

INFANT MORTALITY AND INFLUENZA IN TORONTO, 1917-1921

THE EFFECT OF PANDEMIC INFLUENZA
ON INFANT MORTALITY IN
TORONTO, ONTARIO, 1917-1921

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ABSTRACT

The 1918 influenza pandemic was not a disease of infants. Most research on this disease has focused on young adults, whose excess mortality was most alarming. However, as infant mortality rates are a measure of social health, an analysis of infant death provides another avenue for exploring the declining environmental conditions due to this epidemic. This study investigates infant mortality in Toronto, Canada, from September to December 1918, through the Registered Death Records of the Province of Ontario. A comparison of infant death in 1918 to surrounding years (1917-1921) revealed that infant mortality rates remained relatively stable. However, there were changes in the infant mortality profile. Deaths from influenza did increase slightly and were early for the typical airborne disease season. While infants did not suffer from the drastic rise in excess mortality that was seen in adults, the epidemic altered *who* was dying and *when*. Although a community may be greatly strained by an epidemic and stressful social conditions, the infant mortality rate may be more representative of long-term social stress rather than acute, intensive crises.

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Chapter 1

INTRODUCTION

The influenza epidemic of 1918 represented a period of “crisis mortality” in Ontario and around the world, “defined as unusually high mortalities arising from a common, unusual, causal factor operating for a limited amount of time across a given geographical area” (Bouckaert 1989:218). This epidemic is noted for its unusual mortality profile. Although those most often the victims of infectious diseases were affected by the epidemic (infants and the elderly), the striking anomaly in the pattern of death occurred in the increased mortality of young adults between 20 and 40 years old (City of Toronto Archives, Fonds 200, Series 365, File 21; Harder 1918; Crosby 1976; Pettigrew 1983; Taubenberger 2003). This has been variously attributed to the particular pathology of the disease but also to the global environment in 1918 (Lancet 1918b; Taubenberger et al 2000; Oxford et al 2002; García-Sastre and Whitley 2006). At the close of the First World War, many young people were travelling around the world, to fields of battle, and home to their families. These young men and women had been exposed to many people from around the world, opening a pathway for the spread of infections. Further, the soldiers were weakened from the unhealthy conditions of the war, from injuries, psychological trauma, and diseases such as tuberculosis. The families to whom they returned were stressed from years of war-time rationing and the distress of war-time events (recognizing, as Handwerker (1990) does that “virtually any demographic change thus alters both the physical and mental health of a community” [1990:320]; Lancet 1918b).

Scholarship on the 1918 influenza is slowly increasing, although few books are dedicated to the Canadian situation (including Pettigrew 1983; Herring 2005 for Hamilton; and Jones 2007 for Winnipeg. For articles see, Herring 1993; Sattenspiel and Herring 1998, 2003; Jones 2005; Herring and Sattenspiel 2007; MacDougall 2007). Contemporary articles in the Journal of the Canadian Medical Association and later ones from the Canadian Public Health Journal briefly describe the situation in Toronto and Montreal, focusing mainly on the outbreaks of influenza among soldiers, the symptoms of the disease, and the debate over the identification of the disease as influenza and the real role of Pfeiffer's bacillus, *Bacillus influenzae* (Boucher 1918, CMAJ 1918c, McCullough 1918, Oertel 1919, Robertson 1919, Young 1919, Hare 1937). More modern works mention the Canadian situation intermixed with global accounts (Collier 1974, Crosby 1989) or the flu in passing while addressing other topics (MacDougall 1990, Miller 2002).

Rosenberg explains that “since at least the eighteenth century, physicians and social commentators have used the difference between ‘normal’ and extraordinary levels of sickness as an implicit indictment of pathogenic environmental circumstances” (1989:12). The 1918 influenza pandemic fits this criterion since, in Toronto alone, it has been estimated that over half of the city had the flu in October 1918 (Miller 1999). Globally, between 40 and 100 million people died (Johnson 2003). Yet, as known from contemporary sources, this epidemic did not strike the population equally (The Globe 1918n, The Globe 1918w, Harder 1918; Winternitz et al. 1920). The aggregate published

data, however, give no impression of how social, environmental, and biological factors may have influenced relative risk.

The analysis of the mortality of infants in Toronto during this epidemic, presented in this thesis, is a novel means to approach the situation in Canada in 1918. Asking questions of a group of individuals typically ignored in terms of this illness has the potential to reveal hidden dimensions of this epidemic. This approach to infant mortality in Toronto addresses the question that Nancy Scheper Hughes believes should be asked of all critically interpretive research: "What is being hidden from view in the official statistics" (1997:220)? Although her appeal is to contemporary anthropological demographers, this research examines information that is masked in the official reports of the 1918 flu in Toronto. Further, by describing the historical as well as biomedical background of the epidemic, and suggesting further research into structural inequalities, I place this research in a bio-cultural political-economic perspective, one which focuses on "historically specific social forces, relations, and processes surrounding sickness and health care" (Morsy 1990). This is accomplished by investigating hypotheses of equal rates of infection and death within the historically-bounded context of early twentieth-century Toronto.

To determine the pattern of infant mortality in Toronto from 1917 to 1921 and ascertain whether the 1918 influenza epidemic affected it in any way, I investigate six different aspects of infant death. First, I look at the overall infant mortality rate, both yearly and monthly, to determine the death rates. I then examine the sex-ratio at death to see if the influenza epidemic targeted one sex over the other. The average age at death is

studied for the same reason: to discover if either neonatal (< 1 month) or postneonatal ($\geq$ 1 month) infants were at higher risk. Causes of death are investigated to determine whether influenza did affect infants and to find out if deaths from other causes decreased during the epidemic. The stillbirth rate is analyzed to determine if there is a relationship between stillbirths and neonatal deaths. Finally, the link between maternal and infant mortality is considered to explore another hidden aspect of the effect of the epidemic on families in Toronto.

To explore these issues this thesis uses transcription of the death records for Toronto to determine the infant mortality rate for the five year period, 1917-1921 (Archives of Ontario MS 935, Reels 228-229, 238-240, 251-252, 261-262, 273-274). The purpose of this approach is to explore the patterns of infant death beyond what was reported by the Local Board of Health. Studying the five year period surrounding the epidemic, which includes both pre- and post-war eras, places the experience of 1918 in a wider context and establishes a baseline for comparison. The mortality rates based on the registered infant deaths are tested statistically to determine whether the 1918 influenza epidemic increased infant deaths significantly or if the increase was within the range of normal variation.

Herring and Sattenspiel (2003) note that “while mortality rates from epidemics are useful approximations of *community* devastation, they tell nothing of the social disruption to individual *families* because of illness or death” (2003:170). Further they note that “some families . . . continued to thrive and raise children to maturity, while others in their midst lost all or nearly everyone to the 1918 flu. This is what people remember; this is

what has meaning for them, not the crude mortality rates that we students of the pandemic continue to generate” (2003:171). While this thesis presents infant mortality rates to establish a picture of child loss in Toronto, I also explore the impact on families. This is accomplished by linking infants and adults who died using the names listed in the official death records. By linking the deaths of parents to their children in both 1917 and 1918, I investigate whether 1918 brought an increase in the numbers of families losing both an infant and a mother. Recognition of the experiences of families in the epidemic brings individuals and families back into the epidemiological profile and adds a new dimension, beyond mere aggregations of deaths, to our understanding of the broader social impact of the 1918 influenza outbreaks in Toronto. The Globe newspaper, the Monthly Reports of the Medical Officer of Health for the city of Toronto, and numerous secondary sources add background context to help give a historical understanding to the 1918 influenza epidemic.

As was recognized at the time, influenza did not act alone. Death was a product of infection with influenza as well as *Streptococcus* or *Pneumococcus*, and was hastened by previous tuberculosis infection or pregnancy (CMAJ 1918b, Oertel 1919, Young 1919). The relationship between people, places, and pathogens is important for understanding this epidemic. Young adults between 20 and 40 were at the highest risk of dying (Harder 1918, Taubenberger 2003), while the effects on infants and the elderly were much less pronounced (Crosby 1989). However, these age groups were not isolated entities in society; infants are often under the primary care of adults aged 20 to 40 and are directly affected by the death of their parents or care givers (Pavard et al 2005). This

thesis treats the influenza epidemic of 1918 as part of a syndemic, “a set of intertwined and mutually enhancing epidemics involving disease interactions at the biological level that develop and are sustained in a community/population *because of harmful social conditions and injurious social connections*” (Singer and Clair 2003:429). The interplay between influenza, pneumonia, diarrheal diseases of the summer, the weather, and social stress will be considered to explain the experience of infants during the flu epidemic. This thesis also uses the infant mortality rate of Toronto as a means of understanding the social implications of this pandemic.

Through most of the twentieth century, infant mortality has been used as an indicator of social health (Armstrong 1986). When writing of the effects of the first world war on infant death, *The Lancet*, argued “that the infant mortality-rate is an index of the first importance from a broad medical, social, and economic point of view is not in doubt” (1918a:223). Although *The Lancet* was not sure of what, exactly, the index represented, it is now believed to be a “primary measure of demographic well-being” (Galley and Shelton 2001:65). As infants require the constant care of adults for their welfare, they are susceptible to even slight changes in their environment (Moffat and Herring 1999). Infant mortality is known to be affected by the socio-economic status of caregivers through access to resources and availability of care; religious practices concerning treatment and spacing of children; age, marital status, and reproductive history of the parents; and, the sanitary environment including crowding in houses, number of siblings, and access to clean food and water (Reid 2001, Sawchuk et al 2002, Mercier 2006). As argued by Matteson and colleagues “low economic status and

truncated education lower the likelihood of receiving good nutrition, securing adequate housing, receiving adequate maternal and prenatal health care and developing positive health-related attitudes and life styles” (Matteson et al 1998:1842). The characteristics of the infant also contribute to the risk of infant mortality as male children, low-birth weight babies, and infants from multiple births are known to be at greater risk of death (Reid 2001, Drevenstedt et al 2008). Not only does the infant mortality rate give evidence as to the social condition of infants, it also “frequently serves as a good predictor of the overall life expectancy of a population, as well as the general social well-being” (Sawchuk et al 2002:403-404, Gortmaker and Wise 1997). Therefore, during a pandemic which killed between 40 and 100 million people throughout the world and an estimated 50 000 people in Canada, an increase in infant mortality would be expected to mirror this disturbance to the “general social well-being” (Johnson 2003, Phillips and Killingray 2003).

Knowledge of the rate of infant death in Toronto during the epidemic may give a greater understanding of the level of social disruption experienced by city residents. An increase in the rate of infant mortality resulting from the flu would suggest that this measure of “community health and well-being” applies not only to society under normal conditions, but also during the unusual situation resulting from an epidemic. If infant mortality did not increase, then this would imply that further research is necessary in order to determine how infants managed to avoid the disease and which societal mechanisms may have buffered them from its effects. Understanding how and why infants died during the 1918 influenza pandemic may help contemporary public health officials to mitigate the effects of future pandemics. As asserted by McKeown,

“[influenza] is the only epidemic infectious disease which, in technologically advanced countries, presents today a threat comparable to that experience in early centuries from diseases such as plague and typhus” (1976:83). The flu is not a disease of the past and any knowledge gleaned from historical records should be used to help predict and combat outbreaks in the future.

Infant mortality is represented in a rate as the number of deaths per 1000 live births (Mausner and Bahn 1974). It is divided into neonatal and postneonatal age categories, due to the different causation of death at different ages. The neonatal age group represents infants who died in the first 28 days of life (<28 days) and deaths are generally thought to be caused by “endogenous” biological causes such as “perinatal infections, congenital malformations, and maternal conditions” (Matteson et al 1998:1845). Further, “infants suffering from congenital malformations, low birth weight, and prematurity are at particular risk from neonatal mortality and vulnerable to any physical condition of their mothers that affects their capacity to provide adequate milk and nurturing” (Reid 2001:213). Infants who died between the ages of 29 days and 365 days are classified as postneonatal deaths and are generally caused by “exogenous” environmental causes, such as “most types of infections, nutritional deficiencies, SIDS, violence, and other external causes” (Matteson et al 1998:1845). Although there is some overlap between endogenous and exogenous causes and age at death, generally deaths resulting from the conditions of pregnancy cluster in the first month of life (Knodel 1988, Matteson et al 1998). In terms of the 1918 influenza, epidemic-related neonatal deaths were likely caused by maternal infection with influenza that affected the developing fetus,

pre-term labour, or resulted from maternal-death related starvation. Postneonatal deaths due to the epidemic can include the effects of maternal infection, but were more likely to have resulted from direct infection with influenza and environmental degradation caused by the death or illness of the primary care-givers. According to Knodel (1988), seasonal variation in the patterning of deaths is a product of fluctuating postneonatal infant mortality because neonatal infant mortality remains somewhat stable throughout the year.

Despite seasonal variations in infant death, the 1918 influenza epidemic occurred in the midst of a declining infant mortality rate in Canada. In Ontario, infant mortality dropped from 136.4 deaths per 1000 live births in 1900 to 40.1 deaths in 1942 (McKinnon 1945:287).¹ This decline, however, was not consistent everywhere. Piva notes that in Toronto, infant mortality rose in the 1910s, from 141.4 per 1000 live births in 1902 to 179.7 deaths in 1909 (Piva 1979:123), the causes of which were attacked by a campaign of education and milk purification. McKinnon further explains that “it is perhaps noteworthy that there is no certain evidence of slackening of any of the rates of decline during . . . the war of 1914-1918” (1945:295). Generally, improvements in the infant mortality rate are due to reductions in postneonatal mortality. This is the result of enhancements in implementation of and access to health care, reduction in infectious diseases through vaccination and hospitalization, general advancements in the state of health and nutrition, and improvements in socio-economic status (Pequegnat 1938,

¹ Interestingly, McKinnon explains that “the lack of any great excess in morality [for infants under one year] in 1918 . . . may be noted as contrasted with the other age groups” (1945:288). For the period 1917-1921 the yearly infant mortality rates are presented as 92.4, 98.9, 95.6, 107.6, and 92.1 deaths per 1000 live births, respectively (1944:482).

Pharoah and Morris 1979, Starfield 1985). Specifically for Toronto, Pequegnat attributed the declines in infant mortality prior to 1925 as due to progress in milk pasteurization, water chlorination, and additional public health staff (Pequegnat 1938:479). Although the neonatal infant mortality rate declined more slowly than the total infant mortality rate through most of the twentieth century, it decreased due to technological advancements extending the length of life, increasing birth-weight, and widespread use of biomedical prenatal care (Chapin 1921, Van Ingen 1921, David and Siegel 1983, Knodel 1988, Gortmaker and Wise 1997).

The high rate of infant mortality in Toronto at the turn of the twentieth century was of concern to medical officials. Reports, such as those by Dr. Helen MacMurchy, were written about the causes and consequences of high infant mortality (Chapin 1921, Piva 1979, MacDougall 1990) and drives such as well baby clinics and milk stations were implemented throughout the city (City of Toronto Archives, Fonds 200, Series 365, Files 17-21, MacDougall 1990). Concerns about infant and child health were deemed especially relevant in the face of the low standard of health among recruits for both the Boer and the First World Wars (Dickin McGinnis 1981, MacDougall 1990). The link between poverty and infant mortality was known to MacMurchy (MacDougall 1990), and Mercier has found that in 1901, infant mortality in Toronto was also associated most significantly with religious affiliation (Mercier 2006). He argues that Catholic populations in the city showed high infant mortality, while low rates were found among the Jewish populations. Most infant deaths during the first year of life in 1901 were caused by gastro-intestinal infections during the summer (Mercier 2006).

The First World War did not improve the living conditions of most infants. The social environment changed in ways which would be expected to increase infant mortality: male wage-earners and care-givers disappeared, food and coal were rationed, inflation was rampant, and remaining care-givers were under stress from lowered incomes and fear for the safety of loved ones overseas. Yet, infant mortality, at least in Britain, did not increase automatically. The Lancet reports that, while infant mortality increased in 1915, it declined in 1916. However, the noticeable increase occurred in 1917 (to 97 deaths per 1000 live births, from 91 the year before) (The Lancet 1918a). While the Lancet attributes this increase to stress affecting the ability of mothers to successfully breastfeed, it is likely that the privations of the war were beginning to take their toll on infants and mothers. Interestingly, McKinnon reports the opposite trend for Ontario. He shows that infant mortality declined slightly in 1915, increased in 1916, then dropped in 1917 (from 107.3 in 1916 to 92.4 deaths per 1000 live births in 1917) (McKinnon 1944:482). Long before the influenza epidemic occurred, there were inequalities in risk of death: Did the epidemic continue these trends, or, suddenly, did all infants have an equal chance of dying?

The following chapters will address these questions. Chapter two begins by providing the historical background of the city of Toronto in the early twentieth century and what is known about influenza. Chapter three explains the history of the materials used in this thesis and describes the sources of error which could complicate the analysis. It also describes the methods used, including the transcription, organization, analysis, statistics, and criteria for record linkage. Chapter four gives the results of the analysis, in

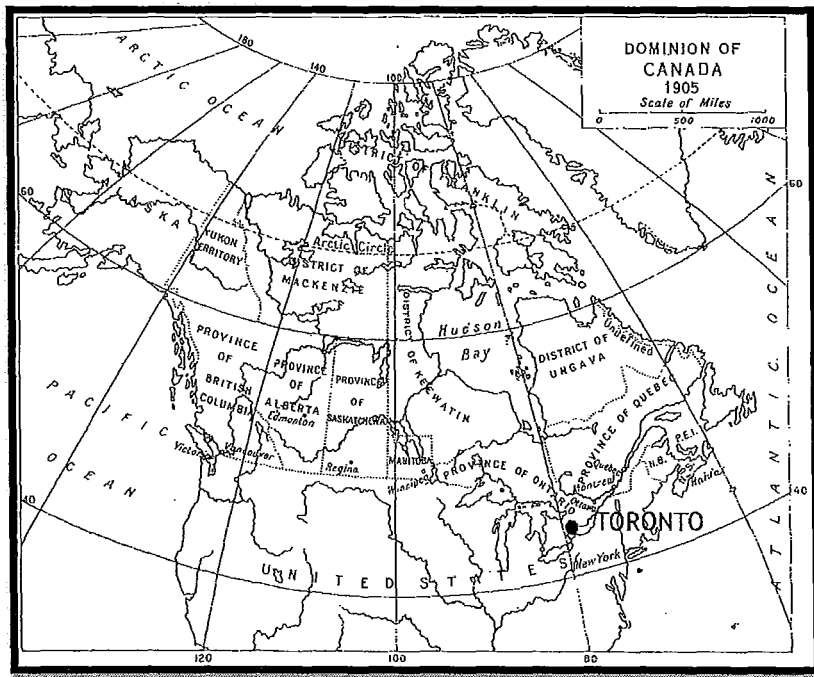
terms of the total infant mortality rate, sex-ratio at death, age at death, cause of death, stillbirth rate, and the findings of the record linkages. Chapter five discusses infant mortality in Toronto over the five year period, 1917-1921, and outlines and explains the continuities and disruptions in infant mortality during the 1918 influenza. The thesis concludes with suggestions for further research into the effects of this epidemic on infants and families.

Chapter 2

BACKGROUND

As stated by Singer and Baer, a critical medical anthropology hopes to be “equally sensitive to bio-environmental factors in health, the experience of suffering among those who are ill, and the primacy of political economy in shaping the impact of bio-environmental factors on disease, sufferer experience, and the character of the health care system deployed in response to disease and illness” (1995:50). In this light, I present the historical and socio-environmental background of the city of Toronto, the effects of the First World War on both the soldiers and the people who stayed at home, and a bio-medical understanding of the influenza virus. As the experience of the influenza pandemic on infants and families was a product of both the features of the virus and the social environment, it is important to have an understanding of the lives of Torontonians at the turn of the last century.

2.1 Toronto



Map 2.1 - Toronto, Canada 1905 (Adapted from John Bartholomew and Son, Ltd [1927: 21]).

The city of Toronto, Canada is the capital of the province of Ontario. It is located in the southwest of the province, along the northwest shore of Lake Ontario (Figure 2.1). Currently, Toronto is the largest city in Canada and at the beginning of the twentieth century it was undergoing the transformation from a small to a large city as vast influxes of people immigrated to Canada. During this period, the population increased from approximately 200 000 in 1900 to 470 000 in 1915, and then to 542 000 by 1924 (City of Toronto Archives, Fonds 200, Series 365, Files 20, 27). The city limits expanded “from 17 to 35 square miles” (Dendy and Kilbourn 1986:154). Toronto was the centre of industry and banking, and was a central node for the expanding railway system (Masters

1947:165), as well as a hub of migration and trade, education, and a locus for improvements in public health.

Social inequalities were a significant aspect of life in Toronto during the early part of the twentieth century. As seen through the records and concerns of the Department of Health (City of Toronto Archives, Fonds 200, Series 365, Files 20-27), Toronto experienced many of the problems of a city trying to accommodate rapid expansion and immigration. The city, especially in slums such as the Ward (Mercier 2006), was overcrowded, with high rents and inadequate housing (Piva 1979, Solomon 2007:17-22). The Department of Health, through its mission to improve basic sanitation and public health, was concerned with the living conditions of the poor and the diseases and infant mortality caused by unclean and overcrowded homes. In an attempt to improve the conditions of the city, the Department destroyed 1600 squalid and decaying homes from 1913 to 1918, yet replaced only one percent (Piva 1979, Solomon 2007:29). Due to the lack of housing and high rents plaguing the city since the nineteenth century, the Toronto Housing Company had begun to build low-income housing in 1913; however, the new homes were out of reach of the most impoverished residents who continued to suffer (Dendy and Kilbourn 1986). In the spring of 1918, Dr. Hastings 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). In October 1918, *The Globe* relates that “Toronto is short ten thousand dwellings” (*The Globe* 1918r:6). The chief Medical Officer of Health of Toronto, Dr. Charles Hastings, believed in interventionist policies to improve public health and argued that a well-structured

department of health could solve any health crisis (City of Toronto Archives, Fonds 200, Series 365, Files 20-27; MacDougall 1990). By destroying unsanitary homes yet avoiding the root cause of the problem, poverty, the Board served merely to amplify the stress, debt load, and homelessness of the most impoverished citizens during a period of increased stress due to the war. Similarly, the board believed that education of mothers could reduce infant mortality (City of Toronto Archives, Fonds 200, Series 365, Files 20-27), but does not acknowledge that poverty reduces access to resources and taxes the ability of even the most well-educated mother. As infant mortality is recognized to vary by socio-economic status (Matteson et al 1998), some infants in Toronto were at greater risk of dying than others.

Dr. Charles Hastings was the chief Medical Officer of Health prior to the First World War and throughout the influenza epidemic (MacDougall 1990). He was dedicated to improving the lives of the people of Toronto through the efforts of public health workers. Dr. Hastings was responsible for instigating milk pasteurization campaigns and for expanding and re-structuring the Health Department, including the creation of the Division of Public Health Nurses in 1914 (MacDougall 1990, MacDougall 2007). His efforts resulted in a professional Department of Public Health with monthly records of vital statistics that provided reports of the activities of the public health nurses and a small write up of the state of health in the city. Dr. Hastings' improvements to the department were occurring in a larger climate of increased medicalization of health care and enhanced belief in scientific medicine in Canada (Van Ingen 1921, Mitchinson 2002). This was part of a global public health and sanitation movement which developed in the

early part of the century that included a combination of increased knowledge of germ theory, medicine, and the benefits of sanitation. That this is true for the Department of Public Health as well is written on the front page of each monthly report from 1917. Dr. Hastings firmly believed that “with a well-organized Department of Public Health, a Municipality may have as much health as it is willing to pay for” (City of Toronto Archives, Fonds 200, Series 365, File 20).

2.2 The Social Environment and War

In 1901, Toronto was a “relatively unhealthy” city in terms of its infant mortality, at 167 deaths per 1000 live births (Mercier 2006:127). Mercier portrays 1901 Toronto as a place highly segregated by neighbourhood and stratified by class and culture (Mercier 2006). These inequalities were reflected in infant mortality rates throughout the city. In his analysis of the location of infant and child death, crowding in houses, socio-economic status, and culture in Toronto, Mercier found that, while all forms of lower socioeconomic status increased infant and child mortality, the greatest variation in the rate of death resulted from religious and cultural differences. He states that “in general, areas of concentration of Catholics were to be associated with high mortality [Corktown, Niagara, and Brocktown Village], and inner-city Jewish ghettos were associated with low levels of childhood mortality, despite the confluence of poor housing, environmental, and socioeconomic conditions in these areas” (Mercier 2006:146). However, Mercier affirms that neighbourhood-based differences do not explain all the variation in infant mortality because of “complex interaction effects” (2006:146).

By 1911, the city was still plagued by socio-economic inequality. Dr. Hastings reported that the Ward, Corktown and Niagara neighbourhoods were slums and that “privies and cesspools, shackhousing, and extreme poverty and crowding predominated in these areas” (Mercier 2006:131-2). When the city was faced with the depression from 1913-1915 (MacDougall 1990), followed by the restrictions and inflation resulting from World War I, it seems unlikely that underlying and persistent variations in health status and access to resources would have translated into an equal experience of epidemic disease.

When war broke out in Europe in August 1914, over 4 000 young men in Toronto went to train in the Canadian Expeditionary Force (Miller 1999:64). From the outset, the people left at home assisted those sent to war, as “the available sources leave no doubt that most women living in Toronto eagerly joined the war effort in any way they could” (Miller 1999:66-7, *The Globe* 1918), giving both time and money (Figure 2.1). While the belief in the necessity of the victory remained strong, by winter 1916, the citizens of Toronto were beginning to complain about the cost of living and coal and food shortages (Miller 1999:142-5). Death, while tragic, became expected and “as the demands of the war grew, the commitment of local residents to winning it increased exponentially. They gave more money, more soldiers, more time, and more of themselves in order to win” (Miller 1999:170-171). When the recruiting drives were no longer successful, Canada legislated conscription in August 1917, to the general acceptance of Torontonians (Miller 1999). But the health of the population was deficient before the war as well, as “typically, for every ten men who volunteered, three or four were rejected for failing to

pass the medical exam, but it was not unusual for six or seven to be turned down” (Miller 1999:199).²



Figure 2.1 - Canadians were urged to do everything possible to win the war (The Globe 1918h).

Disease commonly co-exists with war. Life in the trenches was a perfect breeding ground for epidemic disease. As Sherman states,

war involves overcrowding and intermixing of populations. Resources are diverted, and often famine and malnutrition increase. These co-called enabling factors in turn lead to decreases in personal hygiene and medical

² The Toronto Department of Public Health states that from July 1917 to July 1918 there were “approximately thirteen thousand soldiers discharged from the army for TB in the U.S. and Canada alone” (City of Toronto Archives, Fonds 200, Series 365, File 21)

care and frequently a breakdown of the social structure. During wartime, individuals are subjected to increased stress, they become more susceptible to new diseases, and endemic diseases may become more severe. [Sherman 2006:117]

The First World War not only spread influenza, but typhus and sexually transmitted diseases as well (Sherman 2006:118, Morton 1993). Oxford et al (2002) observe that the daily lives of soldiers in the First World War were conducive to the spread of infection. The soldiers were faced at all times with the stress of potential death. In the front trenches, soldiers made do with little sleep, cold and wet conditions, constant filth, collapsing trenches, diminishing food supplies, and they were surrounded by the dead (Heyman 2002). Soldiers risked being wounded not only from assaults and poison gas, but from water-induced trench foot, drowning in mud-filled trenches, and the bites of lice and rats. The trenches, the warships and submarines, the training camps, and the transports were all overcrowded (Heyman 2002), leading to easily spread respiratory illnesses including influenza, pneumonia, and tuberculosis. These conditions followed the soldiers home. Pettigrew (1983) relates the words of a Canadian soldier returning to Toronto in October, 1918 who lamented “it was a disgrace to Canada to take men right out of hospitals and crowd us into that transport. We didn’t have a bath or a change of clothing for over two weeks” (1983:9).

The war was devastating, not only to the soldiers in the trenches, but also to those supporting them from Canada. Women at home had to care for families alone, were encouraged daily to give more money, more time, to restrict more food and coal, to go into the workforce, and to urge any man not yet enlisted to do so without delay (Miller

1999). These pressures, compounded by inflation, left mothers with more than just a new infant to worry about. Miller rightly assumes that, while these pressures were felt by many women in Toronto, it was the working-class women who felt the rising costs most keenly (1999:317). As Piva explains, after 1915, real earnings in Toronto declined by over 20%, so that 1917 was “the worst year Toronto’s workers had experienced since the turn of the century” (Piva 1979:56). According to the Department of Labor “during October 1918 . . . the cost of living in Canada was still ascending” (The Globe 1918x:5). Jones (2007) agrees that, in Winnipeg, it was the working-class women who suffered the worst of the epidemic.

The first week of August 1918 was filled with civil unrest in Toronto. Ethnic tensions erupted when former soldiers looted and destroyed Greek restaurants due to a conflict a returned soldier had in one such restaurant (Miller 1999:405, Crerar 2005). The arrest of some returned soldiers led to attacks on police and police stations. The tension only ended when the mayor threatened to read the riot act on August 7 (Miller 1999:410). Another coal shortage came on the heels of staggering losses of Torontonians men throughout August and September due to the Amiens offensive (Miller 1999:417-418). The Globe reports that as of September 25th, 1918, there were almost 9000 people without fuel (1918d). This was exacerbated by the influenza epidemic, as coal producers were also affected, drastically reducing production (The Globe 1918s).

In addition to the numerous social conditions favouring the spread of the flu, weather conditions may also have contributed to the spread of disease. Environment Canada lists August 1918 as recording the hottest temperature for the period 1917-1921

(Environment Canada 2008).³ While hot temperatures are normally not associated with respiratory infections, they are associated with summer diarrheal disease in infants (Herring et al 1998). Munoz (2003) suggests that infants who are at greatest risk of hospitalization from influenza are those with underlying medical conditions, most often asthma and congenital heart disease (2003:101). As two factors which influence the physiological response to infection are “nutritional status of the host,” and “pre-existing disease” (Evans 1991), infants with compromised nutrition from diarrhea may have been predisposed to contract influenza in the autumn of 1918: the infants who were at risk of the influenza may also have been those at greatest risk for gastro-intestinal illness during the summer.

Contemporary reports suggest that September 1918 was wet and cold, and blame this for the spread of the flu (The Globe 1918a, The Globe 1918c; The Globe 1918g). The Globe reported that “as the month of September was the coldest and wettest month that the city has experienced in 79 years, Dr. Hastings blamed the epidemic on this” (The Globe 1918g:8). Environment Canada confirms that September 1918 had both the coldest average temperature for the five-year period, as well as more than double the precipitation of the next highest year (Environment Canada 2008).⁴ The clustering of people in heated indoor dwellings to avoid the cold and the rain is known to facilitate the

³ Hottest temperature recorded in August: 1917 = 33.3°C, 1918 = **38.9°C**, 1919 = 33.3°C, 1920 = 31.7°C, and 1921 = 32.8. The mean temperatures for August were: 1917 = 19.9°C, **1918 = 21.7°C**, 1919 = 20.0 °C, 1920 = 21.1°C, and 1921 = 20.1°C.

⁴ Mean temperature for September: 1917 = 14.8°C, **1918 = 13.6°C**, 1919 = 17.5°C, 1920 = 17.7°C, and 1921 = 19.9°C. Total precipitation in September: 1917 = 16.8mm, **1918 = 118.1mm**, 1919 = 48.5mm, 1920 = 50.8mm, and 1921 = 32.3mm

spread of airborne infectious diseases. This is because the possibility of droplet transmission is heightened by crowded conditions and hot, dry air “impairs the protective mechanisms of human mucous surfaces,” allowing the virus to invade the respiratory system (Evans 1991:8).

As noted by Miller, “the story of Toronto during the years 1914-1918 is largely untold” (2002:9). This can be easily extended to 1919 and to the waves of influenza thereafter. By the time of the 1918-1919 influenza epidemics, Torontonians were already sick and stressed from four years of war and from struggling with the disease load associated with immigration and social reform. In May 1918, the Canadian Medical Association Journal reported that hospitals in Toronto were “undergoing a considerable economic strain owing to the conditions enforced by the war” (CMAJ 1918a:460). Even before the epidemic hit the city that autumn, hospitals were in financial distress and had lost doctors, nurses, and staff to the war effort (Dickin McGinnis 1981). Conditions worsened when the medical staff that remained contracted influenza and the hospitals were filled to capacity. Through a combination of unfavourable social, political, martial, and environmental conditions, Toronto in 1918 was extremely vulnerable to a virulent strain of influenza.

2.3 Influenza

The entire continent of America has just passed through the most severe epidemic that has ever visited our nations, and Toronto has had to bear its full share. [City of Toronto Archives, Fonds 200, Series 365, File 21]

The 1918 influenza epidemic did not represent a newly emerging infectious disease. There were epidemics throughout the nineteenth century (the last in 1890) and historians argue that the disease may have been causing death for thousands of years (Pyle 1979, Dickin McGinnis 1981, Phillips and Killingray 2003, Herring 2009). The origins of the 1918 pandemic of “Spanish Influenza” are debated, yet unknown. Credible theories place the origin in either France or Kansas (Dickin McGinnis 1981, Oxford et al 2002, Barry 2004, Humphries 2005). The first wave of the global pandemic began around February or March of 1918 (Dickin McGinnis 1981) but was not as severe as the second wave that autumn. Influenza continued to kill with resurgences throughout 1920 (Dickin McGinnis 1981, Crosby 1989). The epidemic entered Canada from American soldiers journeying to the front lines by way of Canada, and was spread through the country as the Canadian Siberian Expeditionary Force travelled west (Humphries 2005). Influenza came to the Polish Camp at Niagara in mid-September and spread to Toronto from there (Humphries 2005, The Globe 1918c). It is estimated that the flu was responsible for infecting at least one-sixth of the Canadian population and killing anywhere from 30,000 to 50,000 people (Dickin McGinnis 1981, Pettigrew 1983).

The flu first came to Toronto during the second wave of the global pandemic. It ‘officially’ hit the city on October 3, 1918 (the Globe 1918f, Pettigrew 1983:48) and the *Monthly Reports of the Department of Public Health* reveals that 1084 people died from influenza or pneumonia in October (City of Toronto Archives, Fonds 200, Series 365, File 21). Although it peaked in late October, the epidemic continued throughout the rest of 1918 and over the next few years (The Globe 1918y, Crosby 1989). The Department

of Health recorded that a total of 3118 people died from influenza and pneumonia in Toronto in 1918 (City of Toronto Archives, Fonds 200, Series 365, File 21) and Miller estimates that up to half of the city, over 260 000 people, was infected in October (Miller 1999). Pettigrew believes that for the entire epidemic, Ontario had 300 000 people sick with the flu, of which 8 705 died (1983:56). On a global scale, influenza is estimated to have killed between 40 and 100 million people (Johnson 2003).

Influenza is an infectious disease caused by three subtypes of the influenza virus, A, B, and C. The origins and etiology of the 1918-1919 strain are still unidentified and hypotheses change as research continues. In 2000, the virus was identified as a “H1N1-subtype influenza A virus, probably closely related to what is now known as ‘classic swine’ influenza virus” (Taubenberger et al 2000:241). However, new PCR analyses of the entire genome suggest that “the 1918 virus appears to be an avianlike influenza virus derived *in toto* from an unknown source” (Taubenberger and Morens 2006:18). Whatever the cause, the symptoms experienced by those affected are well described. Acute infection with the virus led to a high fever, muscle aches, headaches, and sometimes nausea and vomiting; however, infections with mild or few noticeable symptoms were a factor in the rapid spread of this epidemic (Beveridge 1977:11-13). Young (1919) reports that infection with influenza caused haemorrhages in many parts of the body, including bleeding gums, nosebleeds, and vomiting blood within a day or two of infection (1919:422). Recent research suggests that the clinical presentation of influenza may be different for infants less than one year of age (Kao et al 2000, Munoz 2003). Infants may show only a high fever, or “irritability, decreased oral intake,

vomiting, diarrhea, abdominal pain, lethargy, difficulty breathing, dyspnoea and apnoea” (Munoz 2003:99). The after effects of the flu could include fatigue, “mental apathy, depression, subnormal body temperature, and low blood pressure, which could last for weeks or months” (Ellison 2003:225). Despite the label of *the flu*, uncomplicated influenza was not the major threat during 1918-1919. The high rates of mortality were not caused by influenza alone, but occurred after the development of pneumonia (Beveridge 1977:14, City of Toronto Archives, Fonds 200, Series 365, File 27).

Pneumonia was a significant factor in the lives of the people of Toronto long before 1918. The December 1919 *Monthly Report of the Department of Public Health* shows the increasing rate of pneumonia and broncho-pneumonia from 111 per 100 000 in 1886-1890⁵ to 179 cases per 100 000 in 1917. The pneumonia rate during the 1918 influenza epidemic was 267 cases per 100 000, followed by a decrease to 132 per 100 000 in 1919 (City of Toronto Archives, Fonds 200, Series 365, File 22). Further, the report for January 1917 states that “since 1875 the death rate from pneumonia in infants and children has increased 600 per cent” (City of Toronto Archives, Fonds 200, Series 365, File 20). Kilbourne even suggests that the bacterial infections “enhanced viral virulence through the production of protease enzymes [which has] recently shown to facilitate influenza virus multiplication” (2003:36-7). Singer and Clair (2003) note that “central to the syndemic concept is the assumption that in cases of co-affliction with two or more diseases, actual biological interaction occurs” (2003:427). It is most unfortunate for the

⁵ The rate in 1881-1885 was 129 cases per 100 000, but this was elevated as a result of an influenza epidemic.

people of Toronto (although most likely ecologically significant for the pneumonia virus) that the influenza virus weakened the resistance of the immune system for a disease so highly prevalent in society.⁶ It is likely that one of the reasons for the high mortality of the influenza epidemic was that pneumonia was already an established and increasing illness in the city.

Noymer and Garenne suggest that there is a relationship between tuberculosis and the flu, such that “those with tuberculosis (TB) in 1918 were more likely than others to die from influenza” (2000:565). They also suggest that, since tuberculosis infected young men more often than women, this could explain the unexpected numbers of deaths of young adults, as well as the high rates of male deaths in their study (2000, Noymer 2009). TB was prevalent throughout the world in the early parts of the last century and, as previously noted, from July 1917 to July 1918 there were “approximately thirteen thousand soldiers discharged from the army for TB in the U.S. and Canada alone” (City of Toronto Archives, Fonds 200, Series 365, File 21). Noymer and Garenne explain the relationship between tuberculosis, influenza, and pneumonia:

Tuberculosis infection causes lung cavities to form, which become a breeding ground also for non-TB bacteria, including *Staphylococcus aureus*. This would have had the effect of priming tuberculous individuals for *S. aureus* superinfection in the event of co-infection with influenza. It is highly plausible that TB infection laid the ground for the massive secondary bacterial pneumonias that killed the victims of the flu in 1918. [Noymer and Garenne 2000:577]

⁶ Tashiro et al (1987) note that “in influenza the combined virus–bacterial pneumonia is approximately three times more common than primary viral pneumonia. The bacteria most commonly involved are *Staphylococcus aureus*, *Streptococcus pneumoniae* and *Haemophilus influenzae*. *S. aureus* co-infection is reported to have a fatality rate of up to 42%” (1987:536).

While the interaction between TB and influenza likely increased the risk of death of young adults, it was unlikely to have affected infants directly. However, if TB was predominantly a disease of the poor and those who lived in crowded housing (Frieden et al 1993), the young adults in those households may have been disproportionately affected. This, in turn, may have placed the infants in those households at greater risk of influenza infection, as well as from the negative effects of the death of a parent.

The odd age distribution of the deaths from the 1918-1919 influenza epidemic has yet to be sufficiently explained and is perhaps the most troubling. Mortality rates were significantly raised for people between the ages of 15 and 34 (Taubenberger 2003:40).⁷ Noymer and Garenne (2000) and Noymer (2009) suggest that this is because the young adult age group was also the group at risk for TB. These deaths would have adversely affected the people of Toronto already under the stresses of war-time restrictions. Not only was a terrifying epidemic striking the city, but it attacked those thought to be the strongest.⁸ As Crosby notes “like war, [influenza] preferred young adults as victims” (1976:21). However, not all young adults were equally at risk of death.

⁷ Dr. Harder, a London physician, noted in a December 1918 issue of the *Lancet* that being under the age of 20 or over the age of 40 was beneficial during the time of the epidemic (Harder 1918: 873). Further, the *Monthly Report* for October 1918 notes that “in the decade from 20 to 29 years, there were 455 deaths, or more than one-quarter of the entire mortality” (City of Toronto Archives, Fonds 200, Series 365, File 21)

⁸ This influenza epidemic also killed those who are the usual targets of infectious disease: infants, elderly adults, and those with weakened immune systems. Although there were increases in the rates of mortality for these groups of people, there was an unusual rise in the numbers of deaths of young adults (Crosby 1976:21).

It has long been recognized that pregnant women were hit particularly hard by the 1918-1919 influenza epidemic. Winternitz et al explained in 1920 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 mother, as well as for the child, is definitely increased” and that “the hemorrhagic lesion of influenza seems a plausible explanation for the frequency of abortions” (1920:39). Beveridge also clarifies that “the prognosis was said to be serious for the women who aborted or went into labour” (1977:15). Collier describes a New York obstetrician who recorded a seventy percent mortality rate among pregnant women in 1918 (1974:263). Further, recent studies have shown that women in their third trimester are at greater risk of being hospitalized from influenza and pneumonia (Neuzil et al 1998:1100). Reid’s study of mothers and infants in 1918 Derbyshire, England, found that infection of a mother in the first two trimesters of pregnancy could lead to premature birth which is also associated with increased risk of both infant and maternal mortality (Reid 2005:53). The state of pregnancy may itself be to blame, since Burns et al found that pregnancy is associated with a decrease in immunity, although this reverses at delivery (1999:159). This is known as “pregnancy-associated deficiency syndrome (PAIDS)” and is a “suppression of immunity that allows retention of the foetus” (Reid 2001:220). Jones (2007) shows that in Winnipeg, many impoverished women with influenza died shortly after giving birth, reducing the chance of survival of their infants. Recent studies have found that if the mother survives, the antibodies in her milk can protect the infant for a few months but that “childhood deaths associated with influenza are most frequent in infants under the age of 6 months” (Zaman

et al 2008:2). In contrast, Munoz (2003) observes that infants over six months of age have the highest rate of infection. Children who have older, school-aged siblings and those placed in daily care were at highest risk. This may have been another disadvantage for single working mothers (Munoz 2003:101).

Canadian health officials did not know how to stop the virus. Toronto's response to the flu was similar to other places in Canada (Dickin McGinnis 1981, Pettigrew 1983, Jones 2003) and to other major cities throughout the world (Collier 1974, Crosby 1989, Figure 2.2). The newspapers warned Torontonians of the approach of the Spanish Influenza in military settings, although, in the beginning the reports were optimistic. The first mention of the autumn wave of the epidemic appears in *The Globe* on September 11th, 1918, when readers were made aware of an outbreak among 100 members of the merchant marine in Massachusetts (1918b). Influenza in Canada was reported on September 21st, as 150 soldiers at the Polish Camp at Niagara were counted sick and three deaths were reported (*The Globe* 1918c). Miller notes that the first civilian death, of a girl, occurred on September 29th, 1918 (1999:420); however, as of September 30th, *The Globe* was telling its readers that although there were hundreds of cases, there had been no deaths (1918e). Additionally, those cases were all minor, with Dr. Hastings stating that it was even milder than other fall flu seasons (*The Globe* 1918e).

At the beginning of October, 10 000 students and over 1600 teachers were sick with over 600 people flooding the already understaffed hospitals (Miller 1999:421-2). Yet, it was not until October 3rd that Dr. Hastings reported that "Spanish influenza has officially reached Toronto" and that "there was now little doubt that the disease was the

same as has been spreading through the United States” (The Globe 1918f:6). By October 10th, St. Michael’s Hospital was refusing patients because of overcrowding and the epidemic among the nurses (The Globe 1918l). On October 14th, preparations were being made by the Board of Health to convert two vacant hotel properties, the Arlington and the Mossop, into emergency hospitals (The Globe 1918n, Pettigrew 1983).⁹



Figure 2.2 – Influenza Masks (The Globe 1918k, The Globe 1918m).

At the outset of the epidemic, influenza was not a reportable disease in Ontario and those infected were not subject to quarantine; however, the medical officer of health

⁹ However, only the Arlington Hotel and the un-opened new wing of Women’s College Hospital were used as temporary influenza hospitals as the Mossop was not ready before the epidemic had crested (The Globe 1918u).

had the ability to close places of public congregation (McCullough 1918). Areas of public gathering, such as schools, theatres, and dance halls were eventually closed (Pettigrew 1983) even though both the provincial and local Medical Officers of Health initially saw little use in this action (The Globe 1918i, The Globe 1918j). The Ontario Provincial Board of Health provided information to doctors through pamphlets and to the public through the media (McCullough 1918). When the already war-diminished staff of the hospitals became ill and overworked, volunteers were recruited from medical and nursing students, nursing organizations, the Red Cross, and from regular citizens (The Globe 1918, McCullough 1918, Collier 1974, Pettigrew 1983). The Board of Health produced vaccines for the population at the Connaught Laboratories at the University of Toronto and at Board of Health labs (McCullough 1918), although the effectiveness of such measures is questionable (The Globe 1918t, CMAJ 1918c) as the virus was not identified until 1933 (The Scientific Monthly 1937).

Neither restrictions on public gatherings suggested by the public health office nor the valiant acts of concerned citizens could stop the epidemic from spreading (Miller 1999). Yet, as was the case in many places throughout the world (Crosby 1989), the epidemic could not stop the people of Toronto from celebrating the end of the war. On November 11th, 1918, over 200 000 people celebrated as a parade of veterans marched in jubilation to a gathering at Queen's Park (Miller 1999:429-30). Dr. Hastings saw this as a test to determine whether the epidemic had left the city since everyone must have been exposed during the celebrations. However, due to the waning amount of new cases he was "optimistic and intimate[d] there [was] no cause for alarm" (The Globe 1918v:9).

Dr. Hastings believed that Toronto did well compared to other cities in North America, stating that the crest came in Toronto one week before other cities, and with fewer deaths (The Globe 1918w). The deaths that did occur were blamed on people not resting and following the recommendations of the health department to walk instead of taking the street car and to stop working (The Globe 1918l). Dr. Hastings does not mention that these precautions would have been difficult for many citizens of Toronto.

The recommendations of the Board of Health likely had little effect on the course of the epidemic since the requisite conditions had been set long before influenza arrived. The citizens were suffering from war-time restrictions, social inequalities, infectious diseases, and poor health. Extreme weather caused changes in communal behaviour compounded by fuel restrictions that led to inadequate heating of houses. The epidemic briefly held the social consciousness (The Globe 1918o) but was later subsumed by the numerous difficulties of the 1920s: demobilization of soldiers and their return to work, the quest for suffrage and social equalities, general social reform and improvements in public health, and a rapidly increasing population. This thesis will illuminate but one of those forgotten elements – the plight of infants during the 1918 influenza epidemic.

Chapter 3

MATERIALS AND METHODS

All statistics are socially constructed. What they contain and omit reflects the concerns of their collectors and the particular arrangements for collection. Their meaning also varies according to the social conditions in which they are collected and the purposes for which scholars use them. Because statistics are cultural phenomena, they require social interpretation, not mere technical correction. Yet if the problems of vital statistics are greater than believed, so too is their research potential, provided that scholars build on their social properties. [Emery 1993:3]

3.1 Materials

This research on infant mortality during the 1918 pandemic was conducted using the Registered Death Records of the Province of Ontario, currently publicly available on microfilm at the Archives of Ontario in Toronto (Archives of Ontario MS 935, Reels 228-229, 238-240, 251-252, 261-262, 273-274). In total, the 8952 infant death records from 1917-1921 were transcribed representing the 9016 individuals whose deaths were registered with the Registrar General of the Province of Ontario during this five year period.¹⁰

The death records for 1917-1919 contain the following information: name, sex, age, place of birth, date and location of death, place of burial, occupation, marital status, parents' names (including mother's maiden name), cause of death (both the cause listed by the informant and the causes determined by the physician, including the disease causing death and the immediate reason for death), duration of illness, and physician's

¹⁰ The discrepancy between the number of records and the number of individuals represented by those records results from twins who died at the same time (often stillborn or in the perinatal period) being recorded on the same record.

name and address. The forms changed in 1920, and the 1920 and 1921 records have the additional information of racial origin; length of residence at place of death, in Ontario, and in Canada; birthplace of father and mother; the dates that the physician attended the deceased; the duration of both the primary and contributory cause of death; whether an operation preceded death; and if an autopsy was performed (Appendix A). Data on total population and total numbers of live and stillbirths from 1917-1921 (necessary for the calculation of the infant mortality rate) were drawn from the Sessional Papers of the Legislative Assembly of the Province of Ontario, also available on microfilm at the Archives of Ontario (Series B 97, Reels 149, 154, 159, 164, 169, and 173). Historical data used to contextualize the city from 1917-1921 came from the Globe newspaper and the published reports of the boards of health located in the City of Toronto Archives (Fonds 200, Series 365, Files 21-24). As well, secondary sources were used to describe the city during World War I, leading up to the epidemic, and during the immediate aftermath.

3.1.1 History of the materials

The collection and presentation of vital statistics are products of the cultural system in which they are produced (Emery 1993). They therefore represent a version of events deemed important by those responsible for collecting them. It is necessary to keep this bias in mind as the registered deaths are by no means an impartial and complete record of the deaths which occurred. The nature of the materials influences the

conclusions drawn from them; as such, a brief account of the history of vital registration in Ontario provides context for this study.

Systematic vital statistics collection became the responsibility of the Province of Ontario with the Registration Act of 1869 which was in place until 1920 (Emery 1993:22-30). The act establishes the role of the Registrar General, the Division Registrar and the registration districts that structure how the death records were recorded and collected.

The act:

Cap. 30 – Provides that the Provincial Secretary is Registrar General. Each city, town, county or union of counties forms a registration district and the Clerk of the Peace is Registrar. Each township or union, incorporated village and town and every ward in cities is registration division [sic] . . . The Registrar General procures the necessary books and forms for all the Registrars . . . Division Registrars are to receive their books and forms and make their reports through the District Registrars, on or before the 15th January in each year, and the District Registrars transmit theirs on or before the 1st February. The father, or mother, or person standing in their place, or person in whose house a birth takes place, or the nurse must within 30 days register it, and pay 10cts fee therefor [sic]. If illegitimate, no father's name is to be entered without his consent, the word "illegitimate" being entered in the proper column. Registration may be made within the year. The occupier of the house (or one of them) in which a death takes place, or a person present at, or cognisant of a death must register it within 10 days paying the same fee . . . Physicians must report all deaths and births in cases which they have attended. Errors of registry may be corrected within a year . . . The original returns are to be kept and bound up by the Registrar General and they are also to be transcribed into separate books . . . The Registrar General annually collects and publishes for the use of Parliament a report of the results of all registration. [Harvey 1870:47]

This act was reissued in 1875 with a change that "required householders to register a death *prior* to interment (rather than within ten days), in return for which they were to be issued a certificate of death" (Emery 1993:33). This act also eliminated the role of county registrar. Importantly, the act legislated that "the attending medical

practitioner now was to submit the death certificate to the registrar of the municipality in which the death occurred, not his own municipality of residence” (Emery 1993:33).

The bureaucratic foundation of the public health system was established in the second Ontario Public Health Act of 1882 which created “a permanent central Board of Health and gave it advisory powers over local health boards. The act also provided for a provincial secretary of health” (Emery 1993:37). In 1884 these local boards became established in law, directly responsible to the provincial board of health (Emery 1993:37-38). The third Registration Act of 1896 made it illegal to bury a body without a death certificate (Emery 1993:39). Among other things, municipal registrars were to make two copies of registrations, keeping one for themselves and sending the other to the provincial board (Emery 1993:39). To this day, the death registrations are kept by the Office of the Registrar General for 72 years, at which point they are transferred to the Archives of Ontario for public access (Taylor 2004, Ontario Genealogical Society 2007).

The Dominion Bureau of Statistics was created by the Canada Statistics Act of 1918 which attempted to nationalize and standardize the separate provincial means of collecting vital statistics (Emery 1993:44).¹¹ Ontario passed the Vital Statistics Act in 1919, in the aftermath of the pandemic, to prepare for the new national system in January 1920 (Emery 1993:40). This resulted in the changes to the forms on which the deaths were registered. From 1917-1919, they were identical, with new forms appearing in

¹¹ The Canada Statistics Act forming the Dominion Bureau of Statistics obtained royal assent on May 28th, 1918, before the influenza epidemic affected Canadian consciousness (Worton 1998). It was the Federal Department of Health Act of 1919 which created the precursor to Health Canada that was in direct response to the lack of organization witnessed during the epidemic (Dickin McGinnis 1981, The Globe 1918q).

January 1920 supplied by the Dominion Bureau of Statistics (Emery 1993:45), consistent with the new Vital Statistics Act (Appendix A). Comparable to what Emery found for the change of registration forms in 1935 (1993:134), the change of forms in 1920 brought problems in completeness of the records as some time was needed for officials to become used to the new format. The forms for the early part of 1920, for instance, have information missing from many records.

3.1.2 Sources of error

Consistent with the preceding sixty years of legislation, the Registered Death Records of the Province of Ontario from 1917-1921 are organized into county of death. Within each county, the deaths are further organized into city, town, township, or region *in which the death occurred*. This creates what Emery (1993:137-154) calls the “residence problem” where deaths are listed in the place where they occurred and not in the place of usual residence of the deceased. Although the forms issued by the Dominion Bureau of Statistics after 1920 did ask about the place of residence (Emery 1993:141), the questions were not specific enough to always determine place of habitual residence. This problem was not completely fixed until 1944 when registrations of death were forwarded to the district of residence (Emery 1993:154).

The implication for this project is that the list of deaths classified as having occurred in Toronto, in the county of York, is not *only* a record of resident infants who died in the city. The registrations include infants who died during travel through Toronto (such as one infant who died on a CPR journey) and immigrant families on their way to

other parts of Canada. It also includes infants from surrounding towns who died in Toronto hospitals or maternity homes (especially from New Toronto, Mimico, Etobicoke, and York Township). Further, the records do not include infants who were residents of Toronto but who died when on trips away from the city. Therefore, the death records may not be directly comparable to the registered births as reported in the Sessional Papers of the Legislative Assembly of Ontario for the purposes of the calculation of the infant mortality rate. However, the "residence problem" also affected registered births. Non-residents probably do not account for a large proportion of this study sample because hospital birth was not yet common,¹² but it is an unknown proportion and therefore one source of error. As per Moffat (1992) and Gray (1997), this research assumes that the number of non-resident infants recorded in Toronto is equal to the number of resident infants recorded in other districts, so that together, both totals sum to zero.

The development of ever-increasing legislation to improve vital statistics recording is due to the incompleteness of the records during the 1800s. Emery notes that "officials believed, the returns for deaths and marriages were incomplete for cities until the 1880s and the entire province until 1900. Worse still, the birth returns remained incomplete until the 1920s" (Emery 1993:31, MacDougall 1990). This suggests that the death records were likely reasonably complete by the time of the influenza epidemic in 1918, but that the birth records (used for the calculation of infant mortality rates) may not have been. Further, the birth statistics used in this study were drawn from the Sessional

¹² 16 per cent of the *registered* births in 1920 occurred in hospital (Emery 1993:74).

Papers of the Legislative Assembly of Ontario published for the previous year.¹³ Therefore, the delayed birth registrations are not included in these totals, underestimating the true number of births which occurred in the year. Delayed registrations of birth account for individuals who were not registered at the time of birth and were only registered at the point where it became necessary to prove age to receive government services, for example, drivers licences, old age pensions, or to enlist in the army (Emery 1993:72-97). French (1937) gives an example from 1927 of the effect of delayed registrations of birth, first presented by the Director of Public Health in Chicago, Henry F. Vaughn:

Notwithstanding the fact that the Chicago health authorities have been making strong efforts to secure prompt returning births, the proportion of delayed returns continues to be very high, and the delays refer back in considerable proportion as far as 1916. For instance since 1919 as much as 20.7 per cent 'delayed' reports have been added to the original total of that year. That it is necessary to revise totals year by year over a considerable period of time, if anything approximating correct birth and infant mortality rates are to be had, is obvious. In Chicago original birth rates have been increased as much as three and one half points, and original infant mortality rates decreased more than 23 points for a single year. [French 1937:19-20]

This phenomenon was noticed in 1944 for Ontario by N.E. McKinnon of the Department of Epidemiology and Biometrics, School of Hygiene, University of Toronto. He cautioned that "as birth registrations in the earlier years was somewhat less complete than death registration, it is possible that infant mortality in that period might have been lower than the rates show" (McKinnon 1944:483) and he treats the infant mortality rates based on those registrations before 1920 with some reservation. Later McKinnon states that

¹³ The actual birth registrations will not become available for study until 2012-2017.

“general experience, however, would suggest that the infant mortality rates were actually higher than those recorded” (McKinnon 1945:287). In particular, Emery believes that the provincial record for 1920 was unrepresentative of the births of “females, Central European Immigrants, and [people from] northern Ontario” (1993:156). These absent records account for an unknown number of infant births in Toronto and are therefore a source of error in this project.¹⁴

When dealing with historical records, it is important to consider the contemporary understanding of disease and the conditions surrounding the recording of death. As Emery relates, “the information in the death certificates reflected influences such as the physician’s concept of disease, the design of the death certificate, and the concealment of causes the public stigmatized” (1993:136). Even earlier it was believed that “over the years, changes occur in diagnostic methods, ability, equipment, and even in fashion and therefore in diagnosis itself; in frankness and completeness of certification and even in its vogue” (McKinnon 1945:286). Especially at the beginning of the 1918 influenza epidemic, there was debate over the exact cause of the disease (CMAJ 1918c). In June 1918, English physicians were calling the disease a “pyrexia of unknown origin” (Collier 1974:8). Others thought that the disease could be anything from cholera to typhus, or they refused to identify it (Collier 1974). There was debate over the precise role of the

¹⁴ As an interesting side-note, Emery explains that it is likely that infants who died would have had their births recorded, as “after 1896, however, one had to register the death to obtain a burial permit, and the registration of the death, in turn, presumably alerted the local registrar to the birth. In the circumstances, persons who died near their day of birth probably had a better than average chance of having their birth registered” (Emery 1993:78-79).

organism *Haemophilis influenzae* and whether it could be responsible for the entire epidemic (CMAJ 1918c, Crosby 1989). By the time the second wave of the influenza epidemic hit Toronto, from September to December 1918, the epidemic had been labelled the Spanish Influenza (CMAJ 1918d), so that it is likely that epidemic related deaths would have been properly ascribed to influenza.¹⁵ However, influenza itself was not the primary killer during the pandemic; the great number of deaths resulted from the interaction in the lungs with co-infections such as streptococcus and pneumonococcus (CMAJ 1918c, Oertel 1919, Hare 1937, Horsfall 1937). Noymer writes that

Influenza kills via pneumonia, and therefore many fatal cases of influenza are recorded as pneumonia. For this reason, those who study the demography of influenza mortality look at influenza and pneumonia, combined. Some non-influenza pneumonia is included in the combined measure, but the alternative – looking at influenza, only – is such a vast underestimate of true influenza mortality that looking at the combined measure is the wiser choice. [Noymer 2006:4-5]

For the purposes of this research, deaths listed as being caused by influenza, pneumonia, broncho-pneumonia, or lobar-pneumonia (either as the primary cause of death or the immediate cause) were considered to have been caused by the flu.¹⁶ There was no

¹⁵ Although, as in section 2.2.3, influenza in infants may have presented differently than in adults (Kao et al 2000, Munoz 2003).

¹⁶ By including pneumonia deaths, this study is more directly comparable to the published statistics. In 1933 E.S. MacPhail, (Chief of the Demography Division of the Dominion Bureau of Statistics since 1920 [AJPH 1927]) gave a speech to the Canadian Public Health Association in which he outlined the Bureau's method for assigning cause of death to statistical categories (Emery 1993:170-179). He listed the rules of choice as outlined in the International List of Causes of Death, two of which are of particular importance. The first, that "if one of the two diseases is an immediate and frequent complication of the other, the death should be classified under the head of the primary disease" (Emery 1993:171). And other, that "if one disease is epidemic, and the other is not, choose the

attempt made to quantify deaths which may have been caused by the flu but were attributed to other causes of death, as there is no accurate means to establish which of these deaths were influenza-related.¹⁷

The definition of what constitutes a stillbirth adds to the confusion surrounding the cause of death of live-born infants. In the records linkage portion of this thesis, I link maternal deaths to their stillborn infants. However, the definition of stillbirth is tenuous during this period. Until 1932, the Dominion Bureau of Statistics considered a stillbirth to be any infant with a gestational age of more than six months (Robinson 1947, Emery 1993), but each province had its own legal definition of a viable fetus. In Ontario, in 1930, a stillbirth was any birth that occurred after seven months; any fetus younger than seven months would be considered a miscarriage or abortion (Emery 1993:104-105). Further, the category of live-birth in Ontario was arbitrary. In 1930, in Ontario, there were no specific criteria for declaring a birth a live-birth, and the definition of a birth as live or still was left to the physician (Emery 1993:105). This differs from other provinces, some of which required respiration, presence of a heart-beat, or voluntary movement. In the registered deaths from 1917-1921 some of this variation can be seen in the records of stillbirths where records exist for infants born anywhere between four and nine months gestation, with many between six and seven months. However, it is unlikely

epidemic" (Emery 1993:171). MacPhail states that Ontario followed both of these rules (Emery 1993:172), and both would have ensured that the death was listed as caused by the flu before pneumonia. However, reports in the *Globe* for 1918 speak of both an influenza and a pneumonia epidemic, so that deaths may have been listed as either.

¹⁷ Epidemics can cause deaths to occur from other causes more rapidly than they normally would have, through a process of 'harvesting' or 'mortality displacement' (Smith 2003, Murray et al 2006).

that all miscarriages and abortions before seven months were recorded and the level to which the stillbirth rate approximates all fetal deaths is unknown. For the flu, which was particularly severe among late-term pregnant women (Winternitz et al 1920, Beveridge 1977, Neuzil et al 1998, Reid 2005), many deaths of mothers and their viable infants may not have been recorded if the epidemic killed the women before birth could occur.

Beyond the unrecorded deaths of unborn infants, there were undoubtedly deaths of some live-born infants that were not recorded. Even though Dr. Hastings believed this was not an issue since “no cemetery has authority to open a grave unless the death is registered” (The Globe 1918p:8), unregistered deaths may have affected the quality of the records. In 1918, Wirgman explained 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 till the pressure is over, and so notes of the earlier cases may be lost for lack of leisure” (Wirgman 1918:324). Crises such as epidemics are known to have negative effects on the quality and quantity of vital event recordings due to the overwhelming number of patients per doctor (Johnson and Mueller 2002), the total number of deaths, and the much more pressing concerns of treating patients. As such, it is impossible to be certain that the infant deaths in the death records reflect the true number of infants who died during the epidemic. There is currently no means to address epidemic underreporting (or if the unreported deaths were recorded at a later date) so these infants represent an unknown number of deaths and are another potential source of error in this project

A more material source of error is the condition of the records themselves. In February and March of 1918 the records are soiled with what appears to be ink. Ten records were difficult to read and the transcriptions may contain omissions where the information was obliterated by the ink. The records of five individuals of unknown age were completely destroyed from February 1918. Further, the death registrations for Toronto for 1917-1921 were handwritten, creating problems of legibility. Fortunately, the transcriber was the same throughout all five years, except for some records in the early 1920s.

With the new forms and compilation of records provided by the Dominion Bureau of statistics in 1920, there were newly evident attempts at accuracy and representativeness. In 1920, there was a new section at the end of each quarter-year: infants listed as births but not deaths. Theoretically, these were infants who had died but had only been issued a birth registration and were not registered as deaths at the time of death. Most of these infants represent stillbirths that occurred in hospitals. Many were not transcribed for this project since they were duplicate records of infants who in actuality had been registered at the time of death. These infants were erroneously included twice in the original registers because the information in the records was different, but there were enough similarities that I was able to discern that both listings referred to the same infant. For example, the infant could be recorded with different surnames, one listed under the mother and the other under the father, or there could be spelling errors in the surnames which made them appear to be different individuals.

The change of format of the records in 1920 and 1921 brought other noticeable changes. These two years were the only ones in which the death records were intensely corrected. Many dates, spellings, and ages at death were corrected and stamped with a date of correction. The most commonly seen changes were in age at death, with “stillbirth” becoming “lived a few moments,” or a live-birth being re-defined as a stillbirth. This suggests that the records after 1920 may be more accurate than those prior to 1920 through a complex correction process, likely due to new regulations imposed by the new national Dominion Bureau of Statistics. For the purposes of this research, the corrected version of the record was transcribed as it was more likely to be accurate.

3.2 Methods

3.2.1 Transcription

The majority of the infant death records from 1917-1921 were transcribed from microfilm at the Archives of Ontario into a Microsoft Excel© 2008 spreadsheet. The remainder was transcribed from JPEG copies of the death records previously acquired by Karen Slonim (Department of Anthropology, University of Missouri) and made available for this project. Death records from 1917 were collected in order to establish a pre-epidemic baseline that includes yearly seasonal fluctuations in infant mortality from which to compare the epidemic mortality of the waves of influenza (Spring 1918, September to December 1918, Winter 1919, and the return of influenza in 1920). The infant death records for 1921 were transcribed in order to determine if there were

lingering effects of the epidemic on infant mortality, and to enable a five-year study of infant death. Data on all deaths (including adults) that occurred during September to December, 1917 and 1918 were required to link infant and parental deaths and were obtained from two sources: one provided from the previously transcribed sample of all epidemic deaths from Ann Herring and David Earn (McMaster University) for 1918, and the other from records of women who died between the ages of 15 and 50, for both 1917 and 1918, transcribed by the author.

Death records were transcribed if they met the age criterion. To be included, age at death must have been less than one year (ages listed as one year exactly were excluded). Stillbirths were also transcribed. Where age at death was listed as unknown, or not provided,¹⁸ the death record was transcribed for possible inclusion/exclusion at a later date if there was no indication that the person was not an infant. However, if the death record contained a marital status of 'married' or if the duration of the illness causing death was longer than one year, the record was not transcribed and the individual was not included in the sample. In general, records of unknown individuals were transcribed if the name was 'unknown infant' but not if the name was 'unknown child'.¹⁹

¹⁸ For infants this mostly represented 'foundling' babies who were found dead in the city

¹⁹ However, one male 'unknown child' from 1920 was included as the contributory cause of death was listed as 'cord untied, some haemorrhage,' and he was likely to have been an infant.

3.2.2 Organization

The data were originally transcribed into yearly groupings; from there, the individuals were separated into age categories: Neonatal (live born to <28 days), Postneonatal (≥ 28 days to 365 days), Stillbirth, and Unknown (for names with no age listed, but who were likely to be infants based on some other indication in the death record) (Table 3.1). Babies whose age was listed as 'premature,' 'stillbirth,' or 'newborn' were considered to be stillborn if there was no evidence elsewhere in the death record that would suggest that the infant was live-born. Infants whose age at death was 'a few moments' or 'a few minutes' were included in the neonatal category, since the infant was live-born.²⁰ Once separated, each category was organized chronologically into month of death, and sex. Each individual category was then tallied. For example, In January 1917 there were 31 male neonatal deaths and 17 female neonatal deaths. If no indication of sex was provided in the death record, one was either assigned based on a clearly gendered given name (such as Mary or John), or the individual was placed into a category of unknown sex, most commonly for severely pre-term stillborn infants. The numbers of births were taken from the Sessional Papers of the Legislative Assembly of Ontario (Legislative Assembly of the Province of Ontario 1918-1922), previously organized by month and sex (Table 3.2).

²⁰ Consistent with the arbitrary distinction between still-birth versus live-birth in the Ontario records (Emery 1993).

	Neonatal				Postneonatal				Stillborn				Un- known Age	Total			
Year	M	F	Un- known	Total	M	F	Un- known	Total	M	F	Un- known	Total	Total	M	F	Un- known	Total
1917	281	190	3	474	358	247	0	605	335	207	27	569	1	974	644	31	1649
1918	312	199	1	512	400	291	0	691	290	221	33	544	5	1002	711	39	1752
1919	281	207	5	493	358	264	1	623	335	199	34	568	2	974	670	42	1686
1920	323	230	16	569	489	337	0	826	391	263	55	709	6	1203	830	77	2110
1921	315	222	2	539	402	257	0	659	368	222	29	619	3	1084	701	34	1819
Total	1512	1048	27	2587	2007	1396	1	3404	1719	1112	178	3009	17	5237	3556	223	9016

Table 3.1 - Total Individuals Represented in the Death Registrations of the Province of Ontario for the city of Toronto Divided into Age-at-Death and Sex.

Year	Male	Female	Total
1917	6282	5828	12 110
1918	6131	5648	11 779
1919	5864	5430	11 294
1920	7027	6634	13 661
1921	7044	6334	13 378

Table 3.2 - Total Births per Year by Sex (Legislative Assembly of the Province of Ontario 1918-1922)

3.2.3 Analysis

Once the death records for the five-year period were tallied, the infant mortality profile of the city was established. This was accomplished by calculating the infant mortality rates per year, per month, per sex, per age at death, and per cause of death. The infant mortality rate is calculated as (Mausner and Bahn 1974:186):

$$\text{Infant Mortality Rate (IMR)} = \frac{\text{Total infant deaths per year}}{\text{Total live births per year}}$$

per 1000 live births.²¹

The monthly rates for neonatal mortality and postneonatal mortality were established in order to discover if there were differences in risk of death due to cause of death and age at death. As discussed earlier, neonatal mortality is the death of a live-born infant which occurs between birth and the first twenty-eight days of life and is thought to be related to conditions of pregnancy and health of the mother (WHO 2006). It is indirectly impacted by the 1918 influenza epidemic through infection of the mother. Neonatal mortality is calculated as (Mausner and Bahn 1974:187):

$$\text{Neonatal Mortality Rate} = \frac{\text{Number of deaths in a year of infants <28 days of age}}{\text{Number of live births in the same year}}$$

per 1000 live births.

²¹ The Total Infant Deaths per Year was the total of all individuals transcribed for the year and the Total Live Births per Year was drawn from the total presented in the Sessional Papers of the Legislative Assembly of Ontario for the year following the year in question. This analysis would be strengthened by a comparison with the Registered Births of the Province of Ontario; however, these records are sealed for 97 years and will not become available until 2012-2017 (Ontario Genealogical Society 2007). The infant mortality rates per month were calculated using the monthly totals for both deaths and births.

Postneonatal mortality is the death of a live-born infant which occurs between 28 days and one year of life. It is thought to be most often a result of environmental and social factors not related to birth and gestation (Frisbie 2005). Therefore, it can be affected by the epidemic through direct infection of the infant but also through the decline in the social and physical environment resulting from the infection or death of the parents and the pursuant disruption in the continuity of care. It is calculated as (Mausner and Bahn 1974):

$$\text{Postneonatal Mortality Rate} = \frac{\text{Number of deaths in a year of infants } 28 \text{ days} \geq x < 1 \text{ year of age}}{\text{Number of live births in same year}} \text{ per 1000 live births}$$

Changes in neonatal mortality, mainly caused by conditions of gestation, and increased rates of stillbirths may be a way to investigate the increased risk of infection of pregnant women (Oertel 1919; Young 1919; Winternitz et al 1920). The stillbirth rate is calculated as (Statistics Canada 2006):

$$\text{Stillbirth Rate} = \frac{\text{Number of stillbirths}}{\text{Number of total births (live and still)}} \text{ per 1000 total births}$$

The infant mortality rates were tested statistically to determine whether there were real changes in the risk of infant death. The infant mortality rates were tested statistically to determine whether there were real changes in the risk of infant death.

3.2.4 Statistics

Graphs were created in Microsoft Excel© 2008 and SPSS v.16.0.0 and were evaluated statistically using SPSS. General rates were calculated using a one-way Analysis of Variance (ANOVA) based on the monthly mortality rates. In cases where the conditions of the ANOVA were violated²² the Welch test, which can be used in situations of unequal variance, was performed. To account for outliers, the Brown-Forsythe test was also conducted, modifying the ANOVA so that the results are based on the median rather than the mean. When the ANOVA results were significant, post-hoc tests were used to identify the months of greatest variation through the year: the Tukey test was employed when the variation was homogeneous and the Games-Howell when it was not. The investigation of cause of death was conducted using nested tables in Microsoft Access© 2008.

3.2.5 Record linkage

Registered death records for parents who died during the second wave of the pandemic, September to December 1918, were linked to the deaths of their infants who died during the second wave. This was accomplished through the parental names listed on the death records for the infants, as well as from a secondary piece of identification, such as a matching address. Positive links between death records of adults and infants were defined as those that had a matching parental name and a matching address. Probable links were made when surnames matched (excluding extremely common names)

²²When a significant Levene statistic suggested that variance was not homogeneous.

but there was no secondary matching record. This usually happened when both the putative parent and child died in hospital. Potential links were those where two records likely referred to a parent and an infant, but no categories matched, due to variant spellings of surnames and where one died in hospital and the other at home. The linking of parental and infant records often required decisions on individual cases, due to inaccurate records such as misspelled surnames for the same individual. If it was not obvious that records likely referred to two family members (through surnames and place of death) then those potential links were not made. As a common problem in records linkage (Winchester 1973), this process will undoubtedly have missed some real family links and will have created some links which are not real. This analysis should therefore be considered with caution.

The parental deaths were identified in two ways. During the transcription process, if it was noticed that a parental death was listed immediately beside an infant death, the parental death was transcribed as well. In order to identify the parents who were not listed beside their infant, searches for surnames and addresses were conducted in Microsoft Excel©. These were done from databases of the deaths from 1918 from both a sample constructed by Ann Herring and David Earn and from the transcribed death registrations of women aged 15-50 who died in September to December of 1917 and 1918 collected by the author. In order to determine if there was an increase in infant deaths associated with parental deaths during the pandemic, the same process was conducted for September to December 1917, and then compared to the results for 1918.

Chapter 4

RESULTS

The purpose of this study is to address several questions about infant mortality during the 1918 influenza pandemic. To establish the background against which infant mortality in 1918 can be compared, in this chapter I first describe infant death for a five-year period surrounding the pandemic (1917-1921). In order to ascertain whether patterns of infant mortality during the 1918 epidemic differed from the five year profile, the following questions were asked; Did 1918 differ significantly from the five-year rates in terms of: 1) the infant mortality rate; 2) the sex-ratio of infant deaths; 3) the average age at death; 4) causes of infant death, or; 5) the stillbirth rate? Exploration of these questions suggests that while changes in the total infant mortality rate can be explained as random variation, the 1918 influenza epidemic may have affected the average age at death, the seasonal distribution of causes of deaths, and the number of families losing both a mother and an infant.

4.1 Infant Mortality Rate

4.1.1. Total infant mortality

The infant mortality rate for Toronto from 1917-1921, as calculated from the 6008 Registered Deaths of the Province of Ontario for this period, can be seen in Figure 4.1 as a three-month moving average for total infant mortality, neonatal and postneonatal infant mortality rates. The overall infant mortality rates for each year are presented in Table 4.1.

Year	Infant Mortality Rate (deaths per 1000 live births)	Neonatal Mortality Rate (deaths per 1000 live births)	Postneonatal Mortality Rate (deaths per 1000 live births)
1917	89.18 (n=1080)	39.14 (n=474)	49.96 (n=605)
1918	102.56 (n=1208)	43.47 (n=512)	58.66 (n=691)
1919	98.99 (n=1118)	43.65 (n=493)	55.16 (n=623)
1920	102.55 (n=1401)	41.65 (n=569)	60.26 (n=826)
1921	89.77 (n=1201)	40.29 (n=539)	49.26 (n=659)
TOTAL	96.91 (n=6008)	41.66 (n=2587)	54.98 (n=3404)

Table 4.1 - Annual Infant Mortality Rates, Neonatal and Postneonatal, Toronto, 1917-1921. Calculated from the Registered Deaths of the Province of Ontario and the Sessional Papers of the Legislative Assembly of Ontario (Archives of Ontario MS 935 and Series B 97).

The average infant mortality rate for Toronto for the five year period was 96.91 deaths per 1000 live births (N=6008, SD=19.89). The lowest infant mortality rate recorded over the five year period was in June 1921 at 61.26 deaths per 1000 live births (n=71). The highest rate occurred in October 1918, at 147.84 deaths per 1000 live births (n=144). Figure 4.1 suggests a seasonal pattern for infant mortality. For each year, the highest infant mortality rate was found between August and October with annual peaks noted for September 1917 (114.34 deaths per 1000 live births, n=114), October 1918 (147.84 deaths per 1000 live births, n=144), August 1919 (121.54 deaths per 1000 live births, n=101), October 1920 (135.78 deaths per 1000 live births, n=137), and for September 1921 (137.33 deaths per 1000 live births, n=149). While the month with the lowest infant mortality rate each year was more variable, infant mortality consistently dropped during the summer months of June and July.

The second wave of the influenza epidemic in October 1918 was associated with the highest infant mortality rate over the five year period (147.84 deaths per 1000 live

births), but large increases in infant mortality also occurred in October 1920 (135.78 deaths per 1000 live births) and in September 1921 (137.33 deaths per 1000 live births). A one-way Analysis of Variance (ANOVA) to test the hypothesis that the infant mortality rate did not vary by year (1917 vs. 1918 vs. 1919 vs. 1920 vs. 1921) was accepted: no significant differences were detected in the annual rates of infant death over the five-year period ($p=.238$, $SD=19.887$, $df=59$, 95% CI).

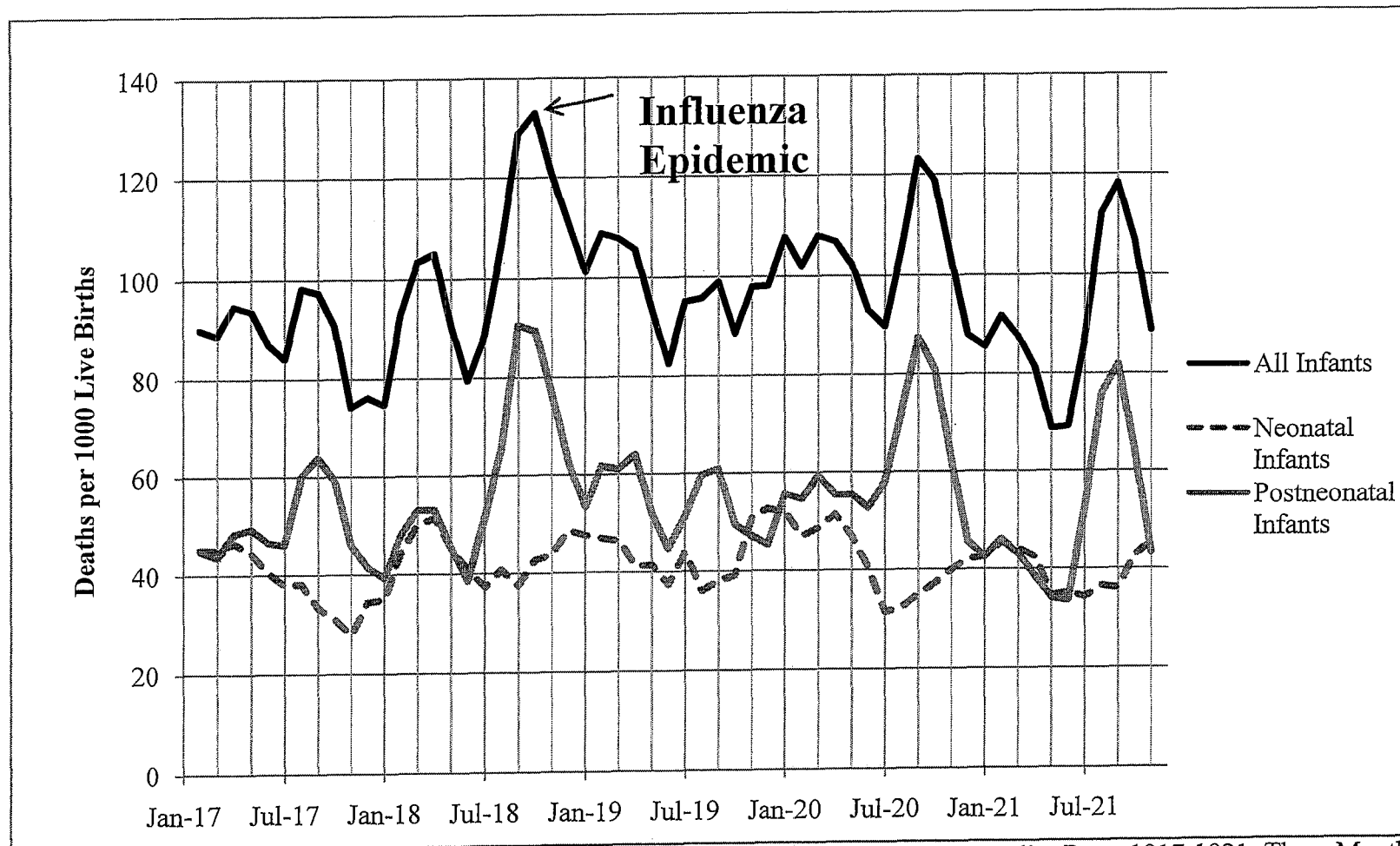


Figure 4.1 – Total Infant Mortality Rate, Postneonatal Mortality Rate, and Neonatal Mortality Rate, 1917-1921: Three Month Moving Average.

The average neonatal infant mortality rate for the five years was 41.66 deaths per 1000 live births ($n=2587$, $SD=8.50$), with a low of 19.50 deaths per 1000 live births in October 1917 ($n=18$) and a high of 61.22 deaths per 1000 live births in December 1919 ($n=57$). The one-way ANOVA analysis of the yearly neonatal infant mortality rate supports the null hypothesis that neonatal infant mortality did not change over the five-year period ($p=.591$, $SD=8.50$, $df=59$, 95% CI).

The postneonatal infant mortality rate more closely mirrors the pattern for total infant mortality and drives the shape of the distribution due to the higher proportion of postneonatal infant deaths in the overall rate (2587 neonatal infant deaths versus 3404 postneonatal infants, a ratio of 0.76 to 1).²³ The average postneonatal infant mortality rate was 54.98 deaths per 1000 live births ($n=3404$, $SD=18.67$), ranging from a low of 31.92 deaths per 1000 live births in May 1921 ($n=37$) to a high of 113.96 deaths per 1000 live births in October 1918 ($n=111$). A one-way ANOVA test failed to reveal significant deviations in postneonatal infant mortality by year ($p=.471$, $SD=18.67$, $df=59$, 95% CI).

4.1.2 Monthly variation in infant mortality

Even though no differences were detected in the annual rate of infant mortality over the five-year study period, seasonal fluctuations in the infant mortality rate, evident in Figure 4.1, proved to be significant when a one-way ANOVA test was performed

²³ The ratio of neonatal infants to postneonatal infants varies per month with the infant mortality rates, ranging from 32 neonatal deaths to 111 postneonatal deaths in October 1918 (ratio=0.29 to 1), to 43 neonatal deaths to 29 postneonatal deaths in June 1919 (ratio=1.48 to 1).

($p=.006$, $SD=19.887$, $df=59$, 95% CI). However, the Levene statistic, which tests for the homogeneity of variance, was significant ($p=.011$, $df=11$, 48), thereby violating a condition of the ANOVA test. The Welch test, which can be used in situations of unequal variance, was therefore performed. The Welch test supported the findings of the ANOVA ($p<.001$, $df=11$, 17.470) indicating that there were significant seasonal differences in infant death. A post-hoc Games-Howell test revealed that the significant differences resided between August and June ($p=.06$) and August and July ($p=.023$).

Figure 4.2 partitions total infant mortality rates into neonatal and postneonatal components, by month and season, for the five-year period (1917-1921).²⁴ The neonatal infant mortality rate remains relatively stable throughout the year, ranging from an average low of 35 per 1000 ($n=174$) live births in October to an average high of 49 per 1000 ($n=277$) live births in March with a yearly average of 41.66 ($n=2587$, $SD=8.43$). In contrast, the postneonatal infant mortality rate by month varies greatly throughout the year. This is not surprising, given that postneonatal mortality accounts for most of the overall pattern of infant death over the year. The lowest postneonatal mortality rate was found in June, at 38.02 per 1000 live births ($n=205$) and the highest in September, at 82.53 per 1000 live births ($n=428$), while the average over the whole year was 54.98 per

²⁴ Knodel (1988) cautions against using infant mortality rates to determine seasonality of death due to the possible confounding variable of the seasonality of births. He explains that "since a high proportion of infant deaths occur within a few weeks after birth, the extent of influence of monthly fluctuations of births on monthly fluctuations in infant deaths can be substantial" (1988:60). For this sample, the raw number of neonatal deaths per month were compared to the raw number of births per month and it was found that the seasonality of births accounts for only 26 percent of the variation in the seasonality of neonatal deaths ($r^2=0.264$, Appendix B). Therefore, the uncorrected infant mortality rates per month have been used in this analysis.

1000 live births ($n=3404$). A one-way ANOVA test of the monthly variation in postneonatal infant mortality rate proved to be significant ($p<.001$, $SD=18.67$, $df=59$, 95% CI). The post-hoc Games-Howell test revealed that the most significant monthly differences were centered on the month of August, with the greatest difference residing between June and August ($p=.014$). Postneonatal infant mortality is elevated from late summer to early autumn.

The total infant, neonatal and postneonatal mortality rates did not change year by year from 1917-1921. This means that infant mortality in 1918 was not significantly different from the years surrounding it. There are, however, yearly idiosyncrasies. In 1918, for example, the total infant mortality rate is elevated in October during the height of the second wave of the influenza epidemic (Figure 4.3).

October 1918 stands out as a time of elevated infant mortality (147.84 per 1000 live births) throughout the five year period, even though it is within two standard deviations of the mean for 1918 ($M=102.82$, $SD=24.2$) (Figure 4.4).

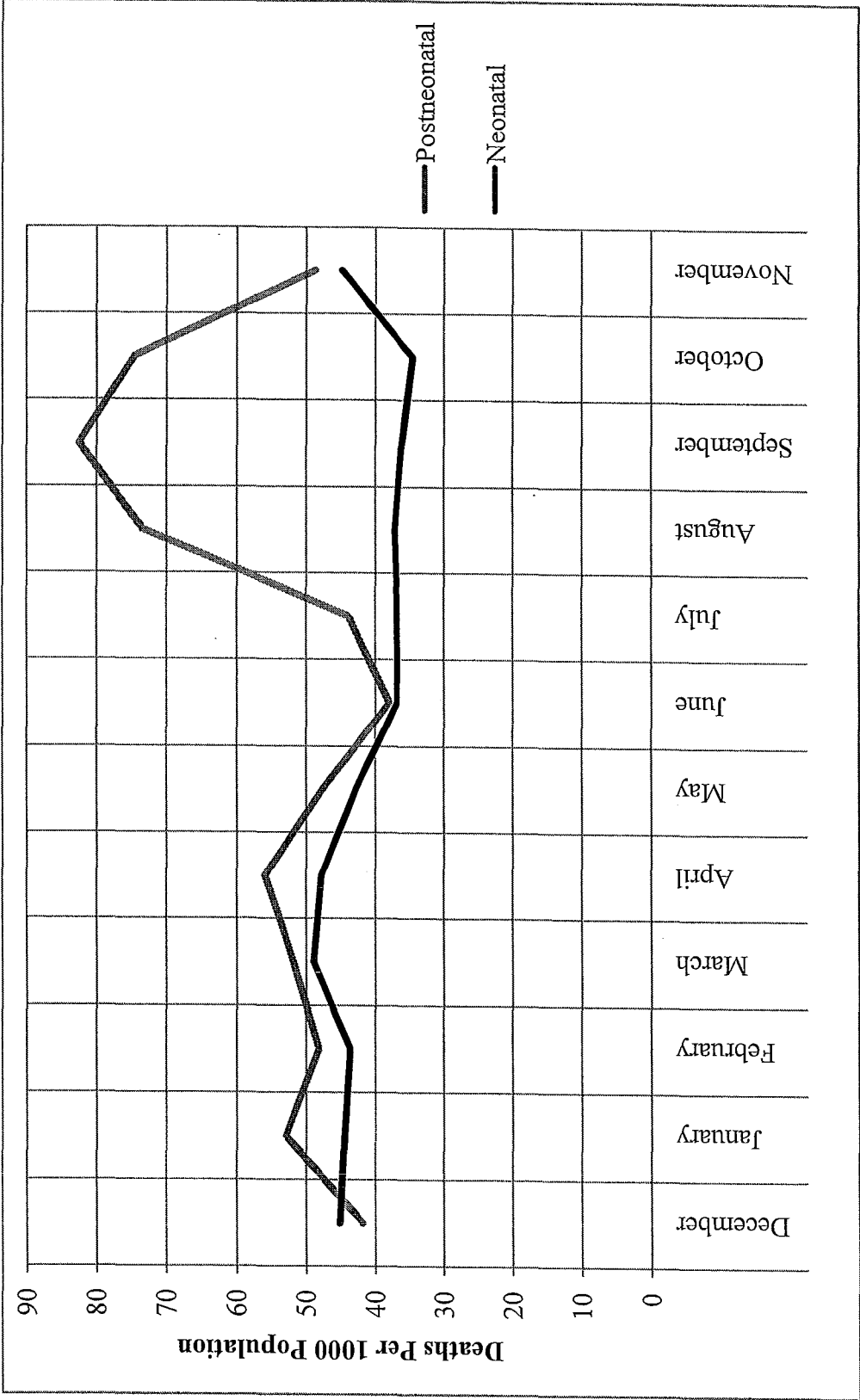


Figure 4.2 - Average Infant Mortality by Month, Postneonatal and Neonatal, 1917-1921.

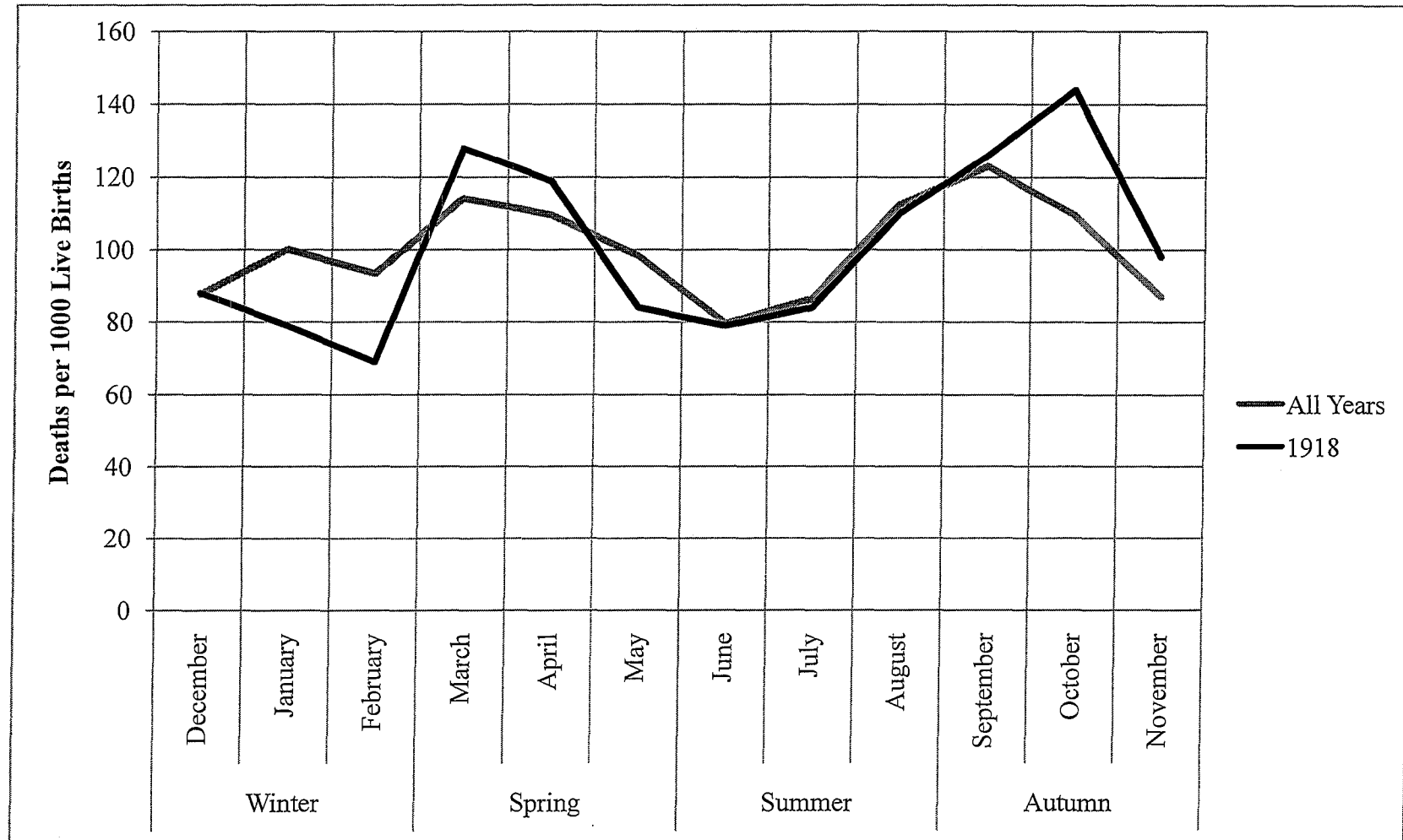


Figure 4.3 - Total Infant Mortality Rate by Month, 1918 compared to an average of 1917-1921.

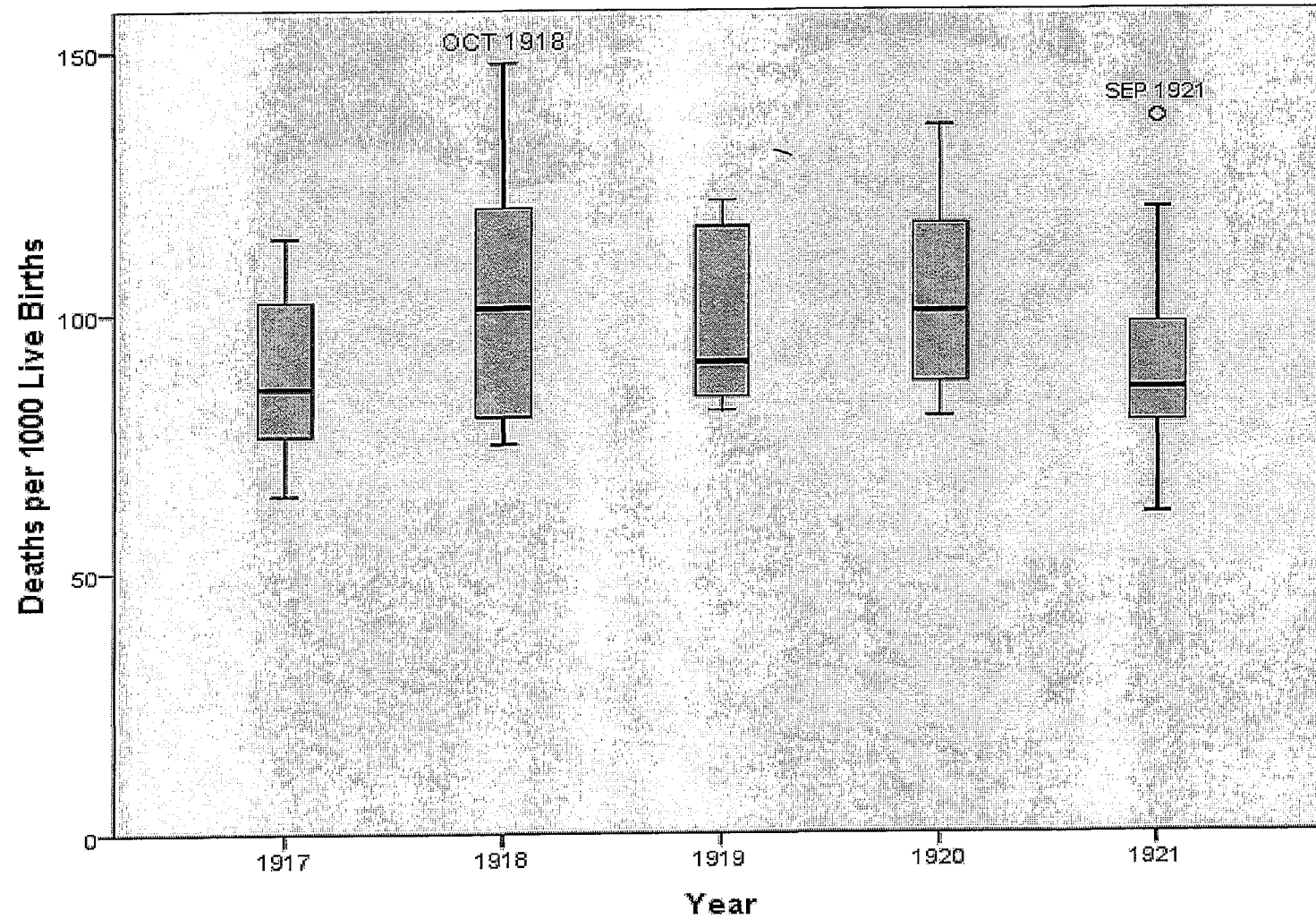


Figure 4.4 - Total Infant Mortality by Year: Box Plot.

4.2 Sex-Ratio at Death

Once it was determined that there were no significant yearly changes in the infant mortality rate in Toronto during the five-year study period, I investigated the data to determine whether there were subtler, yet equally important effects of the epidemic, on other aspects of infant death. I examined the sex-ratio at death to discover if sex-based differences in life led to any inequality in risk of death. To do this, I tested the null hypothesis that the sex-ratio at death did not alter throughout the five-year period; the alternative hypothesis was that there was an unequal risk of death based on sex.

Currently, the living sex-ratio of males to females for “the white population of the United States” is estimated to be 105 males to 100 females (1.05:1) (Cavalli-Sforza and Bodmer 1971, Knodel 1988). Table 4.2 presents the sex-ratio at birth and the sex-ratio at death for Toronto for the five-year study period. The sex-ratio at birth was calculated using the total births found in the Sessional Papers of the Legislative Assembly of Ontario (Legislative Assembly of Ontario 1918-1922). The sex-ratio at death was calculated from the death registrations.

Year	Sex-Ratio at Birth (M:F)	Sex-Ratio at Death (M:F)
1917	1.08:1 (n=12 110)	1.36:1 (n=1080)
1918	1.09:1 (n=11 779)	1.32:1 (n=1208)
1919	1.08:1 (n=11 294)	1.26:1 (n=1118)
1920	1.06:1 (n=13 661)	1.34:1 (n=1401)
1921	1.11:1 (n=13 378)	1.35:1 (n=1201)

Table 4.2 - Live Birth Male-to-Female Sex-Ratios and Sex-Ratios at Death, Toronto, 1917-1921.

As can be seen from the male-to female sex-ratios at death, there was a general selection bias towards males over the five-year period, 1917-1921. This is to be expected, given the generally higher male death rate at all ages of life, and in light of factors influencing infants specifically, such as the stronger female immune system and the higher proportion of male infants born pre-term (Drevenstedt et al 2008). Further, the Canada-wide average sex-ratio at death from 1921-1925, was found to be 126 males to every 100 females, ranging from 122 to 128 males per females (for eight reporting provinces) (MacPhail 1927). The average ratio for Toronto for the five year period was 1.38 male deaths for every one female death ($n=5980$, $SD=0.30$), ranging from a low of 0.8:1 in October 1919 ($n=96$) to a high of 2.35:1 in March 1917 ($n=83$). Although somewhat higher than the later Canadian average, the Toronto figures represent a slightly earlier time period (in a context of improving mortality) and an urban population, while the Canadian figures are both rural and urban. Urban populations had higher rates of infant mortality generally, which could be driving these figures upwards (Williams and Galley 2005). The Toronto sex-ratios at death during the five-year period therefore appear to be consistent with the Canadian average.

The month in which the highest male-to-female ratio was found varied each year.²⁵ However, interestingly, the lowest male-to-female ratio at death was more consistent, occurring in October in three of the years. For two of those years (1918 and

²⁵ 1917: March, 2.35:1, ($n=83$); 1918: February, 1.93:1, ($n=69$); 1919: September, 1.65:1, ($n=88$); 1920: May and November, 1.62:1, ($n=136$, 87); 1921: November, 2.19:1, ($n=89$).

1919) more females died in October than males.²⁶ A one-way ANOVA test found that the differences between the years was not significant ($p=.895$, $SD=0.30$, $df=59$, 95% CI), while a one-way ANOVA test for the monthly sex-ratios at death was found to be significant ($p=.042$, $SD=0.30$, $df=59$, 95% CI). A post-hoc Tukey test revealed that the main difference was found between October and November ($p=.036$).²⁷ However, when October 1918 and 1919 were removed from the analysis, the monthly variation in the sex-ratio at death was not significant ($p=.163$, $SD=0.28$, $df=57$, 95% CI). The 1918 influenza epidemic altered the normal patterning of the sex-ratio at death by increasing the number of females who died in October compared to males.

Separating the total infant mortality rate into neonatal and postneonatal infant deaths does not add information to the sex-ratio at death analysis. Both the neonatal and postneonatal infant mortality varied throughout the months, and generally followed the pattern of total sex-ratio at death. No significant differences were found when either was compared by year or by month. However, it is interesting to note the similar ratios found among neonatal and postneonatal infants during October 1918, the worst month of the epidemic. Specifically, across infant deaths at all ages, more females died than males (Table 4.3, Figure 4.5).

²⁶ 1917: February, 1.04:1, ($n=89$); 1918: October, 0.89:1, ($n=144$); 1919: October, 0.8:1, ($n=96$); 1920: October, 1.04:1, ($n=136$); 1921: December, 1.08:1, ($n=85$). Except for August 1919 (ratio=0.98, $n=101$), October 1918 and October 1919 were the only months in which more females died than males.

²⁷ For the five year combined, the lowest average male-to-female sex-ratio at death was found in October (1.05:1, $n=549$) and the highest in November (1.67:1, $n=434$, Figure 4.6).

Month (1918)	Neonatal Infants	Postneonatal Infants	All Infants
September	1.17:1 (n=45)	1.93:1 (n=80)	1.63:1 (n=126)
October	0.82:1 (n=32)	0.85:1 (n=111)	0.89:1 (n=144)
November	1.20:1 (n=39)	1.26:1 (n=59)	1.33:1 (n=98)
December	1.66:1 (n=80)	1.09:1 (n=41)	1.44:1 (n=88)

Table 4.3 - Sex Ratios at Death, Epidemic Period: September to December, 1918.

The male-to-female sex-ratios at death for Toronto did not alter greatly throughout the five year period 1917-1921 and, although slightly higher (1.38:1), were comparable to ratios for Canada for the subsequent five-year period (1:26:1), reported by MacPhail (1927). As can be seen in Figure 4.6, the pattern of variation in the sex-ratio at death in 1918 was not substantially different from the average of the five-years combined.

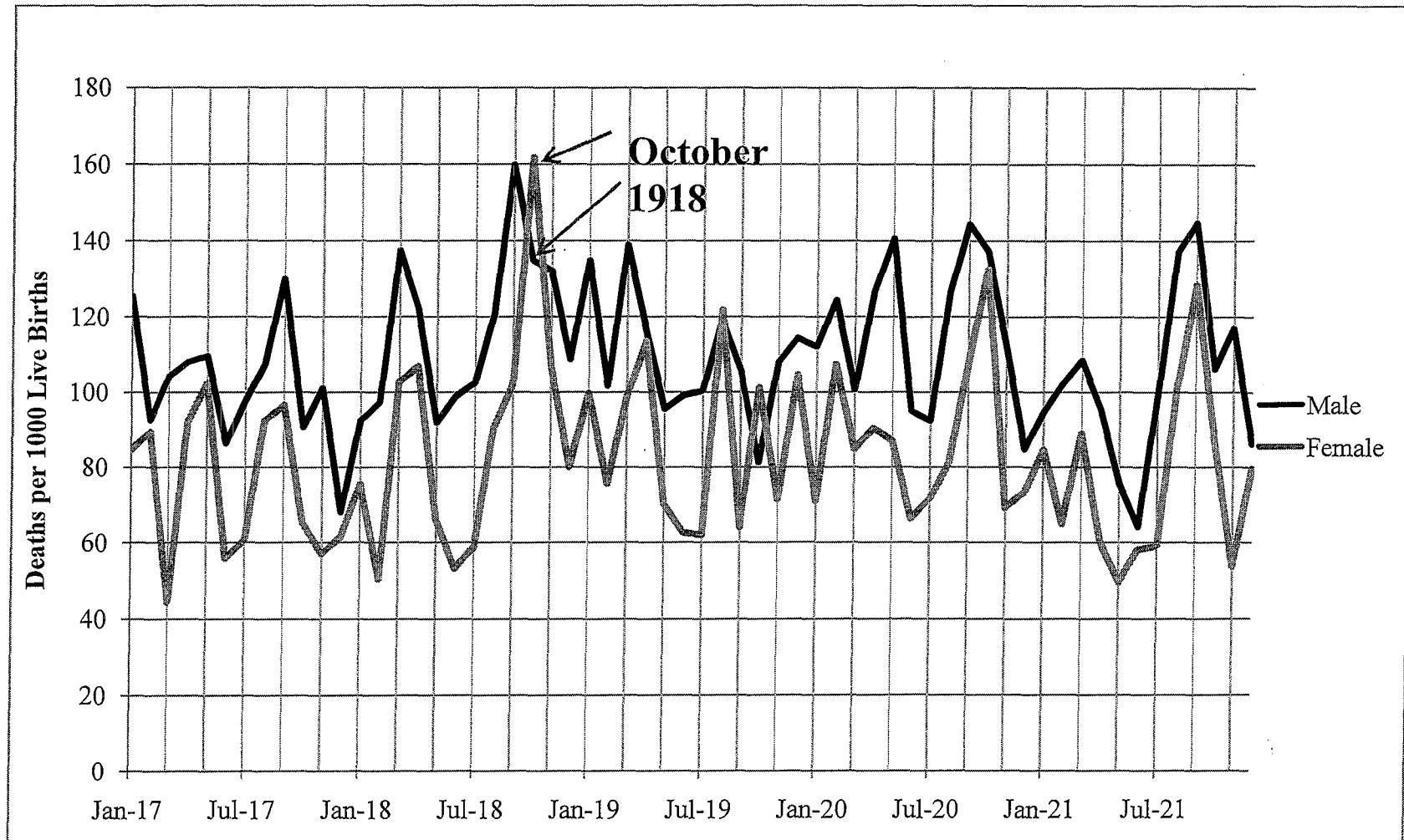


Figure 4.5 - Male and Female Infant Mortality Rates, 1917-1921.

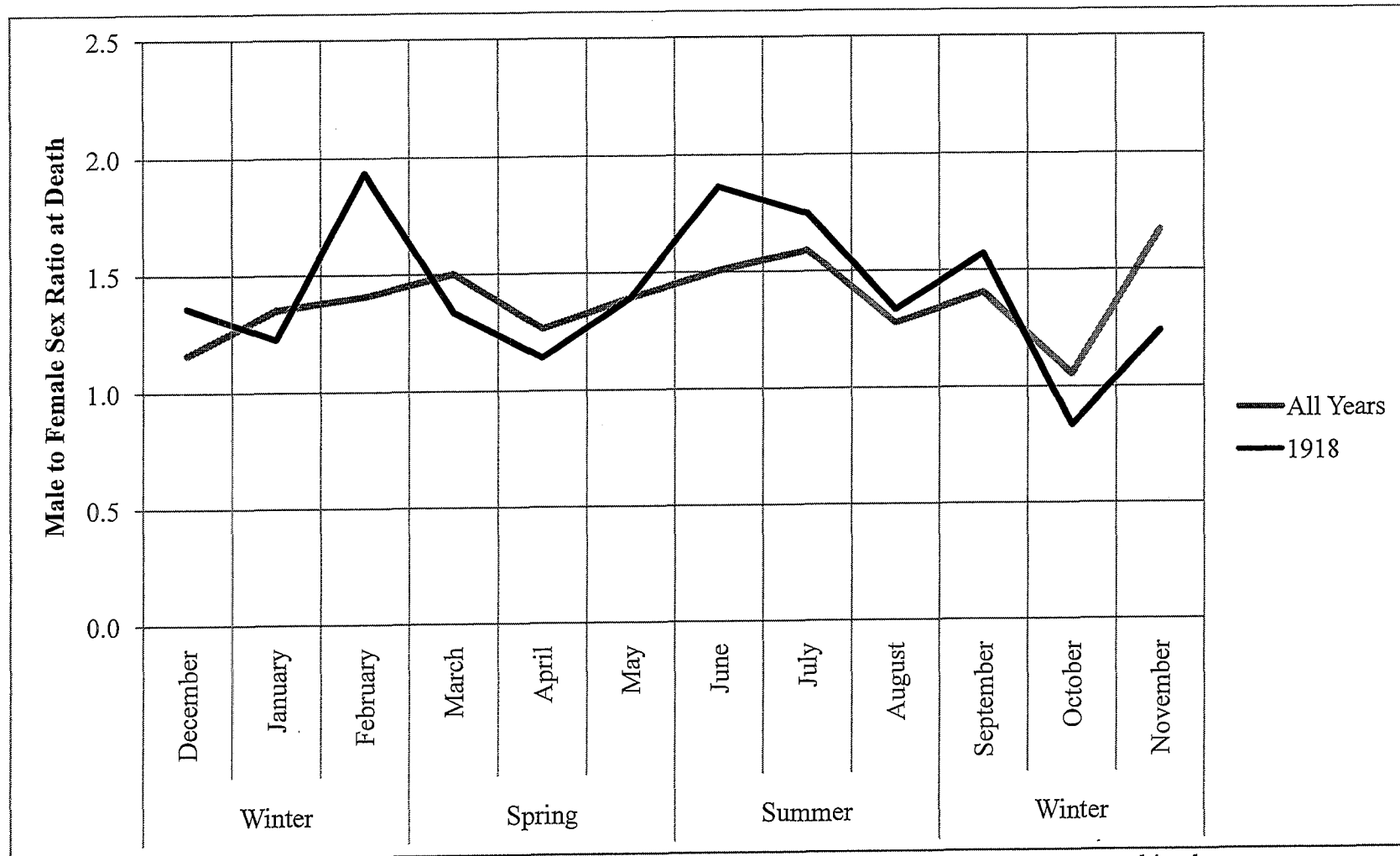


Figure 4.6 – Male to Female Sex Ratio at Death by Month, 1918 compared to average of 1917-1921 combined.

4.3 Age at Death

4.3.1 Total infant age at death

The average age at death for all infants, postneonatal, and neonatal aged infants is shown in Figure 4.7 as a three month moving average. The average age at death for all infants ranged from a low of 59.35 days in November 1921 (n=85) to a high of 132.83 days in September 1921 (n=147), with the average age for the entire five year period being 86.72 days (n=5928, SD=16.61). No significant differences were detected in the average age of infant death from 1917-1921 ($p=.558$, SD=16.610, df=59, 95% CI).

The average neonatal age at death is relatively stable, with death occurring during the first seven days of life (the perinatal period, WHO 2006) for all but three months during the sixty month period.²⁸ The average neonatal age at death for the whole five year period was 6.18 days (n=2521, SD=1.22), ranging from a low of 3.8 days in September 1921 (n=36) to a high of 8.7 days in January 1921 (n=46). A one-way ANOVA revealed that there were no significant yearly differences in neonatal infant mortality over the study period ($p=.463$, SD=1.215, df=59, 95% CI).

The yearly average age at death for the postneonatal infants follows the average age at death for all infants more closely than does the neonatal aged deaths, but not precisely. The average postneonatal age at death for all five years was 148.55 days (n=3407, SD=15.10), ranging from a low of 117.27 days in December 1921 (n=41) to a high of 181.95 days in March 1917 (n=36). Similar to the neonatal yearly age at death, a

²⁸ For the three months with an average age at death of more than eight days, August 1917, April 1919, and January 1921, none exceeded nine days.

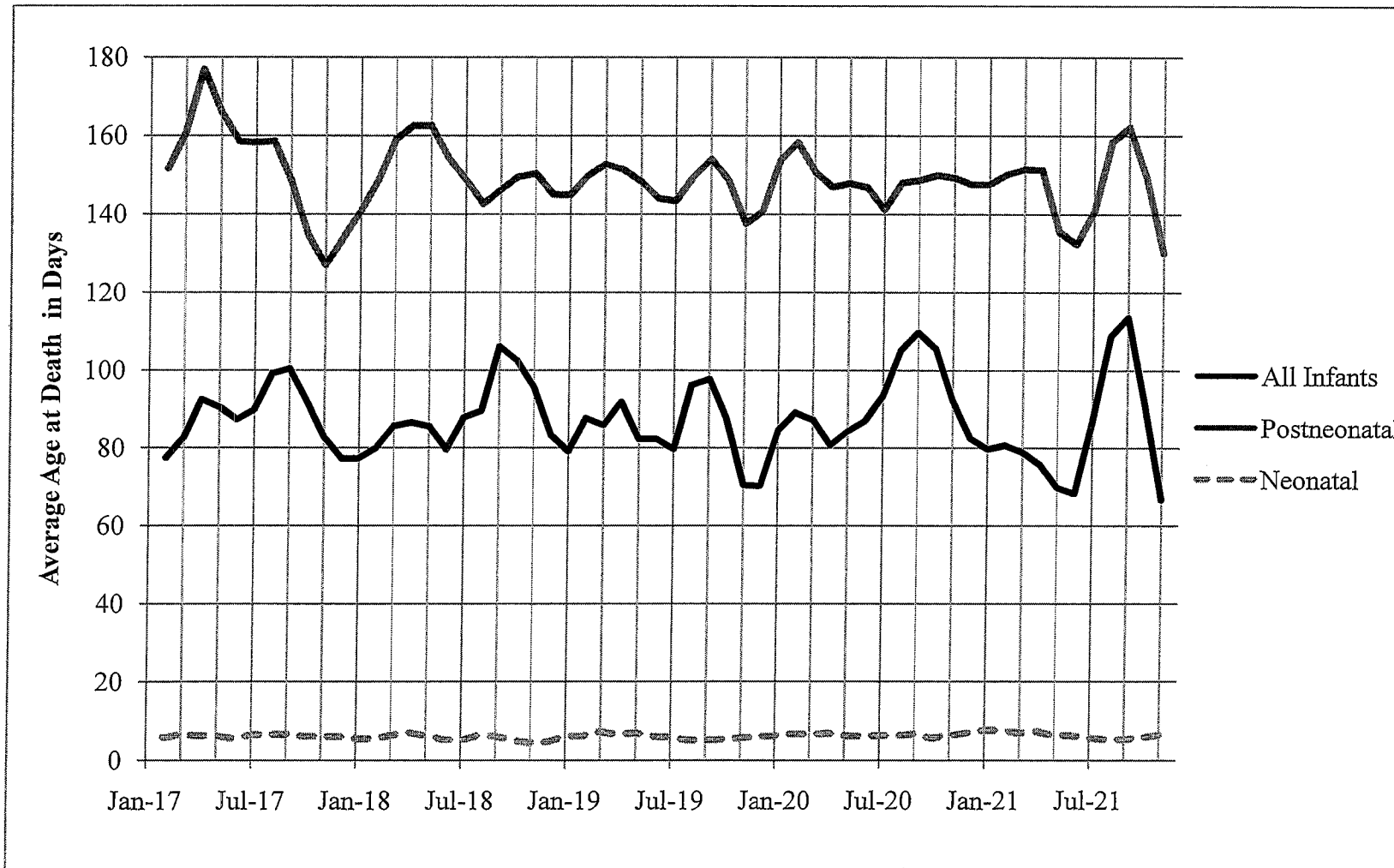


Figure 4.7 – Three month moving average of infant age at death (in days), 1917-1921, All infants, Postneonatal, and Neonatal.

one-way ANOVA test showed no significant variation in postneonatal age at death ($p=.670$, $SD=15.096$, $df=59$, 95% CI).

4.3.2 Monthly variation in age at death

Each year, the highest average age at death for the year occurs in September, except for 1918, when it occurred in October (Table 4.4).²⁹ The month with the lowest average age at death for each year was more variable; however, it always occurred between November and March (Figure 4.8). The average age at death decreased during the winter months, meaning that on average, infants were dying at younger ages in the winter.

Year	Lowest Age at Death (days)	Highest Age at Death (days)
1917	February: 65.01 (n=89)	September: 104.65 (n=114)
1918	December: 65.91 (n=88)	October: 126.58 (n=143)
1919	November: 60.4 (n=87)	September: 112.97 (n=87)
1920	March: 75.6 (n=139)	September: 122.24 (n=135)
1921	November: 59.35 (n=85)	September: 132.83 (n=147)

Table 4.4 - Lowest and Highest Average Age at Death, Toronto, 1917-1921.

When a one-way ANOVA test was conducted comparing the average age of death of each month over the five-year period, a significant difference was found ($p<.001$, $SD=16.610$, $df=59$, 95% CI). A post-hoc Tukey test revealed that the most significant changes in the average age at death occurred during the month of September of each year, with the largest difference occurring between September and December ($p<.001$). This

²⁹ This does not appear so in Figure 4.7 because the chart has been smoothed through a three month moving average. The pattern of 1918 is better seen in Figure 4.8.

supports the alternative hypothesis that the average age at death varied significantly during the year: older infants were more likely to die in the autumn while younger infants were at greater risk of dying in the winter.

There is little consistency in the months with the highest and lowest average age at death for the neonatal age range each year. A one-way ANOVA test showed that this monthly variation was not significant ($p=.298$, $SD=1.215$, $df=59$, 95% CI). Postneonatal mortality also shows considerable variation in monthly highs and lows in ages at death. A one-way ANOVA test found no significant difference in the postneonatal age at death by month ($p=.078$, $SD=15.096$, $df=59$, 95% CI). While the analysis for the average age at death for all infants found significant variation by month, the difference disappeared when age was partitioned into neonatal and postneonatal components. This is a result of the increased postneonatal infant mortality rate (section 4.2.1) and represents a seasonal variation in the total ratio of neonatal to postneonatal deaths, the cause of which will be explored in section 4.4

Figure 4.8 provides the average age at death for 1918 compared to the average for all five years combined. No significant difference was detected ($p=.558$, $SD=16.61$, $df=59$, 95% CI), despite the fact that the month which records the largest increase in the average age at death was different in 1918 relative to the other five years combined: the seasonal pattern was the same until September of 1918.

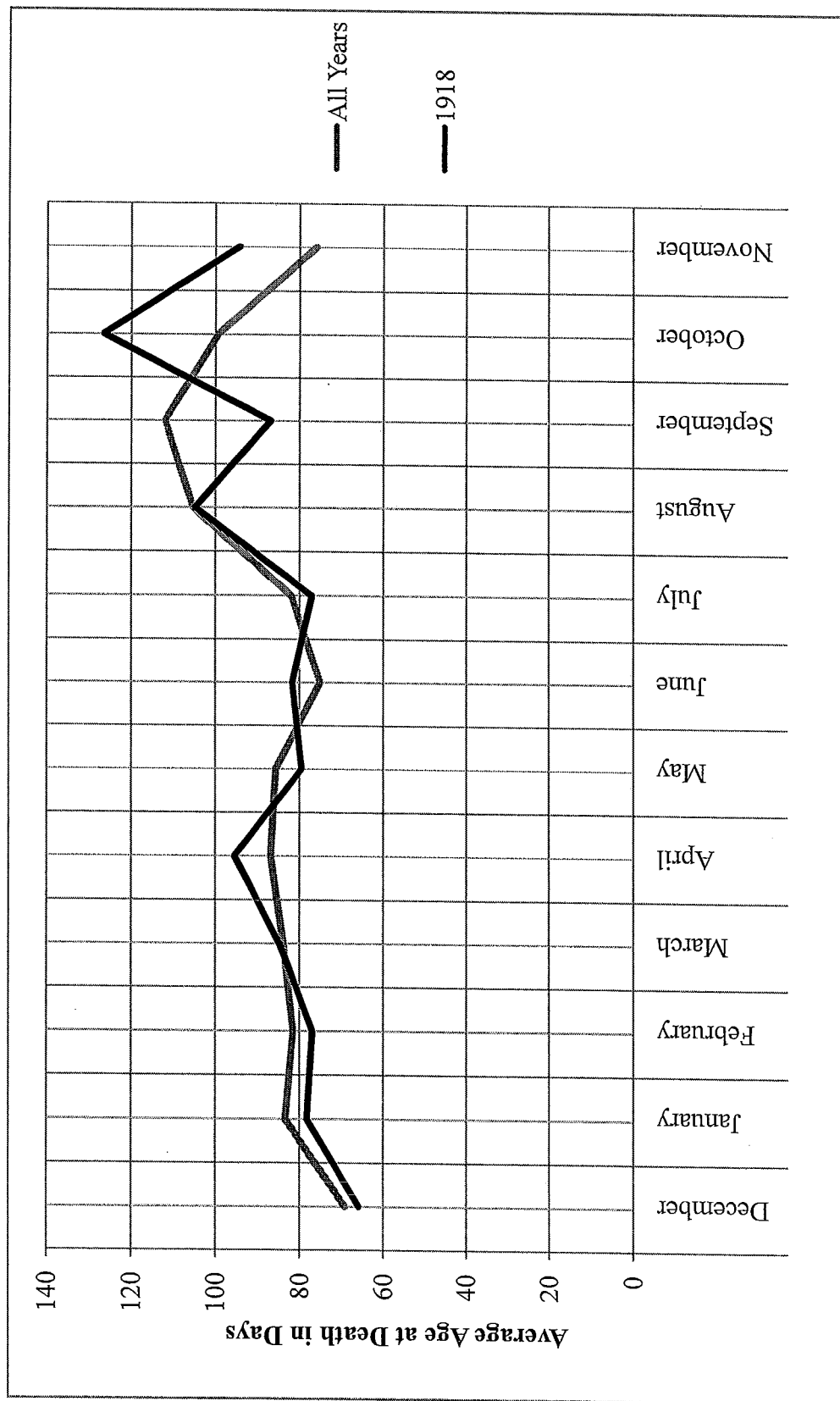


Figure 4.8 - Average Age at Death, by Month. All years (1917-1921) as compared to 1918 alone.

Following a suggestion by Dr. A. Gagnon (Department of Sociology, University of Western Ontario), the pattern of age at infant death is represented in Figure 4.9 as the percent of the population who died in that year who were alive after each month of age,³⁰ plotted on a logarithmic scale. The mortality curve for 1918 is similar to the other years up until the third month of age, after which, at each successive month, there are fewer infants alive than in the other years. The pattern of decline is especially marked for the seventh through ninth months of life. The mortality curve for 1919 is the most similar year to 1918, although more infants lived for a longer period in 1919 than in 1918. This analysis confirms that there was little change in the age at death among the neonatal aged infants and suggests that the 1918 influenza epidemic had the greatest effect on infants older than three months, and especially on those between the ages of seven and nine months. This hints at a possible link between influenza deaths and the weaning process, as will be discussed in Chapter 5.

³⁰ One-hundred percent of the sample is alive at birth and zero are alive at the end of one-year.

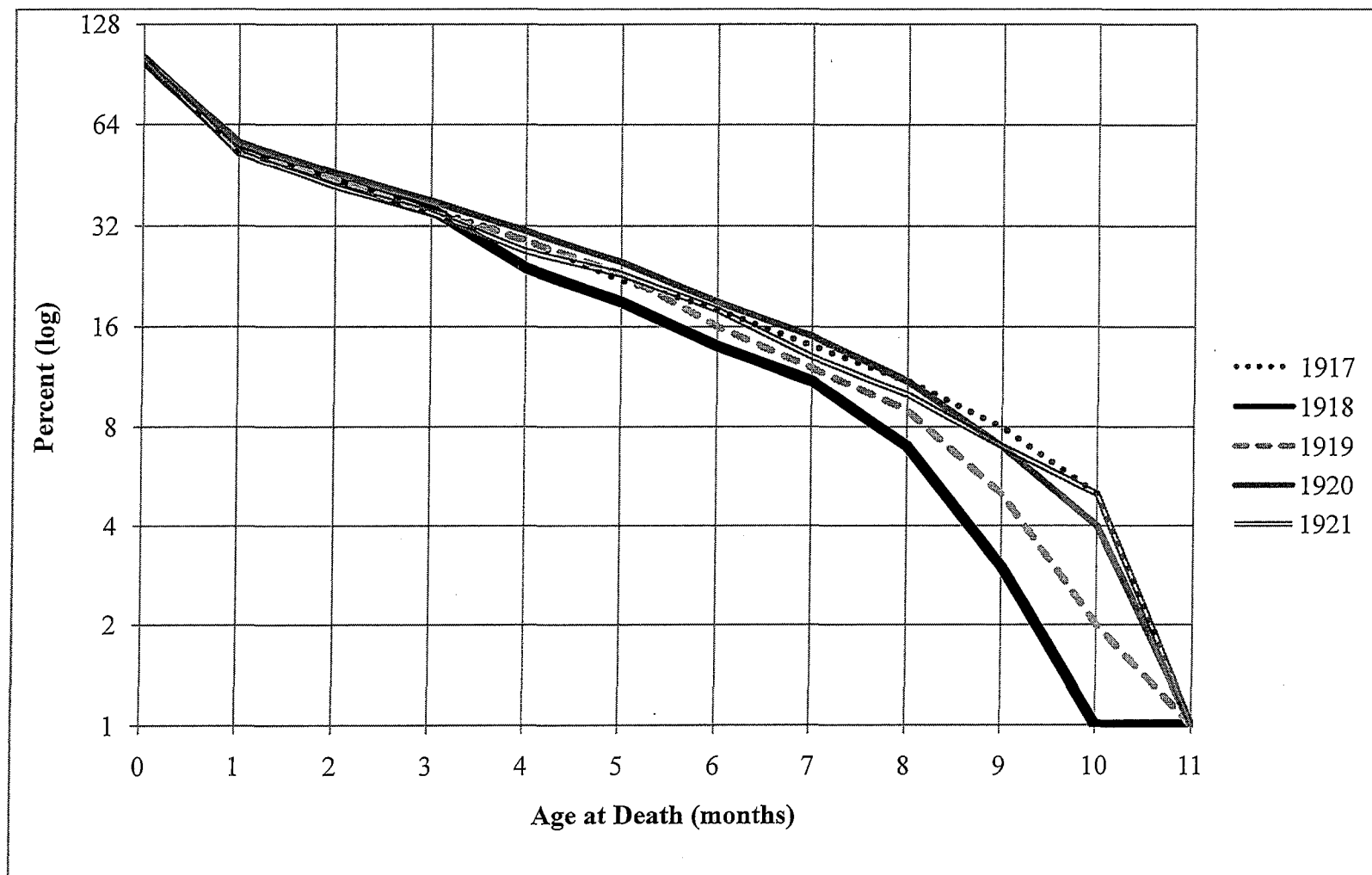


Figure 4.9 - Age at Death in Months - Percent Alive at the End of Each Month after Birth: Logarithmic Scale.

4.4 Cause of Death

4.4.1 All causes of death

Figure 4.10 shows all infant deaths from January 1917 to December 1921 partitioned into infectious causes and all other causes. The results are presented as a cause specific infant mortality rate in a three month moving average.³¹ The average death rate from infectious causes during the five year period was 37.43 deaths per 1000 live births (n=2327, SD=15.23) ranging from a low of 16.59 deaths per 1000 live births in November 1917 (n=15) to a high of 75.98 deaths per 1000 live births in October 1918 (n=74). A one-way ANOVA test for the infectious causes of death showed that there was no significant yearly variation over the five year period (p=.503, SD=15.23, df=59, 95% CI).

The monthly period in which the death rates from infectious causes rose during each five year period is presented in Table 4.5, along with the month which recorded the highest death rate from infectious causes.

³¹ Although it was shown in section 4.1 that the rise in the total infant mortality rate during the epidemic was a result of the increase in the postneonatal infant rate, all infant deaths are included in the cause of death analysis due to the low sample sizes of infectious deaths. Deaths by infectious causes are largely postneonatal deaths and the neonatal deaths do not distort the analysis. The infectious deaths are similarly not divided by sex due to low sample sizes.

Year	Period of Increase in Infectious Causes of Death	Month with Highest Rate of Infectious Causes of Death (rate per 1000 live births)
1917	August - September	August: 51.52 (n=51)
1918	March –April, September-November	October: 75.98 (n=74)
1919	August-October	August: 48.13 (n=40)
1920	February, August-October	February: 69.99 (n=80)
1921	August-September	September: 72.81 (n=79)

Table 4.5 - Period of Annual Increases in Deaths from Infectious Causes and Month with Highest Rate of Death, 1917-1921.

A one-way ANOVA test found this monthly variation in infectious causes of infant mortality to be significant ($p < .001$, $SD = 15.23$, $df = 59$, 95% CI). The Levene statistic was significant ($p = .022$, $df = 11, 48$) violating the requirement of the homogeneity of variables, yet the significant Welch test ($p = .01$, $df = 11, 18.752$) supports the finding of the ANOVA. A post-hoc Games-Howell test found that most of the variation occurred between June and July, and August and September. This supports the alternative hypothesis that there was some variation in the rates of deaths from infectious causes, but suggests a regular seasonality of infectious diseases rather than support for the hypothesis that the 1918 influenza epidemic altered the expected pattern of infant death in Toronto (Figure 4.10). These elevations in the infectious cause of death rate are consistent with the seasonality of infant death suggested in section 4.1 (Figure 4.2).

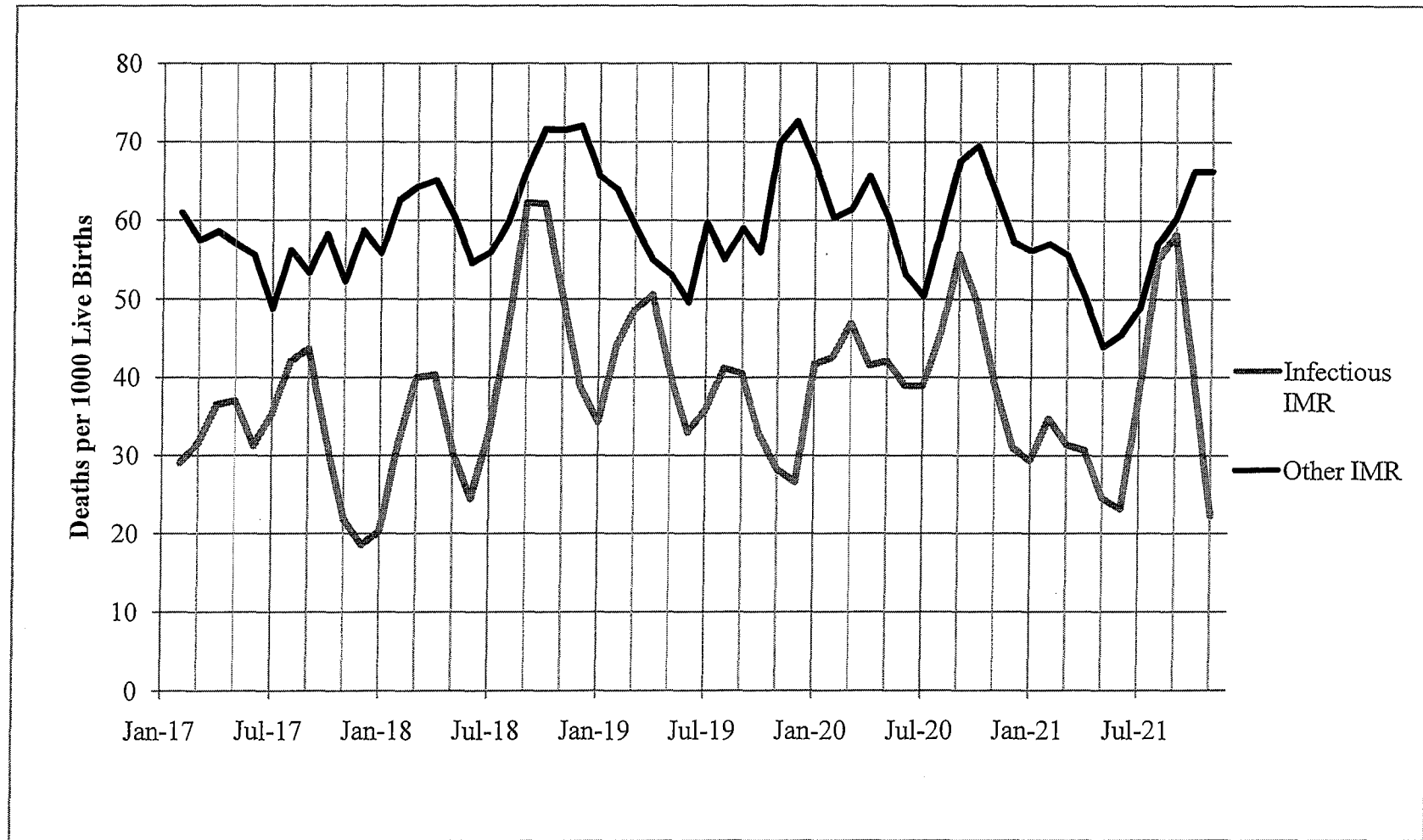


Figure 4.10 - Cause of Death - Infectious versus Other Causes, 1917-1921: Three Month Moving Average.

4.4.2 Infectious causes of death

To explore the infectious causes of death in more detail, I created the three categories shown in Figure 4.11: airborne diseases, food and waterborne diseases, and all other infectious causes of death (see Appendix C).³² The average infant mortality rate for the five year period due to airborne infectious diseases was 20.85 deaths per 1000 live births ($n=1296$, $SD=12.3$),³³ with a low of 3.98 deaths per 1000 live births in October 1921 ($n=4$), to a high of 62.63 deaths per 1000 live births in October 1918 ($n=61$). This variation was not significant (one-way ANOVA: $p=.167$, $SD=12.3$, $df=59$, 95% CI; Welch: $p=.155$, $df=4$, 26.844). This supports the null hypothesis that there was no variation per year in the rates of airborne infectious disease in Toronto. The average infant mortality rate for the five year period due to food and waterborne infectious diseases was 12.21 deaths per 1000 live births ($n=758$, $SD=13.59$), ranging from a low of 0.95 deaths per 1000 live births in May 1918 ($n=1$) to a high of 58.06 deaths per 1000 live births in September 1921 ($n=63$). A one-way ANOVA test showed that the yearly variation in food and waterborne illnesses was not significant ($p=.894$, $SD=13.58$, $df=59$, 95% CI).

³² The infectious causes of death were divided in this manner as per McKeown (1976), Moffat (1992), Gray (1997) and based on the causes of disease in the Forty-Forth Annual Report of the Hospital for Sick Children in Toronto (1919), and the International Statistical Classification of Diseases and Related Health Problems, 10th Revision Version for 2007 (ICD-10 v.2007) (WHO 2007). The analysis was conducted following Padiak (2004).

³³ These results must be interpreted with caution due to the low sample sizes. The range of infant deaths due to airborne infectious diseases spans a low of 4 deaths in October 1921 to a high of 65 in February 1920. Likewise, the total number of infant deaths due to food and waterborne causes ranged from a low of 1 death in May 1918, February 1919, and December 1920 to a high of 64 deaths in August 1921.

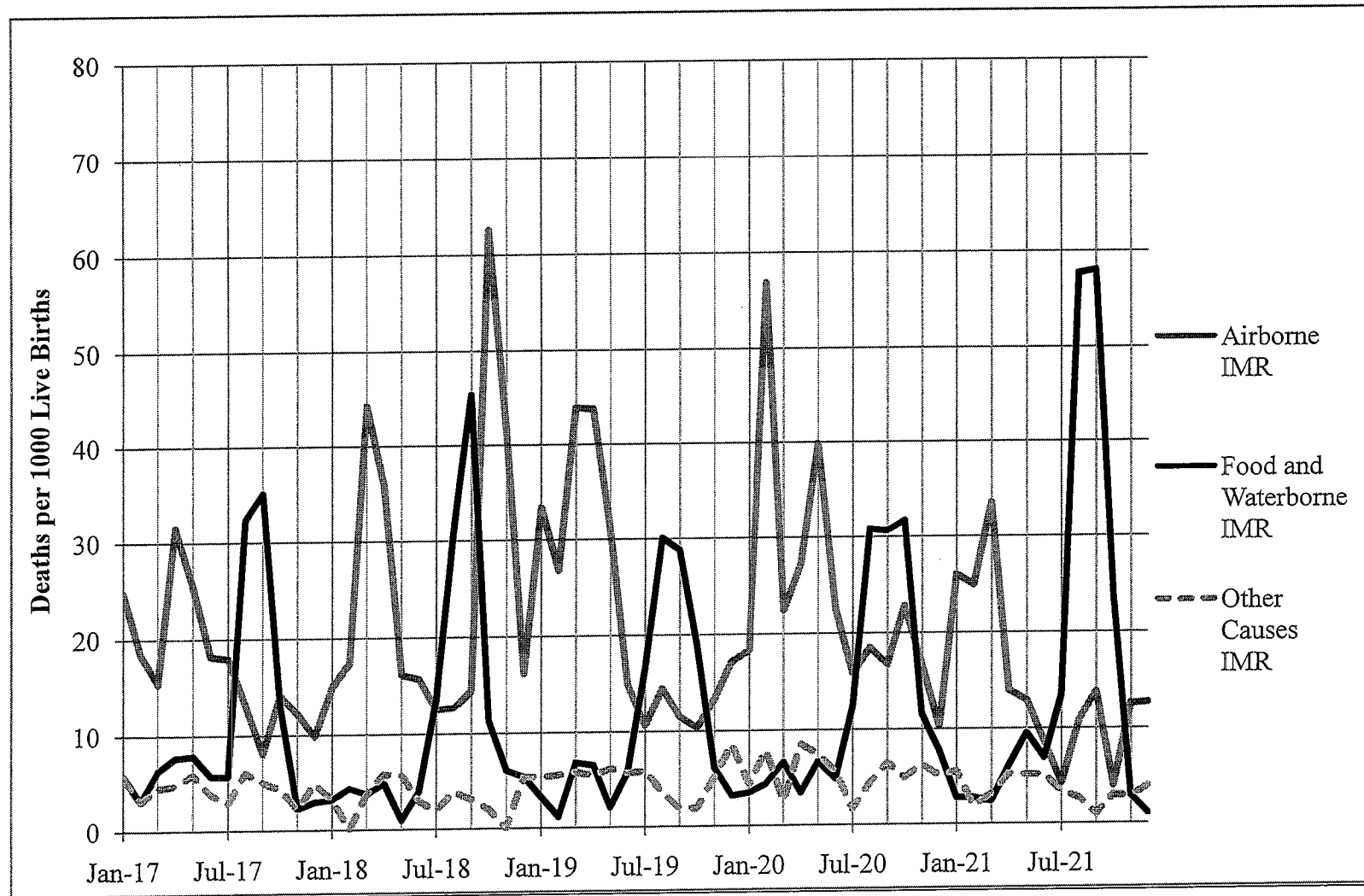


Figure 4.11 - Infectious Causes of Death, 1917-1921: Airborne, Food and Waterborne, and Other Infectious Causes of Death.

Generally, there is an inverse relationship between deaths from airborne infectious diseases and food and waterborne infectious diseases. When there is an increase in deaths from food and waterborne illnesses there is a concomitant decline in deaths from airborne infectious diseases. This reflects the seasonal variability of these diseases, as deaths from diarrheal diseases generally increase during the summer months, while deaths from airborne diseases cause increased mortality during the winter (Collins and Lehmann 1951, Thomas et al 2006, Lofgren et al 2007). Deaths from food and waterborne diseases increase in August of each year and continue to be high until September or October. The months which recorded the most deaths per year from both food and waterborne infectious diseases and airborne illness are shown in Table 4.6. The highest death rate for food and waterborne illness over the entire five year period was recorded in September 1921, mostly from diarrhea and fermentative diarrhea. The monthly variation in food and waterborne illnesses is significant (one-way ANOVA: $p < .001$, $SD = 13.58$, $df = 59$, 95% CI; Welch: $p < .001$, $df = 11$, 18.73).

Year	Month of Most Deaths Due to Food and Waterborne Causes (rate per 1000 live births)	Month of Most Deaths Due to Airborne Causes (rate per 1000 live births)
1917	September: 35.11 (n=35)	April: 31.58 (n=33)
1918	September: 45.41 (n=44)	March: 44.26 (n=47)
1919	August: 30.08 (n=32)	March: 43.95 (n=45)
1920	October: 31.71 (n=32)	February: 56.87 (n=65)
1921	September: 58.06 (n=63)	March: 33.52 (n=42)

Table 4.6 - Month with Most Deaths from Food and Waterborne Infections and Airborne Diseases.

Except for the second wave of the influenza epidemic of October-November 1918, the increases in deaths from airborne diseases occurred in the winter and spring of each year, between February and May. The one-way ANOVA test for monthly variation in the airborne infectious disease rate was significant ($p=.04$, $SD=12.3$, $df=59$, 95% CI). As can be seen in Figure 4.10, the infant mortality rates due to airborne infectious diseases vary significantly by month but the variation is similar each year. October to November 1918 stands out for three reasons: 1) the increase in deaths from airborne infectious diseases was counter to the usual seasonal pattern of disease distribution (occurring in autumn as opposed to winter/spring); 2) the rate of death was highest for the five year period; and, 3) it followed immediately after a particularly severe season of infant deaths from food and waterborne diseases.

Only at two points during the five-year study period were influenza deaths elevated:³⁴ October to April 1918-1919, and February 1920 (Figure 4.12). The average infant influenza death rate for the five year period was 2.32 deaths per 1000 live births ($n=141$, $SD=5.49$), with thirty-three out of the sixty months recording zero deaths from influenza. The death rates from influenza during the second wave of the epidemic period can be seen in Table 4.7.

³⁴ Including deaths listed as cause by influenza, la grippe, influenzal meningitis, Spanish Influenza, epidemic influenza, and weak from influenza of mother.

Month	Infant Death Rate From Influenza (per 1000 live births)
October 1918	23.61 (n=23)
November 1918	22.00 (n=18)
December 1918	9.70 (n=9)
January 1919	10.78 (n=10)
February 1919	3.34 (n=3)
March 1919	6.84 (n=7)
April 1919	4.38 (n=4)

Table 4.7 – Infant Death Rate from Influenza per Month, Second Wave of Epidemic

The influenza death rate for February 1920 was 26.35 deaths per 1000 live births (n=30).³⁵ The influenza deaths do not account for all the epidemic related deaths because many deaths were caused by secondary streptococcal and pneumococcal infections (Tashiro et al 1987) and therefore may likely have been recorded as due to pneumonia,³⁶ or bronchitis. This is particularly true if infants with influenza present with different symptoms than adults (Kao et al 200, Munoz 2003). Further, while there were other airborne diseases which resulted in infant deaths during the study period (tuberculosis, whooping cough, measles, smallpox, chicken pox, diphtheria, German measles, scarlet fever, tonsillitis, and typhoid fever), these diseases did not have high death rates and most infants died from pneumonias. For these reasons, an analysis at the level of airborne diseases expresses the character of this epidemic.

³⁵ The *Monthly Reports* state that there was another influenza epidemic in February 1920, which began January 19th (City of Toronto Archives, Fonds 200, Series 365, File 23).

³⁶ For this study, pneumonia deaths include deaths listed as caused by pneumonia, broncho-pneumonia, lobar-pneumonia, and double pneumonia.

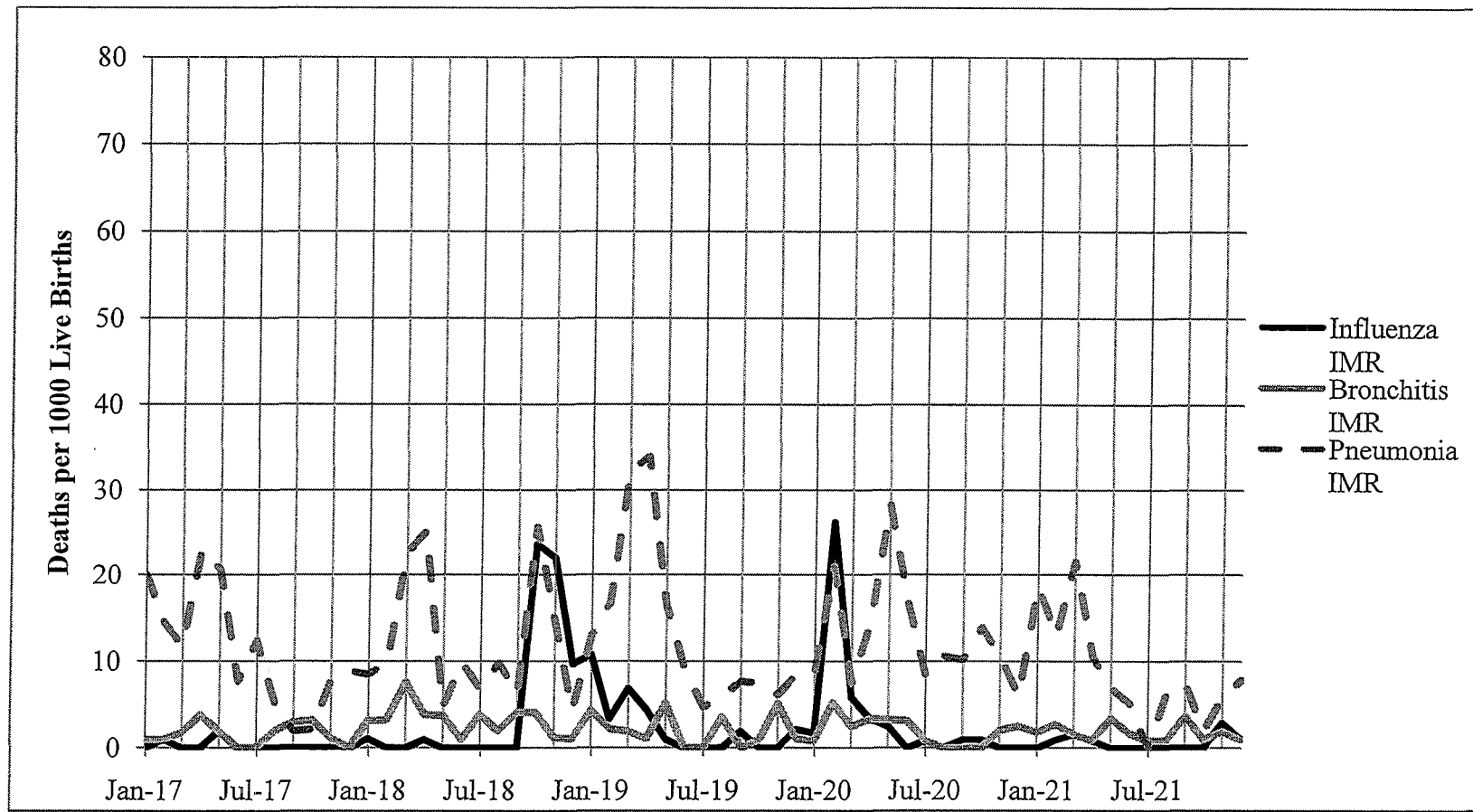


Figure 4.12 – Influenza^a, Bronchitis^b, and Pneumonia Related Deaths^c 1917-1921.

^a Includes deaths from influenza, la grippe, influenzal meningitis, Spanish Influenza, epidemic influenza, and weak from influenza of mother.

^b Includes deaths from bronchitis and capillary bronchitis.

^c Includes deaths from pneumonia, broncho-pneumonia, lobar-pneumonia, and double pneumonia.

When causes of death for 1918 are compared to all other years, it is evident that deaths caused by food and waterborne illnesses were slightly higher in September 1918 (45.41 deaths per 1000 live births) than the average of the other years combined (39.61 deaths per 1000 live births, Figure 4.13). 1918 had the second highest rate in September over the five year period - 1917: 35.11, n=35; **1918: 45.41, n=44**; 1919: 28.79, n=30; 1920: 30.67, n=33; **1921: 58.06, n=63** (see also Figure 4.11). The death rate from food and waterborne illness was not epidemic in proportion in 1918; however, it was followed immediately by the epidemic of influenza and pneumonia (which atypically struck in autumn and not winter/spring). This raises the possibility that some of the infants who died from the flu might have been previously weakened through a summer-time infection with a diarrheal disease.

Figure 4.14 shows airborne infectious diseases in 1918 compared to the average of all five years. The general seasonal pattern of infant deaths found in 1918 was not unusual: there was increased mortality in both the spring and autumn. However, the increase in infant deaths was larger in the autumn than in the spring as opposed to the normal pattern where more deaths occurred in the spring, and the autumn increase was also greatly exaggerated.

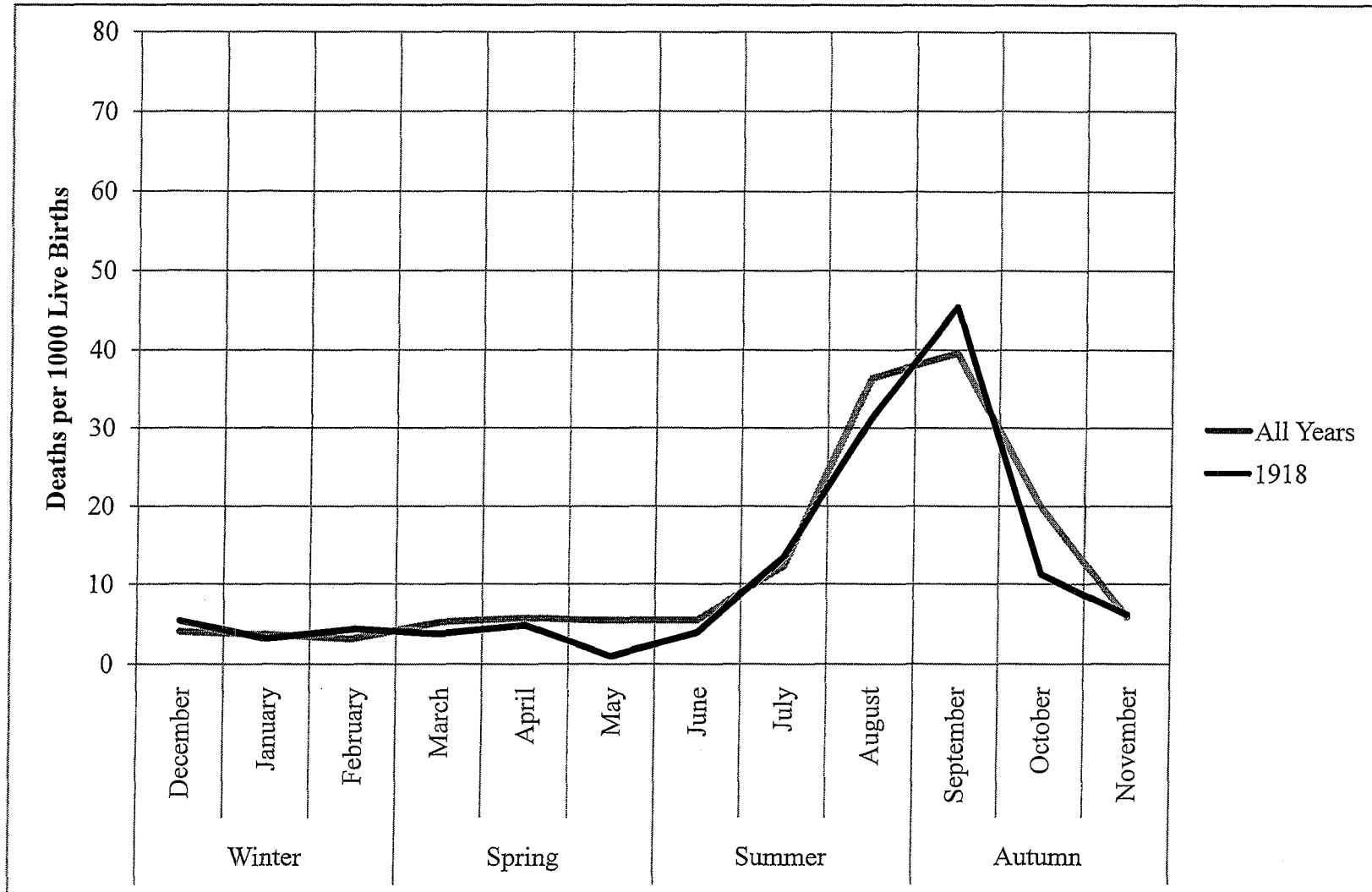


Figure 4.13 - Food and Waterborne Disease Infant Mortality Rate by Month, 1918 Compared to Average of 1917-1921.

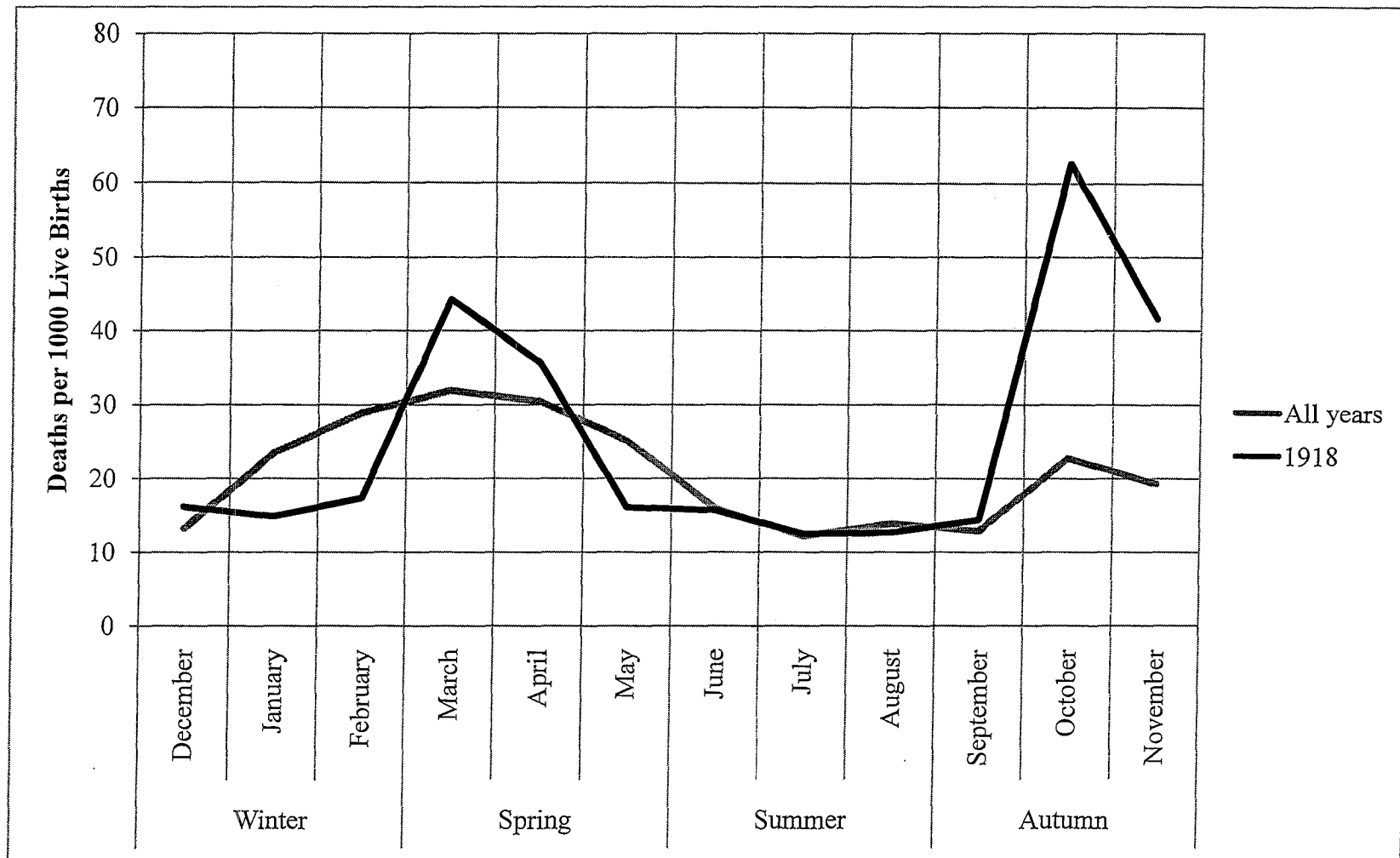


Figure 4.14 - Airborne Diseases Infant Mortality Rate by Month, 1918 Compared to Average of 1917-1921.

4.5 Stillbirths

Infection with influenza was known to have a deleterious effect on pregnant women and in serious cases to result in stillbirth (Winternitz et al 1920:39, Beveridge 1977, Reid 2005). I therefore explored the stillbirth rate in comparison to the neonatal infant death rate to see if any increases in the stillbirth rate lead to decreases in the neonatal death rate. The average stillbirth rate for the entire period was 119.78 deaths per 100 000 population (n=3009, SD=25.23) ranging from a low of 61.31 deaths per 100 000 population in August 1919 (n=26) to a high of 189.5 deaths per 100 000 population February 1920 (n=77). There was little regularity in the pattern of stillbirths and stillbirths and neonatal deaths did not co-vary ($r^2=0.001$).

4.6 Maternal and Infant Mortality

Maternal mortality was investigated for the four month period surrounding the second and worst phase of the 1918-1919 epidemic, September to December 1918. This issue was explored because pregnant women infected with influenza were known to be at higher risk of death and stillbirth (Oertel 1919; Young 1919; Winternitz et al 1920). To do this, the death records of women of child-bearing age (15 to 50) from September to December 1918, are compared to those during the same months during the pre-epidemic season, 1917. The causes of death for the women were analyzed to determine whether their deaths were pregnancy-related.¹ If so, I classified the death as “maternal”. Infant

¹ Pregnancy-related causes of death in this sample were: puerperal eclampsia, parturition, pregnancy, confinement, childbirth, acute nephritis of pregnancy, chorea and pregnancy,

deaths that could be linked to records for their mothers,² but where the death was not related to pregnancy, are also presented and are classified here as incidences of “family” mortality. These links give an indication of the effects of the epidemic at the family level.

Out of 221 women aged 15 to 50 who died from September to December, 1917, 21 deaths were pregnancy-related (0.11%, Table 4.7). Three were linked to infant death records. One set of linked records was for a 26 year old mother who died on September 26th, 1917 from eclampsia and heart failure and her female stillborn infant who was born the previous day. Another pair consisted of a 23 year old mother who died on October 6th, 1917 from parturition and an embolus and a stillborn female infant who was born the same day. The last pair was comprised of a 28 year old mother who died December 5th 1917 from pregnancy and dilation of the heart and a stillborn female infant who was born the same day. The only other linkable records from 1917 were for a 23 year old mother who died November 27th, 1917 and a male stillborn infant who was born the previous day. However, the cause of death for the mother was cardiectasis and therefore was not included in the list of pregnancy-related deaths.

In 1917 there was one probable case of linked mother-infant deaths: a 32 year old mother who died November 12th, 1917 of shock following a thyroidectomy and her three and a half-month old son who died September 22nd, 1917 from marasmus. There was one

toxaemia from tuberculosis and confinement, toxaemia of pregnancy, pernicious voicuture of pregnancy, coma and oturent planta pregnancy, post-partum haemorrhage, anaemia and pregnancy, puerperal septicaemia/sepsis, puerperal gastritis, ruptured ectopic pregnancy, placenta praevia, post-operative shock following caesarean section, abortion, premature labour, miscarriage, influenza (pregnancy), and pneumonia following labour. Any pregnancy-related death which was not listed as such is missing from this analysis.

² One father was also identified.

pair of potentially linked records, which was again an incidence of family mortality rather than maternal mortality. The 41 year 7 month old mother died September 24th, 1917 from uraemia leading to general peritonitis and her 1 month old son died October 19th, 1917 from chronic indigestion. In total, there were 6 pairs of linked, probably linked, or potentially linked records for the four month period. Only three of the mothers in those pairs were identified in their death records as having died from a pregnancy-related cause of death. Of the 24 women (21 pregnancy-related causes of death and 3 other) and the 6 linked infants in the four month period, none had influenza or pneumonia listed as a cause of death or as a contributing factor in death on the death record. Twenty-one women were married, two were single (one died from septic peritonitis following an abortion) and one was a widow.

1917 Total Women = 221, Total Infants = 324, Total Stillbirths = 182						
Month	Linked Records (pairs)	Probably Linked Records (pairs)	Potentially Linked Records (pairs)	Women: % of Total (n)	Infants (n)	Influenza Related Deaths (n: mother, infant)
September	1	0.5 (infant)	0.5 (mother)	0.03 (7)	2	0,0
October	1		0.5 (infant)	0.02(4)	2	0,0
November	1	0.5 (mother)		0.05 (10)	1	0,0
December	1			0.02 (3)	1	0,0
Total	4	1	2	0.11 (24)	6	0,0
1918: Total Women = 860, Total Infants = 456, Total Stillbirths = 180						
September	3	1		0.006 (5)	4	0,0
October	11.5 (mother)	4	1	0.03(24)	15	21,1
November	10.5 (infant)	2	2 ^a	0.02 (16)	17 ^b	13,2
December	2	2		0.008 (7)	4	6,2
Total	27	9	3	0.06 (52)	40	40,5

Table 4.8 - Maternal and Infant Mortality, September to December 1917-1918.

^A One of these was the male/infant link^b Twins

Out of 860 women aged 15 to 50 who died from September 1918 to December 1918, 23 died from pregnancy related causes (0.03%) as determined by a pregnancy related cause of death on the death record (Table 4.8).³ Most of these deaths (14) were not linked to an infant death, possibly because the infant survived, the infant died after December 1918, the death of the mother occurred before the birth, the infant was stillborn

³ Again, it is not possible in this sample to determine the numbers of pregnant women who died of non-pregnancy related causes or who died from pregnancy-related causes but were not listed as such and they are therefore not included in this study.

and not included in the registry, the surnames were different, or information was missing in the registers precluding the linking of records.

The major difference between 1917 and 1918 is the total numbers of linked records and in the causes of death of the mothers, in addition to the increase in deaths of women of reproductive age from 221 to 860. In 1918, 27 pairs of mother-infant death records were linked, 9 were probably linked, and 3 were potentially linked. Of the 27 linked pairs of records, 21 women had influenza or pneumonia listed as a cause of death and eight had pregnancy listed as a cause or contributor to death (with some records having both influenza and pregnancy listed as causes of death). Of the infants, 17 were stillborn, and 10 were neonatal deaths,⁴ and one was postneonatal. Of the 9 possibly linked mother-infant death records, 8 deaths of mothers were due to influenza or pneumonia and only one was caused by (puerperal) gastritis. Five of the infants were stillborn, 3 were neonatal deaths, and one was a postneonatal death (at three months). For the 3 potentially linked records, one was for a father who died November 21st, 1918 from pulmonary tuberculosis, oedema and heart failure, and a premature female infant who died November 4th, 1918 after fifteen minutes of life. The other two records were for mothers who died from influenza and pneumonia and their postneonataally aged sons, one who died from congenital syphilis and the other from influenza. Of the total parents (39 linked and 14 not), 2 of the unlinked records were single mothers, one of the probably linked records was a single mother, and one of the linked mothers was single. All other parents were married.

⁴ One mother was linked to the death of twins.

Interestingly, when the pairs were analyzed according to whether the parent died before the child, if they both died on the same day, or if the infant died first, the proportions between the 1917 and 1918 were approximately equal. In 1917, in 50% percent of the pairs the infant died first, while in 1918, this was true in 48% of the pairs. In both years, 17% of the linked records indicated that the parent died before the infant. The parent and the infant died on the same day in 33% of the pairs in 1917 and in 36% of the pairs in 1918. This would indicate that while the influenza epidemic altered the number of pairs of infant and parent deaths that could be linked as well as the number of those deaths attributed to influenza, the order of death was constant. In both years, two-thirds of the deaths of infants either occurred before the parent or on the same day, revealing that these deaths were more likely caused by the circumstances of birth or the same infectious agent than from lack of care resulting from the death of a care-giver.

4.7 Main Findings

The main inference about infant mortality from 1917-1921 based on statistical analyses is that although the influenza epidemic of 1918 did increase the rate of infant death, the increase was not significant and fell within the range of random yearly variation. Postneonatal infant mortality rates changed seasonally each year, with the highest rates found in late summer and early autumn.

In terms of the sex-ratio at death, males died at a greater rate than females in all months except for October 1918 and 1919, when more females died than males.

The average age at infant death showed no significant yearly fluctuation. The average age at death also varied seasonally, with the youngest age recorded in the winter progressing to the highest age at death in autumn. The 1918 influenza epidemic is visible in that normally, the oldest age at death was found in September, but in 1918 it was recorded in October. Also, the 1918 epidemic generally affected infants older than three months with the greatest effect occurring between seven and nine months of age.

In terms of cause of death, there was again no significant yearly difference but a consistent seasonal pattern. Deaths from food and waterborne infectious diseases increased in late summer and early autumn while deaths from airborne infectious diseases usually increased through the winter to a peak in early spring. This was altered by the influenza epidemic as 1918 showed a higher than normal peak in the early spring, but most deaths occurred in the autumn.

The increased deaths from airborne infectious diseases in autumn of 1918 immediately followed a high rate of deaths from food and waterborne infectious disease. The relationship between these two findings is discussed in Chapter 5. There was no relationship between the stillbirth rate and the neonatal death rate, suggesting that the reason the neonatal death rate did not increase during the epidemic was not because of an increase in stillbirths.

The records linkage revealed that in 1918 more infants and their parents died than in 1917. Further, those parents and children who did die were more likely to have died from influenza. However, the epidemic did not alter the expected chronology of events: in both years, two-thirds of infants died either before their parent or on the same day.

This suggests that the influenza epidemic did not cause an increase in the number of infants dying due to declining social and care-giving conditions due to the death of a mother. Deaths were caused either by the conditions of pregnancy or infection with the same organism.

CHAPTER 5

DISCUSSION AND CONCLUSIONS

To determine the pattern of infant mortality in Toronto from 1917 to 1921 and ascertain whether the 1918 influenza epidemic affected it in any way, I investigated six different aspects of infant death. First, I looked at the overall infant mortality rate, both yearly and monthly, to determine the death rates. I then investigated the sex-ratio at death to see if the influenza epidemic targeted one sex over the other. The average age at death was studied for the same reason: to discover if one subgroup of infants was at higher risk than another. Causes of death were examined to determine whether influenza did affect infants and to find out if deaths from other causes decreased during the epidemic. The stillbirth rate was analyzed to discover if there was any relationship between stillbirths and neonatal deaths but none was found. Finally, maternal and infant mortality were considered to explore another hidden aspect of the effect of the epidemic on families in Toronto.

The 1918 influenza epidemic affected infant mortality rates in some ways, but many patterns remained unchanged. The overall infant mortality rate remained stable over the five year period. The lack of variation in the influenza mortality rate among infants in Toronto is not without precedent. In the United States, Noymer and Garenne found that "at the youngest ages, influenza death rates in 1918 are about the same as in 1917" (2000:567). Further, McKinnon reported that for infants in Ontario "the lack of any great excess in mortality in this age group in 1918 . . . may be noted as contrasted

with other groups” (1945:288). However, although Toronto has been discussed in the larger Canadian context of the influenza epidemic (Pettigrew 1983; MacDougall 1990, 2007; Miller 2002), in terms of World War I (Miller 2002), and the social conditions of the working class (Piva 1979), there has yet to be a comprehensive analysis of the mortality rates and effects of the pandemic on the city. Further, although many studies mention the infant mortality rate from influenza, there are no studies based directly on infant death records for comparison to the results of this project. The findings from this research, based on the death registrations in Toronto, support the evidence of McKinnon (1945) and Noymer and Garenne (2002), but further studies of infant mortality are needed to discover if this result is the same for infants throughout the world. The reasons that the infant mortality rate remained stable in Toronto are still unknown.

Some researchers argue that adults were at greater risk because some aspect of the virus triggered an overactive response by the immune system “causing excessive infiltration of the tissues by immune cells, resulting in tissue destruction” (Loo and Gale 2007:267, Morens and Fauci 2007). As the immune system is more mature and experienced in adults than in infants, this may explain why adults were at higher risk and infants appeared to be protected. Also, the hypothesis of Noymer and Garenne that “those with tuberculosis (TB) in 1918 were more likely than others to die from influenza” (2000:565) and that TB incidence was higher among young adult males (2000:574), may again reveal why infants were relatively untouched. Maternal antibodies may have helped to protect those infants still nursing when the epidemic hit, explaining the

predominance of deaths among older infants in this study who may have been undergoing the weaning process.

The stability of the infant mortality rate in Toronto masks certain effects of the 1918 epidemic on infants. Pursuing “what is being hidden from view in the official statistics” (Scheper-Hughes 1997) revealed an unexpected reversal in the sex-ratio at death for October 1918. This is important because males are known to be at a disadvantage over females generally, but especially in terms of respiratory diseases (Drevenstedt et al 2008). The Toronto pattern for 1918 also differs from that for Canada for the period 1921 to 1925, where male deaths from influenza were 133% greater than for females (MacPhail 1927:480). Male deaths increased in September 1918 while the female death rate did not increase until October 1918. Generally, the infant mortality rate increases in September due to deaths from food and waterborne diseases, of which males are affected more than females. However, 1918 was unusual in that the harsh food- and waterborne season was followed directly by the epidemic of influenza. Perhaps interplay between these two disease clusters contributed to the reversal in the sex-ratio in October.

In this regard, it is possible that the lowered male-to-female sex ratio at death was due to an underreporting of male deaths during the epidemic or to preferential treatment which resulted in male infants receiving better care when ill. Male infants may also have been breastfed longer than females, protecting them with maternal antibodies while females were exposed to a greater risk of many different forms of infection from contaminated water and supplemental foods. However, there is little evidence to suggest that male infants in Toronto were given better care or were fed differently from females.

Further, there is no reason to believe that male infants were selectively underreported where females were not.

The increased death rate of males from diarrheal diseases in September suggests that weaker males may have died in September while the females did not. However, some females were weakened by malnutrition so that they were more susceptible to the flu in October. Also, stronger immune systems were thought to be a factor in the deaths of young adults. Although still much weaker than adults, infant females have stronger immune responses than males (Drevenstedt 2008). This may have protected females from diarrheal diseases in September but also made them susceptible to the fatal consequences of more vigorous immune responses to the influenza and pneumonia in October. However, the numbers of infant deaths that these ratios are based on are small. To be certain that these findings are valid, infant sex-ratios at death for larger cities and geographic areas should be examined.

Another hidden aspect of this disease, which may help to illuminate what was happening in Toronto, was that postneonatally aged infants (especially females) were at greatest risk from the illness. Particularly at risk were those between the ages of seven and nine months who would have begun complementary feeding during the summer months. Infants begin to face challenges from external contaminants between the third and sixth months of age when complementary feeding with solid foods are required to meet their nutritional needs (Wharton 1989, Hendricks and Badruddin 1992). At this time, the infant is susceptible to the “weanling diarrhea syndrome where gastrointestinal disease associated with contaminated water and food combines with other contagious

diseases to take a heavy toll on infants” (Herring et al 1998:433). This is especially devastating during the summer months when hot temperatures lead to the increased consumption of contaminated water: stressed and compromised immune systems are unable to handle the increased disease load and infant mortality increases. The diarrheal diseases have been shown to be highest among bottle fed infants (North 1921, McKeown 1976). The increased rate of food and waterborne illness in September in Toronto gives evidence of the “weanling diarrhea syndrome” and it was also seen in other North American cities in this time period (North 1921, Cheney 1984, Figure 5.1). Well-baby clinics, established in Toronto in 1917, stressed the importance of breast-feeding to mothers (Brown 1931). While breast-feeding did increase, food sources were still contaminated, and many infants were exposed at early ages to unsafe milk and water.

The 1918 influenza epidemic occurred at a time of year when airborne infectious diseases were generally low. Further, the deaths from food and waterborne illnesses during the late summer and early autumn of 1918 were the second highest over the five year period and greater than the average of the five years combined. This may have been related to the extremes of weather found during August and September 1918 (Environment Canada 2008). The infants who were at greatest risk from diarrheal deaths were those in the weaning process, who were consuming water or milk directly, in bottle-based foods, or exposed to contaminated water or milk through incomplete sterilization of the bottles. Infants who survive summer diarrheal diseases can be malnourished and immunologically weakened, leading to easier infection with other diseases (Guerrant et al 1992). This suggests a syndemic relationship between diarrheal diseases, influenza, and

the physical environment including weather extremes and social practices regarding transitional feeding. It is likely that the reversed sex-ratio of death in October reflected the effect of diarrheal diseases in September: the weakest postneonatal infants had already died and malnutrition altered the profile of babies normally susceptible to airborne diseases. Those infants previously at risk from diarrheal diseases may have been the same infants who were later at risk from influenza.

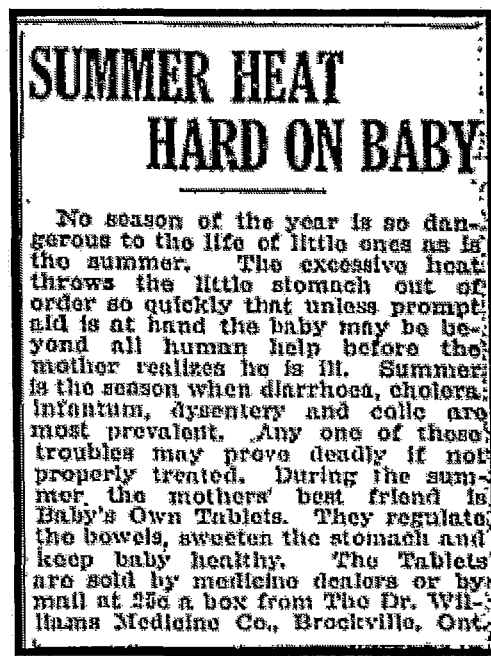


Figure 5.1 - Summer Heat Hard on Baby (The Globe 1921)

The 1918 influenza epidemic has been described as a ‘democratic’ illness in that people of different socio-economic distinctions are equally likely to contract the disease (Crosby 1989, Tomkins 1992, Barrett and Brown 2008). However, this assertion has been questioned by several researchers, including Sydenstricker (1931), Johnson (1993), Phillips and Killingray (2003), Mamelund (2006), Jones (2007) and Herring (2009).

Farmer cautions, moreover, that “critical perspectives on emerging infections must ask how large-scale social forces come to have their effects on unequally positioned individuals in increasingly interconnected populations; a critical epistemology needs to ask what features of disease emergence are obscured by dominant analytic frameworks” (2001:5). In 1931, Sydenstricker wrote

when the [democratic] generalization was subjected to the closer analysis afforded by actual records of influenza incidence in 1918 in enumerated populations, the interesting indication appeared that 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 the factors of color, sex, and age, and certain other conditions. [Sydenstricker 1931:155]

This begs the question: even though the rich were dying, were they doing so at the same rate as the poor? It is clear that malnutrition leads to both higher rates of infection and higher case fatality rates (McKeown 1976, Brown and Inhorn 1990, Phillips and Killingray 2003). Does recognizing that the rich were sick and labelling the disease ‘democratic’ further marginalize the more extensive suffering of the lower-classes? Does the effect of losing one or both parents or one or more children disproportionately affect the poor (Jones 2007)? An analysis of socio-economic inequalities among infants in Toronto in 1918 is beyond the scope of this thesis. However, it is a necessary component which must be understood: As Herring concludes, “the constellation of biosocial conditions that contributed to this diversity [in global mortality rates] has barely been

explored and warrants close scrutiny as the implications are important for future pandemics” (2009:88).

It is known that “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” (Singer and Baer 1995:213). For influenza in particular, infants of lower socio-economic status are five times more likely to need medical care when infected with the flu (Glezen and Couch 1991). Since Mercier (2006) found that the infant mortality rate in Toronto in 1901 varied by religion and ancestry, it is likely that death during the epidemic reflects these previously existing social inequalities. The Medical Officer of Health admitted in October 1918 that the flu was “spreading most rapidly among the poor” (The Globe 1918l:6). With research initiated in this thesis on varying levels of risk based on sex and age-at-death, future work must analyze the role of socio-economic difference in relation to influenza and infant death if comprehensive protective strategies are to be implemented in future outbreaks.

Discovering who was at risk of infection from food and waterborne illness may help to elucidate who was at risk from influenza. Diarrheal deaths are directly subject to the quality of water, access to sewer systems, and prior malnutrition (Guerrant et al 1992, Redlinger et al 2002). Adequate plumbing could be found in some areas throughout Toronto in 1918 but was particularly insufficient in the slums (Piva 1979); this may be an underlying factor in the variation in infant mortality rates. Influenza was also known to have affected lower socioeconomic classes at a greater rate and intensity than higher classes (The Globe 1918n, Sydenstricker 1931), but somehow did not significantly

increase the infant mortality rate in Toronto; it may be that those infants who were weak, malnourished, or socioeconomically disadvantaged were those more likely to die under normal circumstances, and were also those at risk from death during an epidemic. Further research should plot the address of death of the infant located in the death records using Geographic Information Systems technology (GIS) to discover if infant mortality from influenza in 1918 clustered in the impoverished areas of Toronto.

Although record linkage has been attempted for the 1918 influenza epidemic (Herring and Sattenspiel 2007), it has not been used to show the relationship between epidemic-related changes in infant mortality and the deaths of mothers. The 1918 influenza epidemic caused a vast increase in the number of women who died between the ages of 15 and 50. From September to December 1917, 221 women died, while in the same period in 1918, 860 died. Record linkage based on surnames matched in the death records revealed that at least six families lost both a mother and an infant from September to December 1917. For the same period in 1918, a minimum of 39 families lost either a mother or a father, and an infant. Interestingly, even though the numbers of linked pairs increased, the proportion of the infants who died before their parents remained unchanged. In both 1917 and 1918, two-thirds of the infants either died before their parents or on the same day. Infant deaths during this epidemic were not caused by disruptions to the care-giving environment: Infants were much more likely to die from the pregnancy-related consequences of influenza or from the disease itself rather than from malnutrition caused by the mother's death. There may have been other deaths in the family such as those of older children, aunts and uncles, grandparents, and non-related

care-givers, which should be linked in further studies. Record linkage of parents and their children provides an opportunity to explore another hidden aspect of this epidemic: how many families experienced the losses of both a parent and a child?

As Jones wrote, “influenza was not the same epidemic for everyone” (2007:4): why were these families particularly hard hit? Future research should delve into these family associations to determine if there were any commonalities such as socio-economic status, place of employment, health status, and family size. This could be done through family reconstitution using city tax assessment rolls, birth and marriage records, and the 1911 and 1921 censuses. As can be understood for most epidemic diseases, “being infected with influenza was much more than a bodily event in the lives of these men, women, and children. It was a psychic event, and it was often life transforming, altering the contours of their future and changing the nature of their citizenship” (Jones 2007:7). Such analyses would help to clarify why some families suffered disproportionately from the 1918 influenza epidemic and perhaps reveal some of the longer-term implications of this epidemic. Also, as Herring suggests for avian influenza, “failure to explore who is likely to be at greatest risk of acquiring and dying . . . is dangerous from a public health policy perspective” (2009:88). Understanding which infants were at risk from the 1918 influenza epidemic could help protect similar infants in future pandemics.

As stated previously, in order to gain a comprehensive understanding of precisely who was at greatest risk from the 1918 flu, it is important to conduct further research on many areas suggested by this thesis. Infant mortality did not increase in the city of Toronto because of the influenza epidemic, yet it is unlikely that those infants who died

were at equal risk of perishing compared to those who did not. Young adults, the caregivers of those infants, were dying in greatly increased numbers. Even when infants did not die, they were certainly affected by the loss of parents and the cultural upheaval in the face of an ending global war, changes in social structure and social welfare systems, and the constant threat of disease resurgence. The 1918 influenza epidemic hit Toronto after four years of struggle to constantly save, contribute to the war effort, and survive on dwindling food and fuel resources while the cost of living was steadily increasing. Toronto society was heavily stratified, as people with poor health, sanitation, and nutrition were to be found amongst those with great wealth. Although it was necessary to first establish infant mortality rates to determine what happened to infants, *why* these patterns occurred is still unknown. The next step is to continue to question the aggregate data in order to have a complete understanding of risk and mortality in Toronto during the 1918 influenza epidemic.

It is important to realize that, although a community may be greatly strained by an epidemic and stressful social conditions, the infant mortality rate is not always a direct reflection of these social disruptions. Infant mortality has been shown to be an important indicator of social health, such that when a community is under stress from nutritional or water insufficiency, social inequalities and unequal access to resources, or endemic disease, infant mortality is generally higher (Moffat and Herring 1999, Galley and Shelton 2001, Sawchuk et al 2002). As this thesis has shown, infant deaths do not necessarily increase during periods of epidemic stress. Infant mortality may be more of a specific measure of the long-term effects of social strain wearing down the defences of

the most vulnerable rather than a comprehensive universal indicator of social disruption. For all of the reasons discussed, infants can be protected during an epidemic, masking a period of undeniable social stress. That the influenza epidemic struck adults in Toronto is not in doubt. What needs to be understood is what mechanisms prevented this epidemic from killing more infants and how that can be translated to prevent deaths in future pandemics.

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APPENDIX A

DEATHS	
County of	Division of
SURNAME of Deceased. Christian Name. Sex. Age. Date of Death. PLACE OF BIRTH Place of Birth, 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.	SURNAME of Deceased. Christian Name. Sex. Age. Date of Death. PLACE OF BIRTH Place of Birth, 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 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.	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.

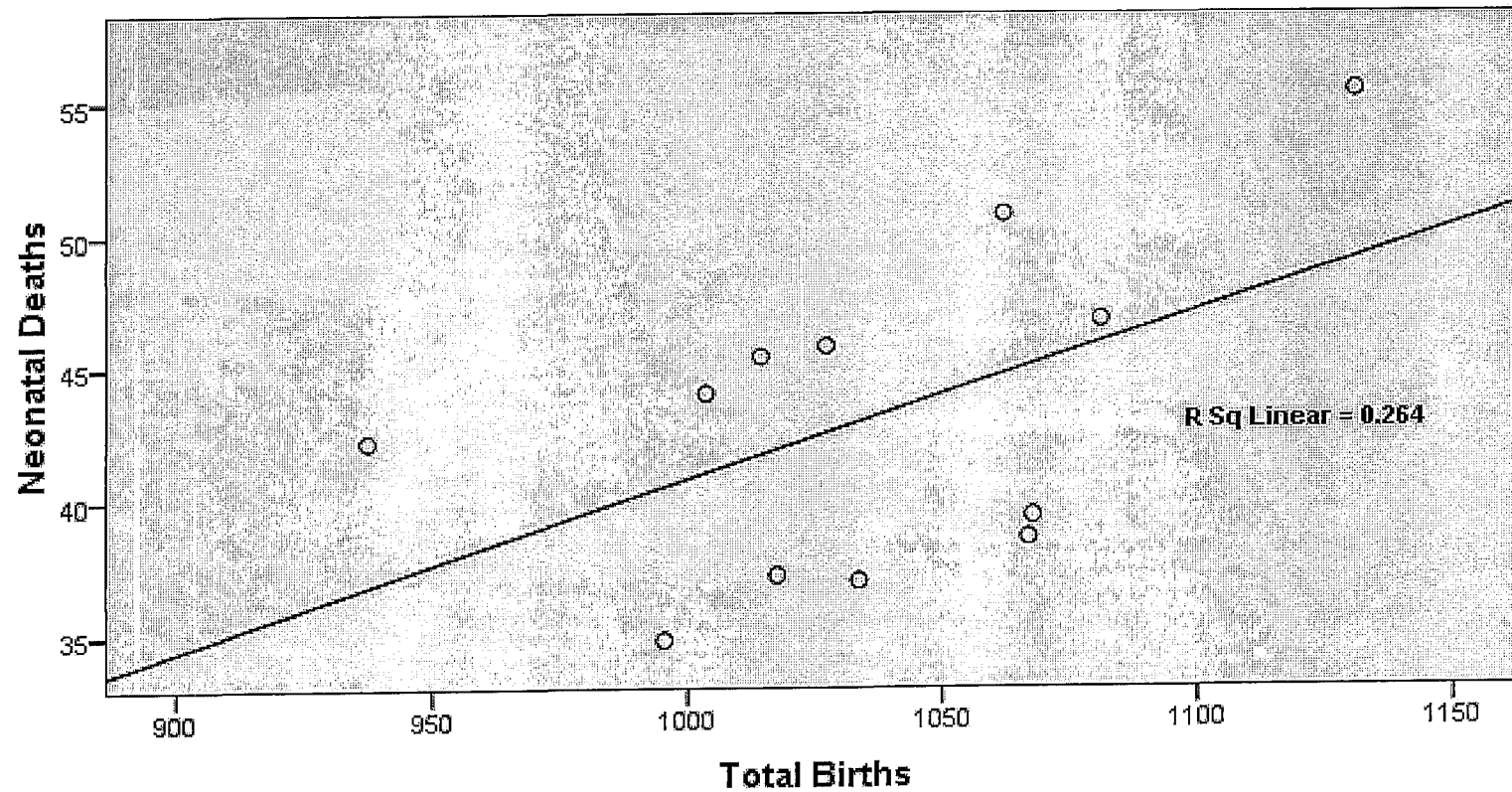
Appendix A.1 - Death Records 1917-1919 (Archives of Ontario MS 935, Reel 227)

County of _____		Division of _____	
DEATHS			
Name of Deceased			
Full given Name			
Place of Death, street and number or			
(a) Sex, (b) Racial Origin			
(c) Single, Married, Widowed			
Age			
(a) Place of Birth			
(b) Date of Birth			
Trade or Occupation			
Kind of Industry			
Date from which to which employed			
Trade or Occupation			
Kind of Industry			
Date from which to which employed			
Length of Residence			
Name of Father			
Birthplace of Father			
Maiden Name of Mother			
Birthplace of Mother			
Name of Informant			
Address			
Relation to Deceased			
Place of Burial			
Date of Burial			
Name of Undertaker			
Address			
Cause of Death if no Physician attended			
Date of Death			
MEDICAL CERTIFICATE OF DEATH			
Name of Deceased			
Date of Death			
Dates from which to which Medical Practitioner Attended Deceased			
CAUSE OF DEATH			
Primary			
Duration			
Contributory			
Duration			
(a) Did an operation precede death?			
(b) Was there an autopsy?			
Name of Physician			
Address			
Date of Return			
Date received by Division Registrar			

Appendix A.2 - Death Records, 1920-1921 (Archives of Ontario MS 935, Reel 261).

APPENDIX B

Births Compared to Neonatal Deaths



Appendix B - Numbers of Births as Compared to the Numbers of Neonatal Deaths, 1917-1921: Covariation.

APPENDIX C

The following cause of death classification is based on the work of McKeown (1976), Moffat (1992), Gray (1997), the Forty-Forth Annual Report of the Hospital for Sick Children in Toronto (1919), and the International Statistical Classification of Diseases and Related Health Problems, 10th Revision Version for 2007 (ICD-10 v.2007) (WHO 2007). The analysis was done using Microsoft Excel© and Microsoft Access© as per Padiak (2004).

Infectious

Airborne: Bronchitis

- capillary bronchitis

Chicken pox

Diphtheria

German measles

Influenza

- epidemic influenza

- flu

- grip

- influenza of mother

- influenzal meningitis

- la grippe

- prenatal flu

- Spanish influenza

Measles

Pertussis/whooping cough

Pneumonia

- broncho pneumonia

- double pneumonia

- lobar pneumonia

Scarlet fever

Smallpox

Tonsillitis

Tuberculosis

- miliary tuberculosis

- phthisis
- pulmonary tuberculosis
- tubercular meningitis

Typhoid fever

Food and Waterborne: Diarrhea

- autointoxication
- bowel poisoning
- cholera infantum
- dysentery
- fermentative diarrhea
- intestinal intoxication
- summer complaint

Digestive inflammation

- colitis
- enteritis
- gastritis
- gastro enteritis
- gastro intestinal ulcerations

Food poisoning

- milk fever
- milk infection

Polio

- Polio encephalitis

Tuberculosis

- tubercular peritonitis
- tuberculosis of the intestines

Other: Abscess

- abscess in head
- abscess of leg
- ear and frontal sinus abscess
- multiple abscess

Arthritis

- septic arthritis
- suppurating arthritis

Aspiration pneumonia

Cellulitis

Dermatitis

Empyemia

Encephalitis

- encephalitis lethargica

Endocarditis
Erysipelas
Exudative diathesis
Furunculosis
Hepatitis
Infection of newborn
Infection umbilical
Mastoiditis
Meningitis
Myocarditis
Myositis
Nephritis
Otitis media
Pemphigus
Peritonitis
Purpura
Pyæmia
Pylitis
Septicæmia
Teething
Tetanus
Toxæmia

Non-infectious

Congenital: Acidosis

Albuminuria
Ascites
Atelectasis
Congenital defect
Eclampsia
Ectopia vesicæ
Embolism from umbilical vein
Euphalitic prenatal
Heart Disease
Heart failure
-foetal heart
-heart defect
-heart lesion
-mitral insufficiency
-patent ductus arteriosus
-patent foramen ovale
-portal obstruction
Hirsprung's disease

Hydronephrosis
Injury at birth
 -dropsy of the amnion
 -dystocia
 -malpresentation
 -maternal deformed pelvis
 -toxaemia
Intestinal Obstruction
Immature development
Imperfect development
Intersusception
Jaundice
 -icterus neonatorum
Malformation
Mongolian idiocy
Monstrosity
Neural tube defects
 -anencephaly
 -encephalocele
 -hydrocephalus
 -meningocele
 -spina bifida
Placenta praevia
Prematurity
Prolapsed fundis
Pulmonary stenosis
Pyloric hypertrophy
Stricture of Oesophagus
Syphilis
 -lues
Tetany
Ulceration in utero
Weakness
 -congenital weakness
 -congenital debility

Non-Specific: Abdominal pain

Accident
Anuria
Asphyxia
Atrophy
Brain tremble
Convulsions

Cranial bleeding

- haematoma
- cranial pressure
- intra cranial haemorrhage
- cerebral embolism

Cyanosis

Dropsy

Exposure

Haemacephalus

Haemorrhage

- basal haemorrhage
- haemorrhage from lungs
- haemorrhage of bowels
- haemorrhage of umbilicus
- haemorrhagica neonatorum
- melaena neonatorum
- purpura haemorrhagica
- renal haemorrhage
- rupture of vein

Hemiplegia

Lung congestion

Lungs full of watery mucus

Miscarriage

Natural Causes

Nostalgia

Obstruction of bowels

Obstruction of stomach

Paralysis

Poisoning

Respiratory failure

Rheumatism

Shock

- collapse following operation
- shock and collapse
- shock from surgery

Stillborn

Weakness

- asthenia
- lack of vitality

Other: Accident

- asphyxia infantide
- burn

- drowning
- gunshot wound
- fractured skull
- infanticide
- neglect
- overlaid
- strangulation
- suffocation

Anaemia

Eczema

Epilepsy

Haemophilia

Heart failure

- acute dilation of heart
- cardiac dropsy
- cardiac syncope
- deficient circulation
- heart disease
- valvular incompetence of heart

Heat prostration

Hepatico duodenal catarrh

Indigestion

- dyspepsia

Infant feeding regulation

- cachexia
- decomposition
- inanition
- malassimilation
- malnutrition
- marasmus
- metabolism oedema
- rickets
- scurvy

Inflammation of liver

- hypertrophy of liver

Intestinal obstruction

- pyloric stenosis
- pyloric spasm

Oedema of lungs

Pulmonary congestion

Pulmonary obstruction

Spasm of glottis

Status lymphaticus

-enlarged thymus
Torn tentorium cerebrai
Uraemia

No Cause of Death Listed

Unknown

Unknown: Arenctsun
Found dead
Unknown