the rest of the CFP Ontario-born population in Ontario in 1901 ($\chi^2=89.0$, $df=9$, $p<.001$). The largest differences between the two groups are found in *Agriculture* and in *Manufacturing*, with there being a smaller percentage of the decedents in *Agriculture* and a larger percentage of the decedents in *Manufacturing* (Figure 6.2). As seen in the urban and rural breakdown, *Agriculture* among the decedents was primarily found in rural environments while 42% of *Manufacturing* jobs were in urban areas and 58% were in rural areas. Of the 816 individuals whose father's occupation at birth was *Agriculture* and who also had an occupation in 1901, 689 (77.2%) were still in *Agriculture*. The largest transfer of those who were born into *Agriculture* was into *Manufacturing*, with 52 individuals (5.8%) making this switch.

Figure 6.2 - Occupational Category of Head of Household in 1901.



Note: Refers to the decedent if head of Household is not working, for those in the WMMIP database as compared to those in the CFP sample. For those linked to a 1901 Census record where the linkage was either “Positive” or “Likely” and who was living in Ontario at the time of the 1901 census (N=2,598). Ontario-born Ontario population from the Canadian Families Project (N= 13,890).

The 1901 census linkages are also subject to many concerns about coverage. In addition to the issues discussed in Chapters 3 and 4, families in which the father was not present often did not have an occupation for any family member. For those individuals who were not married, who were living with their nuclear family members but their father was not present, 46 of 174 (26%) were missing an occupation. In comparison, for those individuals who were not married and were living with their nuclear family and the father was present, only 52 of 2,442 (2.1%) were missing an occupation (only considering PL links). Of the 128 nuclear families without a father and where someone in the household had a listed occupation, 60 (46.9%) were in *Agriculture*, 18 (14%) were in *Domestic and Personal Service*, 29 were in *Manufacturing*, and 11 (8.6%) were in *Trade*. This is important since these families were usually of lower social standing. As stated by

Bradbury, “in 1901, few jobs paid women even two-thirds of a man’s wages. Mothers Pensions and Family Allowances did not yet exist. Most local relief systems, like private charity, made moral judgments that made it much easier for widows than for women who were separated or unmarried to secure support” (2007:288). This analysis of occupations necessarily excludes those families living off of charity or other forms of support that were not recorded in the census.

Although the census does ask about income, this variable is not analyzed due to the lack of consistent coverage. In the 1901 WMMIP database of decedents, only 43.1% of the head of households had any information about income. In the 1901 CFP sample, only 36.7% had earnings listed, whether from their regular occupation or from other sources.

In the 1911 census, there are 2,423 linked individuals (2,381 of which are PL links). Of those 2,381 PL links, 2,037 had information about the occupation of the head of the household (196 of the decedents were themselves the head of household, 8.2%). As with the 1901 census, in families where the head of the household was not working but the decedent was (and the decedent was not the head of the household), the occupation of the decedent is treated as the occupation of the head of the household. This thus provides occupational information for 2,324 families. The decedents in 1911 ranged in age from 16 to 28 (using the reconstructed date of birth, Chapter 4).

The WMMIP database distribution of occupations in 1911 is significantly different from the distribution of occupations in the 1911 census reports (Table 6.6, chi-square test: $\chi^2=165.80$, $df=9$, $p<.001$, Dominion of Canada 1915). It is also significantly different from the distribution as it was in 1901 ($\chi^2=34.68$, $df=9$, $p<.001$) and significantly different from the distribution of the declared occupations at death ($\chi^2=258.54$, $df=9$, $p<.001$). The trends seen when comparing the 1901 occupational distribution of the decedents to the CFP sample continue. Again, the percentage of individuals in *Agriculture* is fewer than in 1901 (but not yet as few as on the death records). All other occupational categories have a higher percentage of the distribution than in 1901 (except for the very small categories of *Mining* and *Gentleman or Lady*). The differences between the distribution of individuals in the WMMIP database in 1911 and in the Canadian-born

census population in 1911 are slight. The largest difference is a greater percentage of the distribution in *Manufacturing* among the decedents than among census population of all Canadian-born Ontarians. Among the decedents as compared to the census, a smaller percentage of the occupations are in *Domestic and Personal Service, Government, Professional, and Trade*. A larger percentage of the decedents are in *Agriculture, Manufacturing, and Transportation*.

Table 6.6 - Occupational Category of Head of Household from the 1911 Census.

Occupational Category	Total		1911 Census ^a		Urban		Rural	
	N	%	N	%	N	%	N	%
Agriculture	881	38.4	260,407	36.4	30	3.6	862	57.7
Construction	165	7.2	51,453	7.2	98	11.8	71	4.8
Service ^b	92	4.0	49,420	6.9	52	6.3	41	2.7
Government	27	1.2	17,073	2.4	21	2.5	6	0.4
Forestry	36	1.6	12,202	1.7	8	1.0	28	1.9
Manufacturing	621	27.0	149,995	21.0	313	37.7	309	20.7
Mining	4	0.2	8,682	1.2	1	0.1	3	0.2
Professional	72	3.1	37,150	5.2	51	6.1	24	1.6
Trade	192	8.4	83,691	11.7	121	14.6	76	5.1
Transportation	197	8.6	44,886	6.3	132	15.9	67	4.5
Gentleman or Lady	1	0.04	0	0.0	0	0.0	1	0.1
Student	9	0.4	0	0.0	3	0.4	6	0.4
Total	2,297	100.0	714,959	100.0	830	100.0	1,494	100.0
<i>Missing and Maybe</i>	84				38		61	
χ^2			165.80*				713.12*	
df			9				9	

Note: Refers to the decedent if head of household is Not Working. For those linked to a 1911 Census record where the linkage was either "Positive" or "Likely" and for those living in Ontario at the time of the 1911 census (N=2,297).

Source: The occupational information on the Canadian born population is from Dominion of Canada (1915).

*p<0.05

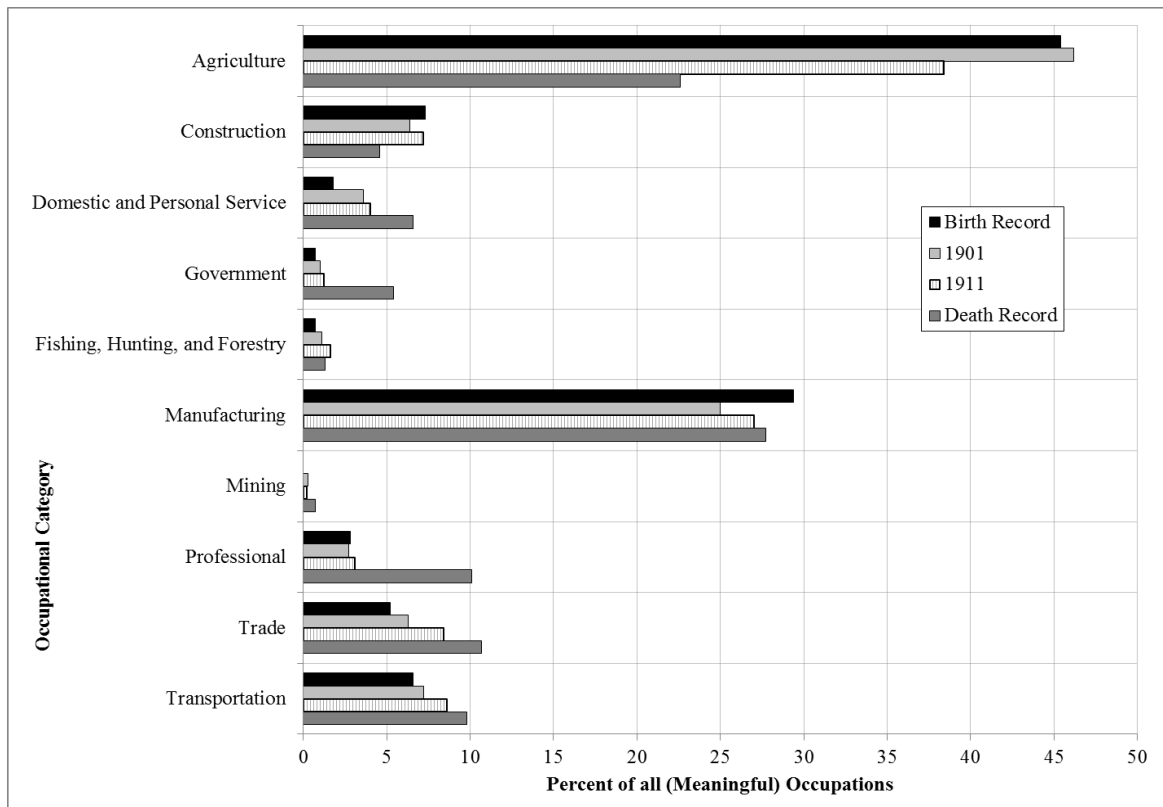
^a. Refers to Canadian-born Ontarians.

^b. Service = Domestic and Personal Service. Forestry = Fishing, Hunting, and Forestry

The distribution of occupations at all four time points can be seen in Figure 6.3. The decline in the percent of the distribution in *Agriculture* can be seen to have started after 1901. For *Domestic and Personal Service*, *Government*, *Professional*, *Trade*, and

Transportation, the greatest increase in the distribution in these occupations occurred between 1911 and 1918. This is to be expected, since the 1911 census was taken at the point before many of the individuals in the sample had started their own families and had their own occupations. The differences between the occupational categories at birth and at death speak to the intergenerational transfer of status whereby there was an increase in the occupational category which might have been of lower income and status (*Domestic and Personal Service*), but there were increases in many categories of higher incomes (such as *Government*, *Professional*, and *Trade*). Only the categories of *Construction* and *Manufacturing* did not show a consistent pattern of change, however, the percentage in *Manufacturing* did increase steadily after 1901.

Figure 6.3 - Distributional of Occupational Categories of the Decedents at Four Time Periods.



Note: Occupational environment at birth (N=1,966), Occupational environment in 1901 (N=2,598) and 1911 (2,297), and declared occupation at death (N=1,829).

Two notable patterns emerge from an analysis of the occupational environments (or occupations) of the decedents at the four different time points. The first is the vast difference in the percentage of individuals who were in *Agriculture* at the time of birth as compared to the time of death. While there was a higher percentage of the decedents in *Agriculture* in 1911 than in the census population (38.4% as compared to 36.4%), the drop to 22.6% was steeper than the declines noted in the census (in 1921, the percentage of the employed Ontario population over 10 years of age in *Agriculture* was 26.4% and in 1931, the percentage had finally dropped to 22.7%, Dominion of Canada 1931:12-13). The categories used in this analysis do not allow for the investigation of a gradient in differences in groups, but greater than expected numbers of individuals in *Domestic and Personal Service* and *Manufacturing* may indicate lower status as a fundamental cause of death. The fewer than expected individuals in *Agriculture* might indicate a protective effect of rural, less crowded environments and conversely, a deleterious effect of overcrowding. The implications of this change are discussed in the following section.

The second pattern to emerge is that each of the occupational distributions of the decedents is statistically different from the next and also statistically different from the comparison samples in 1901 and 1911. This shows change over time in occupational categories, but also that the decedents varied from the rest of the population in significant ways at two different points in their lives. This provides support for the decedents being different from the rest of society in certain ways, since, if the 1918 influenza pandemic killed equally among all individuals, the individuals should not represent a distinct subset within the rest of the Ontario population.

It is possible that society changed so rapidly between 1911 and 1918 that the population at risk in the province had significantly altered, such that the sample of decedents actually does represent the distribution of occupations as they were in 1918. This cannot be solved through this analysis since that exact distribution in 1918 is not known. However, the records linkage process has provided new and valuable information. It is now possible to include in the analysis those individuals who did not have a declared occupation on the death record, including the vast majority of women among the decedents. It is also possible to discover change through time in the environment in

which they lived and the direction of that change. Although not ideal, the snapshot of these individuals in 1901 and 1911 allows for a comparison sample of occupations at the same time point that had not been previously possible. The following sections investigate other new forms of information on social differences that is now possible using the records linkage process.

6.2 Region

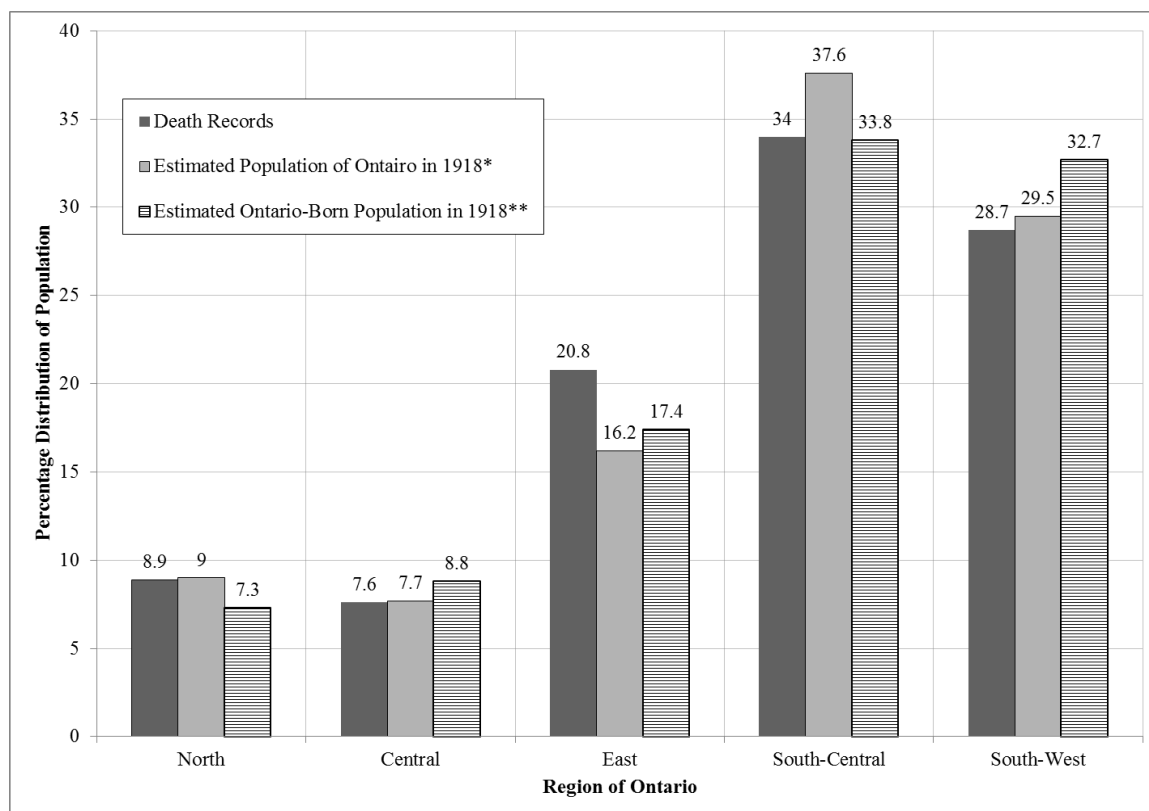
As in previous analyses, Ontario has been divided into five general regions. This is to account for the different migratory patterns, cultural practices, and economic basis of such a large province. A condensed version of Table 4.1 can be seen in Figure 6.4 that shows the number and percentage of death records in 1918 from each region of Ontario. This is shown compared to both the 1918 population as estimated from 1918 Report for the Registration of Births Marriages and Deaths (as in Table 4.1), as well as the estimated Ontario-born population for each region based on the Ontario-born populations in the 1911 and 1921 censuses.^{75,76} Comparing the death records to the estimated total population using a chi-square test, the differences between the deaths from each region and the population in each region is statistically significant ($\chi^2=55.75$, $df=4$, $p<.001$), with there being more deaths from the East region than would be expected and fewer deaths in the South-Central area. If the estimated Ontario-born population is used in comparison, the results are statistically significant ($\chi^2=56.20$, $df=4$, $p<.001$), yet the pattern is slightly different. There are more deaths than expected in the North and the East, while deaths in South-Central region are at similar levels, and deaths in the Central

⁷⁵ There is an error in the population totals for the district of Middlesex North in the 1911 census: the total male population born in Ontario (N=6,959) is recorded as higher than the population born in Canada (N=6,934) (Dominion of Canada 1913:397). While I have attempted to correct for this discrepancy, the population totals of all the summed districts for males are in excess of 201 individuals over the provincial total (955,772 versus 955,571, 1913:442). Similarly, in 1921, the summed totals for the districts is 2,168,594, while the total Ontario-born population for the province is declared to be 2,175,365 (1925:313), a difference of 6,771 individuals. I have used the summed district totals in this analysis.

⁷⁶ Estimate created using Waring's two-point formula (Judson and Popoff 2004, Appendix B) as described in Section 3.2.

and South-West regions are fewer than would be expected, supposing an equal distribution of deaths among the population.

Figure 6.4 - Distribution of Death Records in Final Database and Total Population by Region in 1918.



Note: WMMIP database, N=3,316.

Source: The estimated population in 1918 for the individual counties was taken from the 1918 Report of the Registration of Births Marriages and Deaths (Legislative Assembly of Ontario 1919a). The populations were combined from tables 1 and 2, N=2,798,970 (1919a:1-12).

** Estimate created using Waring's two-point formula (Judson and Popoff 2004, Appendix B) as described in Section 3.2, based on the Ontario-born population from the 1911 census (Dominion of Canada 1913:388-406) and the Ontario-born population from the 1921 census, N=2 097 594 (Dominion of Canada 1925:328-334).

Since these data only include individuals who died in Ontario and who were also born in Ontario, it is more appropriate to consider only the Ontario-born population rather than the entire population, taking into account the different immigration and settlement

patterns of the province. The data on the Ontario-born population only is directly available in the 1901 and 1911 census reports, so it is useful to compare the location of residence in 1901 and 1911 of those who died in 1918 as compared to the rest of the population, without the use of estimated populations. This also accounts for potential unequal migration of individuals towards urban centres after 1911 (for example, if there was a tendency for one group over another to migrate to cities).

Table 6.8 shows the difference between the Canadian-born and specifically Ontario-born individuals in Ontario in both 1901 and 1911, according to the censuses (Dominion of Canada 1913:416-436, 1925:387-406, 422). In both 1901 and 1911, there is only a slight difference between the Canadian-born and Ontario-born populations of the Central, South-Central, and South-West regions, suggesting that there was not a large amount of inter-provincial migration into these areas. However, there was a larger difference between the Canadian-born and Ontario-born populations in the North and East regions. In both instances, this was almost entirely due to immigration from Quebec.⁷⁷ In both 1901 and 1911, the East region of Ontario had the highest percentage of individuals who had been born in Canada. The South-Central region being a centre for immigration (particularly to Toronto and Hamilton, Dominion of Canada 1925) can be seen in the relatively lower percentage of individuals born in Canada in both 1901 and 1911. The equally low percentage of Canadian-born individuals in the North in 1911 is likely due to the same work-related forces driving immigrants to this region that were also drawing young men from both Ontario and Quebec (Weaver 1913).

⁷⁷ In the North in 1901, 11.4% of the population had been born in Quebec and in the East, 8.2% had been born in Quebec. In 1911, 10.5% of the population of the North had been born in Quebec, and 6.8% of the population of the East was born in Quebec. The 1911 census separates place of birth by sex. In the East, the percentage of male and female migration to the East was equal (3.3% male and 3.4% female), while migration to the North was more of a process for men (6.8% male and 3.8% female). This might suggest that immigration to the East from adjacent Quebec was more due to cultural and familial ties, while immigration to the North resulted more from male migration for work in the mines, forestry, and the railroad. Choquette reports that “the scarcity of arable land in Quebec’s Saint Lawrence River valley coupled with the density of the area’s population and periodic economic crises, prompted French-speaking Canadians, during the last three quarters of the nineteenth century, to migrate in ever increasing numbers to the adjacent province of Ontario” (1975:1). In Eastern Ontario, French-Canadian immigration began to increase in the 1880s (1975:56).

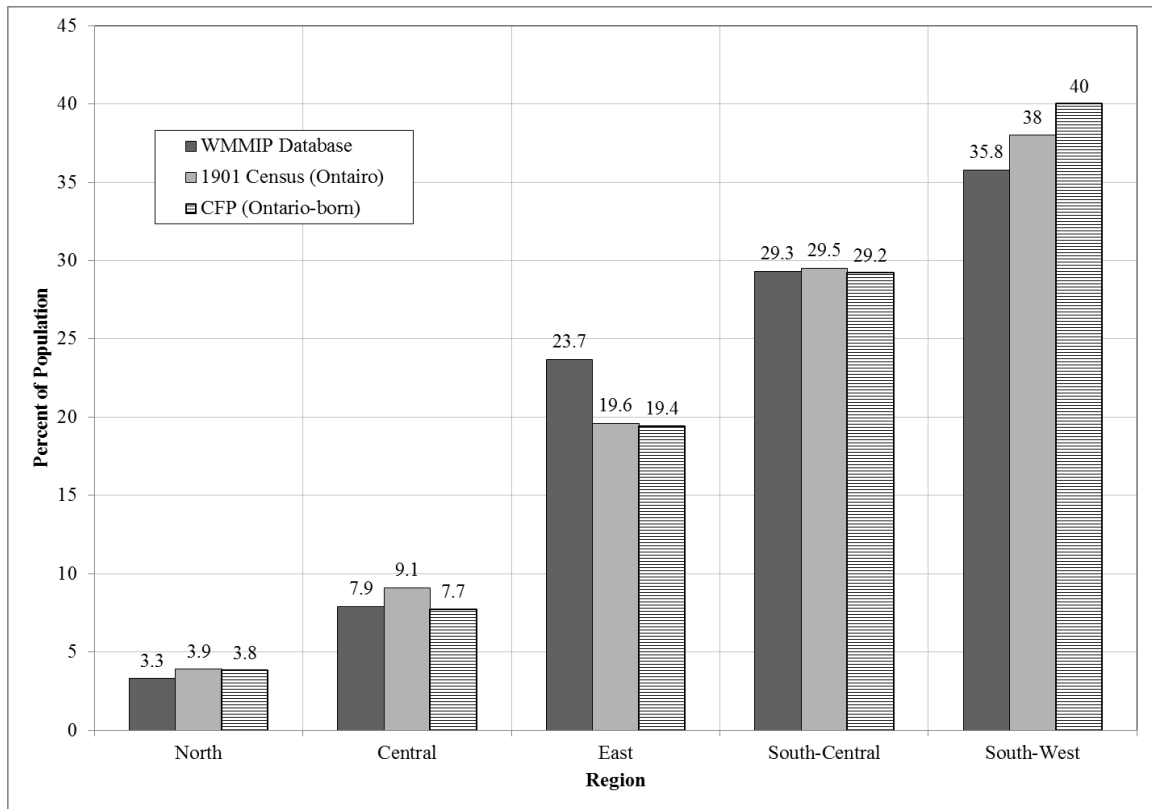
Table 6.7 – Percent Population within each Region in Ontario by Place of Birth, 1901 and 1911.

Region	1901 Census		1911 Census	
	Canadian-Born	Ontario-Born	Canadian-Born	Ontario-Born
North	83.6	69.9	70.0	57.4
Central	88.4	86.7	87.4	86.0
East	90.0	81.3	89.6	82.3
South-Central	80.7	79.1	70.6	69.1
South-West	85.7	84.6	85.1	84.1

Source: Dominion of Canada 1913, 1925

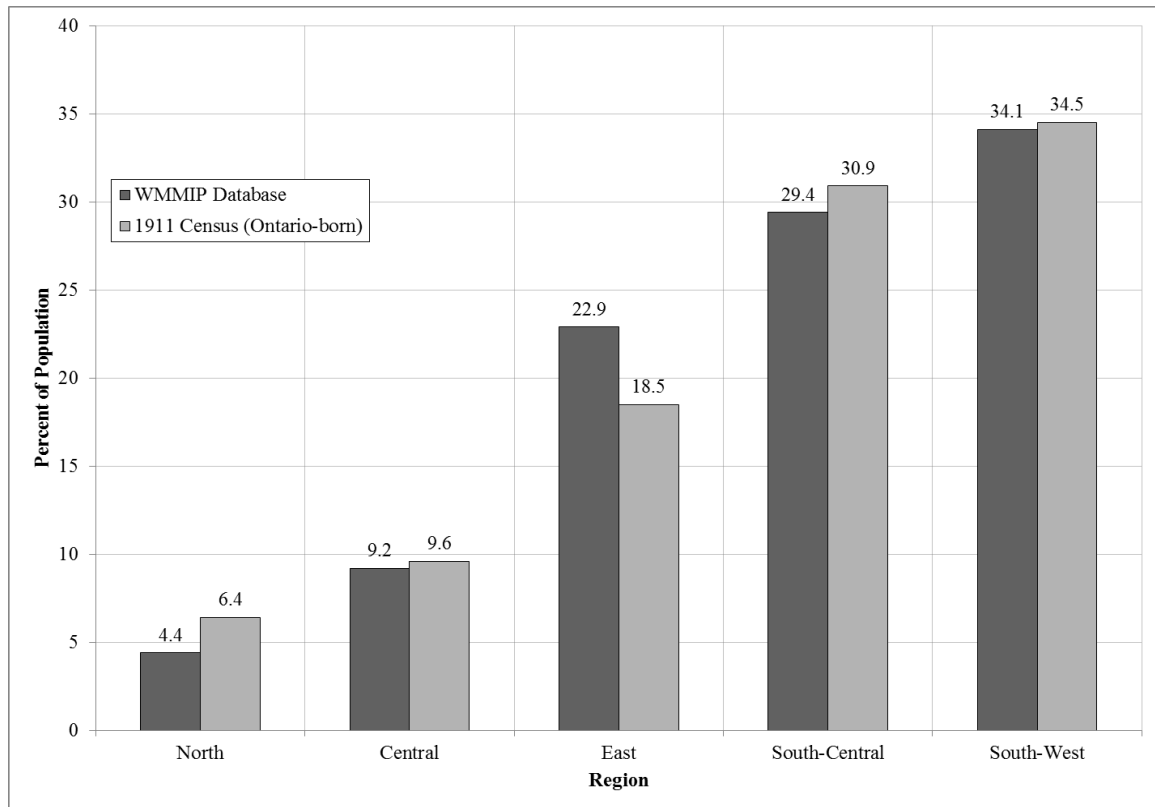
Figure 6.5 shows the distribution of individuals who were found in the 1901 census and who were living in Ontario in 1901 with PL links (N=2,687) as well as the total Ontario-born population in that region in 1901 (N=1,784,760). This is also shown next to the regional distribution of population from the CFP sample (N=15,178). The difference between the regional distribution in the database and the census distribution of population is statistically significant using a chi-square test ($\chi^2=33.32$, $df=4$, $p<.001$) with more decedents living in the East than expected and fewer decedents living in the South-West or in Central Ontario in 1901. The regional distribution of the CFP sample follows fairly closely the distribution from the 1901 census.⁷⁸ It is between both the census and the CFP as compared to the WMMIP database of the decedents where the largest differences occur.

⁷⁸ However, the differences between the Ontario-born population from the census and the Ontario-born sample from the CFP is statistically significant ($\chi^2=51.57$, $df=4$, $p<.001$)

Figure 6.5 – Location of Residence in 1901

Note: For those individuals who died during the 1918 Pandemic and who were “positively” or “likely” linked and who lived in Ontario at the time of the 1901 census (N=2,686) as compared to the entire Ontario-Born Population of Ontario in 1901 (N=1,784,760) and the Ontario-born population from the CFP sample (N=15,178). Data on the Ontario Born Population from Dominion of Canada (1902).

Similar results are found when the decedents’ place of residence in 1911 was compared to the rest of the Ontario-born residents of the province. Once again, the differences are statistically significant ($\chi^2=40.61$, $df=4$, $p<.001$) with the major differences being fewer than expected deaths among those who lived in the North in 1911 while there were more deaths among those who lived in Eastern Ontario in 1911. It is important to note that among those deaths that could not be linked to any record, 118 (39.7%) occurred in Northern Ontario (Table 3.3). The dearth of decedents who were living in the North in 1911 may reflect the inability to link these records rather than a protective effect of living in the North.

Figure 6.6 - Location of Residence in 1911.

Note: For those individuals who died during the 1918 pandemic, were living in Ontario in 1911 and were “Positively” or “Likely” linked (N=2,354) as compared to the entire Ontario-Born Population of Ontario in 1911 (N=1,931,927). Data on the Ontario Born Population from Dominion of Canada (1913).

Ontario was experiencing rural-to-urban migration around the turn of the twentieth century, with the urban population outnumbering the rural population by 1911 (Dominion of Canada 1912:528, Chapter 2). The vast growth of the cities is thus partially explained by the abandonment of farming and rural life. However, drawing conclusions from the CFP sample, Sylvester cautions that a “wholesale displacement of working families from the countryside awaited the development of a mature industrial economy, with steadier employment, and a mid-century expansion of housing stock. Until then, emigration from rural life remained modest because the kind of independence rural émigrés found in urban life was not so simple” (2007:166). Additionally, the rural emigrants were not going to larger urban areas, but to smaller cities of closer to 10,000 in population (2007:157). The process of migration among the decedents can be examined two ways

using this database: through migration to and from rural and urban areas and from migration within regions of the province.

The location of death by rural and urban area as compared to the urban and rural estimated population in Ontario in 1918 is presented in Table 3.1. To summarize, 48.7% of the deaths occurred in rural areas, while 56.1% of the population was estimated to be living in the same areas. While 51.3% of deaths occurred in urban areas, 43.9% of the population was estimated to be urban, revealing more deaths among urban dwellers than would be expected.⁷⁹

Table 6.8 shows the migratory experiences of individuals in the WMMIP database at three different time periods, through presence in an urban or a rural location. It compares the location of their birth record to the location of their death, their location in the 1901 census to the location of death, and from their location in the 1911 census and the location of death. The most common experience for these individuals was not to move, especially to stay within rural locations. Among the people who died, 44.7% were born in a rural location and died in a rural location. A further 20.7% were born in an urban location and died in an urban location, so that 65.4% of the individuals died in the same environment in which they were born. Of the two kinds of possible migration, rural to urban was by far the most common (30.7%), while some individuals did migrate from urban areas to rural (3.8%). Hypothesizing that an individual migrated only once, much of this migration was completed by 1911, since at this time 47.1% of individuals were in a rural location and would also die in a rural location and 32% of the database were living in urban locations and would also die in urban locations. This leaves 20.9% of individuals who were not living in the type of environment in which they died.

It is important to keep in mind that the death record only captures a moment in the individual's life. During this epidemic, individuals would travel to larger centres to obtain medical care, for example, in hospitals (Dickin McGinnis 1981). It is quite

⁷⁹ This classification scheme of rural and urban places is based on the 1918 Report of the Registration of Births, Marriages, and Deaths (Legislative Assembly of Ontario 1919a:12) and the definition of urban is more conservative than in the census (see Section 3.3.1).

possible that some of the rural to urban migration can be explained through this process as well as the rural to urban dislocation of young men upon entry into the military.

Table 6.8 – Pathways of Interprovincial Migration within Ontario.

Pathway	Birth-Death		1901-Death		1911-Death	
	N	%	N	%	N	%
Rural to Rural	917	44.7	1,256	46.5	1,121	47.1
Rural to Urban	631	30.7	697	25.8	408	17.1
Urban to Rural	79	3.8	92	3.4	89	3.7
Urban to Urban	426	20.8	658	24.3	763	32.0
Total	2,053	100.0	2,703	100.0	2,381	100.0

Note: To and from urban and rural locations for those with “positive” or “likely” links for those. Birth to Death (N=2,053), 1901 to Death (N=2,703), and 1911-Death (N=2,381).

Table 6.9 separates these migratory pathways based on the location of origin. This is done in order to facilitate comparison with Sylvester’s estimates of migration based on the declared location of birth and presence in the 1901 census from the CFP sample for the Ontario-born population of working age, defined as over age 15 (2007:156, Table 5.3).⁸⁰ The comparison may not be exact, since the location of origin is based on the declared place of birth on the census return. Further, an urban location in 1901 is based on cities over 1,000 population. The differences between the decedents at every time point and the Sylvester CFP sample is for there to either be more individuals living in urban areas or moving to urban areas among the decedents. This is in accordance with the reduction in *Agriculture* jobs among the decedents reported in Section 6.1. This suggests a deleterious effect of living in cities upon mortality in 1918 (which is to be expected from an airborne infectious disease), but may indicate an additional risk for those who had moved to an urban centre after being born in a rural area and thus the question of whether there were socio-economic differences among those who moved as opposed to those who did not.

⁸⁰ This is done in order to compare the amount of individuals among the decedents who were migrating to and from urban areas with a comparison sample taken from the 1901 census.

Table 6.9 - Pathways of Interprovincial Migration within Ontario separated by Urban and Rural Locations.

Pathway	Birth-Death	1901-Death	1911-Death	Birth-1901 ^a
	%			
Rural to Rural	59.2	64.3	73.3	77.7
Rural to Urban	40.8	35.7	26.7	22.3
Total	100.0	100.0	100.0	100.0
Urban to Rural	15.6	12.3	10.4	21.1
Urban to Urban	84.4	87.7	89.6	78.9
Total	100.0	100.0	100.0	100.0

Note: See Chapter 3 for a definition of urban and rural. For those with PL links: Birth to Death (N=2,053), 1901-Death (N=2,703), 1911-Death (N=2,381).

^a. Ontario-born Work Age Population of Ontario from the 1901 census (N=40,722), from Sylvester (2007:156, Table 5.3).

There is no statistically significant difference between the pathway in each type of record (birth record, 1901 census, and 1911 census) and the sex of the individual using chi-square tests. This may also be important since both Leslie (1974) and Sylvester (2007) suggest that the process of urbanization in Ontario was more common for women. As stated by Sylvester, “with reference to men, rural-born women were 28 percent more likely to make the transition to urban life [and] that the risk of urbanization was as great in Quebec as in Ontario” (2007:160). The greater female rural to urban migration as reported for Ontario may have been countered by the military-based male migration to urban centres from 1914-1918 and the prevalence of influenza among military camps (Crosby 1989). However, the lack of a difference by sex among those who migrated is a way in which the decedents varied from the rest of the Ontario population.

Thus far, migration has been treated as a single event; however, migration may have been recurrent, or circular. Of those individuals who had at least two records to match for location (and they were PL links), the locations were different on at least one time point for 1,077 individuals (36.3% of the linked individuals, for example, birth in an urban area, while the 1911 census and the death records were from rural areas). Of those 1,077, 922 (85.6%) were a one-way migration, of either the rural-to-urban pathway (N=822,

76.3%) or the urban-to-rural pathway (N=100, 9.3%). This leaves 155 individuals (14.4%) who migrated two or more times, to and from urban and rural locations. As the overwhelming majority of those who migrated did so only once (as far as can be determined from these sources and in terms of urban and rural locations), it appears as though movement among the decedents was a permanent event.

Within the province, migration can also be examined in terms of region, as is presented in Table 6.10. As with rural and urban migration, the most common experience for the individuals who died during the pandemic and who were born in Ontario was to die in the same region in which they were born: 79.8% of individuals died in the same region in which they were born, while 4.2% moved to the North, 2.3% moved to Central Ontario, 1.4% moved to Eastern Ontario, 9.8% of individuals moved to South-Central Ontario, and 2.6% of individuals moved to South-Western Ontario. Of those who moved, the most common location was to South-Central Ontario, which included Toronto. By 1911, 87% of the individuals in the WMMIP database were living in the region in which they would die, suggesting that most migration was completed by this point (at this time, the individuals ranged from ages 16 to 28). Deaths occurred among those who lived in the East more frequently than would be expected. Since not many individuals moved to the East after having been born elsewhere in Ontario, it can be assumed that most of those who died in the East had lived there their entire lives.

Table 6.10 – Pathways of Interprovincial Migration by Region of Ontario.

Pathway	Birth-Death		1901-Death		1911-Death	
	N	%	N	%	N	%
Non-Movers	1,644	79.8	2,199	81.9	2,047	87.0
Move to the North	86	4.2	75	2.8	42	1.8
Move to Central	4	2.3	81	3.0	46	2.0
Move to East	28	1.5	27	1.0	27	1.1
Move to South-Central	202	9.8	237	8.8	149	6.3
Move to South-West	53	2.6	67	2.5	43	1.8
Total	2,060	100.0	2,686	100.0	2,354	100.0

Note: For those with “Positive” or “Likely” links. For those individuals with locations at both time points and who were living in Ontario, Birth (N=2,060), 1901-Death (N=2,686), and 1911-Death (N=2,354).

This analysis shows that more individuals than expected died in Eastern Ontario and that they had likely lived there their entire lives. Further, rural to urban migration and being born and dying in an urban centre were more common pathways than has been found by other authors. A notable feature is that, among those who died in 1918, the sex of the individual choosing a particular pathway was not relevant. This chapter so far has looked for difference among those who moved by looking at their occupation and their place of residence as they changed throughout their lives in order to determine how, if at all, the decedents were different from the rest of the population of Ontario. The rest of this chapter will examine if cultural factors such as religion and family size may partially explain the differences found so far, as potential fundamental causes.

6.3 Religion

The death records do not contain information on religion of the decedent; therefore, it is difficult to examine the religious affiliation of the individuals who died in Ontario in

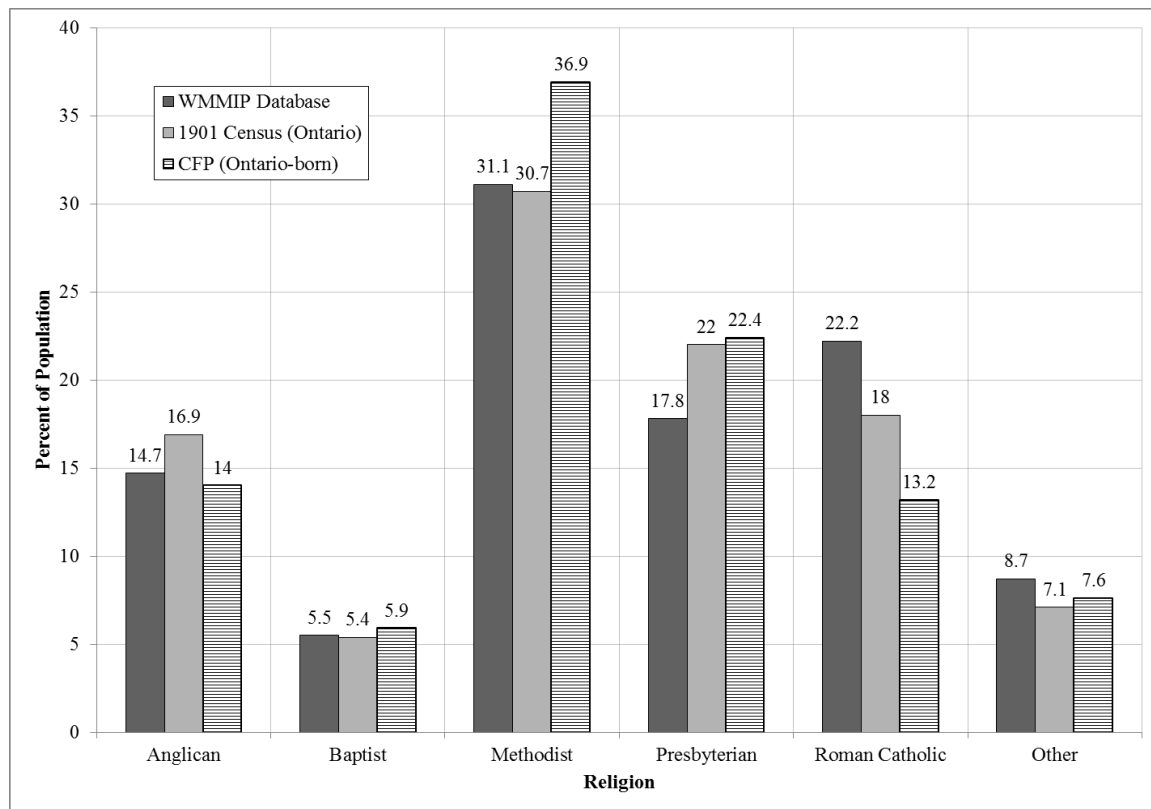
1918. Among the sources used in this research, data on religion is only available from the census and marriage records. It is not possible to compare the decedents to people who did not die in 1918. However, their declared religion from the 1901 and 1911 censuses can be compared to aggregate data from the whole province at concurrent time points.

The religion of the decedents in 1901 was recorded for 2,697 of the 2,704 PL links (99.7%). Where there was discordance between the religion of the individual and the religion of the household, the religion of the individual was used. The distribution of the religions in 1901 is seen in Figure 6.7 and has been condensed for the purposes of analysis into the major denominations (any that consisted of more than 5% of the population in the database. The 17 individuals who were not living in Ontario at the time of the 1901 census are excluded).⁸¹ The major differences between the database population and the rest of Ontario from the 1901 census is that more Catholic individuals died than would be expected and fewer Presbyterians and Anglicans died ($\chi^2=66.00$, $df=5$, $p<.001$).⁸² When the database sample is compared to the CFP sample of individuals who were born in Ontario, the differences are in the same direction (except for Anglicans), but more striking and with the additional difference that there is a higher percentage of Methodists in the CFP sample ($\chi^2=172.41$, $df=5$, $p<.001$).

⁸¹ The category of *Other* includes the following religions: Amish, Apostolic, Believer in Christ, Buddhist, Christadelphian, Christian, Christian Science, Christian Worker, Church of Christ, Church of God, Congregationalist, Disciple, Dunkard, Episcopalian, Evangelical, Free Church, Greek, Hutterite, Independent, Jewish, Liberal, Lutheran, Member of Gospel Hall, Mennonite, Messiah, Moravian, Mormon, Pagan, Plymouth Brethren, Protestant, Quaker, Salvation Army, Secularist, Seventh Day Adventist, Spiritualist, Swedenborgian, Unitarian, United Brethren, and Zionist Reincarnation.

⁸² It is not possible to separate the Ontario-born from the rest of the province in the 1901 or 1911 census. Therefore, the comparison includes immigrants and the comparison between the decedents and the census reports may be less accurate than the comparison between the decedents and the CFP sample.

Figure 6.7 - Major Religious Denomination in 1901.



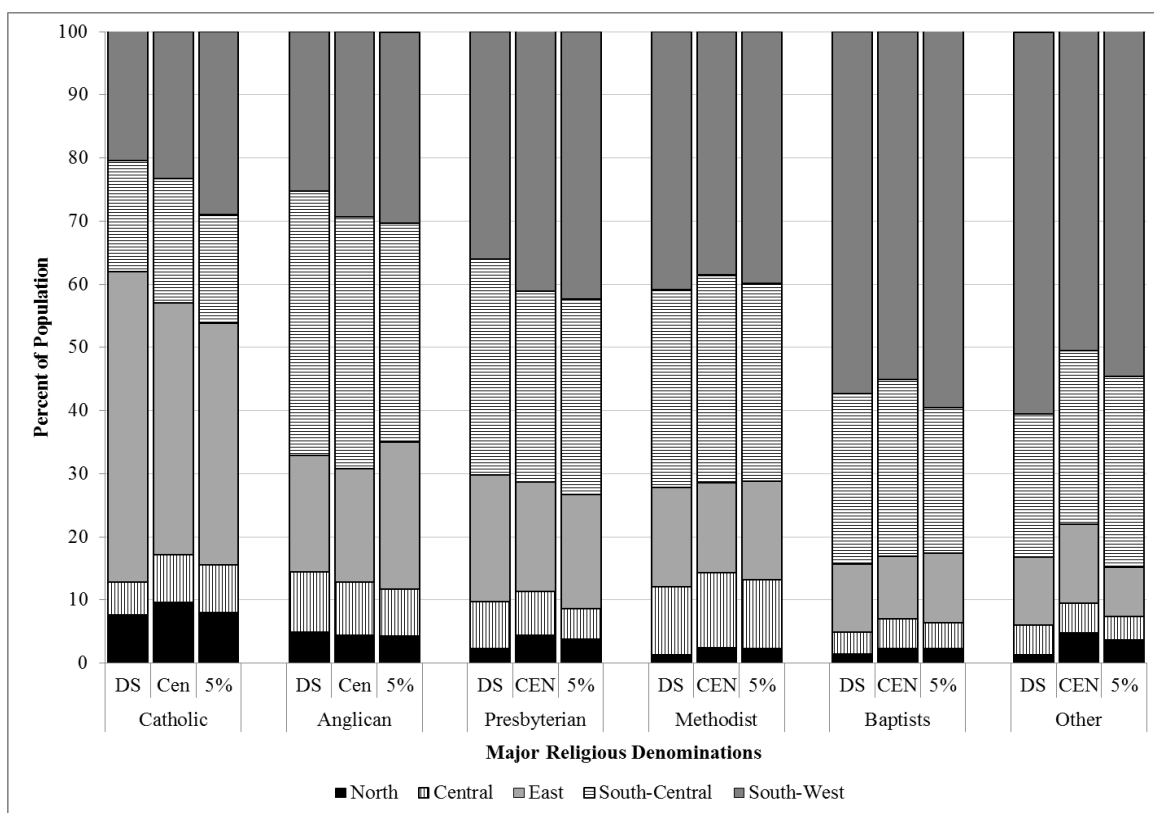
Note: For individuals living in Ontario in 1901 (with “positive” or “likely” links, N=2,680) as compared to the entire Ontario population from the 1901 census (N=2,172,321) and the Ontario-born population from the five percent sample created by the CFP (N=15,051).

As it has been established that more individuals died in the east than was expected, a breakdown of religion by region can analyze whether the greater number of Catholic individuals were also dying in Eastern Ontario. Figure 6.9 shows the WMMIP distribution of religion by region (for those with PL links and who were living in Ontario at the time of the 1901 census), as well as the comparison sample of the 1901 census and the CFP sample.⁸³ Graphically, a higher percentage of Catholic individuals lived in the East among the decedents than among the rest of the population as seen in the 1901 census sample and the CFP sample and fewer in the South-West.

⁸³ 17 individuals were living in other provinces at the time of the 1901 Census: 1 in British Columbia (Catholic), 1 in Alberta (Anglican), 4 in Manitoba (1 Anglican, 1 Methodist, 2 Presbyterians), and 11 in Quebec (2 Anglican, 7 Catholic, 2 Methodist).

Although there were more Methodists in the CFP sample, the distribution of where they lived in the province is fairly consistent between decedents, the census, and the CFP sample, a finding that is also true for Baptists. The distribution of Anglicans was also fairly consistent, with there being more Anglicans in the East region in the CFP sample and fewer in the South-West among the decedents than either the census or the CFP sample. There were more Presbyterians in the East and in Central Ontario among the decedents, while there were fewer in the South-West. The Other category is harder to parse, since this includes all of the aboriginal religions, especially in the North (which were harder to link, Chapter 3).

Figure 6.8 - Distribution of Religions by Region of Residence in 1901.



Note: For those who died from September to December 1918 who were found in the 1901 census and were living in Ontario (and “positive” or “likely” links, N=2,686) as compared to the distribution of religions by region for the whole of Ontario in the 1901 census (N=2,182,947) as well as the Ontario-born sample as found in the CFP 5% sample (N=15,051). Data on the 1901 census from Dominion of Canada (1902).

The distribution of religions among the decedents as listed in the 1911 census is similar in distribution as to the findings in the 1901 census, meaning that it was uncommon for these individuals to change religions over their lives (Table 6.11). Of the 2,281 individuals with PL links who had both a religion in the 1901 and the 1911 censuses, 474 (20.9%) had changed religions, but 432 (91%) of those changes were from one protestant denomination to another. The distribution of religions of all people who lived in Ontario is also similar, except that in 1901, 16.9% of the population was Anglican and in 1911 this had increased to 19.5% of the population. The Methodist population had decreased from 30.7% in 1901 to 26.8% in 1911. There was a less than two percent fluctuation in the distributions of the other religions. These differences remain statistically significant ($\chi^2=51.54$, $df=5$, $p<.001$). Due to these similarities and the existence of the Ontario-born only CFP sample in 1901, 1911 will not be analyzed separately.

Table 6.11 – Major Religious Denominations in 1911.

Religion	Database		Census Total	
	N	%	N	%
Anglican	366	15.6	489,704	19.5
Baptist	138	5.9	132,809	5.3
Methodist	703	30.0	671,727	26.8
Presbyterian	421	17.9	524,603	20.9
Roman Catholic	530	22.6	484,997	19.4
Other	189	8.1	202,131	8.5
Total	2,347	100.0	2,505,971	100.0
<i>Missing or Unspecified</i>	76 ⁸⁴		6,403	

Note: For those Individuals living in Ontario in 1911 (with “Positive” or “Likely” Links, N=2,423) as compared to the Entire Ontario Population from the 1911 Census (N=2,389,134).

⁸⁴ 27 individuals were living in other provinces at the time of the 1911 Census: 4 in British Columbia (1 Baptist, 2 Presbyterian, 1 Methodist), 4 in Alberta (Methodists), 8 in Saskatchewan (2 Catholic, 4 Methodist, 2 Presbyterian), 6 in Manitoba (1 Catholic, 3 Methodist, 2 Presbyterians), and 5 in Quebec (2 Anglican, 1 Baptist, 2 Catholic).

There were more individuals among the decedents who declared themselves to be Catholic in 1901 and 1911 than would be expected, given the distributions of religions as shown in the censuses and in the sample of Ontario-born individuals from the CFP. There were also fewer individuals who were Presbyterian and, compared to the CFP sample, far fewer who identified as Methodist. This analysis in itself cannot account for the reasons behind these differences. However, it is possible that religious differences speak to larger cultural inequalities that might have altered the risk of contracting and dying of influenza.

As Gossage and Gauvreau note “the Roman Catholic faith was strongly associated with high child-woman ratios in almost every province” (2007:82). The cultural-specific behaviours that go beyond the belief system that might influenza mortality could be a higher number of individuals in Catholic families. This could have an effect on poverty, in that scarce resources are required to go further and not all members of the family may be adequately nourished. A larger number of individuals in the household may also factor into increased spread of the virus, since airborne infectious diseases like influenza spread rapidly in crowded environments (Glezen and Couch 1997). Since there were more individuals among the decedents who were Catholic and who were living in the East, the following sections dissect these religious differences into other potential contributing factors, including ethnicity and family size.

6.4 Ethnicity

As with religion, the ethnic background of the individuals in the WMMIP database is only available on the 1901 and 1911 censuses, through the question of “Racial or Tribal Origin”. Since there has been a slight overrepresentation of those in Eastern Ontario and Catholic individuals among the decedents, this section will analyze whether this was potentially a result of ethno-cultural differences.

In addition to Catholicism being a determinant of infant and childhood mortality in Toronto in 1901, Mercier also found that being of West European ancestry also increased

mortality rates (2006:143). This is not surprising, since religion and ethnicity were so deeply intertwined in Ontario in this time period and that the major adherents of (Roman) Catholicism in Ontario were of Irish, French, Scottish, and German origins (Choquette 1975:55).⁸⁵ A higher reliance upon artificial feeding as opposed to breast feeding has been suggested as reason for the higher rates of infant mortality among French Canadians (Alter 1997, McInnis 1997, Thornton and Olson 1997). Thornton and Olson (1997) state that weaning occurred earlier for the French-Canadians than for those of Irish descent, whether Catholic or Protestant. As detailed in Chapter 4, infants are protected to a certain amount by maternal antibodies at the time of birth (Hanson et al. 1985, Holsapple 2003) and if French-Ontarian women were not breastfeeding as long as English-Ontarians, this might have altered the rates of infection in early infancy, and thus the ability of the immune system to respond to a different strain in 1918 (Gagnon et al. 2013). Many of the risk factors associated with adults in regards to ethnicity are the same as those detailed for religion in Section 6.3, due to the close overlap between ethnicity and religion.

Table 6.12 displays the distribution of major ethnicities in the database as found in the 1901 census⁸⁶ (for those living in Ontario) as compared to the rest of the Ontario population in 1901 as well as in the Ontario-born CFP sample. Using a chi-square test, the differences between the database and the census for the entire Ontario population is statistically significant ($\chi^2=87.87$, $df=5$, $p<.001$), as are the differences between the database and the CFP sample selecting only the Ontario-born population living in Ontario ($\chi^2=183.14$, $df=5$, $p<.001$). The major differences between the groups are the greater percentage of French and English among the decedents as well as the smaller percentages of those of Irish and Scottish descent. This is shown graphically in Figure 6.9.

⁸⁵ Italian immigration to Ontario was not significant until later in the twentieth century. There were 378 people in Ontario in 1880-81 who had been born in Italy (Dominion of Canada 1882:395) which had increased to only 3,301 by 1901 (Dominion of Canada 1902:417). In 1921 there were 17,918 Italian-born individuals in Ontario (Dominion of Canada 1925:298) while this number almost doubled to 34,809 by 1951 (Dominion of Canada 1953:47-14).

⁸⁶ For ethnicities that comprise greater than 5% of the database sample. Included in the “Other” Category are: First Nations (N=58, 2.2%), other European ethnicities (N=54, 2%), African (N=12, 0.5%), and other British ethnicities (including Welsh, N=9, 0.3%).

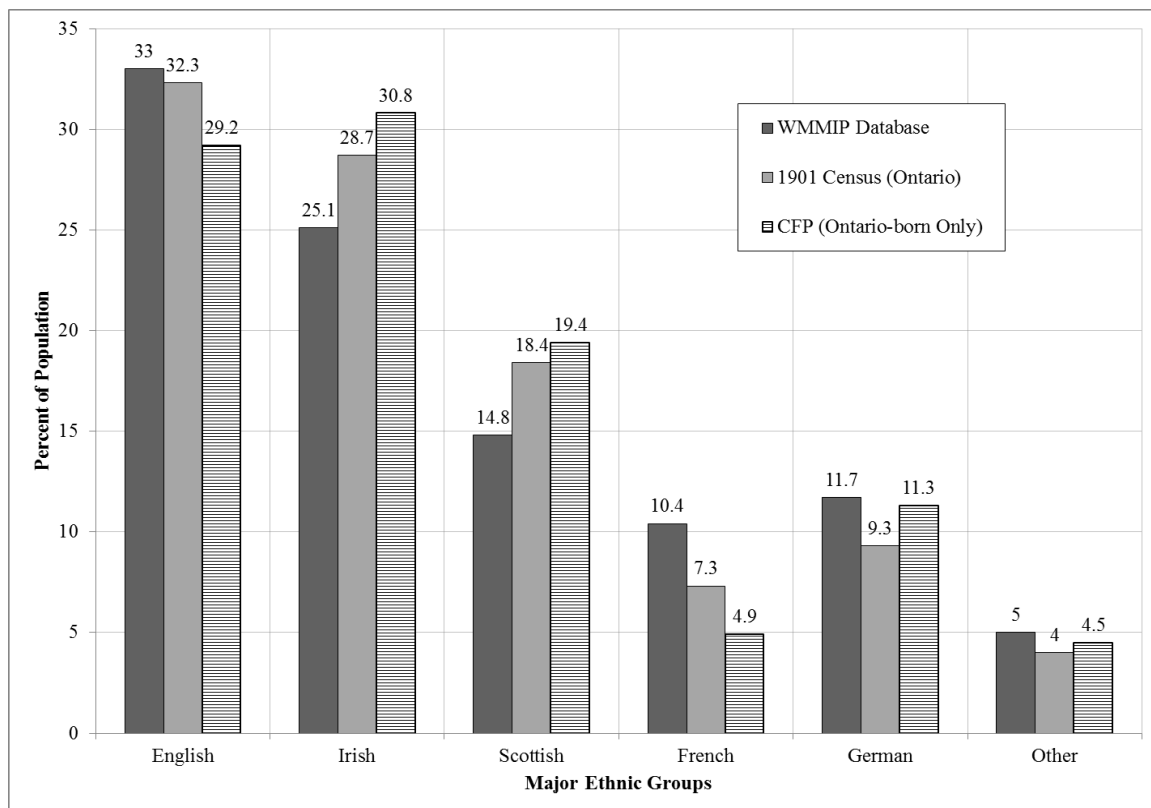
Table 6.12 - Major Ethnic Categories in 1901.

Ethnic Groups	Database		Census		CFP ^a	
	N	%	N	%	N	%
English	885	33.0	701,413	32.3	4,420	29.2
Irish	675	25.1	624,332	28.7	4,655	30.8
Scottish	398	14.8	399,530	18.4	2,928	19.4
French	280	10.4	158,671	7.3	741	4.9
German	313	11.7	203,319	9.3	1,709	11.3
Other	133	5.0	87,411	4.0	676	4.5
Total	2,684	100.0	2,174,676	100.0	15,129	100.0
<i>Missing or Unspecified</i>	53		8,271		49	
χ^2			87.87*		183.14*	
df			5		5	

Note: For Individuals living in Ontario in 1901 (with “Positive” or “Likely” Links, N=2,684) as compared to the entire Ontario population from the 1901 Census (N=2,174,676) and the Ontario-born population from the Canadian Families Project (N=15,129) for ethnicities that comprise greater than 5% of the WMMIP database.

*p<0.05 ^a. Ontario-born only

Figure 6.9 – Major Ethnic Categories in the WMMIP database for 1901

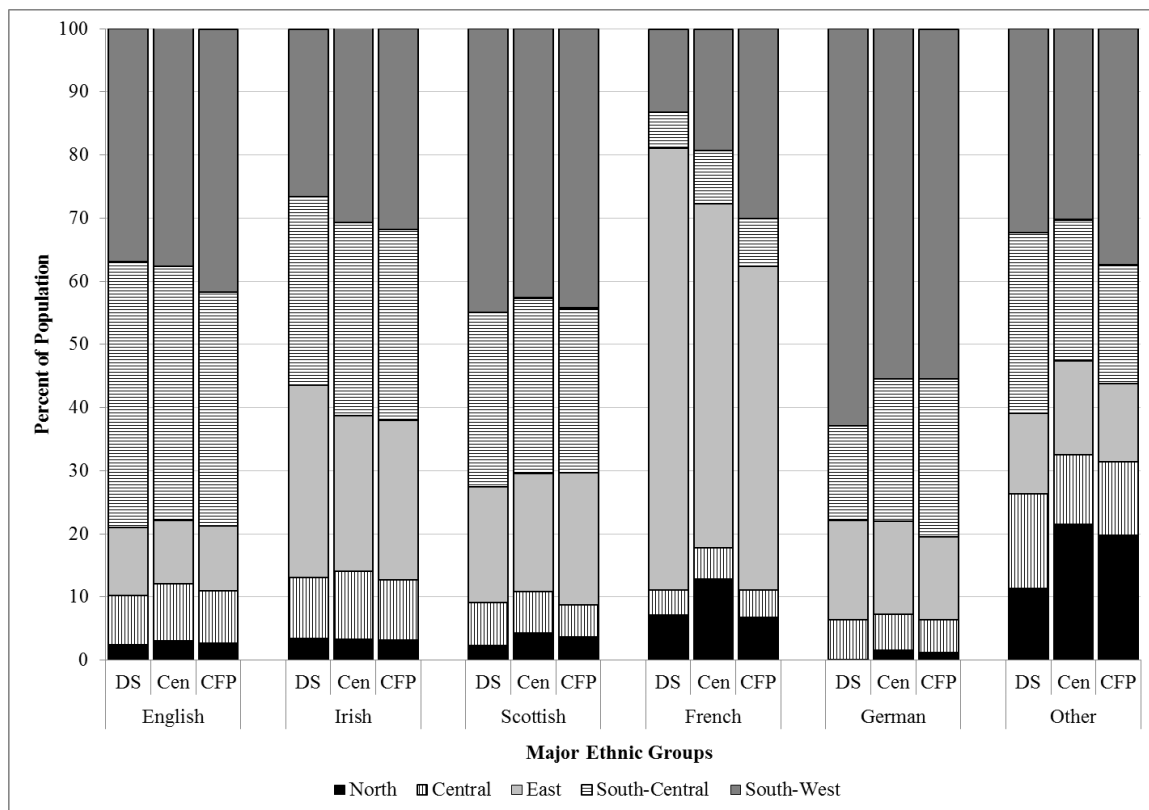


Note: For Individuals living in Ontario in 1901 (with “Positive” or “Likely” Links, N=2,684) as compared to the Entire Ontario Population from the 1901 Census (N=2,174,676) and the Ontario-born Population from the 5 percent sample created by the Canadian Families Project (N=15,129) for ethnicities that comprise greater than 5% of the database.

Figure 6.10 shows the distribution of the 1901 religions in Ontario for the database sample, the 1901 census and the 1901 CFP sample of Canadian-born individuals by region of residence. The distributions of the regions of residence are quite similar for the English and the Scottish across the three samples. They are fairly close for the Irish, with a slight preponderance among the decedents in Eastern Ontario. While the percentage of those of French ancestry is the highest in Eastern Ontario over all 3 samples, the percentage is greater among the decedents than among either the census or the CFP, with far fewer of those of French ancestry dying in South-Western Ontario than would be expected from population-levels. There are more German individuals in South-Western Ontario and fewer in Eastern Ontario. The Other category is once again hard to parse. The greater proportion in the North among both the census and the CFP sample than

among the decedents likely represents the lower success rate of linkages for those who lived and died in Northern Ontario, as well as that this category contains many individuals of First Nations descent, who were also difficult to link due to the many spelling and demographic errors across all types of records (Chapter 3).

Figure 6.10 - Distribution of Ethnicities by Region of Residence in 1901.



Note: For those who died from September to December 1918 who were found in the 1901 Census (N=2,722) as compared to the distribution of religions by ethnicities for the whole of Ontario in the 1901 census (N=2,182,947). Data on the 1901 Census from Dominion of Canada (1902).

Like religion, ethnicity does not tend to change over the lifecourse, as enumerators were instructed in the 1901 census that “among whites the racial or tribal origin is traced through the father” (Dominion of Canada 1902:xviii). For this reason, the distribution of ethnicities for those found in the 1911 census is similar to the distribution in the 1901 census (Table 6.13). Among the decedents living in Ontario in 1911 as compared to the rest of the Ontario population, there were fewer individuals of English and Scottish descent and Other ethnicities (again, possibly reflecting the lower success rate linking

first nations individuals in the north), while there were more individuals of French and German descent. This results are statistically significant using a chi-square test ($\chi^2=81.32$, $df=5$, $p<.001$).

Table 6.13 - Major Ethnic Categories in 1911.

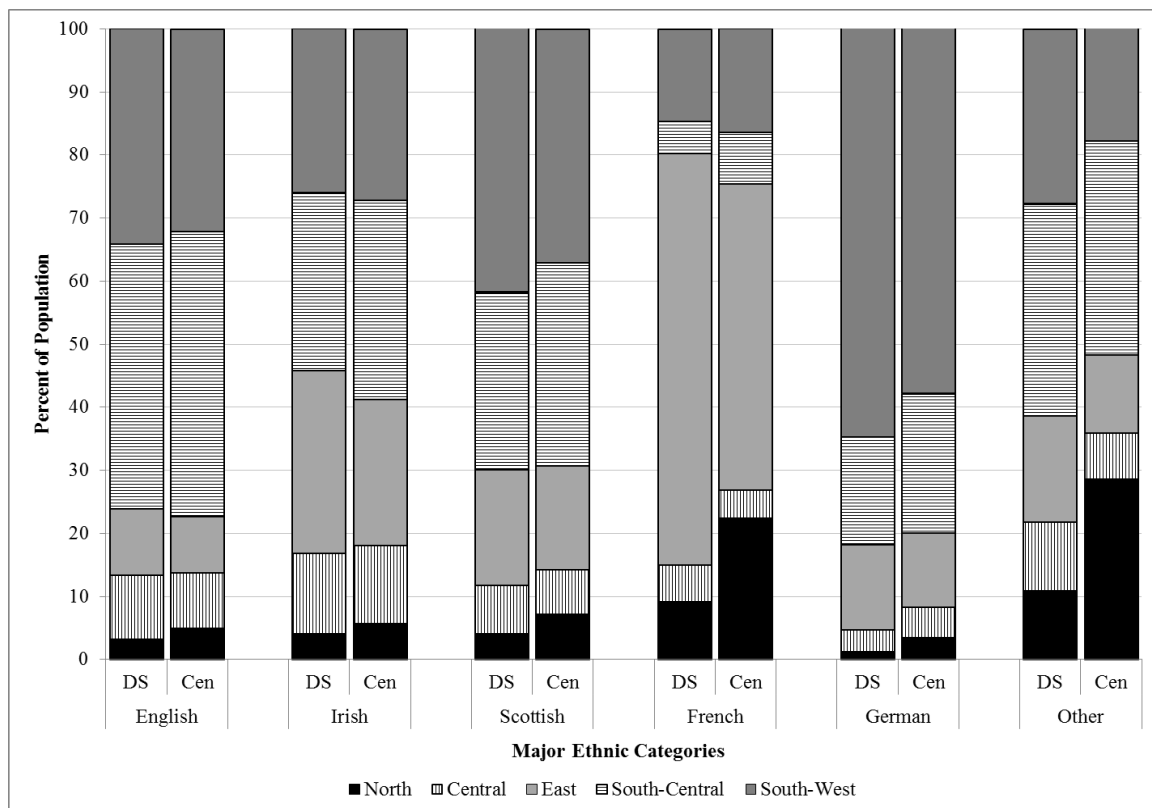
	Database		Census	
	N	%	N	%
English	760	32.7	884,432	35.6
Irish	589	25.3	608,137	24.5
Scottish	344	14.8	424,873	17.1
French	253	10.9	202,442	8.1
German	259	11.1	192,320	7.7
Other	119	5.1	174,104	7.0
Total	2,324	100.0	2,486,308	100.0
<i>Missing or Unspecified</i>	99		36,926	
χ^2			81.31*	
df			5	

Note: For Individuals living in Ontario in 1911 (with “positive” or “likely” links, N=2,324) as compared to the entire Ontario population from the 1911 census (N=2,486,308) for ethnicities that comprise greater than 5% of the WMMIP database.

* $p<0.05$

The distribution by region is shown in Figure 6.12. Is it also quite similar to the distribution in 1901, with there being more Irish individuals in Eastern Ontario and even more French individuals in the East. There are also more Scottish individuals in the South-West. The Other category shows more explicitly the difficulties in finding individuals in Northern Ontario, especially if they were single men who migrated for work (since there was less information on which to link the records).

Figure 6.11 - Distribution of Ethnicities by Region of Residence in 1911.



Note: For those who died from September to December 1918 who were found in the 1911 Census (N=2,394) as compared to the distribution of Ethnicities by Region for the whole of Ontario in the 1911 Census (N= 2,523,234 – There are 40 individuals missing from district breakdowns as compared to the county totals). Data on the 1911 Census from Dominion of Canada (1913).

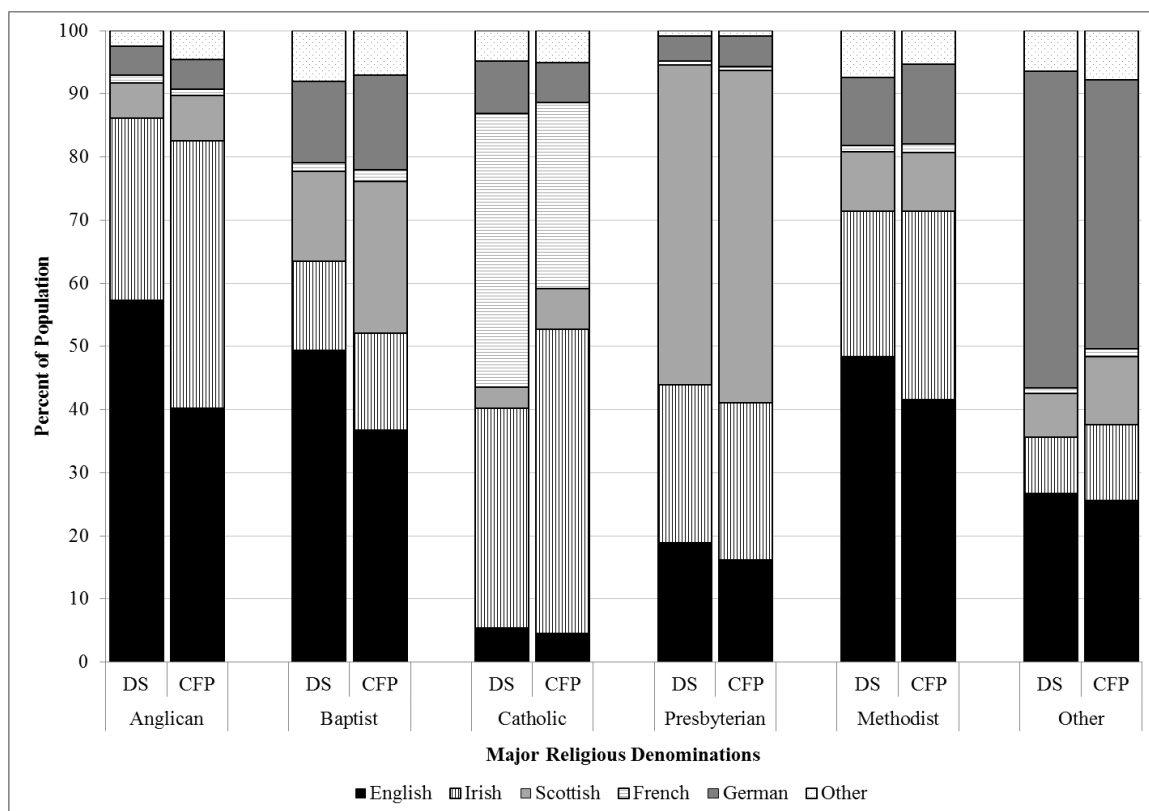
Since more individuals died in the East, more were French, and more were Catholic, it is instructive to analyze how close the accordance was between Catholicism and French descent among the decedents as compared to a census sample. In order to compare the Ontario-born to the Ontario-born, this is done using the linked records from the 1901 census for the decedents and compared to the five percent CFP sample, selecting the Ontario-born (Figure 6.13).

Among Anglicans, Baptists, and Methodists, the proportion of English is greater among the decedents than in the census. For Anglicans and Methodists, this increase comes with a decrease in the percentage of Irish individuals of those denominations. However, for the Baptists, there are fewer Scottish among the decedents than in the census. The

distribution of those of Other religions by ethnicity is similar for the decedents in 1901 as compared to the rest of the 1901 Ontario population.

The differences among the distributions of the Catholic religion by ethnicity are instructive to understanding the relationship between religion, ethnicity, politics, and mortality. As seen in the CFP sample for all residents of Ontario, the largest groups of Catholics at the time were of Irish (43.9%) and French (36.5%) origins. However, when this is restricted to only the Ontario-born CFP sample the proportions change to 48.3% Irish and 29.5% French. Looking at the 1901 sample of decedents, the largest groups are still Irish and French, but their proportions have switched to 43.6% French and 34.8% Irish. Among the decedents in Eastern Ontario in 1901, 293 (46.3%) are Catholic and of those, 189 (64.5%) are French and 78 (26.6%) are Irish.

Figure 6.12 - Ethnicity by Religion in 1901



Note: For everyone with a "positive" or "likely" link who was living in Ontario at the time of the 1901 census (N=2,678) as compared to the CFP sample of the Ontario-born (N=15,004).

The distribution of those who died in 1918 as compared to the censuses of 1901 and 1911 show that more individuals died in Eastern Ontario than expected, there were a greater proportion of Catholic individuals, and more than expected French-Canadians. This research does not examine the causes of these trends, but I hypothesize that it could be related to social differences among cultural groups that may have biological consequences. A higher level of artificial feeding of infants among French-Canadians may have affected risk of exposure in 1890. Further, higher fertility among French-Canadians may have led to higher family sizes, reduced access to resources, and more crowded living environments. Socially, higher fertility among French-Catholics than Irish-Catholics may have caused higher mortality among the French for the same reasons. Also, generalized resistance to the First World War among the French and disagreement with the *Military Service Act, 1917* may have altered the composition of the Ontario population sufficiently from the 1911 census, that the distribution found among the decedents actually does represent the true distribution of the population of the province. With the current data, it is only possible to examine one of these hypothesized causes, that of family size. However, it is likely that a complex interweaving of all of these conditions may have occurred

6.5 Number of People in the Household

As with religion and ethnicity, the information on the number in the household can only be found in the census. The family size of the decedents in 1901 and 1911 was calculated by counting the number of individuals listed as living in the same “Family or House” in the census. It includes all lodgers, boarders, and relatives, unless they are specifically listed with a separate house number. The average number of individuals in the household from the censuses was taken by dividing the number of families by the total population for each district (Dominion of Canada 1902:6-8, 1912:311-358). The value for the average number of individuals in a dwelling from the Ontario-born sample from the CFP was taken from the variable “Count of Persons in Dwelling House”. In both the sample of decedents and CFP sample only those living with 20 or fewer other individuals were included to account for those living in large institutions.

Table 6.14 shows the distribution of average number in the household by region of Ontario for the decedents as found in the 1901 census, as well as from the 1901 census report and from the Ontario-born sample from the CFP. Across all regions, the average number in the household among the decedents was higher, as was the overall average. The highest average in the decedents was in the East, while in the census report and the CFP, the largest household sizes were in the North. The differences are large; however, some of it may be due to the record linkage process. It was easier to link those in large families because there was more information on which to base links. Yet, it is doubtful that this accounts for all the difference. The differences between total families sizes as seen in the 1901 sample of decedents and the Ontario-born sample of the CFP is seen in Figure 6.13

Table 6.14 – Average Number of Individuals in Household in 1901 by Region of Residence.

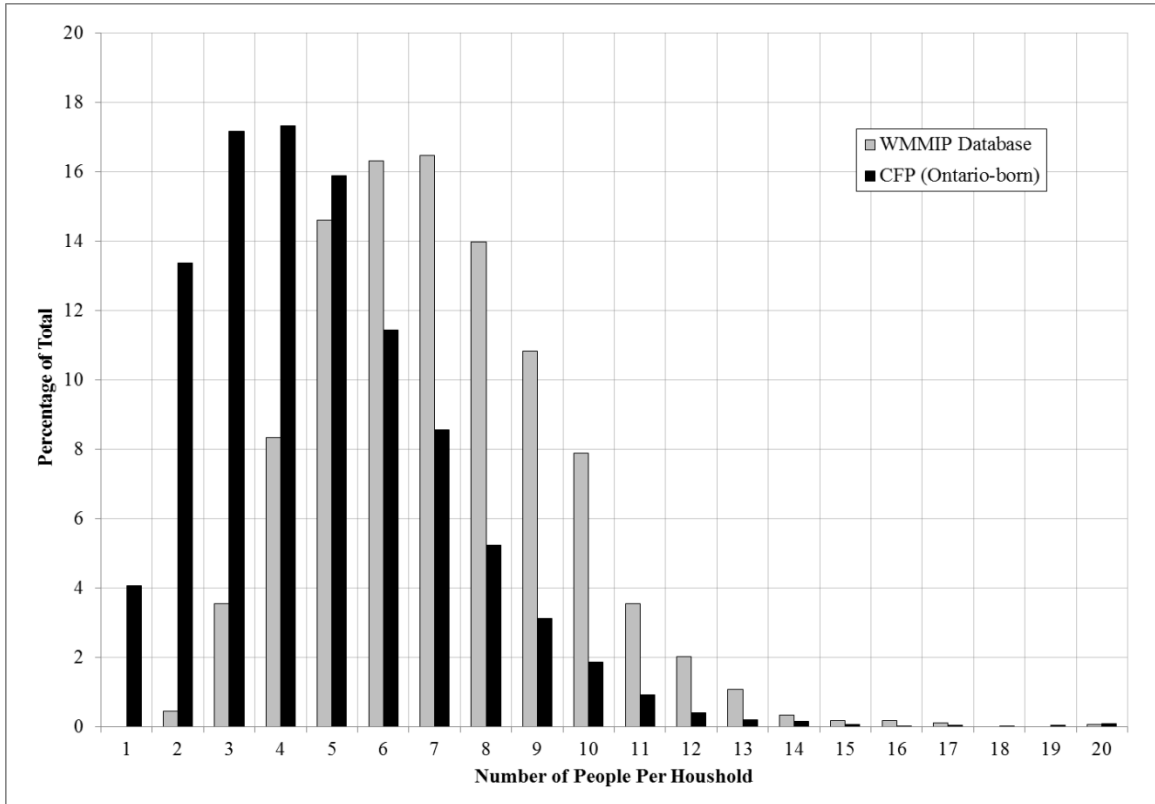
	WMMIP	1901 Census ^a	CFP ^b
		%	
North	7.4	5.7	5.5
Central	7.2	4.7	4.7
East	7.6	5.2	5.0
South-Central	6.8	4.9	4.8
South-West	6.9	4.7	4.8
Total	7.1	4.9	4.9

Note: WMMIP database individuals who were living in Ontario at the time of the 1901 census and who were “Positive” or “Likely” links, (N=2,676). 1901 Census information from Dominion of Canada (1902:6-8) and the Ontario-born sample from the CFP (N=15,157).

a. Average number of people per house

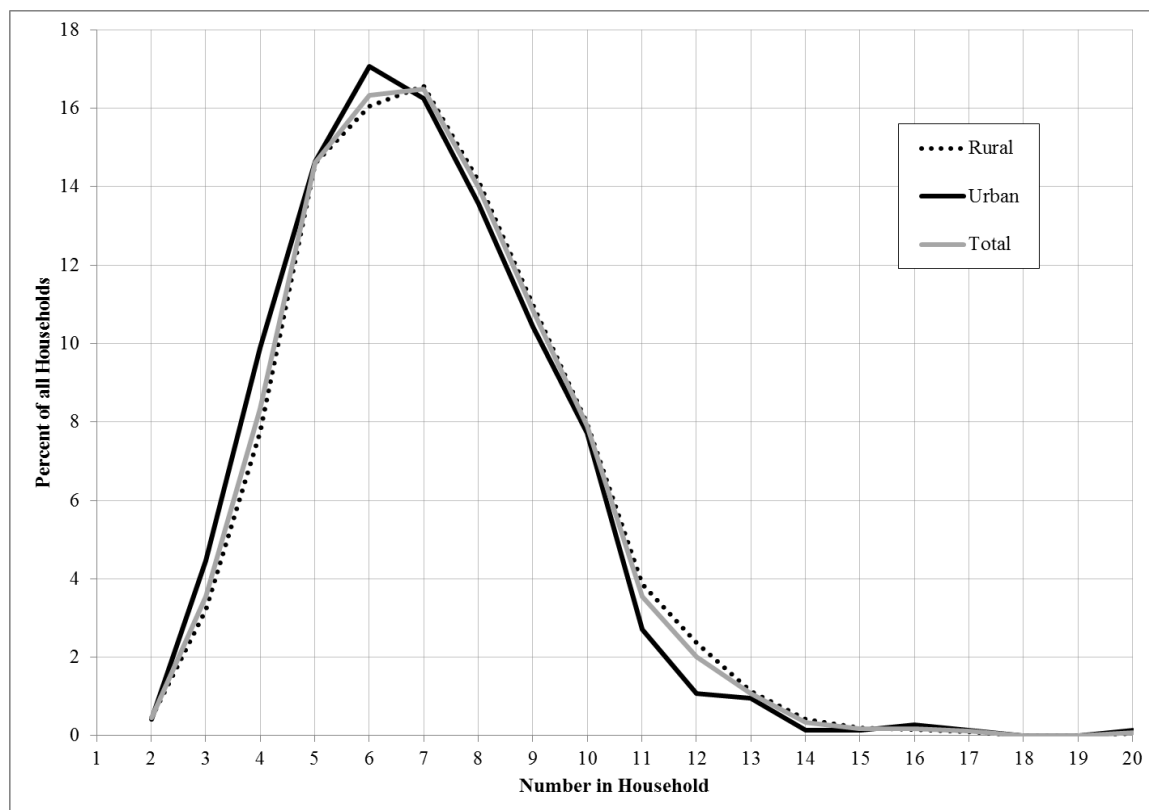
b. Average number of people per dwelling.

Figure 6.13 – Average Number of Individuals in the Household in 1901.



Note: For those with “positive” or “likely” links and who were living in Ontario at the time of the 1901 census (N=2,676) and the Ontario-born sample of the CFP (N=15,157).

Figure 6.14 – Number of Individuals in the Household of the Decedents in 1901 by Urban and Rural Status.



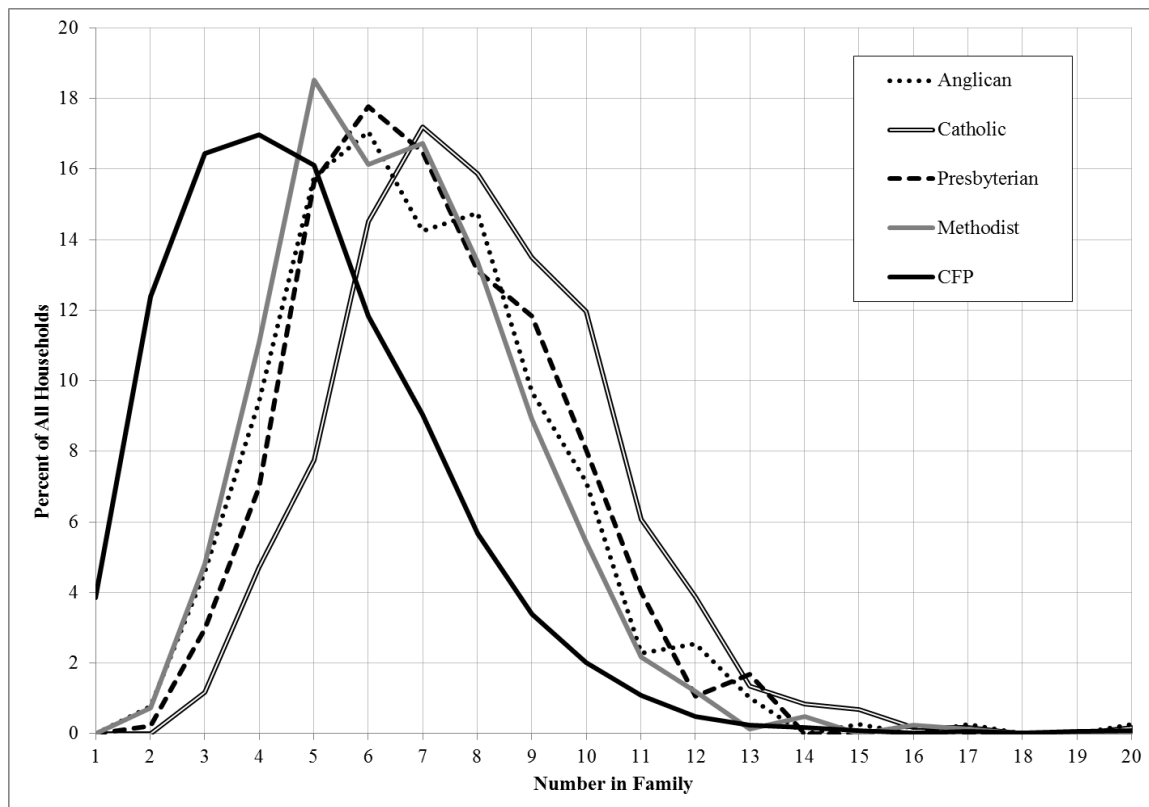
Note: For individuals with “positive” or “likely” links and who were living in Ontario at the time of the 1901 census (N=2,676, urban n=738, rural n=1,938).

Since individuals in the database have larger households on average than the entirety of the province taken together and household sizes are larger in the east, this raises a question: could this be related to urban poverty or is it more an issue of religiously-based fertility differences? Figure 6.14 shows the number of individuals in the household of the decedents in 1901 separated by rural or urban status. There is little, if any difference between the urban or rural status of the household in 1901 and household size. The average household size for rural families is 7.18 and the average household size for urban families is 6.9 (the overall average is 7.1).

While the place of residence does not have an effect on household size, the impact of religion is more pronounced (Figure 6.15). Compared to the household size as found in the CFP, the religions that were found to be different among the decedents (Section 6.3)

universally show larger households. Of the four religions compared, Catholics have the largest family sizes, and Methodists have slightly smaller. Anglican and Presbyterian family sizes are similar (the average family size for the four religions are: Anglican = 6.9, Catholic = 8.0, Presbyterian 7.1, and Methodist = 6.6).

Figure 6.15 - Number of Individuals in the Household of the Decedents in 1901 by Religion.



Note: For the religions with the biggest differences in Section 6.3. Anglican (n=393), Catholic (n=593), Presbyterian (n=473), and Methodist (n=831).

As in 1901, the average household size among the decedents in 1911 was larger in every region than in the census report (Table 6.15). Also, the East has the largest family sizes among the decedents, while in the census, this is found in the North. The census represents all individuals in Ontario, not just the Ontario-born.

Table 6.15 – Average Number of Individuals in the Household in 1911.

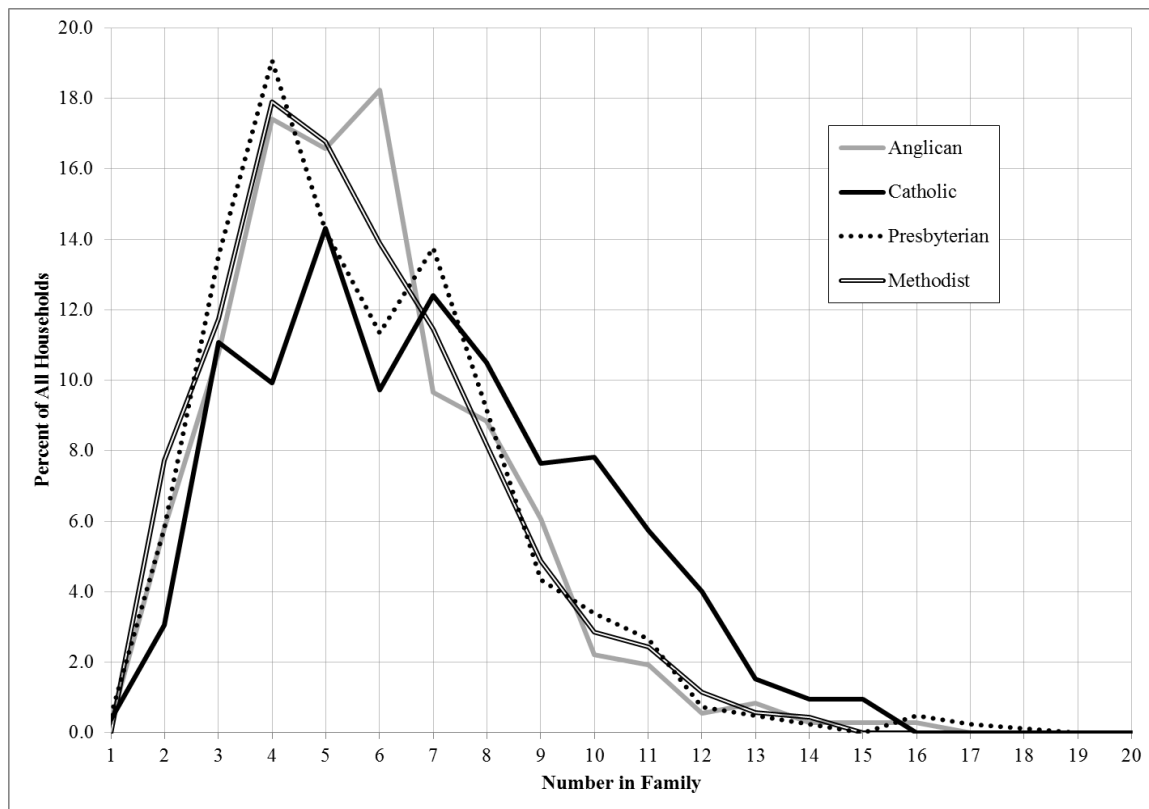
	WMMIP	1911 Census ^a
	Average	
North	6.0	6.8
Central	5.8	4.5
East	6.4	5.4
South-Central	5.9	4.5
South-West	5.7	4.5
Total	5.9	4.8

Note: For those with “positive” or “likely” links and who were living in Ontario at the time of the 1911 census (N=2,328). Data from the 1911 census from Dominion of Canada (1912:311-358).

a. Average number of people per house

Similar urban and rural results were found in 1911. However, there were more differences by religion (Figure 6.16). Greater variability in the curves for household size may be due to the fewer linked records in the 1911 census, but also because more individuals had left their natal homes to start their own families at this point (In 1901, 2,620 (97.6%) of the individuals were living in their natal homes, while 64 (2.4 %) were not. By 1911, likely the result of the increasing age of the decedents, 1,665 (70.3%) were living in their natal homes, while 702 (29.7%) were not). The average household sizes had changed by 1911 to: Methodist=5.6, Anglican=5.7, Presbyterian=5.7, and Catholic=6.9. With some variability among households with more than four individuals, the curves for Anglicans, Presbyterians, and Methodists are fairly similar. Catholic families have fewer smaller families and a greater proportion of larger families.

Figure 6.16 - Number of Individuals in the Household in 1911, by Religion.



Note: For the religions with the biggest differences in Section 6.3. Anglican (n=362), Catholic (n=524), Presbyterian (n=414), and Methodist (n=698).

The finding of greater family sizes in Eastern Ontario is not consistent with research on fertility in Ontario in 1901 from the CFP. Gossage and Gauvreau found that a “rather clear pattern in Ontario of declining fertility as one proceeds from north to south; indeed, the low child-woman ratios encountered in dozens of mainly rural counties bordering the St. Lawrence and the Great Lakes are one of the most striking features of [their] map” (2007:75). However, they did find higher family sizes among Catholic families, farmers, and those born in rural areas. The larger family sizes in the East among the decedents is likely due to the higher than expected proportion of French Catholics in the region.

Household sizes in both 1901 and 1911 among the decedents were higher than in the comparison groups for the rest of the province. Family sizes were found to be higher among Catholic families, while location of residence, either rural or urban had no impact.

This suggests that cultural differences among individuals that could lead to sharing of resources were likely an important factor, as was the spread of influenza through crowded dwellings. The overcrowding and poverty of cities did not have an additional impact, suggesting that cultural factors amplified the effects of having a large family and functioned as a fundamental cause of disease.

6.6 Multivariate Analysis

The factors that have been suggested to be important socio-cultural differences are occupation, region of residence, religion, ethnicity, and number in the household. These variables are analyzed together in this section with logistic regression using the 1901 census linkages. The outcome variable (group membership) was created by combining the Ontario sample of the CFP with the WMMIP for the given variables, where 0=a census case and 1=a WMMIP case. Both of these samples are taken from the Ontario population of the 1901 Canadian census and therefore may not be fully independent. However, the sample taken from the CFP contains 15,178 individuals and the WMMIP contains 3,316 individuals. Using the given population of Ontario in the 1901 census of 2,172,321, the percentage of the total population in the CFP sample is 0.70% and in the WMMIP is 0.15%. There is unlikely to be much overlap between the two groups; however, it is possible and is a limitation of this analysis.

The results of the sequential logistic regressions are found in Table 6.16. The independent variables are Occupation (reference category: *Agriculture*), region (reference category: East), religion (reference category: Catholic), ethnicity (reference category: French), and number in the household. The reference categories reflect the important groups identified in Sections 6.1 to 6.5 (Section 6.1).

Model 1 includes the occupational categories discuss in Section 6.1. *Construction, Government, Domestic and Personal Service, Manufacturing, and Transportation* all have statistically significantly higher odds of being in the WMMIP than those in *Agriculture* families. The *Mining, Professional and Trade* categories all have lower odds

than *Agriculture* of being in the WMMIP; however, this is only significant for the *Professional* category. This emphasizes the retreat from *Agriculture* as suggested in Section 6.1. Controlling for region in Model 2 has little effect on the odds of being in each occupational category. When controlling for occupation, people in all areas of the province have lower odds than those in the East of being in the WMMIP. This is significant for those in the South-Central and South-West regions, and is significant at $\alpha=0.1$ in the North. Since the greatest problems in linkage occurred with individuals in the North (Chapter 4), this difference throughout the models likely reflects a problem in the linkage process. The South-West of Ontario was primarily an agricultural area (Chapter 2). Since there were fewer than expected individuals in agriculture in the WMMIP at all time periods after birth, this explains why there are significantly fewer individuals in the South-West in the WMMIP across all models. The lower odds of being in the South-Central region disappear when controlling for religion, ethnicity, and size of the household.

Model 3 introduces religion into the logistic regression. All religions have lower odds than Catholics of being in the WMMIP when controlling for region of residence and occupational category. These differences remain significant when controlling for ethnicity in Model 4, but only remain significant for Baptists, Presbyterians, and Methodists when controlling for size of the household.

The Irish, Scottish, and Germans had significantly lower odds of being in the WMMIP than the French and this remains while accounting for size of the household in Model 5. The odds for the English and the Other categories are similar to the French and are not statistically significant.

Table 6.16 - Logistic Regression of Group Membership by Socioeconomic Variables in 1901

	Model 1	Model 2	Model 3	Model 4	Model 5
OR					
Occupation					
(Agriculture) ^a					
Construction	1.28***	1.28***	1.26***	1.24***	1.23**
Government	1.74***	1.70***	1.58***	1.56***	1.34**
Service ^b	1.56**	1.50*	1.40	1.45	1.59*
Forestry	1.31	1.27	1.12	1.04	0.99
Manufacturing	1.37***	1.37***	1.31***	1.27***	1.34***
Mining	0.76	0.77	0.90	0.91	0.99
Professional	0.69***	0.69***	0.71***	0.72***	0.82
Trade	0.92	0.92	0.92	0.94	1.03
Transportation	1.54***	1.52***	1.47***	1.48***	1.68***
Region					
(East)					
North		0.79*	0.79*	0.75**	0.67***
Central		0.87	0.98	0.99	1.09
South-Central		0.81***	0.93	0.93	0.99
South-West		0.73***	0.83***	0.82***	0.88**
Religion					
(Catholic)					
Anglican			0.65***	0.68***	0.88
Baptist			0.62***	0.60***	0.80**
Presbyterian			0.50***	0.56***	0.71*
Methodist			0.52***	0.51***	0.72*
Other			0.75***	0.72***	0.92
Ethnicity					
(French)					
English				0.89	0.98
Irish				0.56*	0.60*
Scottish				0.63*	0.69***
German				0.77*	0.79***
Other				0.94	1.01
Size of Household					1.54***
Model χ^2	91.10***	119.15***	248.05***	341.51***	2044.29***
Pseudo R Square	0.006	0.008	0.017	0.024	0.142
AIC	14,439.75	14,361.03	14,179.36	14,078.10	12,337.31
BIC	14,516.85	14,468.96	14,325.70	14,262.90	12,569.81
N	16,486	16,470	16,347	16,315	16,315

^a. Reference categories in parentheses.

^b. Service = Domestic and Personal Service

***p<0.01, **p<0.05, *p<0.1

The multivariate analysis reinforces the findings of the individual analyses – there is an important reduction in agricultural occupations and there are more individuals of French-Catholic origin in 1901 in the WMMIP than would be expected based on the CFP sample. This is partially, but not fully, accounted for by differences in household size. There were important social differences between those who died in the 1918 flu and the rest of the Ontario population as measured in 1901.

6.7 Conclusion

This chapter addressed the following research question:

4. *Among young adults, were there unequal mortality experiences resulting from socio-cultural or demographic differences among individuals or groups?*

Those individuals who were born in Ontario between 1883 and 1895 and who died in the province between September and December 1918 do show some differences from the population of Ontario in 1901 and 1911, both the Canadian and the Ontario-born. While a large proportion of the decedents were born into agricultural families, far fewer were farming at the end of their lives. Most individuals stayed in their region of residence from birth to death and stayed in the same rural or urban environment, yet more were living in urban environments than in the census populations. A greater proportion of the decedents were living in Eastern Ontario than would otherwise be expected, fewer were Methodist and Presbyterian, and more were Catholic. Across all regions of the province, the decedents were living in larger homes in 1901 and 1911, with there being larger households among Catholic individuals, but urban and rural status was not important. Regarding the research question, the decedents as they were in 1901 and 1911 did not represent the rest of the province at the same time points.

These differences may be a result of many factors, the examination of which is beyond the scope of this dissertation. Since it is not possible to get an accurate depiction of the population as it was in August 1918, it may be that the demographic characteristics of the province were significantly altered from 1911, due to differential enlistment in the First

World War. Thus, any differences found between the decedents and the population may be reflective of how society at large had changed. However, these cultural differences may also reflect fertility behaviours that lead to larger family sizes, and thus potentially increased poverty or greater spread of influenza through crowding. Mortality among groups in 1918 does have correlates with earlier infant (and adult) mortality patterns in the province, whereby French-Catholic mortality was higher than Anglo-Protestant mortality (Taylor 1986, Thornton and Olson 1997, Mercier and Boone 2002, Mercier 2006). In this regard, the higher than expected mortality among French-Catholic individuals from Eastern Ontario is not as anomalous. While it is not possible to determine whether the socioeconomic differences among the decedents represent a gradient in risk of death, it is possible to state that there were differences among groups. This suggests that cultural factors, such as Catholic religion and French ethnicity were fundamental causes of disease.

The impact of the records linkage process must also be examined (Chapter 4). The WMMIP consistently have larger households than other areas in the province; however, these were the households that were easiest to link. While there was a high linkage success rate (89.4%), it is possible that those among the unlinked had smaller household sizes and different socio-cultural characteristics.

The reasons behind these differences must be ascertained in future analyses; however, this research has shown characteristics of the decedents that were impossible from the analysis of the death records alone. Use of birth and census records provides data on those who would otherwise have been excluded from the analysis, a disproportionate amount of whom were women. Having data from various time points throughout their lives allows analysis of the various movements and change that would otherwise have remained hidden. Although the exact composition of those who were at risk of dying is not known, we now have greater, and important, information about those who did die.

Chapter 7

7 Conclusion

The unexpected excess mortality among young adults during the 1918 influenza pandemic has been discussed since the time of the pandemic itself. Various hypotheses have been proposed that could account for this, but most failed to analyze mortality beyond five-year age categories. Once this is done, there emerges a distinct pattern of mortality, with larger than expected numbers of deaths at the age of 28 in Canada (Gagnon et al. 2013, Hallman and Gagnon 2014, see also Oeppen and Wilson 2006, Viboud 2013, Yang 2013). The data necessary to answer these questions had not yet been compiled into a useable format; therefore, the first step in this dissertation was the creation of the novel Western, McMaster, Montreal Influenza Pandemic (WMMIP) database. This database utilized the death records of 3,316 individuals who died in the province of Ontario between the ages of 23 and 35 from September to December, 1918, and who were also born in Ontario, as provided by the International Infectious Disease Data Archive (IIDDA) at McMaster University. I created a records linkage protocol (Appendix A) that gave instructions on how to link the death records to birth records, the 1901 and 1911 Canadian censuses, marriage records, and attestation papers. This protocol was taught to research assistants at Western University, McMaster University, and the Université de Montréal. Together, with my supervision, we created a rich new database that allows us unprecedented access to the lives of individuals who died during the 1918 influenza pandemic.

Once this database had been created, I used it to answer the following research questions:

1. *Through a historical demographic lens, are the extant historical records in Ontario suitable for demographic analyses of past infectious disease? (Chapter 4)*
2. *Were all ages among young adults in Ontario at equal risk of death from the 1918 flu pandemic? (Chapter 5)*

3. *Since young adults have already been shown to be at an unusually high risk of death, could this be due to: (Chapter 5)*
 - a. *Previous exposure to influenza, resulting in physiological impairments or immunological conditioning?*
 - b. *Co-morbidity with tuberculosis?*
4. *Among young adults, were there unequal mortality experiences resulting from socio-cultural or demographic differences among individuals or groups? (Chapter 6).*

While acknowledging that there are unique limitations to each type of record used in this study (Chapters 3 and 4) and that they are best analyzed in concert, the use of archival data that had previously been underutilized helps to broaden our understanding of an important period in the history of Ontario. Death records during epidemics may be incomplete due to the extraordinary efforts of doctors and nurses to attend to the living, or because individuals died quickly, far from their homes, where the decedent was unknown to the informant of the death. Because of this, and because women in 1918 were often not employed outside the home, death records for the purposes of historical demographic analyses are of limited utility by themselves. Linked to birth records, the age at death in whole years becomes a much more accurate and useful exact date of birth, which allows for the type of age at epidemic exposure analysis as conducted in Chapter 5. Further, birth records from 1883-1895 are almost always completely filled out, meaning that it is possible to have a picture of the type of socioeconomic environment into which an individual was born (Chapter 6). The main limitation being that birth records during this period were not exhaustive, with their being systematic biases against aboriginal Canadians, immigrant mothers, and single mothers. The 1901 and 1911 Canadian censuses provide rich data concerning the home environment of individuals, and both provide information on date of birth. The 1901 census asked for the month, day, year of birth and the age at last birthday, while the 1911 census asked for the month and year of birth, and the age at last birthday. In both cases, the age at last birthday was the most inaccurate piece of information, and the declared dates of birth for individuals linked to

both records was not consistent (Chapter 4). However, used together, it is possible to get a fairly accurate estimation of the exact date of birth (through the birth record seconded with the 1901 census), and also socioeconomic status at birth, in childhood or adolescence (1901 census), in adolescence or young adulthood (1911 census), and at the time of death (mainly for men). The records linkage process thus allows for a limited understanding of the circumstances under which an individual died to become more rounded picture of conditions throughout the lifecourse. Although these data would not meet the requirements of modern demographic analyses, they provide a great deal of previously unknown information, the kind that is essential to historical demographic research.

By using the birth record, verified with the census records, we created a reconstructed date of birth for each individual. Since a distinct peak in mortality was noted at the age of 28 (Chapter 5) and the 1890 Russian influenza pandemic occurred 28 years previous to the 1918 Spanish influenza pandemic, an exact date of birth allows for analysis of the date of potential exposure in 1890 (the age in whole years as declared on the death record does not allow for a sufficiently precise analysis of an epidemic that was limited to a few months duration). Although the reconstructed age of individuals diluted the peak at age 28 for men (and reduced the overall sample size), the pattern remains for women. Those who were *in utero* at the time of potential maternal exposure died in greater numbers than would be expected if mortality was equally distributed over the age categories, and those in the first trimester of gestation had an unusual sex-ratio at death, possibly reflecting a lowered sex-ratio at birth resulting from maternal stresses. Of the various hypotheses proposed to account for the unexpectedly high young adult mortality in 1918 (Chapter 2), these data most closely support that of antigenic imprinting, whereby exposure to a different strain of influenza early in life can lead to an insufficient immune response to a highly virulent strain in later life. There is cautious support for the fetal growth restrictions hypothesis, but these data do not support the scarring mechanism.

It has been proposed that there was a relationship between tuberculosis and influenza co-infection, that both resulted in higher mortality for men during the 1918 influenza pandemic and also a reduction in mortality from tuberculosis in the ensuing years, since

individuals who would have died later from tuberculosis died in 1918 (Noymer and Garenne 2000, Noymer 2009, 2010, Chapters 2 and 5). Data from the Registrar General for Ontario (Legislative Assembly of Ontario 1911-1932) do not support this hypothesis, since tuberculosis mortality in Ontario was declining long before 1918 and tuberculosis in Ontario was not primarily a disease of men, nor did the 1918 influenza pandemic in the province disproportionately target men over women.

Finally, the records linkage process allows for an analysis of the characteristics of the individuals who died, in greater detail than was previously available using death records alone. Death records provide occupational information for only about half of the decedents (Chapter 6), and over 80% of that information pertains only to men. It becomes almost impossible to understand the socioeconomic conditions of the women who died. It is less than ideally accurate to discern ethnic origins from given or surnames alone, and it is not possible to obtain information concerning religion, migration, or number of individuals in a household from death records. These data are amply available in the WMMIP database. Through an analysis of the linked records, we now know that more individuals left agricultural homes of origin among the decedents than among the Ontario population in general. There were proportionally more French Canadians among the decedents, more catholic individuals, and more people from Eastern Ontario. The sex-ratios were approximately the same, but the decedents came from larger families than were found in the greater population (for a discussion of the possibility that this was an artifact of the records linkage process see Chapter 6). There has been a long-standing debate in the literature over whether the 1918 influenza pandemic was so virulent that all individuals in society were at equal risk of contracting and dying from the disease (Chapter 2). Recent research questions this hypothesis, suggesting that those individuals who are typically at higher risk of death (such as the impoverished, the discriminated, and those living in overcrowded dwellings) were at greater risk of dying (for example, see Mamelund 2006 and Herring and Korol 2012). This research reveals that the mortality pattern in Ontario during the pandemic was similar to what it was previously: mortality continued along the fault lines in society and did not equalize risk in a “democratic” manner.

This research is the first analysis of the newly created WMMIP database. However, it is by no means exhaustive. There is much that can still be analyzed, demographically, sociologically, and anthropologically. While marriage records were obtained during the records linkage process, they were only cursorily used in this research, but provide valuable information about marriage processes, partner selection, and age at marriage. Further, attestation papers were collected, but only found for 200 of the 3,316 decedents. Although limited, these records provide anthropometric data that is not available from other sources. This dissertation focused on the creation of the WMMIP database and the descriptive analysis in terms of four specific research questions. Many avenues exist to extend this research and widen our understanding of a very interesting period in the epidemiological history of Ontario.

References

Acheson, E. D.

1967 *Medical Record Linkage*. Toronto: Oxford University Press.

Acton, Janice, Penny Goldsmith, and Bonnie Shepard, eds.

1974 *Women at Work: Ontario, 1850-1930*. Toronto: Canadian Women's Educational Press.

Adams, Annemarie and Stacie Burke

2006 "Not a Shack in the Woods": Architecture for Tuberculosis in Muskoka and Toronto. *Canadian Bulletin of Medical History* 23(2):429-455.

Adams, Tracey L.

2011 Making a Living: African Canadian Workers in London, Ontario 1861-1901. *Labour* 67:9-43.

2012 The Rise and Fall of Osteopathic Medicine in Ontario, 1900-1930s. *Social History* 45(89):51-79.

Adams, W., R.E. Kendell, E. H. Hare and P. Munk-Jørgensen

1993 Epidemiological Evidence that Maternal Influenza Contributes to the Aetiology of Schizophrenia. *British Journal of Psychiatry* 163:522-534.

Almond, Douglas

2006 Is the 1918 Influenza Pandemic Over? Long-Term Effects of In Utero Influenza Exposure in the Post-1940 U.S. Population. *Journal of Political Economy* 114(4):672-712.

Almond, Douglas and Bhashkar Mazumder

2005 The 1918 Influenza Pandemic and Subsequent Health Outcomes: An Analysis of SIPP Data. *The American Economic Review* 95(2): 258-262.

Alter, George

1997 Infant and Child Mortality in the United States and Canada. *In* *Infant and Child Mortality in the Past*. Alain Bideau, Bertrand Desjardins, and Héctor Pérez Brignoli, eds. Oxford: Clarendon Press. Pp. 92-108.

Archives of Ontario.

- 2004 The Changing Shape of Ontario: The Districts of Northern Ontario. Electronic Document, <http://www.archives.gov.on.ca/en/maps/ontario-north.aspx>, accessed April 25, 2014.
- N.d. Series RG 80-8. Ontario Death Registrations.
- 2014 Tracing Your Family History, Electronic Document, http://www.archives.gov.on.ca/en/tracing/the_records.aspx, accessed June 16, 2014.

Baltagi, Sirine A., Michael Shoykhet, Kathryn Felmet, Patrick M. Kochanek, and Michael J. Bell

- 2010 Neurological Sequelae of 2009 Influenza A (H1N1) in children: A Case Series Observed During a Pandemic. *Pediatric Critical Care Medicine* 11(2):179-184.

Barker, David J. P.

- 1992 The Fetal Origins of Diseases of Old Age. *European Journal of Clinical Nutrition* 46(Suppl 3):S3-S9.
- 1995 Fetal Origins of Coronary Heart Disease. *British Medical Journal* 311:171-174.
- 2006 Adult Consequences of Fetal Growth Restriction. *Clinical Obstetrics and Gynecology* 49(2):270-283.

Barrett, Ron and Peter J. Brown

- 2008 Stigma in the Time of Influenza: Social and Institutional Responses to Pandemic Emergencies. *Journal of Infectious Diseases* 197:S34-7.

Barry, John M.

- 2004 The Site of Origin of the 1918 Influenza Pandemic and its Public Health Implications. *Journal of Translational Medicine* 2:3.

Baskerville, Peter A.

- 2005 *Sites of Power: A Concise History of Ontario*. Toronto: Oxford University Press.

Basler, Christopher F., Ann H. Reid, Jody K. Dybing, Thomas A. Janczewski, Thomas G. Fanning, Hongyong Zheng, Mirella Salvatore, Michael L. Perdue, David E. Swayne, Adolfo Garcia-Sastre, Peter Palese, and Jeffery K. Taubenberger

- 2001 Sequence of the 1918 Pandemic Influenza Virus Nonstructural Gene (NS) Segment and Characterization of Recombinant Viruses Bearing the 1918 NS Genes. *PNAS* 98(5):2746-2751.

Beaujot, Roderic and Don Kerr

- 2004 *Population Change in Canada*, 2nd edition. Toronto: Oxford University Press.

Ben-Shlomo, Y and D. Kuh

- 2002 A Life Course Approach to Chronic Disease Epidemiology: Conceptual Models, Empirical Challenges and Interdisciplinary Perspectives. *International Journal of Epidemiology* 31:285-93.

Bengtsson, Tommy and Jonas Helgertz

- 2013 Long-term Income Effects from Early-Life Exposure to the 1918/1919 Influenza Pandemic: The Case of Southern Sweden. Paper presented at the XXVII IUSSP Conference, Busan, South Korea, August 25-21 2013.

Bengtsson, Tommy and Martin Lindström

- 2003 Airborne Infectious Disease during Infancy and Mortality in Later Life in Southern Sweden, 1766-1894. *International Journal of Epidemiology* 32:286-294.

Bengtsson, Tommy and Frans van Poppel

- 2011 Socioeconomic Inequalities in Death from Past to Present: An Introduction. *Explorations in Economic History* 48:343-356.

Berkner, Lutz K.

- 1975 The Use and Misuse of Census Data for the Historical Analysis of Family Structure. *Journal of Interdisciplinary History* 5(4):721-738.

Beveridge, W.I.B.

- 1977 *Influenza: The Last Great Plague: An Unfinished Story of Discovery*. London: Heinemann Educational Books.

Bilodeau Bertrand, Marianne

- 2014 L'influence sur la longévité d l'exposition très tôt dans la vie à une épidémie au Québec à la fin du XIX^e siècle. M.Sc. thesis, Department of Demography, University of Montreal.

Bintliff, J. L.

- 1991 The Contribution of and Annaliste/Structural History Approach to Archaeology. *In* The Annales School and Archaeology. J. L. Bintliff, ed. Pp. 1-33. Leicester, London: Leicester University.

Blum, Alain, Jacques Houdaille, and Marc Lamouche

- 1990 Mortality Differentials in France during the Late 18th and Early 19th Centuries. *Population (English selection)* 2:163-185.

Bogaert, Kandace

- 2012 Trains, Tents and the Red Triangles: Spanish Flu & the Polish Army at Niagara-on-the-Lake, 1917-1919. Paper presented at the Historical Inequality and Mobility: New Perspectives in the Digital Era Workshop, Guelph, Ontario, May 26.

Bothwell, Robert

- 1986 A Short History of Ontario. Edmonton: Hurtig Publishers Ltd.

Bodewes, R., G. de Mutsert, F. R. M. van der Klis, M. Ventresca, S. Wilks, D. J. Smith, M. Koopmans, R. A. M. Fouchier, A. D. M. E. Osterhaus, and G. F. Rimmelzwaan

- 2011 Prevalence of Antibodies against Seasonal Influenza A and B Viruses in Children in Netherlands. *Clinical and Vaccine Immunology* 18(3):469-476.

Bourbeau, R. and J. Légaré

- 1982 Evolution de la mortalité au Canada et au Québec, 1831-1931. Essai de mesure par generation. Montréal: Les presses de l'Université de Montréal.

Boyle, Coleen A. and José Cordero

- 2005 Birth Defects and Disabilities: A Public Health Issue for the 21st Century. *American Journal of Public Health* 95(11):1884-1886.

Bradbury, Bettina

- 2007 Canadian Children who Lived with One Parent in 1901. *In Household Counts: Canadian Households and Families in 1901*, Eric W. Sager and Peter Baskerville, eds. Pp. 247-301. Toronto: University of Toronto Press.

The British Medical Journal (BMJ)

- 1890 Correspondence: The Epidemic in Russia. *The British Medical Journal* 1(1514, January 4, 1890):46-47.

Brown, Alan S., Melissa D. Begg, Stefan Gravenstein, Catherine A. Schaefer, Richard J. Wyatt, Michaeline Bresnahan, Vicki P. Babulas, and Ezra S. Susser

- 2004 Serologic Evidence of Prenatal Influenza in the Etiology of Schizophrenia. *Archives of General Psychiatry* 61(8):774-780.

Bruckner, Tim A., Ralph Catalano, and Jennifer Ahern

- 2010 Male Fetal Loss in the United States Following the Terrorist Attacks of September 11, 2001. *BMC Public Health* 10:273-278.

Bryan, Thomas

- 2004 Population Estimates. *In The Methods and Materials of Demography*, 2nd edition. Jacob S. Siegel and David A. Swanson, eds. Pp. 523-560. New York: Elsevier Academic Press.

Bryce, Peter H

- 1921 The Story of Public Health in Canada. *In A Half Century of Public Health: Jubilee Historical Volume of the American Public Health Association. In Commemoration of the Fiftieth Anniversary Celebration of its Foundation New York City, November 14-18, 1921.* Mazÿck P. Ravenel, ed. New York: American Public Health Association. Pp. 56-65.

Burguière, André

- 2009 *The Annales School: An Intellectual History.* Jane Marie Todd, trans. Ithaca: Cornell University Press.

Burke, Stacie D. A.

- 2007 Transitions in Household and Family Structure: Canada in 1901 and 1911. *In Household Counts: Canadian Household and Families in 1901.* Eric W. Sager and Peter Baskerville, eds. Pp. 17-58. Toronto: University of Toronto Press.

Byrne, Julianne and Dorothy Warburton

- 1987 Male Excess among Anatomically Normal Fetuses in Spontaneous Abortions. *American Journal of Medical Genetics* 26:605:611.

Catalano, Ralph, Tim Bruckner, Elizabeth Anderson, and Jeffrey B, Gould

- 2005 Fetal Death Sex Ratios: A Test of the Economic Stress Hypothesis. *International Journal of Epidemiology* 3:944-948.

The Canadian Medical Association Journal (CMAJ)

- 1918a Miscellany. *The Canadian Medical Association Journal* 8(5):460.
- 1918b Editorial: The Need for Conscription of Canadian Doctors. *The Canadian Medical Association Journal* 8(10):933-935
- 1918c Editorial: The Present Epidemic. *The Canadian Medical Association Journal* 8(11):1028-1030.

Careless, J. M. S.

- 1984 *Toronto to 1918: An Illustrated History*. Toronto: James Lorimer and Company.

Chambers, Lori

- 2007 *Misconceptions: Unmarried Motherhood and the Ontario Children of Unmarried Parents Act, 1921-1969*. Toronto: Osgoode Society for Canadian Legal History.

Chapin, Charles V.

- 1921 History of State and Municipal Control of Disease. In *A Half Century of Public Health: Jubilee Historical Volume of the American Public Health Association*. In Commemoration of the Fiftieth Anniversary Celebration of its Foundation New York City, November 14-18, 1921. Mazÿck P. Ravenel, ed. Pp.133-160. New York: American Public Health Association.

Charbonneau, Hubert, Bertrand Dejardins, Jacques Légaré, and Hubert Denis

- 2000 The Population of the St. Lawrence Valley, 1608-1760. In *A Population History of North America*. Michael R. Haines and Richard H. Steckel, eds. Pp.99-142.

Chen, Ying-Chu, Chung-Ping Lo, and Tzu-Pu Chang

- 2010 Novel Influenza A(H1N1)-Associated Encephalopathy/Encephalitis with Severe Neurological Sequelae and Unique Image Features – A Case Report. *Journal of Neurological Science* 298(1-2):110-113.

Choquette, Robert

- 1975 *Language and Religion: A History of English-French Conflict in Ontario*. Ottawa: University of Ottawa Press.

City of Toronto Archives

- 1917 *Monthly Reports of the Medical Officer of Health Adopted by the Local Board of Health. Monthly Report of the Department of Public Health of the city of Toronto*. Archived Material, City of Toronto Archives, Toronto, Ontario. Second Floor Stacks: Fonds 200, Series 365, Folio 3, Box 225022, File 20.

Cohen, Marjorie Griffin

- 1988 *Women's Work, Markets, and Economic Development in Nineteenth-Century Ontario*. Toronto: University of Toronto Press.

Cohen, Alan, John Tillinghast, and Vladimir Canudas-Romo

- 2010 *No Consistent Effects of Prenatal or Neonatal Exposure to Spanish Flu on Late-Life Mortality in 24 Developed Countries*. *Demographic Research* 22(20):579-634.

Collier, Richard

- 1974 *The Plague of the Spanish Lady: The Influenza Pandemic of 1918-1919*. London: Macmillan.

Comacchio, Cynthia R.

- 1992 'The Infant Soldier': Early Child Welfare Efforts in Ontario. *In Women and Children First: International Maternal and Infant Welfare 1870-1945*. Valarie Fildes, Lara Marks, and Hilary Marland, eds. New York: Routledge

Crerar, Adam

- 2005 *Ontario and the Great War*. *In Canada and the First World War: Essays in Honour of Robert Craig Brown*. David MacKenzie, ed. Pp. 230-271. Toronto: Toronto University Press.

Crosby, Alfred W.

- 1989 *America's Forgotten Pandemic: The Influenza of 1918*. New York: Cambridge University Press.

Crowley, Terry

- 1988 Introduction. *In* *Clio's Craft: A Primer of Historical Methods*. Terry Crowley, ed. Pp. 1-9. Toronto: Clop, Clark, Pitman, Ltd,

Dannefer, D.

- 2003 Cumulative Advantage/disadvantage and the Life Course: Cross-Fertilizing Age and Social Science Theory. *Journal of Gerontology: Social Sciences* 58B, S327-S337.

Darroch, Gordon

- 2007 Families, Fostering, and Flying the Coop: Lessons in Liberal Cultural Formation, 1871-1901. *In* *Household Counts: Canadian Households and Families in 1901*, Eric W. Sager and Peter Baskerville, eds. Pp. 198-246. Toronto: University of Toronto Press.

Davenport, Fred M. and Albert V. Hennessy

- 1956 A Serologic Recapitulation of Past Experiences with Influenza A; Antibody Response to Monovalent Vaccine. *Journal of Experimental Medicine* 104(1):85-97.

Davenport, Fred M., Albert V. Hennessy, and Thomas Francis Jr.

- 1953 Epidemiologic and Immunologic Significance of Age Distribution of Antibody to Antigenic Variants of Influenza Virus. *Journal of Experimental Medicine* 98(6):641-56.

Dean, W. G., ed.

- 1969 *Economic Atlas of Ontario*. Toronto: University of Toronto Press.

Delorme, Laurent and Peter J. Middleton

- 1979 Influenza A Virus Associated with Acute Encephalopathy. *JAMA American Journal of the Diseases of Children* 133:822-824.

Department of Commerce, Bureau of the Census

- 1920 *Special Tables of Mortality from Influenza and Pneumonia, Indiana, Kansas, and Philadelphia, PA*. Washington: Government Printing Office.

Dickin McGinnis, Janice P.

- 1981 The Impact of Epidemic Influenza: Canada, 1918-1919. *In* *Medicine in Canadian Society*, S.E. Shortt, ed. Pp. 446-77. Montreal and Kingston: McGill-Queen's University Press.

Doblhammer, Gabriele

2004 *The Late Life Legacy of Very Early Life*. New York: Springer.

Dominion of Canada

- 1882 *Census of Canada, 1880-81. Volume I – General Table of Subjects*. Ottawa: MacLean, Roger & Co., Wellington Street.
- 1902 *Fourth Census of Canada, 1901. Volume I – Population*. Ottawa: S. E. Dawson.
- 1910 *Census and Statistics, Bulletin XI: Occupations of the People*. Ottawa C. H Parmelee.
- 1912 *Fifth Census of Canada, 1911. Volume I – Areas and Population by Provinces, Districts, and Subdistricts*. Ottawa: C. H. Parmelee.
- 1913 *Fifth Census of Canada, 1911. Volume II – Religions, Origins, Birthplace, Citizenship, Literacy and Infirmities, by Provinces, Districts, and Sub-Districts*. Ottawa: C. H. Parmelee.
- 1915 *Fifth Census of Canada, 1911. Volume VI – Occupations of the People*. Ottawa: J. de L. Taché.
- 1919 *Report of the Director of the Military Service Branch to the Honourable the Minister of Justice on the Operation of the Military Service Act, 1917*. Ottawa: J. de Labroquerie Taché.
- 1924 *Sixth Census of Canada, 1921. Volume I - Number, Sex and Distribution, Racial Origins, Religions*. Ottawa: F.A. Acland.
- 1925 *Sixth Census of Canada, 1921. Volume II – Population: Age Conjugal Condition, Birthplace, Immigration, Citizenship, Language, Educational Status, School Attendance, Blindness and Deaf Mutism*. Ottawa: F.A. Acland.
- 1953 *Ninth Census of Canada, 1951. Volume I - Population, General Characteristics*. Ottawa: Edmond Cloutier.

Dowdle, W. R.

- 1999 *Influenza A Virus Recycling Revisited*. *Bulletin of the World Health Organization* 77(10):820-828.

Elder, Glen H., Jr, Monica Kirkpatrick Johnson, and Robert Crosnoe.

- 2003 The Emergence and Development of Life Course Theory.” *In Handbook of the Life Course*. J. Mortimer and M. Shanahan, eds. New York: Kluwer. Pp. 3-19.

Emery, George

- 1993 *The Facts of Life: The Social Construction of Vital Statistics, Ontario 1869-1952*. Montreal and Kingston: McGill-Queen’s University Press.

Emery, George and Kevin McQuillan

- 1993 Death in Ingersoll, 1880-1972: A Case-Study Approach to the Revision of Defective Mortality Statistics. *In The Facts of Life: The Social Construction of Vital Statistics, Ontario 1869-1952*. Pp. 50-71. Montreal and Kingston: McGill-Queen’s University Press.

Eversley, D. E. C.

- 1966 Population History and Local History. *In An Introduction to English Historical Demography: From the Sixteen to the Nineteenth Century*, E. A. Wrigley, ed. Pp. 14-43. New York: Basic Books, Inc., Publishers.

Farr, William

- 1856 Report on the Nomenclature and Statistical Classification of Diseases for Statistical Returns. Report on the Nomenclature and Statistical Classification of Diseases for Statistical Returns. London: 16th Annual Report of the Registrar-General of Births, Deaths, and Marriages in England.

Francis, Thomas Jr.

- 1953 Influenza: The New Acquaintance. *Annals of Internal Medicine* 39(2):203-221.
- 1955 The Current Statue of the Control of Influenza. *Annals of Internal Medicine* 43(3):534-538.

Fridlizijs, Gunnar

- 1989 The Deformation of Cohorts: Nineteenth Century Mortality in a Generational Perspective. *Scandinavian Economic History Review* 37:3-17.

Fujimoto, Shinji, Masanori Kobayashi, Osamu Uemura, Mitsuji Iwasa, Tsunesaburo Ando, Toshiyuki Katoh, Chie Nakamura, Noriei Maki, Hajime Togari, and Yoshiro Wada,

- 1998 PCR on Cerebrospinal Fluid to Show Influenza-Associated Acute Encephalopathy or Encephalitis, *The Lancet* 352(9131):873-875.

Gadfield, Chad

- 1988 Theory and Method in Canadian Historical Demography. *In Clio's Craft: A Primer of Historical Methods*. Terry Crowley, ed. Pp. 163-178. Toronto: Copp, Clark, Pitman Ltd.

Gadoury, Lorraine

- 1992 La noblesse de Nouvelle-France: famille et alliances. LaSalle: Huturbise.

Gagnon, Alain,

- 2012 Effect of Birth Season on Longevity: Thrifty and Hopeful Phenotypes in Historical Quebec. *American Journal of Human Biology* 24:654-660.

Gagnon, Alain, J. Enrique Acosta, Joaquin Madrenas, and Matthew S. Miller

- 2015 Is Antigenic Sin Always "Original?" Re-examining the Evidence Regarding Circulation of a Human H1 Influenza Virus Immediately Prior to the 1918 Spanish Flu. *PLoS Pathogens* 11(3):e1004615.

Gagnon, A., M. S. Miller, S. Hallman, R. Bourbeau, D. A. Herring, D. J. D. Earn, and J. Madrenas.

- 2013 Age-Specific Mortality during the 1918 Influenza Pandemic: Unraveling the Mystery of High Young Adult Mortality. *PLoS ONE* 8(8): e69586

Gauvreau, Danielle

- 2006 Religious Diversity and the Onset of the Fertility Transition: Canada, 1870-1900. *In Religion and the Decline of Fertility in the Western World*. Dordrecht, the Netherlands: Springer.

Gauvreau, Danielle and Peter Gossage

- 2001 Canadian Fertility Transitions: Quebec and Ontario at the Turn of the Twentieth Century. *Journal of Family History* 26(2):162-188

Glezen, W. Paul

- 1996 Emerging Infections: Pandemic Influenza. *Epidemiologic Reviews* 18(1):64-76.

Glezen, W.P. and R.B. Couch

- 1997 Influenza viruses. *In* *Viral infections of humans: Epidemiology and control*, 4th edition, Alfred S. Evans and Richard A. Kaslow, eds. Pp. 473-505. New York: Plenum Medical Book Company.

The Globe

- 1918a Sunshine Aid to Combat Flu. *The Globe*, October 10:6.
- 1918b No Epidemic in Toronto. *The Globe*, December 30:8.
- 1918c Houses are Toronto's Need. *The Globe*, October 28:6.
- 1918d Fifty-Five Deaths from Influenza and Pneumonia. *The Globe*, October 18:8.

Gossage, Peter and Danielle Gauvreau

- 2007 Canadian Fertility in 1901. *In* *Household Counts: Canadian Households and Families in 1901*, Eric W. Sager and Peter Baskerville, eds. Pp. 58-109. Toronto: University of Toronto Press.

Gulati, U., K. Kumari, W. Wu, W.A. Keitel, and G.M. Air

- 2005 Amount and Avidity of Serum Antibodies Against Native Glycoproteins and Denatured Virus After Repeated Influenza Whole-Virus Vaccination. *Vaccine* 23(11):1414-25.

Hall, W. J., R. G. Douglas Jr., R. W. Hyde, F. K. Roth, A. S. Cross, and D. M. Speers

- 1976 Pulmonary Mechanics After Uncomplicated Influenza A Infection. *The American Review of Respiratory Disease* 113(2):141-148.

Hallman, Stacey

- 2009 The Effect of Pandemic Influenza on Infant Mortality in Toronto, Ontario, 1917-1921. M.A. thesis, Department of Anthropology, McMaster University.
- 2012 An Exploration of the Effects of Pandemic Influenza on Infant Mortality in Toronto, 1917–1921. *Canadian Studies in Population* 39(3-4):35-48

Hallman, Stacey and Alain Gagnon

- 2014 Does Exposure to Influenza Very Early in Life Affect Mortality Risk During a Subsequent Outbreak? The 1890 and 1918 Pandemics in Canada. *In Are Modern Environments Bad for Human Health? Revisiting the Second Epidemiological Transition*, Molly K. Zuckerman, ed. Pp. 123-138. Toronto: Wiley-Blackwell.

Hanson, Lars Å., Staffan Ahlstedt, Bengt Andersson, Barbro Carlsson, Sven P. Fällström, Lotta Mellander, Oscar Porrás, Tommy Söderström, and Catharina Svanborg Edén

- 1985 Protective Factors in Milk and the Development of the Immune System. *Pediatrics* 75(1):172-176.

Harris, Richard

- 1992 The End Justified the Means: Boarding and Rooming in a City of Homes 1890-1951. *Journal of Social History* 26(2):331-358

Health Canada

- 2009 Canada's Health Care System. Electronic document, <http://www.hc-sc.gc.ca/hcs-sss/pubs/system-regime/2011-hcs-sss/index-eng.php>, accessed July 22, 2013.

Hendricks, K. M. and S.H. Badruddin

- 1992 Weaning Recommendations: The Scientific Basis. *Nutritional Review* 50(5):126-133.

Herlihy, David

- 1973 Problems of Record Linkages in Tuscan Fiscal Records of the Fifteenth Century. *In Identifying People in the Past*. E. A. Wrigley, ed. Pp. 41-56. London: Edward Arnold.

Herring, D. Ann

- 1993 "There Were Young People and Old People and Babies Dying Every Week": The 1918-1919 Influenza Pandemic at Norway House. *Ethnohistory* 41(1):73-105.
- 2009 Viral Panic, Vulnerability, and the Next Pandemic. *In Health, Risk, and Adversity*. Catherine Panter-Brick and Agustín Fuentes, eds. Pp. 78-97. Oxford: Berghan Press.

Herring, D. Ann, ed.

- 2005 *Anatomy of a Pandemic: The 1918 Influenza in Hamilton*. Hamilton: Allegra Print and Imaging.

Herring, D. Ann and Sally Carraher, eds.

- 2011 Miasma to Microscopes: The Russian Influenza Pandemic in Hamilton. Anthropology Publications. Paper 5. Electronic document, http://digitalcommons.mcmaster.ca/anthro_coll/5, accessed August 12, 2012.

Herring, D. Ann and Ellen Korol

- 2012 The North-South Divide: Social Inequality and Mortality from the 1918 Influenza Pandemic in Hamilton, Ontario. *In Epidemic Encounters: Influenza, Society, and Culture in Canada, 1918-20*. Magda Fahrni and Esyllt W. Jones, eds. Pp. 97-112. Toronto: UBC Press.

Herring, D. Ann and L. Sattenspiel

- 2003 Death in Winter: The Spanish Flu in the Canadian Subarctic. *In The Spanish Influenza Pandemic of 1918-19*. Howard Phillips and David Killingray, eds. Pp.156-172. London: Routledge.

Henry, Louis and Michel Fleury

- 1956 Des Registres Paroissiaux à l'Histoire de la Population: Manuel de Dépouillement et d'Exploitation de l'Etat-Civil Ancien. Paris: National d'Etudes Démographic.

Hill, Kenneth

- 2011 Influenza in India 1918: Excess Mortality Reassessed. *Genus* 67(2):9-29.

Hobbs, Frank B.

- 2004 Age and Sex Composition. *In The Methods and Materials of Demography, 2nd Edition*. Jacob S. Siegel and David A. Swanson, eds. Pp. 125-173. New York: Elsevier Academic Press.

Hollingsworth, T. H.

- 1969 Historical Demography. Ithaca, New York: Cornell University Press.

Holsapple, Michael P., Lori J. West, and Kenneth S. Landreth

- 2003 Species Comparison of Anatomical and Functional Immune System Development. *Developmental and Reproductive Toxicology* 68(4):321-334.

Honigsbaum, Mark

- 2010 The Great Dread: Cultural and Psychological Impacts and Responses to the 'Russia' Influenza in the United Kingdom, 1889-1893. *Social History of Medicine* 23(2):299-319.

Hopkins, J. Castell

- 1919 The Province of Ontario in the War: A Record of Government and People. Toronto: Warwick Bros. & Rutter, Limited.

Horder, Thomas

- 1918 Some Observations on the More Severe Cases of Influenza Occurring during the Present Epidemic. *The Lancet* December 28, 1918: 871-3.

Humphries, Mark Osborne

- 2005 The Horror at Home: the Canadian Military and the Great Influenza Pandemic of 1918. *Journal of the Canadian Historical Association* 16:235-260.
- 2013 The Last Plague: Spanish Influenza and the Politics of Public Health in Canada. Toronto: University of Toronto Press.

Jackson, Kelly M. and Andrew M. Nazar

- 2006 Breastfeeding, the Immune Response, and Long-Term Health. *The Journal of the Osteopathic Association* 106(4):203-207.

Jarratt, Melynda

- 2008 Captured Hearts: New Brunswick's War Brides.

Jenness, Diamond

- 1937 The Indian Background of Canadian History. Ottawa: J. O. Patenaude,

Jensen, Keith E., Fred M. Davenport, Albert V. Hennessy, and Thomas Francis Jr.

- 1956 Characterization of Influenza Antibodies by Serum Absorption. *Journal of Experimental Medicine* 104(2): 199-209.

Johansen, Anders and Didier Sornette

- 2001 Finite-Time Singularity in the Dynamics of the World Population, Economic and Financial Indices. *Physica A: Statistical Mechanics and its Applications* 294(3-4):465-502

Johnson, Leo

- 1974 The Political Economy of Ontario Women in the Nineteenth Century. *In* Women at Work: Ontario 1850-1930, Janice Acton, Penny Goldsmith, and Bonnie Shepard, eds. Toronto: The Canadian Women's Educational Press. Pp. 13-31.

Johnson, N.P.A.S.

- 1993 Pandemic influenza: An Analysis of the Spread of Influenza in Kitchener, October 1918. MA thesis, Department of Geography, Wilfrid Laurier University.
- 2003 The Overshadowed Killer: Influenza in Britain. *In* The Spanish Influenza Pandemic of 1918-1919: New Perspectives, H. Phillips and D. Killingray, eds. Pp. 132-155. New York: Routledge.

Johnson, N.P.A.S. and Juergen Mueller

- 2002 Updating the Accounts: Global Mortality of the 1918-1920 "Spanish" Influenza Pandemic. *Bulletin of the History of Medicine* 76(1):105-115.

Jones, Esyllt W.

- 2007 Influenza 1918: Disease, Death, and Struggle in Winnipeg. Toronto: University of Toronto Press.

Judson, D. H. and Carole L. Popoff

- 2004 Appendix C: Selected General Methods. *In* The Methods and Materials of Demography, 2nd edition. Jacob S. Siegel and David A. Swanson, eds. Pp. 677-732. New York: Elsevier Academic Press.

Kaslow, Richard A. and Alfred S. Evans

- 1997 Epidemiologic Concepts and Methods. *In* Viral Infections of Human: Epidemiology and Control, 4th Edition. Alfred S. Evans and Richard A. Kaslow, eds. Pp. 3-58. New York: Plenum Medical Book Company

Katona, P, and J. Katona-Apte

- 2008 The Interaction between Nutrition and Infection. *Clinical Infectious Diseases* 46(10):1582-8.

Katz, Michael B.

- 1972 Occupational Classification in History. *The Journal of Interdisciplinary History* 3(1):63-88.

Kelley, Ninette and Michael J. Trebilcock

- 2010 *The Making of the Mosaic: A History of Canadian Immigration Policy*. Toronto: University of Toronto Press.

Kim, Jin Hyang, Ioanna Skountzou, Richard Compans, and Joshy Jacob

- 2009 Original Antigenic Sin Responses to Influenza Viruses. *The Journal of Immunology* 183:3294-3301.

Knodel, John E.

- 1988 *Demographic Behaviour in the Past: A Study of Fourteen German Village Populations in the Eighteenth and Nineteenth Centuries*. New York: Cambridge University Press.

Kok, Jan, Frans van Poppel and Ellen Kruse

- 1997 Mortality among Illegitimate Children in Mid-Nineteenth-Century The Hague. *In The Decline of Infant and Child Mortality: The European Experience, 1750-1990*. Carlo A Corsini and Pier Paolo Viazzo, eds. Pp. 193-212. Cambridge, MA: Martinus Nuhoff.

Kolata, Gina

- 1999 *Flu: The Story of the Great Influenza Pandemic of 1918 and the Search for the Virus that Caused It*. New York: Touchstone.

Korol, Ellen

- 2011 *The 1918 Influenza Pandemic in Ontario: Impact of the Herald Wave on Autumn Mortality*. M.A. thesis, Department of Anthropology, McMaster University.

Kosteniuk, J. G. and H. D. Dickinson

- 2003 Tracing the Social Gradient in the Health of Canadians: Primary and Secondary Determinants. *Social Science and Medicine* 57:263-276.

Kreijtz, J. H. C. M., R. A. M. Fouchier and G. F. Rimmelzwaan

- 2011 Immune Responses to Influenza Virus Infection. *Virus Research* 162(1-2):19-30.

Kuczynski, Robert R.

- 1930 *Birth Registration and Birth Statistics in Canada*. Washington, D.C.: The Brookings Institution.

The Lancet

- 1918 Annotations. Unmarried Motherhood. *Lancet* 4930 (February 23, 1918):303-304.

Laraya-Cuasay, L.R, A. DeForest, D. Huff, H. Lischner, and N. N. Huang

- 1977 Chronic Pulmonary Complications of Early Influenza Virus Infection in Children. *The American Review of Respiratory Disease* 116(4):617-625.

Laslett, Peter

- 1966 Introduction: The Numerical Study of Society. *In An Introduction to English Historical Demography*, E. A. Wrigley, ed. Pp.1-13. New York: Basic Books Inc., Publishers.

Lawlor, D.A.

- 2004 Commentary: The Art and Science of Epidemiology: Governed by the Seasons? *International Journal of Epidemiology* 33:144-146.

Le Goff, Jean-Marie

- 2011 Diffusion of Influenza during the Winter of 1889-90 in Switzerland. *Genus: Journal of Population Sciences* 67(2):77-99.

Leck, Ian and J. K. Steward

- 1972 Incidence of Neoplasms in Children Born after Influenza Epidemics. *British Medical Journal* 4:631-634.

Legislative Assembly of Ontario

- 1883 Report Relating to the Registration of Births, Marriages, and Deaths in the Province of Ontario for the Year Ending 31st December 1882. Toronto: Printed by C. Blackett Robinson, 5 Jordan Street.
- 1884 Report Relating to the Registration of Births, Marriages, and Deaths in the Province of Ontario for the year ending 31st December 1883. Toronto: Printed by "Grip" Printing & Publishing Co., 26 & 28 Front St.
- 1885 Report Relating to the Registration of Births, Marriages and Deaths in the Province of Ontario for the Year Ending 31st December 1884. Toronto: Printed by Warwick & Sons, 26 & 28 Front Street West.

- 1887a Report Relating to the Registration of Births, Marriages, and Deaths in the Province of Ontario for the Year Ending 31st December 1885. Toronto: Printed by Warwick & Sons, 26 and 28 Front Street West.
- 1887b Report Relating to the Registration of Births, Marriages, and Deaths in the Province of Ontario for the Year Ending 31st December 1886. Toronto: Printed by Warwick & Sons, 26 and 28 Front Street West.
- 1888 Report Relating to the Registration of Births, Marriages and Deaths in the Province of Ontario for the year ending 31st December 1887. Toronto: Printed by Warwick & Sons, 68 and 70 Front Street West.
- 1889 Report Relating to the Registration of Births, Marriages and Deaths in the Province of Ontario for the Year Ending 31st December 1888. Toronto: Printed by Warwick & Sons, 68 and 70 Front Street West.
- 1891a Report Relating to the registration of Births Marriages and Deaths in the Province of Ontario for the year ending 31st December, 1889. Toronto: Printed by Warwick & Sons, 68 and 70 Front Street West.
- 1891b The Ninth Annual Report of the Provincial Board of Health of Ontario Being for the Year 1890. Toronto, Printed by Warwick & Sons, 68 and 70 Front Street West.
- 1892 Report Relating to the Registration of Births, Marriages, and Deaths in the Province of Ontario for the Year Ending 31st December 1890. Toronto: Warwick & Sons.
- 1893 Report Relating to the Registration of Births, Marriages and Deaths in the Province of Ontario for the Year Ending 31 December, 1891. Toronto: Printed by Warwick & Sons, 68 & 70 Front St, West.
- 1894 Report Relating to the Registration of Births, Marriage and Deaths in the Province of Ontario for the Year Ending 31 December 1892. Toronto: Warwick Bros & Rutter, Printers &c., 68 and 70 Front Street West.
- 1895 Report Relating to the Registration of Births, Marriage and Deaths in the Province of Ontario for the Year Ending 31 December 1893. Toronto: Warwick Bros & Rutter, Printers &c., 68 and 70 Front Street West.
- 1896 Report Relating to the Registration of Births, Marriages, and Deaths in the Province of Ontario for the Year Ending 31 December 1894. Toronto: Warwick Bros & Rutter, Printers 68 and 70 Front St West.
- 1897 Report Relating to the Registration of Births, Marriages, and Deaths in the Province of Ontario for the Year Ending 31st December 1895. Toronto: Warwick Bro's & Rutter, Printers &c., 68 and 70 Front Street West.

- 1898 Report Relating to the Registration of Births, Marriages and Deaths in the Province of Ontario for the Year Ending 31st December 1896. Toronto: Warwick Bro's & Rutter, Printers &c., 68 and 70 Front Street West.
- 1900 Report Relating to the Registration of Births, Marriages, and Deaths in the Province of Ontario for the Year Ending 31st December 1898. Toronto: Warwick Bro's & Rutter, Printers &c., 68 and 70 Front Street West.
- 1901a Ontario, Canada: A Statement Concerning the Extent Resources Climate and Industrial Development of the Province of Ontario. Prepared by Direction of the Hon. J. E. Davis, Commissioner of Crown Lands. Toronto: L. K. Cameron.
- 1901b Report Relating to the Registration of Births, Marriages, and Deaths in the Province of Ontario for the Year Ending 31st December 1899. Toronto: L. K. Cameron.
- 1901c Nineteenth Annual Report of the Provincial Board of Health of Ontario, being for the Year 1900. Toronto: L. K. Cameron.
- 1902 Report Relating to the Registration of Births, Marriages, and Deaths in the Province of Ontario for the Year Ending 31st December 1900. Toronto: L. K. Cameron.
- 1911 Report Relating to the Registration of Births, Marriages and Deaths in the Province of Ontario for the Year Ending 31st December, 1910. Toronto: L. E. Cameron.
- 1918 Report Relating to the Registration of Births, Marriages, and Deaths in the Province of Ontario for the Year Ending 31st December 1917. Toronto: A. T. Wilgress.
- 1919a Report Relating to the Registration of Births, Marriages, and Deaths in the Province of Ontario for the Year Ending 31st December 1918. Toronto: A. T. Wilgress.
- 1919b Thirty-Seventh Annual Report of the Provincial Board of Health of Ontario, Canada for the Year 1918. Toronto: Clarkson W. James.
- 1922 Report Relating to the Registration of Births, Marriages, and Deaths in the Province of Ontario for the Year Ending 31st December 1920. Toronto: A. T. Wilgress.
- 1924 Forty-Second Annual Report of the Provincial Board of Health of Ontario, Canada for the Year 1923. Toronto: Clarkson W. James.

- 1932 Report Relating to the Registration of Births, Marriages, and Deaths in the Province of Ontario for the Year Ending 31st December 1930. Toronto: Herbert H. Ball.

Leroux, Marc

- 2010 The Canadian Great War Project.
<http://www.canadiangreatwarproject.com/>. Accessed April 16, 2014.

Leslie, Genevieve

- 1974 Domestic Service in Canada, 1880-1920. *In Women at Work: Ontario 1850-1930*, Janice Acton, Penny Goldsmith, and Bonnie Shepard, eds. Toronto: The Canadian Women's Educational Press. Pp. 71-125.

Lessler, Justin, Steven Riley, Jonathan M. Read, Shuying Wang, Huachen Zhu, Gavin J.D. Smith, Yi Guan, Chao Qiang Jiang, and Derek A. T. Cummings

- 2012 Evidence for Antigenic Seniority in Influenza A (H3N2) Antibody Responses in Southern China. *PLoS Pathogens* 8(7):e1002802.

Lilienfeld, D. E.

- 2007 Celebration: William Farr (1897-1883) – An Appreciation on the 200th Anniversary of his Birth. *International Journal of Epidemiology* 36(5): 985-987.

Link, Bruce and Jo Phelan

- 1995 Social Conditions as Fundamental Causes of Disease. *Journal of Health and Social Behaviour* 35:80-94

Loo, Yueh-Ming and Michael Gale Jr.

- 2007 Fatal Immunity and the 1918 Virus. *Nature* 445(18):267-268.

Lynch, Scott M, ed.

- 2008 Special Issue: Race, Socioeconomic Status, and Health in Life-Course Perspective. *Research on Aging* 30(2).

Lynch, John and George Kaplan

- 2000 Socioeconomic Position. *In Social Epidemiology*. Lisa F. Berkman and Ichiro Kawachi, eds. Pp. 13-35. Toronto: Oxford University Press.

Ma, Junling, Jonathan Dushoff, and David J.D. Earn

- 2011 Age-Specific Mortality Risk from Pandemic Influenza. *Journal of Theoretical Biology* 288:29-34.

MacDougall, Heather

- 1990 *Activists and Advocates: Toronto's Health Department, 1883-1983*. Toronto: Dundurn Press.
- 2007 Toronto's Health Department in Action: Influenza in 1918 and SARS in 2003. *Journal of the History of Medicine and Allied Sciences* 61(1):56-89.

MacMurchy, Helen.

- 1910 *Infant Mortality: Special Report*. Toronto: L. K. Cameron.

Mamelund, Sverre-Erik

- 2006 A Socially Neutral Disease? Individual Social Class, Household Wealth and Mortality from Spanish Influenza in Two Socially Contrasting Parishes in Kristiania 1918-19. *Social Science and Medicine* 62:923-940.

Marion, Nicole and Joseph Scanlon

- 2011 Mass Death and Mass Illness in an Isolated Canadian Town: Coping with Pandemic Influenza in Kenora, Ontario, in 1918-1920. *Mortality* 16(4):325-342.

Maris, Natasha K.

- 2011 The Impact of Influenza: A Global Perspective. *In Miasma to Microscopes: The Russian Influenza in Hamilton*, Ann Herring and Sally Carraher, eds. Pp. 30-39. Anthropology Publications. Paper 5. Electronic document, http://digitalcommons.mcmaster.ca/anthro_coll/5, accessed August 12, 2012.

Marmot, Michael

- 1993 Epidemiological Approach to the Explanation of Social Differentiation in Mortality: The Whitehall Studies. *International Journal of Public Health* 38(5):271-279.
- 2004 *Status Syndrome: How Your Social Standing Directly Affects Your Health and Life Expectancy*. London: Bloomsbury Publishing.

Martel, Kelly A.

- 2011 Pandemic Influenza in Hamilton. *In* Miasma to Microscopes: The Russian Influenza in Hamilton, Ann Herring and Sally Carraher, eds. Pp. 50-58. Anthropology Publications. Paper 5. Electronic document, http://digitalcommons.mcmaster.ca/anthro_coll/5, accessed August 12, 2012

Masters, D. C.

- 1947 *The Rise of Toronto: 1850-1890*. Toronto: The University of Toronto Press.

Mattock, C., M. Marmot, and G. Stern

- 1988 Could Parkinson's Disease Follow Intra-Uterine Influenza? A Speculative Hypothesis. *Journal of Neurology, Neurosurgery, and Psychiatry* 51:753-756.

Mazumder, B., D. Almond, K. Park, E. M. Crimmins, and C. E. Finch

- 2010 Linger Prenatal Effects of the 1918 Influenza Pandemic on Cardiovascular Disease. *Journal of Developmental Origins of Health and Disease* 1(1):26-34.

McCullough, John W. S.

- 1918 *The Control of Influenza in Ontario*. *The Canadian Medical Association Journal* 8(12):1084-1085.
- 1920 *1910-20: A Review of Ten Years Progress*. Ontario: Provincial Board of Health.

McDade, T., M. Beck, C. Kuzawa, and L. Adair

- 2001 Prenatal Undernutrition and Postnatal Growth are Associated with Adolescent Thymic Function. *The Journal of Nutrition* 131:1225-1231.

McDonough, Peggy and Pat Berglund

- 2003 Histories of Poverty and Self-Rated Health Trajectories. *Journal of Health and Social Behavior* 44:198-214.

McInnis, Marvin

- 1997 Infant Mortality in Late Nineteenth Century Canada. *In* *Infant and Child Mortality in the Past*. Alain Bideau, Bertrand Desjardins, and Héctor Pérez Brignoli, eds. Pp. 262-275. Toronto: Clarendon Press.

- 2000 The Population of Canada in the Nineteenth Century. *In A Population History of North America*. Michael R. Haines and Richard H. Steckel, eds. Pp.371-432.

McKinnon, N.E.

- 1944 Mortality Reductions in Ontario, 1900-1942. *Canadian Journal of Public Health* 35:481-484.

McNeill, William H.

- 1976 *Plagues and peoples*. Toronto: Anchor Books, Doubleday.

McQuillan, Kevin

- 1985 Ontario Mortality Patterns, 1861-1921. *Canadian Studies in Population* 1(2):31-48.

McVey, Wayne W. Jr. and Warren E. Kalbach

- 1995 *Canadian Population*. Toronto: Nelson Canada.

Meckel, Richard A.

- 1990 *Save the Babies: American Public Health Reform and the Prevention of Infant Mortality*. Baltimore: The Johns Hopkins University Press.

Mercier, Michael E.

- 2006 The Social Geography of Childhood Mortality, Toronto, 1901. *Urban Geography* 27(2):126-151.

Mercier M. E. and C. G. Boone

- 2002 Economy, Culture and Environment: The Determinants of Infant Mortality in Ottawa, 1901. *Journal of Historical Geography* 28(4):486-507.

Middleton, Jesse Edgar and Fred Landon

- 1927 *The Province of Ontario – A History: 1615-1927. Volume II*. Toronto: The Dominion Publishing Company, Limited.

Miller, Ian Hugh MacLean

- 2002 *Our Glory and Our Grief: Torontonians and the Great War*. Toronto: University of Toronto Press.

Miller, Matthew S., Thomas J. Gardner, Florian Krammer, Lauren C. Aguado, Domenico Tortorella, Christopher F. Basler and Peter Palese.

- 2013 Neutralizing Antibodies against Previously Encountered Influenza Virus Strains Increase over Time: A Longitudinal Analysis. *Science Translational Medicine* 5(198):198ra107.

Moffat, Tina

- 1992 Infant Mortality in an Aboriginal Community: An Historical and Biocultural Analysis. Unpublished MA thesis, Department of Anthropology, McMaster University.

Moore, Sophie E., Timothy J. Cole, Andrew C. Collinson, Elizabeth M.E. Poskitt, Ian A. McGregor, and Andrew M. Prentice

- 1999 Prenatal or Early Postnatal Events Predict Infectious Deaths in Young Adulthood in Rural Africa. *International Journal of Epidemiology* 28:1088-1095.

Morens, David M. and Anthony S. Fauci

- 2007 The 1918 Influenza Pandemic: Insights for the 21st Century. *Journal of Infectious Diseases* 195:1018-1028

Morens, David D. and Jeffrey K. Taubenberger

- 2012 1918 Influenza, A Puzzle with Missing Pieces. *Emerging Infectious Diseases* 18(2):332-335.

Myrskylä, Mikko, Neil K. Mehta, and Virginia W. Chang

- 2013 Early Life Exposure to the 1918 Influenza Pandemic and Old-Age Mortality by Cause of Deaths. *American Journal of Public Health* 103(7):e83-e90.

The National Archives

- n.d. Royal Air Force Personnel. Electronic Document, <http://www.nationalarchives.gov.uk>, accessed 13 November 2013.

Naeye, Richard L, Leslie S. Burt, David L. Wright, William A. Blanc, and Dorothy Tatter

- 1971 Neonatal Mortality and the Male Disadvantage. *Pediatrics* 48:902-906.

Neuzil, Kathleen M., George W. Reed, Edward Mitchel, Lone Simonsen, and Marie R. Griffin

- 1998 Impact of Influenza on Acute Cardiopulmonary Hospitalization in Pregnant Women. *American Journal of Epidemiology* 148(11):1094-1102.

Norton, Susan L.

- 1980 The Vital Question: Are Reconstituted Families Representative of the General Population? *In Genealogical Demography*. Bennett Dyke and Warren T. Morill, eds. Pp.11-22. Toronto: Academic Press.

Noymer, Andrew

- 2009 Testing the Influenza-Tuberculosis Selective Mortality Hypothesis with Union Army Data. *Social Science and Medicine* 68:1599-1608.
- 2010 The 1918 Influenza Pandemic Affected Sex Differentials in Mortality: Comment on Sawchuk. *American Journal of Physical Anthropology* 143:499-500.

Noymer, Andrew and Michel Garenne

- 2000 The 1918 Influenza Epidemic's Effects on Sex Differentials in Mortality in the United States. *Population and Development Review* 26:565-581.

O'Malley, Kimberly J., Karon F. Cook, Matt D. Price, Kimberly Raiford Wildes, John F. Hurdle, and Carol M. Ashton

- 2005 Measuring Diagnoses: ICD Code Accuracy. *Health Services Research* 40(5): 1620-1639.

O'Rand, A. M

- 1995 The Cumulative Stratification of the Life Course. *In Handbook of Aging and the Social Sciences*, 4th Edition. R. Binstock and L. K. George, eds. Pp. 188-207. New York: Academic Press.

O'Rand, A. M. and Jenifer Hamil-Luker

- 2005 Processes of Cumulative Adversity Linking Childhood Disadvantage to Increased Risk of Heart Attack Across the Life Course. *Journal of Gerontology: Social Sciences* 60(2):117-24.

Oeppen J., and C. Wilson

- 2006 Epidemiological evidence for viral exposure in childhood as a risk-factor in subsequent influenza pandemics. Paper Presented at the Population Association of America, Los Angeles, March 30-April 1 2006.

Oertel, Horst

- 1919 Anatomical and Bacteriological Findings in the Recent Epidemic Pneumonia. *Canadian Medical Association Journal* 9(4):339-344.

Oris, Michel, Renzo Derosas, and Marco Breschi

- 2004 Infant and Childhood Mortality. *In Life Under Pressure: Mortality and Living Standards in Europe and Asia, 1700-1900.* Tommy Bengtsson, Cameron Campbell, and James Z. Lee, eds. Pp. 359-398. Cambridge: The MIT Press.

Ornstein, Michael

- 2000 Analysis of Household Samples: The 1901 Census of Canada. *Historical Methods* 33(4): 195-198.

Oxford, J.S., A. Sefton, R. Jackson, W. Innes, R.S. Daniels, and N.P.A.S Johnson

- 2002 World War 1 May Have Allowed the Emergence of "Spanish" Influenza. *Lancet Infectious Diseases* 2:111-114.

Parsons, H. Franklin

- 1891 The Influenza Epidemics of 1889-90 and 1891 and their Distribution in England and Wales. *The British Medical Journal* 2(1597, August 8, 1891):303-308.

Patterson, K. David

- 1986 *Pandemic Influenza, 1700-1900: A Study in Historical Epidemiology.* Totowa, New Jersey: Rowman & Littlefield Publishers.

Pettigrew, Eileen

- 1983 *The Silent Enemy: Canada and the Deadly Flu of 1918.* Saskatoon: Western Producer Prairie Books.

Piva, Michael J.

- 1979 *The conditions of the Working Class in Toronto 1900-1921.* Ottawa: University of Ottawa Press.

Phelan, J.C., B. Link, A. Diez-Roux, I. Kawachi, and B. Levin.

- 2004 Fundamental Causes of Social Inequalities in Mortality: A Test of the Theory. *Journal of Health and Social Behavior* 45:265-285.

Phillips, Howard and David Killingray

- 2003 Introduction. *In The Spanish Influenza Pandemic of 1918-19: New Perspectives*. Howard Phillips and David Killingray, eds. Pp. 1-26. New York: Routledge.

Preston, Samuel H., Patrick Heuveline, and Michel Guillot

- 2001 *Demography: Measuring and Modeling Population Processes*. Malden, Massachusetts: Blackwell Publishers, Inc.

Preston, Samuel H., Mark E. Hill, and Greg L. Drevenstedt

- 1998 Childhood Conditions that Predict Survival to Advanced Ages among African-Americans. *Social Science and Medicine* 47(9):1231-1246.

Quinn, Tom

- 2008 *Flu: A Social History of Influenza*. London: New Holland.

Ramanna, Mridula

- 2003 Coping with the Influenza Pandemic: The Bombay Experience. *In The Spanish Influenza Pandemic of 1918-1919: New Perspectives*, H. Phillips and D. Killingray, eds. Pp. 86-98. New York: Routledge.

Ramkhalawansingh, Ceta

- 1974 Women during the Great War. *In Women at Work: Ontario 1850-1930*, Janice Acton, Penny Goldsmith, and Bonnie Shepard, eds. Toronto: The Canadian Women's Educational Press. Pp. 261-307.

Rankin, John

- 2012 The Reporting of the Influenza Pandemic, 1918-1920 in Hamilton, Ontario. *Health* 4(12): 137-1327.

Rasmussen, K.M.

- 2001 The "Fetal Origins" Hypothesis: Challenges and Opportunities for Maternal and Child Nutrition. *Annual Review of Nutrition* 21:73-95.

Rau, Roland

- 2007 *Seasonality in Human Mortality: A Demographic Account*. New York: Springer.

Ravenholt, R. T. and William H. Foege

- 1892 1918 Influenza, Encephalitis Lethargica, Parkinsonism. *The Lancet* October 16, 1982: 860-864

Razzell, P. and C. Spence

- 2006 *The Hazards of Wealth: Adult Mortality in Pre-Twentieth-Century England*. *Social History of Medicine* 19:381–405

Reid, Alice

- 2001 Neonatal Mortality and Stillbirths in Early Twentieth Century Derbyshire, England. *Population Studies* 55(3):213-232.
- 2005 The Effects of the 1918-1919 Influenza Pandemic on Infant and Child Health in Derbyshire. *Medical History* 49:29-54.

Reid, Ann H., Jeffery K. Taubenberger, Thomas G. Fanning

- 2001 The 1918 Spanish Influenza: Integrating History and Biology. *Microbes and Infection* 3(1):81-87.

Rewegan, Alex, Kandace Bogaert, Melissa Yan, Alain Gagnon, and D. Ann Herring

- 2015 The First Wave of the 1918 Influenza Pandemic Among Soldiers of the Canadian Expeditionary Force. *American Journal of Human Biology* doi: 10.1002/ajhb.22713

Ross, C.E. and J. Mirowsky

- 2001 Neighborhood Disadvantage, Disorder, and Health. *Journal of Health and Social Behavior* 42:258-276.

Rotberg, Robert I. and Theodore K. Rabb, eds.

- 1983 *Hunger and History: The Impact of Changing Food Production and Consumption Patterns on Society*. New York: Cambridge University Press.

Rotenberg, Lori

- 1974 The Wayward Worker: Toronto's Prostitute at the Turn of the Century. *In* Women at Work: Ontario 1850-1930, Janice Acton, Penny Goldsmith, and Bonnie Shepard, eds. Toronto: The Canadian Women's Educational Press. Pp. 33-69.

Sager, Eric W.

- 2007 Inequality, Earnings, and the Canadian Working Class in 1901. *In* Household Counts: Canadian Households and Families in 1901, Eric W. Sager and Peter Baskerville, eds. Pp. 339-370. Toronto: University of Toronto Press.

Sager, Eric W. and Peter Baskerville

- 2007 Introduction. *In* Household Counts: Canadian Households and Families in 1901, Eric W. Sager and Peter Baskerville, eds. Pp. 3-13. Toronto: University of Toronto Press.

Sarna, Jonathan D.

- 1976 Jewish Immigration to North America: The Canadian Experience. *Jewish Journal of Sociology* 18:31-41.

Sawchuk, L. A.

- 2009 Brief Communication: Rethinking the Impact of the 1918 Influenza Pandemic on Sex Differentials in Mortality. *American Journal of Physical Anthropology* 139:584-590.

Scholtz, William C.

- 1890 The Influenza Epidemic at the "Cape". *The British Medical Journal* 1(1524):600.

Seeman, T.E., B.H. Singer, J. W. Rowe, R. I Horwitz, and B. S. McEwen

- 1997 Price of Adaptation – Allostatic Load and its Health Consequences. *Archives of Internal Medicine* 157:2259-2268.

Shah, Snehal, Anthony Keil, Kieren Gara, and Lakshmi Nagarajan

- 2013 Neurologic Complications of Influenza. *Journal of Child Neurology* September 25 2013. DOI: 10.1177/0883073813499610

Shanks, G. Dennis and John F. Brundage

- 2012 Pathogenic Responses among Young Adults during the 1918 Influenza Pandemic. *Emerging Infectious Diseases* 18(2):201-207.

Sherman, Irwin W.

2006 *The Power of Plagues*. Washington, D.C.: ASM Press.

Singer, Merrill and Hans Baer

1995 *Critical Medical Anthropology*. Amityville, NY: Baywood Press.

Slonim, Karen

2010 "Send Only Your Serious Cases." Delivering Flu to Toronto: An Anthropological Analysis of the 1918-19 Influenza Epidemic in Toronto, Ontario, Canada. Ph.D. dissertation, Department of Anthropology, University of Missouri-Columbia.

Smith, Daniel Scott

1973 Parental Power and Marriage Patterns: An Analysis of Historical Trends in Hingham, Massachusetts. *Journal of Marriage and Family* 35(3):406-418.

Smith, F. B.

1995 The Russian Influenza in the United Kingdom, 1889-1894. *Social History of Medicine* 8(1):55-73.

Smith, Gordon C. S.

2001 Use of Time to Event Analysis to Estimate the Normal Duration of Human Pregnancy. *Human Reproduction* 16(7):1497-1500.

Statistics Canada

2009 Estimated Population of Canada, 1605 to Present. Electronic document, <http://www.statcan.gc.ca/pub/98-187-x/4151287-eng.htm#ii>, accessed August 5, 2012.

2011 2011 Census of Population: Population, Urban and Rural, by Province and Territory (Ontario). Electronic document, <http://www.statcan.gc.ca/tables-tableaux/sum-som/l01/cst01/demo62g-eng.htm>, accessed July 13, 2013.

2012 Population and Family Estimations at Statistics Canada. Electronic Document, <http://www5.statcan.gc.ca/olc-cel/olc.action?objId=91-528-X&objType=2&lang=en&limit=0>, accessed April 18, 2014.

2013 Infant Mortality Rates, By Province and Territory (Both Sexes). Electronic Document, <http://www.statcan.gc.ca/tables-tableaux/sum-som/l01/cst01/health21a-eng.htm>, accessed June 16, 2014.

Steenhock, Meghan

- 2011 Flu Talk: The Language of Russian Influenza in Hamilton. *In Miasma to Microscopes: The Russian Influenza in Hamilton*, Ann Herring and Sally Carraher, eds. Pp. 122-129. Anthropology Publications. Paper 5. Electronic document, http://digitalcommons.mcmaster.ca/anthro_coll/5, accessed August 12, 2012.

Storey, William Kelleher

- 2009 *The First World War: A Concise Global History*. Toronto: Rowman & Littlefield Publishers, Inc.

Susser, Mervyn and Zena Stein

- 2009 *Eras in Epidemiology: The Evolution of Ideas*. Toronto: Oxford University Press.

Swanson, David A. and Jacob S. Siegel

- 2004 Introduction. *In The Methods and Materials of Demography*, 2nd edition. Jacob S. Siegel and David A. Swanson, eds. Pp. 1-8. New York: Elsevier Academic Press.

Swedlund, Alan C., Richard S. Meindl, and Margaret I. Gradie

- 1980 Family Reconstitution in the Connecticut Valley: Progress on Record Linkage and the Mortality Survey *In Genealogical Demography*. Bennett Dyke and Warren T. Morill, eds. Pp.139-155. Toronto: Academic Press.

Sydenstricker, Edgar

- 1931 The Incidence of Influenza among Persons of different Economic Status during the Epidemic of 1918. *Public Health Reports* 46(4):154-170.

Sylvester, Kenneth M.

- 2007 Rural to Urban Migration: Finding Household Complexity in a New World Environment. *In Household Counts: Canadian Households and Families in 1901*, Eric W. Sager and Peter Baskerville, eds. Pp:147-179.

Tabachnick, Barbara G. and Linda S. Fidell

- 2007 *Using Multivariate Statistics*, 5th Edition. Toronto: Pearson Education, Inc.

Tackett, Timothy

- 2009 Forward. *In The Annales School: An Intellectual History*. Jane Marie Todd, trans. Pp. ix-xiv. Ithaca: Cornell University Press.

Takei, N., P. Sham, E O'Callaghan, G Murray, G. Glover, and R. Murray

- 1994 Prenatal Exposure to Influenza and the Development of Schizophrenia: Is the Effect Confined to Females? *American Journal of Psychiatry* 151(1):117-119

Takei, Noriyoshi, Graham Murray, Eadbhard O'Callaghan, Pak C. Sham, Gyles Glover, and Robin M. Murray

- 1995 Prenatal Exposure to Influenza Epidemics and the Risk of Mental Retardation. *European Archives of Psychiatry and Clinical Neuroscience* 245:255-259.

Taubenberger, Jeffrey K.

- 2006 The Origin and Virulence of the 1918 "Spanish" Influenza Virus. *Proceedings of the American Philosophical Society* 150(1): 86–112.

Taubenberger, Jeffrey K. and David M. Morens

- 2006 1918 Influenza: The Mother of All Pandemics. *Emerging Infectious Diseases* 12(1): 15-22.

Taubenberger, Jeffrey K., David M. Morens, and Anthony S. Fauci

- 2007 The Next Influenza Pandemic: Can It Be Predicted? *JAMA* 297(18):2025-2027.

Taylor, Carl E.

- 1983 Synergy among Mass Infections, Famines, and Poverty. *In Hunger and History: The Impact of Changing Food Production and Consumption Patterns on Society*. Robert I. Rotberg and Theodore K. Rabb, eds. New York: Cambridge University Press. Pp. 285-303.

Taylor, John H.

- 1986 *Ottawa: An Illustrated History*. Toronto: James Lorimer & Company.

Thompson, Samantha

- 2011 The Russian Flu Rushes to Hamilton. *In Miasma to Microscopes: The Russian Influenza in Hamilton*, Ann Herring and Sally Carraher, eds. Pp.40-49. Anthropology Publications. Paper 5. Electronic document, http://digitalcommons.mcmaster.ca/anthro_coll/5, accessed August 12, 2012.

Thornton, Patricia and Sherry Olson

- 1997 Infant Vulnerability in Three Cultural Settings in Montreal, 1880. *In* Infant and Child Mortality in the Past. Alain Bideau, Bertrand Desjardins, and Héctor Pérez Brignoli, eds. Oxford: Clarendon Press. Pp. 216-241.

Tomkins, Sandra M.

- 1992 The Failure of Expertise: Public Health Policy in Britain during the 1918-19 Influenza Epidemic. *Social History of Medicine* 5(3):435-454.

Toovey, Stephen

- 2008 Influenza-Associated Central Nervous System Dysfunction: A Literature Review. *Travel Medicine and Infectious Disease* 6(3):114-124.

Trovato, Frank

- 2009 Canada's Population in a Global Context: An Introduction to Social Demography. Toronto: Oxford University Press.

Valleron, Alain-Jacques, Anne Cori, Sophie Valtat, Sofia Meurisse, Fabrice Carrat, and Pierre-Yves Boëlle

- 2010 Transmissibility and Geographic Spread of the 1889 Influenza Pandemic. *PNAS* 107(19):8778-8781.

Valtat, Sophie, Anne Cori, Fabrice Carrat, Alain-Jacques Valleron

- 2011 Age Distribution of Cases and Deaths during the 1889 Influenza Pandemic. *Vaccine* 29S:B6-B10.

Van Die, Marguerite

- 1989 An Evangelical Mind: Nathanael Burwash and the Methodist Tradition in Canada, 1839-1918. Kingston: McGill-Queen's University Press.

Viboud, Cécile, Jana Eisenstein, Ann H. Reid, Thomas A. Janczewski, David M. Morens, and Jeffery K. Taubenberger

- 2013 Age- and Sex-Specific Mortality Associated with the 1918-1919 Influenza Pandemic in Kentucky. *Journal of Infectious Diseases* 207(5):721-729.

Walter, S. D.

- 1977 The Power of a Test for Seasonality. *British Journal of Preventive and Social Medicine* 31:137-140.

Wang, Gefei F., Weizhong Li, and Kangshen Li

- 2010 Acute Encephalopathy and Encephalitis Caused by Influenza Virus Infection. *Inflammatory Diseases and Infection* 23(3):306-311.

Weaver, Emily P.

- 1913 *The Story of the Counties of Ontario*. Toronto: Bell and Cockburn.

Webster, Robert G., William J. Bean, Owen T. Gorman, Thomas M. Chambers, and Yoshihiro Kawaoka

- 1992 Evolution and Ecology of Influenza A Viruses. *Microbiological Reviews* 56(1):152-179.

Wharton, Brian

- 1989 Weaning and Child Health. *Annual Review of Nutrition* 9:377-394.

Winchester, Ian

- 1973a A Brief Survey of the Algorithmic, Mathematical and Philosophical Literature Relevant to Historical Record Linkage. *In Identifying People in the Past*. E. A. Wrigley, ed. Pp. 128-150. London: Edward Arnold.
- 1973b On Referring to Ordinary Historical Persons. *In Identifying People in the Past*. E. A. Wrigley, ed. Pp. 17-40. London: Edward Arnold.

Willson, Andrea

- 2009 "Fundamental Causes" of Health Disparities: A Comparative Analysis of Canada and the United States. *International Sociology* 2009 24:93-113.

Wilson, Barbara M.

- 1977 *Ontario and the First World War 1914-1918: A Collection of Documents*. Toronto: University of Toronto Press.

Wilson, Nick, Jane Oliver, Geoff Rice, Jennifer A. Summers, Michael G. Baker, Michael Waller, and G. Dennis Shanks

- 2014 Age-Specific Mortality During the 1918-19 Influenza Pandemic and Possible Relationship to the 1889-92 Influenza Pandemic. *Journal of Infectious Diseases* 2010(6):993-995.

Winternitz, M.C., Isabel Watson, and Frank McNamara

- 1920 *The Pathology of Influenza*. New Haven: Yale University Press.

Wirgman, C.W.

- 1918 An "Influenza" Outbreak. *Lancet* 4958 (7 September 1918):324-325.

World Health Organization

- 2014 Tuberculosis. Electronic Document, <http://www.who.int/mediacentre/factsheets/fs104/en/>, accessed July 2, 2014.

Worobey, Michael, Guan-Zhu Han, and Andrew Rambaut

- 2014 Genesis and Pathogenesis of the 1918 Pandemic H1N1 Influenza A Virus. *PNAS* 111(22):8107-8112).

Wrammert, J., K. Smith, J. Miller, W.A. Langley, K. Kokko, C. Larsen, N.Y. Zheng, I. Mays, L. Garman, C. Helms, J. James, G.M. Air, J.D. Capra, R. Ahmed, and P.C. Wilson

- 2008 Rapid Cloning of High-Affinity Human Monoclonal Antibodies Against Influenza Virus. *Nature* 453(7195):667-71.

Wrigley, E. A.

- 1973 Introduction. *In Identifying People in the Past*. E. A. Wrigley, ed. Pp. 1-16. London: Edward Arnold.

Wrigley, E. A., ed.

- 1966 *An Introduction to English Historical Demography: From the Sixteen to the Nineteenth Century*. New York: Basic Books, Inc., Publishers.

Wrigley, E. A. and R. S. Schofield

- 1973 Nominal Record Linkage by Computer and the Logic of Family Reconstitution. *In Identifying People in the Past*. E. A. Wrigley, ed. Pp. 64-101. London: Edward Arnold.

Wrigley, E. A., R. S. Davies, J. E. Oeppen, and R. S. Schofield

- 1997 *English Population History from Family Reconstitution*. Cambridge: Cambridge University Press.

Yang, Wan, Elisaveta Petkova, Jeffrey Shaman

- 2013 The 1918 Influenza Pandemic in New York City: Age-Specific Timing, Mortality, and Transmission Dynamics. *Influenza and Other Respiratory Viruses* doi: 10.1111/irv.12217

Young, George S.

- 1919 The Recent Epidemic of Pneumonia – Bedside Findings and Some Inferences. *Canadian Medical Association Journal* 9(5):421-426.

Zarit, Steven H., Leonard I. Pearlin, and Jon Hendricks, eds.

- 2005 Health Inequalities Across the Life Course. *The Journals of Gerontology: SERIES B. Psychological Sciences and Social Sciences* 60B: Special Issue II:5-139.

Zaslow, Morris

- 1967 The Ontario Boundary Question. *In Profiles of a Province: Studies in the History of Ontario. A Collection of Essays Commissioned by the Ontario Historical Society to Commemorate the Centennial of Ontario.* Pp. 107-117. Toronto: Ontario Historical Society.

Zhang, Shenghai, Ping Yan, Brian Winchester, and Jun Wang

- 2010 Transmissibility of the 1918 Pandemic Influenza in Montreal and Winnipeg of Canada. *Influenza and Other Respiratory Viruses* 4(1):27-31.

Zylberman, Patrick

- 2003 Holocaust in a Holocaust: The Great War and the 1918 ‘Spanish’ Influenza Epidemic in France. *In The Spanish Influenza Pandemic of 1918-1919: New Perspectives*, H. Phillips and D. Killingray, eds. Pp. 191-201. New York: Routledge.

Appendices

Appendix A: Records Linkage Project Instructions to Research Assistants

Purpose

This research is examining mortality surrounding the influenza pandemic in 1918 in Ontario. I am interested in determining two things: 1) the effect of the 1890 flu on death in 1918, through either *in utero* or early life exposure, and 2) the effect of socioeconomic circumstances over the life course on death in 1918. Although age of death is provided in the death records, it is not specific enough to determine timing of exposure to the 1890 flu, which was at its peak of virulence only in January 1890. It is therefore necessary to obtain the exact date of birth from other historical records. The socioeconomic status throughout the lifecourse must be obtained from records taken at various points between birth and death.

Data:

Main Source

The source of data on which this project is based is the Registered Death Records for the Province of Ontario, from September to December 1918. This epidemic reached Ontario at the very end of September 1918 and had decreased in virulence by December. These records have been transcribed and provided by the International Infectious Disease Data Archive (IIDDA) run by Dr. David Earn of the Department of Mathematics and Statistics at McMaster University.

Sources to be linked to

The three main websites that are used are:

1. www.ancestry.ca.
2. <http://automatedgenealogy.com/>
3. www.collectionscanada.gc.ca

- a. <http://www.collectionscanada.gc.ca/databases/cef/001042-100.01-e.php>

At these sites you will be looking for:

1. Birth Records
2. Marriage Records
3. Death Records
4. Attestation Records
5. 1901 Census
6. 1911 Census
7. 1891 Census (if necessary) (also if necessary, the 1916 census of the Prairie Provinces)
8. Misc. Sources that can provide useful information
 - a. Rootsweb
 - b. Genweb
 - c. Ontario Genealogical Society
 - d. Family Trees
 - e. Obituaries
 - f. <http://www.canadiangreatwarproject.com/>
 - g. <http://www.ontarioroots.com/> (to locate individuals in the census using their address)
 - h. Toronto city directories (such as from 1918, found at: http://ia600201.us.archive.org/load_djvu_applet.php?file=11/items/torontodirec191800midiuoft/torontodirec191800midiuoft.djvu)

Detailed Description of Sources:

1. **Birth Records:** These are found at ancestry.ca. The best way to look for these is through the search function. I have found that the easiest way to locate people is to start with their names and the names of their parents. Keep in mind that the age at death may be off by a few years and the spellings of both the names of the parents and the individual may be variants (or, they may be listed by their middle names). Adding more information to the search may help, such as location or

year. If you cannot find an individual, but you think you know place of birth and the day that they were born (for example, from the 1901 census), you can search for the birth records for the county. If, you know a person was born in Toronto, and suspect that they were born in March of 1890, you can locate the records for York for 1890 and do a manual search of the records picking out Toronto, then March.

2. Marriage Records: These are also on ancestry.ca. They can be searched by either the bride or the groom and also typing in the names of the parents helps. These come in different formats as the years change, and you may pull up either the marriage certificate itself or the registration of the registrar general. Both of these provide the information necessary, so just select what is needed from the record and put it into the database, where you think it should go.
3. Death Records: If there is something in the death record from the IIDDA that you are not certain about, you can look up the individual record itself on ancestry.ca to double check.
4. Attestation Records: These are the forms that people filled out when they either enlisted or were conscripted into the First World War. They are kept by Libraries and Archives Canada at <http://www.collectionscanada.gc.ca/databases/cef/001042-100.01-e.php>. These are searched by name only, but you can use the wildcard of (*). For example. If you know someone's name is Francis Thompson, but sometimes it is spelled Frances Thomson, you could search for Franc*s Thom*son, or Fran* Thom* or any such variation. For uncommon names, it is sometimes best to search by the surname alone, then scroll through the results to find the individual you are looking for. Very few individuals in this sample have attestation records (and even less of the women), so do not be overly concerned if you cannot find someone.
 - a. The Canadian Great War Project (CGWP) can also be quite useful, at <http://www.canadiangreatwarproject.com/searches/soldierSearch.asp>. This can help you to link soldiers (only those who died) to their attestation records, through regimental numbers. The CGWP lists date of death, if this matches the date of death in the death record, you can be fairly certain

of the link to the attestation paper with the same regimental number and same name.

5. 1901 Census: These records can be found at ancestry.ca, but I prefer to use automatedgenealogy.com, for both the 1901 and 1911 censuses. These are searched by surname, then by first name, but can filtered by sex and age and sorted by location. You can also search by location (ward and sub-district) if known, and can look up what electoral district a specific address is in by using <http://www.ontarioroots.com/> (for Toronto only). The records in ancestry.ca do not have as good of a zoom function (it can be hard to read handwriting), but the census records may come up when doing the general search for the individual.
6. 1911 Census: Same as for the 1901 census.
7. 1891 Census (also 1916 census of the Prairie Provinces): Only use for those individuals for whom you are uncertain of their age. For example, if there is a discrepancy between the 1901 census and the birth record, and you are not sure if someone was born in 1888 or 1889, you can check to see how old they were in 1891. This census does not add any new information and many people in the sample had not been born in 1891, so there is no need to transcribe for every individual.
8. Miscellaneous Sources: It may be that there is an individual who you cannot find in the census or birth record, but you feel that you should be able to. In ancestry.ca, it is possible to search family trees, where you can link by parents or spouses names. Do not transcribe this information, but it can be useful to help you identify more information about the person, which can then be linked to the records of interest. For example, it is possible to do general Google searches, to use the cemetery finder at <http://ogs.andornot.com/CemeteryIndex.aspx>, to use the LDS database at <https://www.familysearch.org/>, or to look up family history discussions at [genforum](http://genforum.com) (although there is great potential here, I've actually found this source to be of little use).

Database

The database for this project is in Microsoft Access.

Procedure:

1. Select individual to link from the master file (either Toronto Deaths DAVID, or Ontario Deaths David). Look at the characteristics of the individual, useful things to link on can be name, sometimes day of death, place of birth/death/burial, occupation, marital status, name of father/mother, name/address of informant. If the individual is a married woman, pay special attention to her father's surname, in case it is different from her own. You will probably need to start with her maiden name, as most people who are married were not married by 1911 and are still in their parents' household at that time.
2. Select which piece of information you think most likely to link and start there. For example, if the individual was a soldier, it may be most useful to start with the Attestation papers or the Canadian Great War Project.
3. Once you have the record that you think is a match, fill out the information in the proper table in Access. For each table you will use the ID number from the master table and record it in the column DR_ID so that people can be linked at a later date.
4. You will then make your best judgement on the quality of the link (RL_RANK), whether you are Positive of the link, whether you think it is Likely, or whether it is only Maybe a link (for instances where you have a hunch that the records relate to the same person. You are not certain, but are certain enough that you do not feel comfortable not recording it at all). You will also be asked to record a score to the records linkage (RL_SCORE). This is not something to worry about and should be done quite quickly – I am not so much interested in the actual value of the number, just whether it is relatively high or low. The guidelines to determine a score are:
 - a. An exact match receives 10 points
 - b. A close match (for example, the correct name but different spelling or a short-form, or an opposite name order) receives 5 points.

- c. Missing data should not be scored (0 points)
- d. 5 points are to be subtracted for data that is wrong (for example, mother's name is wrong, year of birth wrong).
- e. These data are matched according to what is listed in the death record. However, since multiple records are being utilized, information that is not recorded in the death records, but is present in two other linked records will receive a score of 5 (for example, name of spouse's mother in the marriage record and name of resident mother-in-law in the 1911 census).

NB: The score an individual record receives will be at least partially a result of the order in which the records are accessed. This is fine and to be expected. Do not go back once you have analyzed all records to revise the score, it is not meant to be that specific (should take only about 10-30 seconds to devise score).

5. Once you have either linked a person or determined that they cannot be linked, either put a YES or a NO in the LINKED column of the master table and check which records you have found in the following boxes.
6. Then, open the table Study AAD (stands for age-at-death). Locate the individual by their ID number. Then, using the records you have just found, enter their date of birth in the columns DOB_YEAR, DOB_MOS, and DOB_DAY. It may be that the dates are not the same and you have to use your best judgement. In these cases, I tend to use the birth record first, then the attestation records, then the 1901 census, then the 1911. I have found that, in general, the 1911 census tends to be off by about one year. To account for this, at the end of each record in the Study AAD table, you should check the box either All_Ages_Same or Not_All_Agree so that I know there was a potential problem. It may be that all you have is a year, if so, that is all you can record. Also, for individuals that you cannot link, do not put anything into the Study AAD table.
7. Finally, in the last box in the table, LINKED_BY, record your initials.

8. On all tables there is a Comments_PROJECT column where you can record anything about that record that you think should be noted.
- When there is a question mark in the records, record as ?. However, when there is something that you are unsure of, but there is writing in the record, please write a question mark in brackets (?).

Useful Information

Sometimes it can be useful to have a rough estimation of what year an individual ‘should’ have been born in. You may find this chart useful as a guide:

Age at Death (in 1918)	Year of Birth	Age in 1911 Census	Age in 1901 Census
23	1894/1895	15/16	5/6
24	1893/1894	16/17	6/7
25	1892/1893	17/18	7/8
26	1891/1892	18/19	8/9
27	1890/1891	19/20	9/10
28	1889/1890	20/21	10/11
29	1888/1889	21/22	11/12
30	1887/1888	22/23	12/13
31	1886/1887	23/24	13/14
32	1885/1886	24/25	14/15
33	1884/1885	25/26	15/16
34	1883/1884	26/27	16/17
35	1882/1883	27/28	17/18

Appendix B: Ontario Death Record, 1918.

Table B0.1 - Death Records, 1918

The diagram illustrates the structure of an Ontario Death Record form from 1918. It is divided into two main sections: **DEATHS** and **Physician's Return of Death**.

DEATHS Section:

- Header: 148, DEATHS, County of _____, Division of _____
- Fields: SURNAME of Deceased, Christian Name, Sex, Age, Date of Death, PLACE OF BIRTH (Place of Death, City, Town, Village, or Concession and Lot), Occupation, Single, Widowed or Divorced, Name of Father, Maiden Name of Mother, Cause of Death, if known, Name of Physician who attended Deceased, Name of Informant, Address, Date of Return.

Physician's Return of Death Section:

- Fields: Surname of Deceased, Christian Name, Date of Death, DISEASE CAUSING DEATH, Duration, Immediate Cause of Death, Duration, Physician's Name, Address, Date of Return, Remarks.

Physician's Return of Death Section (Side Box):

- Fields: SURNAME of Deceased, Christian Name, Sex, Age, Date of Death, PLACE OF BIRTH (Place of Death, City, Town, Village, or Concession and Lot), Occupation, Single, Widowed or Divorced, Name of Father, Maiden Name of Mother, Cause of Death, if known, Name of Physician who attended Deceased, Name of Informant, Address, Date of Return.

Arrows indicate the flow of information: from the 'DEATHS' section to the 'Physician's Return of Death' section, and from the 'Physician's Return of Death' section back to the 'DEATHS' section.

Source: Archives of Ontario MS 935, Reel 261. Figure from Hallman (2009:126).

Appendix C: Age-Specific Population Totals in 1918.

These were created using Waring's 2-point formula and based on the 1911 and 1921 age-specific population totals created using Sprague's Fifth Difference Equation (Dominion of Canada 1925, Judson and Popoff 2004).

	Male	Female	Total
Under 1	28,379.99	27,343.09	55,723.08
1	29,146.67	28,227.24	57,373.91
2	29,653.37	28,826.19	58,479.56
3	29,928.06	29,170.75	59,098.81
4	29,998.72	29,291.72	59,290.44
5	29,893.31	29,219.94	59,113.25
6	29,639.82	28,986.21	58,626.02
7	29,266.19	28,621.34	57,887.53
8	28,800.42	28,156.15	56,956.57
9	28,270.46	27,621.46	55,891.92
10	27,666.94	27,013.24	54,680.18
11	26,980.48	26,327.49	53,307.97
12	26,425.80	25,769.16	52,194.96
13	26,105.57	25,438.72	51,544.29
14	25,935.71	25,262.49	51,198.20
15	25,738.24	25,053.90	50,792.14
16	25,559.29	24,835.30	50,394.59
17	25,336.07	24,679.71	50,015.78
18	25,022.88	24,599.99	49,622.87
19	24,664.81	24,566.81	49,231.62
20	24,331.46	24,534.85	48,866.31
21	23,983.50	24,509.44	48,492.94
22	23,761.25	24,439.91	48,201.16
23	23,733.38	24,295.14	48,028.51
24	23,818.82	24,090.67	47,909.49
25	23,888.05	23,888.85	47,776.91
26	23,991.17	23,696.06	47,687.23
27	23,930.31	23,405.74	47,336.05
28	23,601.62	22,977.15	46,578.77
29	23,107.95	22,463.61	45,571.55
30	22,629.25	21,941.22	44,570.47
31	22,097.52	21,373.18	43,470.69
32	21,725.81	20,924.79	42,650.59
33	21,627.98	20,679.12	42,307.09
34	21,683.65	20,550.81	42,234.46
35	21,683.59	20,385.83	42,069.42
36	21,699.62	20,232.28	41,931.89
37	21,493.59	19,943.88	41,437.47

38	20,935.48	19,437.87	40,373.36
39	20,153.62	18,793.44	38,947.06
40	19,401.79	18,174.41	37,576.21
41	18,623.82	17,550.28	36,174.10
42	17,940.53	16,965.68	34,906.21
43	17,435.91	16,460.07	33,895.98
44	17,046.44	16,010.27	33,056.71
45	16,616.09	15,539.38	32,155.47
46	16,165.86	15,045.65	31,211.52
47	15,740.68	14,624.23	30,364.92
48	15,344.01	14,309.29	29,653.30
49	14,959.35	14,054.25	29,013.60
50	14,584.41	13,798.47	28,382.88
51	14,240.81	13,578.18	27,818.99
52	13,793.29	13,223.47	27,016.76
53	13,181.06	12,651.35	25,832.41
54	12,474.04	11,950.62	24,424.66
55	11,785.48	11,274.41	23,059.89
56	11,071.76	10,569.70	21,641.46
57	10,499.97	10,015.96	20,515.93
58	10,158.02	9,710.77	19,868.79
59	9,958.16	9,557.97	19,516.13
60	9,734.37	9,376.25	19,110.62
61	9,531.34	9,216.80	18,748.14
62	9,231.35	8,944.24	18,175.59
63	8,762.75	8,477.73	17,240.48
64	8,189.29	7,891.89	16,081.17
65	7,645.77	7,338.51	14,984.28
66	7,104.55	6,782.86	13,887.41
67	6,598.94	6,286.65	12,885.59
68	6,158.82	5,894.47	12,053.29
69	5,763.32	5,570.51	11,333.83
70	5,362.81	5,236.42	10,599.22
71	4,968.57	4,910.28	9,878.85
72	4,581.66	4,576.20	9,157.87
73	4,198.11	4,219.22	8,417.33
74	3,822.46	3,852.68	7,675.14
75	3,463.28	3,504.69	6,967.97
76	3,118.90	3,169.38	6,288.28
77	2,794.30	2,854.20	5,648.50
78	2,492.86	2,565.60	5,058.45
79	2,212.37	2,298.93	4,511.30
80	1,947.21	2,043.91	3,991.12
81	1,698.40	1,803.54	3,501.94
82	1,467.63	1,574.88	3,042.51
83	1,255.13	1,355.75	2,610.88

84	1,060.62	1,148.92	2,209.54
85	883.49	960.13	1,843.62
86	723.76	789.18	1,512.95
87	581.48	635.87	1,217.35
88	456.55	500.99	957.54
89	348.92	385.33	734.24
90	258.49	289.67	548.16
91	185.19	214.80	400.00
92	128.96	161.53	290.48
93	89.70	130.62	220.33
94	67.36	122.88	190.23
TOTAL	1,424,200.60	1,381,724.60	2,805,925.20

Appendix D: Descriptive Statistics for Linkages

Table D0.2 - Descriptive Statistics of Records Linked and Not Linked to a Birth Record.

		Birth Record		No Birth Record		χ^2	df	p
Demographic Features		N	%	N	%			
Total		2,079	100.0	1,237	100.0			
Sex	M	1,080	62.0	663	38.0	0.85	1	.358
	F	999	63.5	574	36.5			
Declared Age	Mode	26		28		1.56	3314	.12
	Median	28		28				
	Mean	28.6		28.8				
	SD	3.6		3.5				
Marital Status	Single	737	36.2	405	35.2	0.33	1	.567
	Ever-Married	1,299	63.8	746	64.8			
Indigenous	Y	2	0.1	80	6.5	130.53	1	<.001*
	N	2,077	99.9	1,157	93.5			
Soldier	Y	68	3.3	47	3.8	0.65	1	.42
	N	2,011	96.7	1,190	93.2			
Itinerant Occupation	Y	744	46.0	435	48.1	8.15	1	0.004*
	N ^b	873	54.0	470	51.9			
Conditions of Death								
Flu	Y	1,773	85.3	1,043	84.3	0.56	1	.45
	N	306	14.7	194	15.7			
Tuberculosis	Y	121	5.8	79	6.4	0.44	1	.51
	N	1,958	94.2	1158	93.6			
Urban	Y	1,071	51.5	629	50.9	0.14	1	.71
	N	1,008	48.5	608	49.2			
Toronto	Y	291	14.0	196	15.8	2.113	1	.15
	N	1,788	86.0	1,041	84.2			
Institution	Y	502	24.2	362	29.3	10.55	1	.001*
	N	1,577	75.9	875	70.7			
Region	North	118	5.7	178	14.4	72.44	1	<.001*
	Not-North	1,961	94.3	1,059	85.6			
State of Death Record								
Occupation	Y	1,167	56.1	662	53.5	2.15	1	.14
	N	912	43.9	575	46.5			
Father's Name	Y	1,889	90.9	932	75.3	147.1	1	<.001*
	N	190	9.2	305	25.7			
Mother's Name	Y	1,689	81.2	765	61.8	151.7	1	<.001*
	N	390	18.8	472	38.2			

*P<0.5

Table D0.3 - Descriptive Statistics of Records Linked and Not Linked to the 1901 Census.

		1901 Record		No 1901 Record		χ^2	df	p
Demographic Features		N	%	N	%			
Total		2,737	100.0	579	100.0			
Sex	M	1,432	52.3	311	53.7	0.37	1	.54
	F	1,305	47.7	268	46.3			
Declared Age	Mode	28		30		1.11	3314	.002*
	Median	28		29				
	Mean	28.6		29.1				
	SD	3.5		3.6				
Marital Status	Single	972	36.2	170	33.7	1.23	1	.27
	Ever-Married	1,710	63.8	335	66.3			
Indigenous	Y	46	1.7	36	6.2	40.79	1	<.001*
	N	2,691	98.3	543	93.8			
Soldier	Y	90	3.3	25	4.3	1.5	1	.22
	N	2,647	96.7	554	95.7			
Itinerant Occupation	Y	977	45.5	237	63.0	39.3	1	<.001*
	N ^b	1,169	54.5	139	37.0			
Conditions of Death								
Flu	Y	2334	85.3	482	83.3	1.54	1	.22
	N	403	14.7	97	16.8			
Tuberculosis	Y	164	6.0	36	6.2	0.04	1	.84
	N	2,573	94.0	543	93.8			
Urban	Y	1,374	49.8	326	56.3	7.1	1	.008*
	N	1,363	50.2	253	43.7			
Toronto	Y	376	13.7	111	19.2	11.26	1	.001*
	N	2,361	86.3	468	80.8			
Institution	Y	657	24.0	207	35.8	34.23	1	<.001*
	N	2,080	76.0	372	64.3			
Region	North	156	5.7	140	24.2	200.76	1	<.001*
	Not-North	2,581	94.3	439	75.8			
State of Death Record								
Occupation	Y	1,547	56.5	282	48.7	11.81	1	.001*
	N	1,190	43.5	297	51.3			
Father's Name	Y	2,496	91.2	325	56.1	462.67	1	<.001*
	N	241	8.8	254	43.9			
Mother's Name	Y	2,210	80.8	244	42.1	370.21	1	<.001*
	N	527	19.3	335	57.9			

*P<0.5

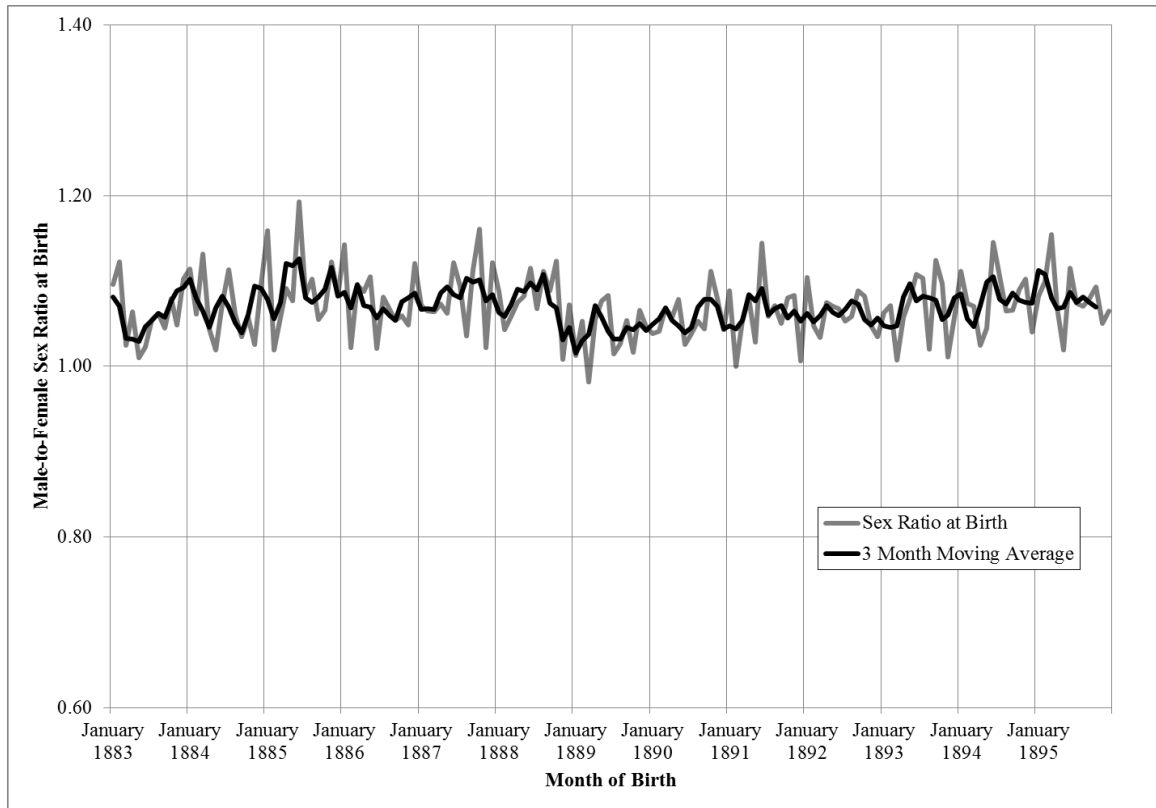
Table D0.4 - Descriptive Statistics of Records Linked to a Birth Record, the 1901 and 1911 Censuses, and those Not Linked to all 3 Records.

		All 3 Records		At Least One Missing		χ^2	df	p
Demographic Features		N	%	N	%			
Total		1,667	100.0	1,649	100.0			
Sex	M	844	50.6	899	54.5	5.03	1	.02*
	F	823	49.4	750	45.5			
Declared Age	Mode	25		28		4.94	3314	<0.01*
	Median	28		29				
	Mean	28.4		29.0				
	SD	3.5		3.5				
Marital Status	Single	610	37.3	532	34.3	3.09	1	.08**
	Ever-Married	1,026	62.7	1,019	65.7			
Indigenous	Y	1	.1	81	4.9	80.92	1	<.001*
	N	1,666	99.9	1,568	95.3			
Soldier	Y	42	2.5	73	4.4	9.0	1	.003*
	N	1,625	97.5	1,576	95.6			
Itinerant Occupation	Y	558	42.7	656	54.0	32.64	1	<.001*
	N ^b	750	57.3	558	46.0			
Conditions of Death								
Flu	Y	1,415	84.9	1,401	85.0	0.004	1	.95
	N	252	15.1	248	15.0			
Tuberculosis	Y	96	5.8	104	6.3	0.44	1	.51
	N	1,571	94.2	1,545	93.7			
Urban	Y	825	49.5	875	53.1	4.23	1	.04*
	N	842	50.5	774	46.9			
Toronto	Y	214	12.8	273	16.6	9.15	1	.002*
	N	1,453	87.2	1,376	83.4			
Institution	Y	376	22.6	488	34.0	21.31	1	<.001*
	N	1,291	77.4	1,161	66.1			
Region	North	86	5.2	210	12.7	58.53	1	<.001*
	Not-North	1,581	94.8	1,439	87.3			
State of Death Record								
Occupation	Y	926	55.6	903	54.8	.21	1	.65
	N	741	44.5	746	45.2			
Father's Name	Y	1,522	91.3	1,299	78.8	102.43	1	<.001*
	N	145	8.7	350	21.2			
Mother's Name	Y	1,368	82.1	1,086	65.9	113.17	1	<.001*
	N	299	17.9	563	34.1			

*P<0.5

Appendix E: Male-to Female Sex-Ratios at Birth, January 1883-December 1885

Table E0.5 – Male-to-Female Sex Ratio at Birth, January, 1883, to December, 1885.



Source: Calculated from Legislative Assembly of Ontario (1883-1898).

Curriculum Vitae

Name: Stacey A Hallman

Post-Secondary Education and Degrees:

University of Victoria
Victoria, British Columbia, Canada
2003-2007 B.A. Hons.

McMaster University
Hamilton, Ontario, Canada
2007-2009 M.A.

The University of Western Ontario
London, Ontario, Canada
2009-2015 Ph.D.

Honours and Awards: (External)

Ontario Graduate Scholarship
2009-2011; 2008-2009 & 2011-2012 (declined)

Social Science and Humanities Research Council (SSHRC)
Joseph Armand Bombardier Canada Graduate Scholarship
2008-2009

Social Science and Humanities Research Council (SSHRC)
Doctoral Fellowship
2011-2013

Honours and Awards: (Internal)

University of Victoria:
President's Entrance Scholarship, 2003
President's Scholarship, 2005
Faculty Scholarship, 2005, 2006
Alumni Undergraduate Scholarship, 2006
Dean's List, 2007

McMaster University
Entrance Scholarship, 2007
Ontario Graduate Fellowship, 2009

University of Western Ontario
Entrance Scholarship, 2009

Related Work Experience Sessional Lecturer: Statistics for Sociology, Research Methods in Sociology
The University of Western Ontario
2012-2014

Publications:

Hallman, Stacey and Alain Gagnon

- 2014 Does Exposure to Influenza Very Early in Life Affect Mortality Risk During a Subsequent Outbreak? The 1890 and 1918 Pandemics in Canada. *In Are Modern Environments Bad for Human Health? Revisiting the Second Epidemiological Transition*, Molly K. Zuckerman, ed. Pp. 123-138. Toronto: Wiley-Blackwell.

Gagnon, A., M. S. Miller, S. Hallman, R. Bourbeau, D. A. Herring, D. J. D. Earn, and J. Madrenas.

- 2013 Age-Specific Mortality during the 1918 Influenza Pandemic: Unraveling the Mystery of High Young Adult Mortality. *PLoS ONE* 8(8): e69586

Holt, Sarah, S. Hallman, M. Powell, and M. Langley

- 2013 The ‘Other’ Burials at Torre de Palma: Childhood as Special Death in a Medieval Portuguese Site”. *In Tracing Childhood: Bioarchaeological Investigations of Early Lives in Antiquity*, edited by J. Thompson, M. Alfonso-Durruty, and J. Crandall. Gainesville: University Press of Florida. Pp. 75-98

Hallman, Stacey

- 2012 An Exploration of the Effects of Pandemic Influenza on Infant Mortality in Toronto, 1917–1921. *Canadian Studies in Population* 39(3-4):35-48