An exploration of the effects of pandemic influenza on infant mortality in Toronto, 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 which infants were dying and at what time, as can be seen through a male-to-female sex-ratio at death of 0.89 during the worst month of the epidemic. 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.

Introduction

The influenza epidemic of 1918 represented a period of "crisis mortality" in Ontario and around the world (Bouckaert 1989:218). This epidemic is noted for its unusual mortality profile. Although those most often the victims of infectious diseases were also 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 1918; 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 thus been exposed to many people from around the world which opened 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 1918).

Research 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 1918, McCullough 1918, Oertel 1919, Robertson 1919, Young 1919, Hare 1937). More modern works address the Canadian situation intermixed with global accounts (Collier 1974, Crosby 1989) or mention 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 1918c, The Globe 1918d, Harder 1918; Winternitz et al. 1920). The aggregate published data, however, give no impression of how social, environmental, and biological factors may have influenced the risk.

The analysis of the mortality of infants in Toronto during this epidemic is a novel means to approach the situation in Canada in 1918. This approach 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, this research is placed in a perspective that 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, five different aspects of infant death were investigated. First, the overall infant mortality rate was considered, both yearly and monthly, to determine the death rates. Then the sex-ratio at death was examined 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 (≥ 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. 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.

Location and Socioeconomic Context

This paper concerns infant mortality in Toronto, the capital city of the province of Ontario, Canada. Social inequalities were a significant aspect of life in the city during the early part of the twentieth century. As the largest city in Ontario and a centre for immigration, Toronto saw its population grow from approximately 200,000 in 1900 to 470,000 in 1915, to 540,000 by 1924 (Department of Health 1917, 1924). As seen through the records and concerns of the Department of Health (1917-1924), Toronto experienced many of the problems of a city trying to accommodate rapid expansion and immigration. The city, especially the slums such as the Ward (Mercier 2006), was crowded, with high rents and inadequate housing (Solomon 2007:17-22).

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. However, Mercier affirms that neighbourhood-based differences do not explain all the variation in infant mortality because of "complex interaction effects" (2006:146).

In 1911, the city was still plagued by socio-economic inequalities, which continue to this day. Dr. Hastings (the Medical Officer of Health) 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.

Materials and Methods

This research 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 8,952 infant death records from 1917-1921 were transcribed representing the 9016 still and live born infants who died and were registered

with the Registrar General of the Province of Ontario during this five year period (Table 1).[1] As noted by Emery (1993), by 1917 the death records in Ontario were almost complete, making the materials of this study (the Registered Deaths of the Province of Ontario) a suitable means to attempt a description of infant mortality in the city. Data on total population and total numbers of live and stillbirths from 1917-1921 were found in the Sessional Papers of the Legislative Assembly of the Province of Ontario, also located on microfilm at the Archives of Ontario (Table 2).

The majority of the infant death records from 1917-1921 were transcribed at the archives. The remainder were transcribed from JPEG copies of the death records previously created 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 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. This paper is based on my MA thesis, completed at McMaster University in August 2009.

[1] 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.

	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

Table1 - 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	6634	13 378

Table 2 - Total Births per Year by Sex (Legislative Assembly of the Province of Ontario 1918-1922)

Results

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 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 3.

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 3 - 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 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). There was no significant differences in the annual rates of infant death over the five year period (1917 vs. 1918 vs. 1919 vs. 1920 vs. 1921) (ANOVA p=.238, SD=19.887, df=59, 95% CI).

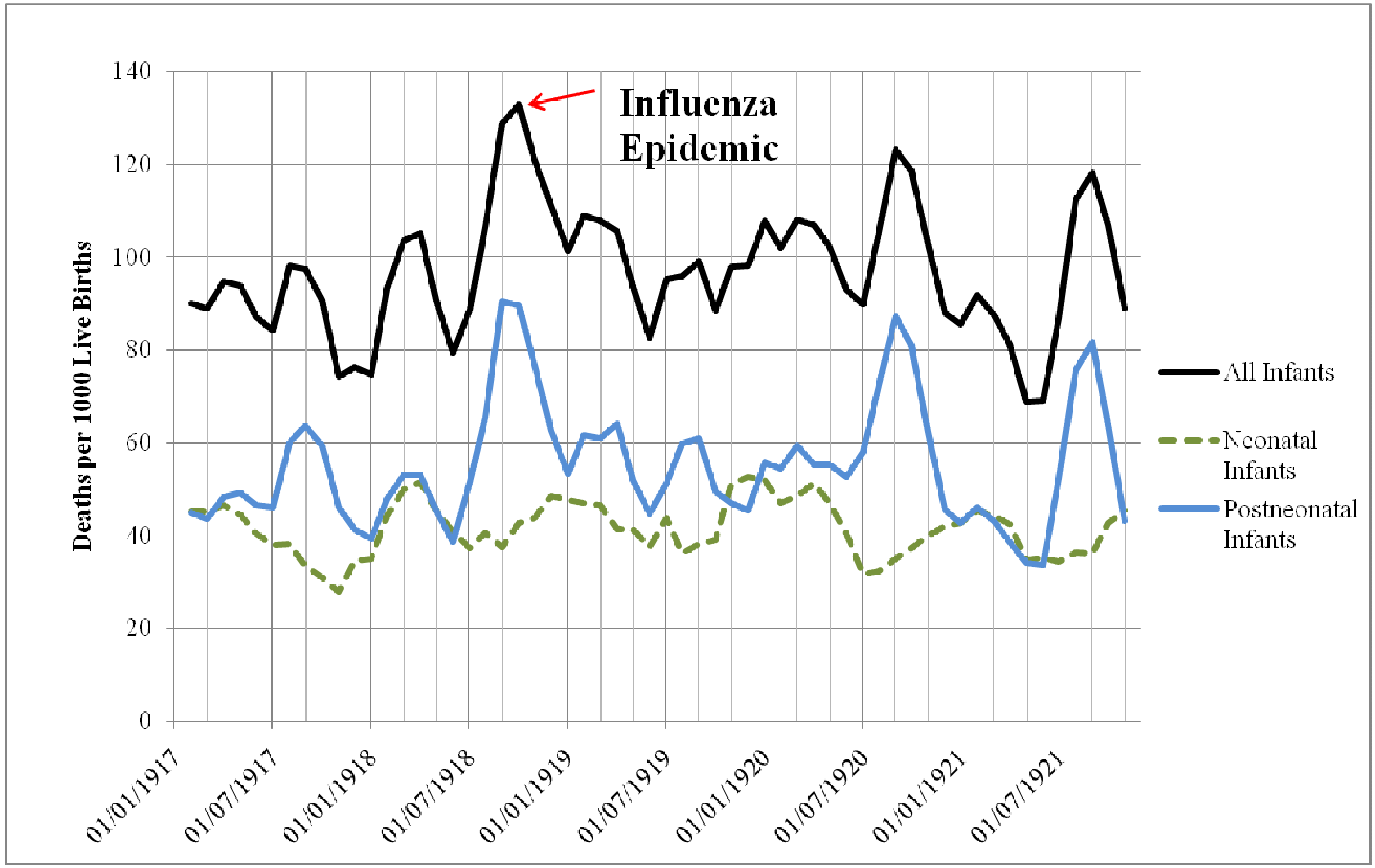

Figure 1 – Total Infant Mortality Rate, Postneonatal Mortality Rate, and Neonatal Mortality Rate, 1917-1921: Three Month Moving Average.

In 2000, the sex-ratio at births of males to females in Canada was 1056 males to 1000 females (1.06:1) (Mathews and Hamilton 2005:5). Table 4 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 - 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.[2] 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 1919) more females died in October than males.[3] The differences between the years was not significant (AVOVA p=.895, SD=0.30, df=59, 95% CI), while the monthly sex-ratios at death were significant (ANOVA p=.042, SD=0.30, df=59, 95% CI). 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). This indicates that 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 during this month (Table 5, Figure 2).

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[2] 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).

[3] 1917: February, 1.04:1, (n=89); 1918: October, 0.83:1, (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.

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 5 – Infant 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).

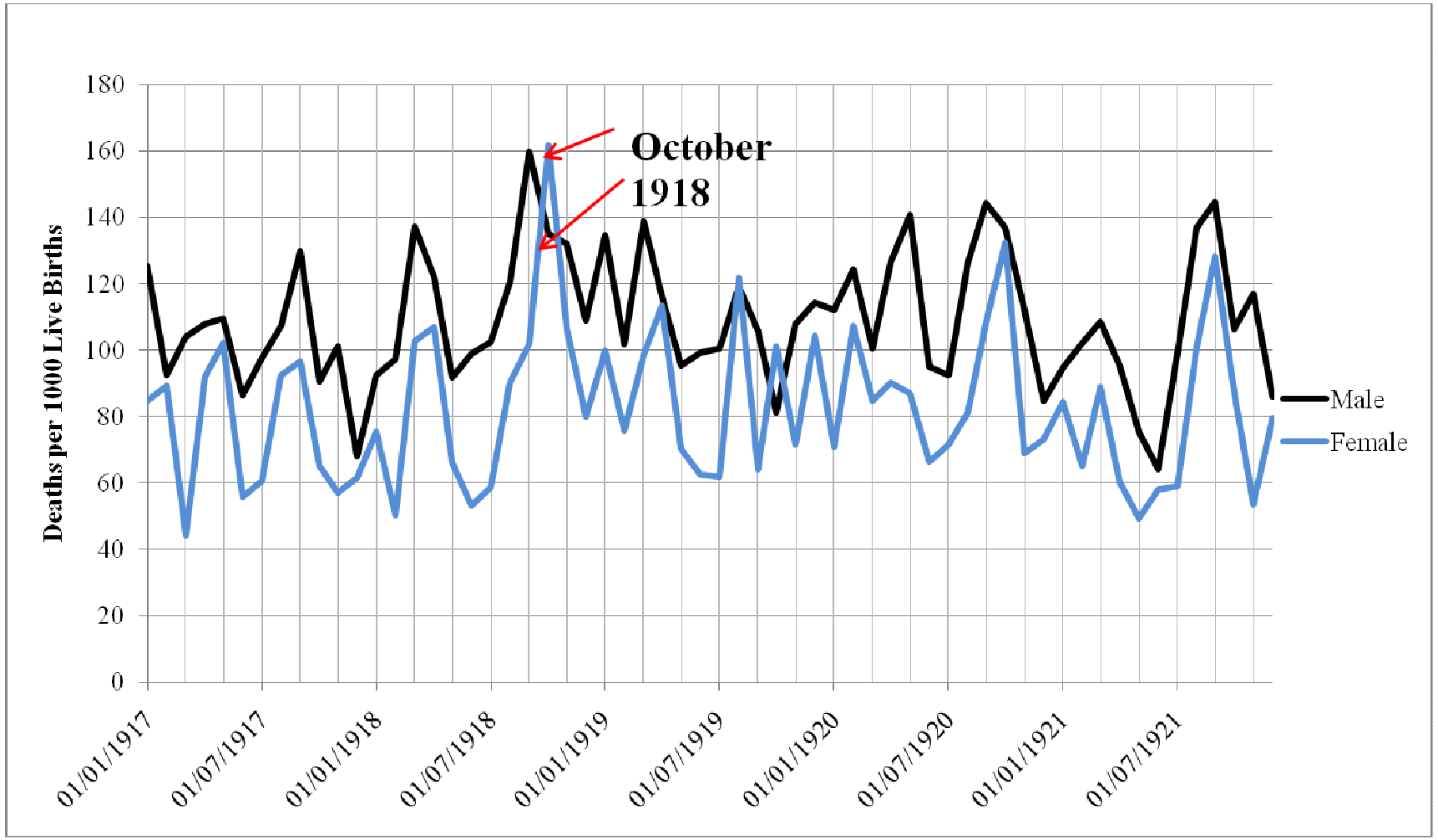

Figure 2 - Male and Female Infant Mortality Rates, 1917-1921.

Infectious causes of death

To explore the infectious causes of death in more detail, the three categories shown in Figure 3 were utilized: airborne diseases, food and waterborne diseases, and all other infectious causes of death.[4] 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),[5] 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 according to a one-way ANOVA test (p=.167, SD=12.3, df=59, 95% CI; Welch: p=.155, df=4, 26.844). 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). The yearly variation in food and waterborne illnesses was not significant (ANOVA p=.894, SD=13.58, df=59, 95% CI).

[4] 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).

[5] 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.

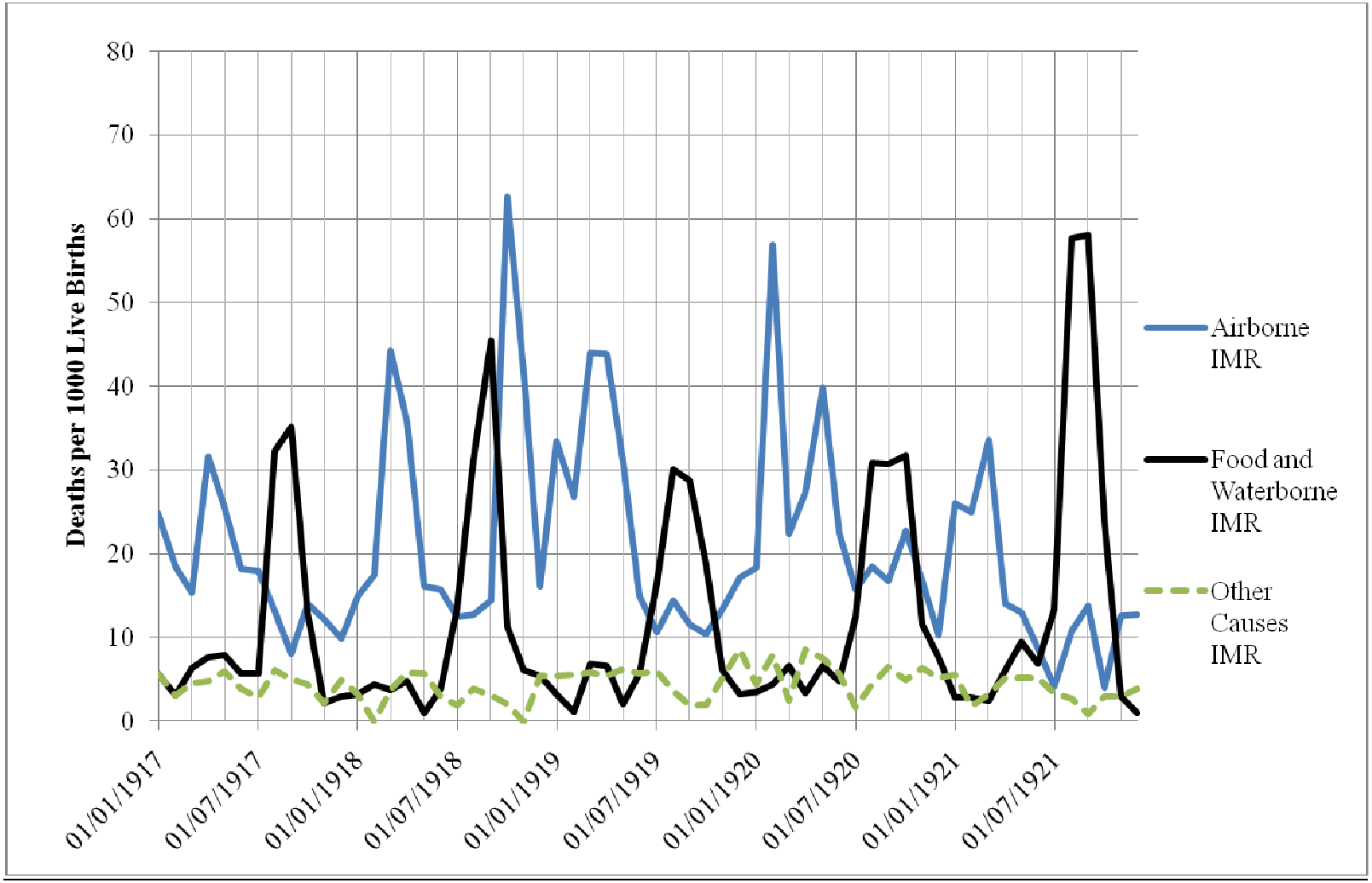

Figure 3 - Infectious Causes of Death, 1917-1921: Airborne, Food and Waterborne, and Other Infectious Causes of Death.

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 generally increase in 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 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 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 monthly variation in the airborne infectious disease rate was significant (ANOVA $p=.04$, SD=12.3, df=59, 95% CI). 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:[6] October to April 1918-1919, and February 1920. 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 7.

Month	Infant Death Rate From Influenza (per 1000 live births)
September 1918	0.00 (n=0)
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 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).[7] 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

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

[7] 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).

therefore may likely have been recorded as due to pneumonia,[8] 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, 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 is sufficient to explain this epidemic.

Conclusions

The 1918 influenza epidemic affected infant mortality rates in some ways, but many patterns remained unchanged. The overall infant mortality rate did not change significantly due to the influenza epidemic and this lack of variation 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

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

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. In general, diarrheal diseases have been shown to be higher for bottle fed infants than for breastfed 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). 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).[9] 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.

Discovering who was at risk of infection from food and waterborne illness may help to determine who was at risk from influenza. Diarrheal deaths are directly subject to the quality of water, access to sewer systems, and prior malnutrion (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 1918c, Sydenstricker 1931), but somehow had little effect on infant mortality rate in Toronto; it may be that those infants who were weak, malnourished, or socioeconomically disadvantaged were those

[9] August 1918 recorded both the highest peak temperature as well as the highest mean temperature of any August in the five year study period. September 1918 "was the coldest and wettest month that the city has experienced in 79 years" (The Globe 1918a:8, Environment Canada 2008).

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. This 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).

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 paper. 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 dying compared to those who did not. Young adults, the care-givers 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 as well as 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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