Does exposure to influenza very early in life affect mortality risk during a subsequent outbreak? The 1890 and 1918 pandemics in Canada

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Abstract

Using Canadian data, we explore how exposure to influenza very early in life during the pandemic of 1890 may have influenced mortality risk in the subsequent pandemic of 1918, twenty eight years later. As mortality in 1918 peaked at age 28 in Toronto and in other Canadian cities, we posit that infection with influenza in critical periods of development can result in physiological or immunological impairments that increase risk of death from influenza later in life. The peak at age 28 was most evident in large Canadian cities, while the pattern was still present in a less extreme form in rural areas. The 1918 influenza pandemic occurred during the health transition and, through enduring links to the 1890 pandemic, shows that experiences before the transition may have directly influenced the course of the most severe pandemic of this time period. This study provides new empirical insights connecting early physiological insults and immunological experiences to later life mortality.
The second demographic transition in North America involved a gradual shift from epidemic infectious diseases to those of a degenerative nature (which may themselves be of an infectious origin) during the 19th and early 20th centuries (Omran, 1971; Barrett et al., 1998). This long and progressive transition was briefly interrupted by the 1890 Russian influenza pandemic as well as the 1918 Spanish pandemic, two of the latest and most notable exceptions. At what is considered the end of “the Age of Receding Pandemics” (Omran, 1971) in North America came an epidemic in 1918 that has been estimated to have killed between 40 to 100 million people worldwide (Johnson, 2003), far more than the 9 million soldiers killed during the First World War (Patterson, 1986; Quinn, 2008; Storey, 2009). As explained by Crosby, “nothing else – no infection, no war, no famine – has ever killed so many in so short a period” (1989:311). Despite the vast number of casualties, the Spanish influenza pandemic rapidly left the public consciousness and often receives only a cursory mention in histories of the First World War (Crosby, 1989). This is even more applicable to the 1890 pandemic, which is all but forgotten in the collective memory and is seldom the topic of scholarly interest (Herring and Carraher, 2011a). The lack of attention given to the 1890 and 1918 pandemics may have influenced the theorizing about the stages and progression of the epidemiologic transition. As Barrett and colleagues suggest, “had the historical precedents of influenza been given closer consideration, previous projections for the continued decline in infectious diseases might not have been so optimistic” (Barrett et al., 1998:262).
The Spanish flu pandemic is notable for its unusual pattern of death as it killed unprecedented numbers of young adults between the ages of 20-40. This pattern has been well noted in the literature (Luk et al., 2001; Crosby, 1989) and confirmed recently in India, a country with a less developed statistical system (Hill, 2011). However, debate remains as to the exact reason for this unusual occurrence. Several scenarios have been proposed. First, it has been suggested that the excess deaths among young adults could be due to a negative interaction between tuberculosis and influenza, since young adult males were at greatest risk of tuberculosis, such that those individuals with a prior infection with and lung damage from tuberculosis were at greater risk of dying from influenza (Noymer and Garenne, 2000; Noymer, 2009). Contrastingly, the idea has been advanced that deaths among young adults resulted from an overactive immune response which caused the lungs to fill with fluid, known as a cytokine storm, causing death from drowning. As young adults are at the height of immunocompetency, this would have placed them at highest risk from the pandemic (Loo and Gale, 2007). Third, acquired immunity has also been hypothesized as a means by which older adults could have been protected through previous infection from a strain similar to the 1918 flu that would have been in circulation prior to 1890 (Ahmed et al., 2007). All of these explanations have been based on the general finding that young adults between the ages of 20-40 were at greatest risk, often with the highest unexpected mortality being found among those aged 25-29. However, it is generally agreed that key elements of the modern environment, including the rapid movement of people, the gathering of previously more isolated and thus more susceptible individuals, and the unsanitary conditions of the war were elements that contributed to the astounding spread and impact of this disease.

This chapter presents the results of preliminary research into the age-distribution of deaths among young adults during the 1918 pandemic. We show that, among various cities and regions in
Canada, it was not merely an entire age range that was at greatest risk, but that mortality centered on the specific age of 28. We use the hypotheses of fetal origins (Barker, 2006), scarring during critical periods of development (Preston et al., 1998), and original antigenic sin (Francis, 1953; Kim et al., 2009) to explain why these younger individuals at this particular age were at heightened risk. Is it possible that mortality risk in 1918 was conditioned by exposure to influenza 28 years earlier, during the other forgotten influenza pandemic in 1890? Was it not only the modern environment in 1918, in the mist of the second epidemiological transition, that allowed the spread of the influenza, but were certain people at greater risk because of the environmental conditions at the time of their birth?

Theoretical framework

Understanding the complex interrelationship between early life conditions and adult mortality has become an area of concern in the development of strategies for the protection of population health. Following the work by Barker on the fetal origins hypothesis, it is now established that inadequate nutrition during growth in utero results in compensatory physiological restrictions that increase the risk of metabolic and cardiovascular disease in later life (Barker, 2006). In the critical period model, exposures acting during specific windows of time have irreversible effects on health. There is also evidence that maternal exposure to a virulent epidemic disease can divert resources to the maternal immune response at the detriment of fetal maturation, leading to higher cardiovascular disease prevalence and mortality in the offspring at older ages (Almond and Mazumder, 2005; Myrskylä et al., 2010). More directly, infection with airborne infectious diseases in critical periods of development can harm lung tissue (causing scarring during critical periods of development), leading to greater susceptibility to (and mortality from) later airborne
infectious diseases (Preston et al., 1998; Bengtsson and Lindstrom, 2003). Critical period models also connect prenatal undernutrition to infectious death in adolescence. What has yet to be addressed is the possibility that exposure to a virulent agent such as influenza early in life may lead to increased susceptibility from later outbreaks of the same disease. It is unknown, for instance, whether sequential pandemics of different strains of the same disease are related, whether risk of death during a pandemic relates not only to health and immune status but also to exposure during the last major pandemic.

We first hypothesize that exposure to influenza during early development results in physiological impairments that increase risk of death later in life from airborne infectious diseases. According to the fetal origins hypothesis, we should expect to find a mortality peak at the age of 28 years during the pandemic of 1918 since 28 years separate this pandemic from the previous one in 1890. As this hypothesis posits detriments to long term health based on the gestational environment, those individuals who were exposed to a severe manifestation of the Russian influenza strain in utero in January 1890 would have experienced developmental impairments that would have increased their mortality in 1918, at the age of 28, potentially explaining the peak of deaths at the age of 28 (see below).

It is also possible that an immune response to the 1890 flu resulted in higher mortality in 1918, rather than it being the result of physiological sequelae from maternal infection or exposure early in life. According to the “original antigenic sin” model (Francis, 1953; Kim et al., 2009; Ma et al., 2011), the first influenza virus strain encountered during childhood conditions the immune response to that specific variant. Having built antibodies to this initial strain, the immune system will respond inadequately to a highly virulent and antigenically novel one. We posit as a second
hypothesis that “commitment” to a specific strain depends on both virulence and on the age at which it is first encountered. Since maternally derived antibodies provide protection against influenza in the first 6 months after birth (Beaudry et al, 1995; Munoz, 2003), infants do not have the capacity to mount an immune defence prior to that age. If the antigenic sin hypothesis holds true, then people born a year or two before 1890 were at a higher risk of death during the 1918 pandemic because they first encountered (and developed an immune response to) the 1890 strain at the youngest possible age and at the time of its maximum virulence. Important to this hypothesis, the pandemic strain in 1890 (thought to be either a subtype of influenza A/H2 or H3N8) had a quite dissimilar surface antigen than the strain in 1918 (influenza A/H1N1) (Dowdle, 1999; Taubenberger and Morens, 2006; Kolte et al., 2008; Valleron et al., 2010). Thus, this second overreaching hypothesis implies higher death tolls at ages slightly older than 28.

Scarring from infection in the first years of life could also cause deaths at age 28 as well as at slightly older ages. Using the information available to us, it would not be possible to separate the effects of scarring from original antigenic sin or gestational impairments, but scarring would not be expected to result in such a clear peak in mortality at age 28 (due to the higher risk of mortality at all ages following the first infection).

The 1890 and 1918 Pandemics in Canada

The age of exposure to the 1890 pandemic strain can be known with high precision because of the short duration of this pandemic, known as the “Russian flu.” Through newspapers accounts we have determined that its transmission in Canada was as swift as elsewhere (Valleron et al., 2010; Le Goff, 2011), leaving an impression of simultaneity. On the eve of the New Year, December 31st, 1889, a few cases were reported in North America in the Toronto Globe
newspaper but none, as of yet, in Canada (However, Patterson (1986) reports cases in December in Montreal). Then, on January 8, 1890, the disease was raging in most of Eastern and Central Canada. But after January 15 few new cases are reported in the Globe and practically none in the following months. Maris (2011) reports that the pandemic in Canada had peaked by the first week in January, although it continued along the Eastern coast of the United States until the end of the month. In Toronto, the highest numbers of deaths occurred between the end of January and mid-February, consistent with Patterson’s assertion that influenza deaths are normally first noticed to be elevated approximately four weeks after the start of an epidemic (Patterson, 1986; Ancestry.com). The disease is believed to have entered Canada from the port of Halifax, Nova Scotia and to have spread westward to the rest of the country along the transportation networks, but may also have entered the country through the railways linking New York and Chicago with Ontario (Thompson, 2011). It returned in the spring of the following year when new cases were reported in New York City. However, no mention of the affliction is found for Canada in 1891, although the disease could have very well been present in a milder form and Patterson (1986) does report cases in Canada in both the second wave (January to June 1891) and in the third wave (September 1891 to February 1892). It appears from studies conducted in England and Wales (Smith, 1995; Langford, 2002) that this pandemic strain was endemic for many years after 1890. More detailed research establishing the exact mortality curves from death records in Ontario from the 1890 flu will help to establish the true extent of this pandemic in Canada. However, all indications at present, including the case study of the Russian pandemic Hamilton by Herring and Carraher (2011b), are that the spread of the flu in Canada was similar to reports from other parts of the world. The Russian influenza was followed by a much milder pandemic in 1899-1900, reported to have been present in Ontario, that has been theorized as varying little from the previous strain, only on the H antigen, and was not nearly as virulent as either the 1890 or 1918
pandemics (Patterson, 1986; Dowdle, 1999). As is typical of any influenza variant, virulence most likely decreased over time from its pandemic peak until it was fully replaced by a novel strain in 1918.

Similarly, the Spanish flu pandemic was also of short duration. While the exact geographic origins of the 1918 epidemic have not been established (theories suggest either the battlefields of France or an American training base in Kansas, see Oxford et al., 2002; Barry, 2004; Humphries, 2005), in Canada, the disease is thought to have entered the country through a military training base in southern Ontario and from there to have spread westward with the Siberian Expeditionary Forces (Humphries, 2005). It appears to have first struck during the second global wave in the fall of 1918. Deaths were most numerous in October, although the epidemic continued throughout November and December, followed by a less severe resurgence during the third wave in the spring of 1919. The flu was initially thought to have entered Canada in late September 1918 through a military camp in Lincoln County, Ontario (Humphries, 2005).

The Russian and Spanish influenza pandemics are similar in that they are both described as completely global and reliant on rapid trade and transit for their almost instantaneous spread (Patterson, 1986; Crosby, 1989; Le Goff, 2011). Yet, there are two major differences between the pandemics in 1890 and 1918: the case-fatality rate and the age-structure of mortality. Valleron and colleagues report that the Russian influenza had a clinical attack rate of 60% (interquartile range of 45-70%), while the case-fatality rate ranged from 0.1% to 0.28% (2010:8778). This suggests that approximately 60% of any given population would have contracted the flu during the pandemic, but of those who became ill, only 0.1% would die from the disease, resulting in relatively few deaths being attributed to the pandemic. This is significant
for this study since high morbidity and low mortality in 1890 means that many individuals would have been exposed early in life and would have survived to meet the pandemic in 1918, thus giving us a sample of susceptible individuals. It is typical of influenza epidemics and pandemics to infect far more people than those who die (Patterson, 1986; Glezen and Couch, 1997), but those who usually die are those most at risk: infants, the elderly, and the immunocompromised. This results in the typical ‘U’ shaped mortality curve, where the two highest rates of death are among the very young and the very old (Crosby, 1989; Valtat et al., 2011). While 1890 pandemic followed the classical influenza pattern (Morens and Fauci, 2007; Valtat et al., 2011), the 1918 Spanish influenza is known for the ‘W’ shaped age-distribution of deaths, where young adults were at an unexpectedly high risk of death from the disease. In contrast to the case-fatality rate of 0.1 to 0.28% in 1890, the 1918 pandemic had a case-fatality rate greater than 2.5% (Taubenberger and Morens, 2006), while the clinical attack rate ranged from 20 to 60% (Morens and Fauci, 2007).

**Materials and Methods**

For our analysis, we utilized the registered death records from September to December 1918 for the Ontario cities of Toronto, Ottawa, London, and Hamilton, and the counties of Welland and Lincoln in southern Ontario. We focus on Ontario since the microfilmed death records for the pandemic period have been digitized and are available online (www.ancestry.ca). However, we also utilized the indexes of the registered death records for Winnipeg, Manitoba, and Vancouver, British Columbia, in order to compare experiences in major Canadian cities covering a large geographic distance. From the data available in the complete death records for Ontario, information on sex, age at death, date of death, and cause of death were recorded. The death
records for Manitoba and British Columbia are not available online, but the indexes containing sex, date of death, and age at death are available through the Vital Statistics Agency of Manitoba (http://vitalstats.gov.mb.ca/index.html) and the British Columbia Archives (http://www.bcarchives.gov.bc.ca/index.htm). Therefore, for the cities and counties in Ontario we have information on both total deaths for the four month period as well as death specifically from pandemic-related causes (considered to be influenza, pneumonia, and bronchitis). These three causes of death have been grouped together as ‘influenza-related’ since death during the pandemic was generally caused by secondary bacterial infections of the lungs (Harder, 1918; Crosby, 1989). Selecting individuals who only had a cause of death of ‘influenza’ would miss a substantial proportion of pandemic-related deaths. For Winnipeg and Vancouver, however, we are limited to an analysis of deaths from all causes. As population totals are as of yet unavailable for these cities in 1918, we are unable to calculate rates of death. However, as a comparison, the total numbers of deaths were recorded for each city for September, 1918, before the epidemic had entered Canada (see Table 1 for the populations and densities for each city from the 1921 Census). As can be seen in Figure 1, the mortality pattern in Toronto was drastically different during the epidemic than for the preceding, non-epidemic month.

[Table 1 about here. Table 1 Caption: 1921 population size and population density (Canada, 1924).]

Death registration is subject to different forms of error for various reasons. Although death records for Ontario are deemed to have become complete by around 1920 (Emery, 1993) due to careful legislation requiring a death certificate before a grave can be dug, it is possible that the deaths of some people were not recorded. Those individuals not present in the records are
probably a select group (more likely to be immigrants or the impoverished), but as this is not a nominal analysis, individuals not missing at random are not of a large concern as it is unlikely that they would over-represent any specific age.

The deaths records themselves have been transcribed multiple times, beginning with the initial individual who filled out the record. From there, the records were sent to the Registrar General for Ontario where they were transcribed into a comprehensive register (Emery, 1993). Much later, the records were transcribed onto microfilm, then digitized and made available on the internet. At any stage, inaccuracies in transcription may have occurred. The extent to which this may have influenced the results will be assessed in a future records linkage project meaning to confirm exact date of birth of the individuals who died during the pandemic in Ontario.

**Results**

Figure 1 reports the total number of deaths by age recorded during the deadliest wave of the Spanish flu in Toronto in October 1918 and in the surrounding months. Strikingly, mortality peaked at age 28, precisely for the generation born at the time of the peak of the previous influenza pandemic, in January 1890.

[Figure 1 about here. Figure 1 Caption: Number of deaths by age from all causes in the City of Toronto, September to December 1918 (September, n=441; October, n=1885, November n=731, December n=618. Total n=3675). The vertical line indicates age 28.]
Also apparent in Figure 1 is the elevated number of deaths among young adults aged 20-40, which was noted in contemporary accounts and that has been reinforced by more modern analyses (Harder, 1918; Oertel, 1919; Luk and Gross, 2001; Crosby, 1989; Phillips and Killingray, 2003; Taubenberger and Morens, 2006). Due to available population data, however, these previous investigations have collapsed yearly ages at death into age-groups (20-24, 25-29, 30-34, etc.) and, as a result, were not able to reveal if there was a noteworthy peak at any specific age. Yet, in Toronto, death totals at age 28 were higher than in any single age between the ages of 20 and 25 or between 31 and 35, which were abnormally high as well. Figure 1 also shows a secondary peak in deaths at the age of 30, which would represent those individuals who met the 1890 pandemic at the earliest ages to mount an immune response. This can also be seen for deaths from pandemic related causes in other cities in Ontario.

It is possible that some of our results are influenced by age-heaping at ages 28 and 30, with correspondingly less deaths attributed to age 29 and to age 31 (see Figure 1 for heaping evident in older ages). Age heaping may occur in historical data because age declaration often tends to be rounded up or down to the nearest number that ends in 0, 2, 5, or 8, especially among the elderly or the uneducated. The level of such bias can be estimated with Myers’s summary index (Myers, 1940), which measures the amount of preference for a specific terminal digit while accounting for the effect of mortality, such that there would be expected to have more individuals in a specific decade with a terminal digit of 0, decreasing from 1 to 9 (for example, there are more people aged 80 than aged 89, simply due to the increased mortality rate with age) (Hobbs, 2004). Using this method, a summary index with a value close to 0 represents no heaping and 90 would indicate all deaths being reported at the same terminal digit (Hobbs, 2004). In our data, the summary index for all cities and regions pooled together for the four month period is 4.21, a
relatively low figure. Redistribution of the deaths between the ages of 25-34 would not be of such a magnitude as to reduce the pattern evident. Yet, it is possible that deaths at age 29 were slightly underreported, causing deaths at the ages of 28 and 30 to be increased.

To show that this pattern is due to pandemic-related causes of death, Figure 2 shows deaths from influenza, pneumonia, and bronchitis for the cities of Toronto, Ottawa, Hamilton, London, and the counties of Welland and Lincoln, Ontario. Again, the deaths are elevated for those aged approximately 25 to 34, with the peak coming at the age of 28. The same pattern of ages at death can also be seen when looking at two major Canadian cities outside of Ontario (according to the 1921 census the populations of the four major cities we analyzed were: Toronto = 521 893; Ottawa = 107 843; Vancouver = 117 217; and Winnipeg = 179 087) (Canada, Dominion Bureau of Statistics, 1921). Figure 3 shows the age-distribution of deaths from all causes from September to December 1918. Among young adults, the most deaths occurred at age 28 in Winnipeg and there are peaks at both age 28 and 30 for Vancouver, with age 30 having the higher number of deaths.

[Figure 2 about here. Figure 2 caption: Deaths from influenza, pneumonia, and bronchitis, September to December 1918 in selected Ontario cities. Toronto n=2195. The vertical line indicates age 28.

*Includes London (n=290), Hamilton (n=536), Ottawa (n=640), and Lincoln and Welland Counties (n=550).]
[Figure 3 about here. Figure 3 caption: Deaths from all causes, Winnipeg (n=1193) and Vancouver (n=1141), September to December 1918 (excluding infants). The vertical line indicates age 28.]

To highlight the experience of young adults, Figure 4 shows the deaths at each age from 15-45 as the percentage of the total mortality. It displays mortality from all causes from every city combined as well as the pooled flu fatalities from all cities and regions in Ontario for the four month period of September to December, 1918 (the only province from which we had cause of death data readily available). Clearly, the age of 28 accounted for the highest percentage of total young-adult mortality during this period, with a secondary peak at age 30. This is more pronounced for those individuals whose listed cause of death was pandemic related, revealing that this phenomenon was specifically the result of the 1918 influenza epidemic.

[Figure 4 about here. Figure 4 caption: Percentage of deaths from all causes and from influenza from ages 15-45, September to December 1918. The vertical line indicates age 28.


**Deaths from influenza, bronchitis, and pneumonia from Toronto, Ottawa, Hamilton, London, and Welland and Lincoln Counties.]

Discussion

Our research into selected Canadian cities confirms the finding that young adults, specifically those between the ages of 25 and 35 were at greatest risk of death from the 1918 Spanish
influenza pandemic (Crosby, 1989; Luk and Gross, 2001; Loo and Gale, 2007). Previous investigators proposed mechanisms that would explain the higher death tolls among young adults, such as tuberculosis or an overactive immune response (cytokine storm) (see above). But, none of these mechanisms can account for the concentration of deaths at a specific age. Significantly, we found that those at the ages of 28 and 30 were most at risk. Future research will need to use dates of birth (through linking birth and death records) to determine exactly the timing of potential exposure during in utero development and infancy, to the month or even to the day. This will help to distinguish whether the observed patterns are the product of scarring during critical periods of development of very young individuals (deaths at age 28) or the result of early exposure to an antigenically different strain of influenza, our main working hypotheses (deaths at ages slightly older than 28).

For the time being, we note that even though our study does not allow us to distinguish between the two scenarios for the increased death toll at age 30, the situation for age 28 is much clearer. As these individuals would have been in utero or the majority still exclusively breastfeeding (thereby protected by the mother’s antibodies and not producing their own) at the time of the 1890 pandemic (Beaudry et al., 1995; Munoz, 2003), their response to the 1918 flu would have resulted from an insult during a critical period of development and not primarily from antigenic sin.

As seen in Figure 1, mortality was increasing from around the age of 10 to the peak at 28. According to our reading of the theory of antigenic sin, this could be the result of exposure to less and less virulent forms of the 1890 flu that continued to circulate in the population in the years after 1890. Likewise, mortality was declining after the age of 28, which may have been the result
of children who had encountered the 1890 pandemic at slightly older ages and who had presumably already developed antibodies to either another influenza strain, or to the 1890 virus but in a less virulent form. Their response would then have been less specific to the 1890 pandemic variant, allowing for better protection from the 1918 strain because they were less compromised. Patterson reports that the last flu epidemic to hit North America occurred in 1873-74 (including “the United States, France, Germany, Australia, and Sweden”) (1986:51). The individuals born during this period would have been 16-17 in 1890 and 44-45 in 1918, while anyone born between 1874 and 1889 would probably have been exposed to this flu variant early in life, through the circulation of seasonal influenza strains. Alternatively, if we suppose that an H1N1-like virus was circulating and drifting during the years leading up to the 1890 antigenic shift (Palese, 2004), then it is possible that those born say, around 1878 were exposed early in life to a strain that was closer antigenically to the 1918 strain than those born, say in 1888. If so, the susceptibility and mortality of those aged 30 would have been higher in the 1918 pandemic than for those aged 40. At the other end of the spectrum, some elderly people (60-65+) may have been spared in 1918 by earlier exposure to an influenza virus similar to the 1918 strain that circulated prior to 1890 (Luk and Gross, 2001), although mortality at older ages was also high during this pandemic (Crosby, 1989)

The second epidemiologic transition is associated with industrialization and urbanization. However, these were exactly the conditions that aid in the spread of airborne infectious diseases. Influenza spreads quite easily in the winter, since people cluster in heated indoor environments and hot, dry air weakens the mucosal surfaces of the nasal passages, making it easier for the virus to enter the lungs (Kaslow and Evans, 1997). Through rapid migration, major Canadian cities, such as Toronto, were overcrowded with cramped living conditions, especially among
immigrants and the most impoverished members of society (Mercier, 2006). Not only were these conditions conducive to the spread of influenza, they also helped to propagate tuberculosis infections (Sherman, 2006). And, as hypothesized by Noymer and Garenne (2000) and Noymer (2009), tuberculosis infection may have intensified deaths among young adults from the flu in 1918, since both diseases targeted these populations and previous lung damage from tuberculosis allowed for earlier and more intensive infections with influenza and the ensuing secondary bacterial infections of the lungs. However, the early life exposure to influenza is the preferable hypothesis, as it is not clear as to how prior infection with tuberculosis could lead to higher death tolls at specific ages.

The very modern and novel ‘Great’ War brought about the conditions of spread for this pandemic. Technological advances and an increasingly interconnected world allowed the four-year long stagnant war with its resulting horrific conditions in the trenches throughout northern Europe. Many soldiers came from rural areas, such that they would have had limited previous exposure to crowd diseases (simulating the effects of virgin soil epidemics) and their immune systems would have been greatly strained when hit by the such a highly virulent influenza virus in 1918 (the modern development of trench warfare not only allowed the spread of the flu but was directly responsible for its rapid, global, spread) (Crosby, 1989). Beyond the cold, wet, and disease-ridden conditions faced by the soldiers, the war led directly to food and energy rationing and stressed civilian populations, complete with the concomitant immune-compromising effects of deprivation and psychosocial stress (Livi-Bacci, 1991). Thus, it was modern conditions that allowed for both decreases in infant and maternal mortality and the resulting extensions of the average life-expectancy but also facilitated the widespread devastation of the 1918 influenza pandemic (medical and technological advances, respectively). Yet, reduction in exposure to
epidemic diseases early in life starting at the end of the 19th century, coupled with the further reductions in mortality in the first quarter of the 20th century, could have made the early life signal of the 1890 pandemic “stronger” and more decipherable in 1918. In previous historical periods, early life effects were likely blurred by overall higher levels of infant and adult mortality (Gagnon and Mazan, 2009). When overall mortality decreases, it becomes possible to detect more subtle changes in typical mortality patterns. Perhaps the epidemiological transition was the necessary historical prerequisite for a clear manifestation (and thus understanding) of early life exposure to influenza on later life mortality from the same disease.

**Conclusion**

Previous research has been valuable in highlighting the alarming experience of young adults during the 1918 flu. Those who are generally the healthiest and most productive in society, who had just survived 4 years of global war, were dying in unprecedented numbers. This could not be adequately explained in 1918 and a universally approved hypothesis has yet to be proposed. Consistent with current literature, we propose that individuals who died in 1918 cannot be analyzed in isolation: their deaths were a product of their experiences in the world around them, extending from conception to the last moments of their lives. The exact age at which they met the previous pandemic strain of influenza in 1890 could have been the deciding factor as to whether they survived the pandemic of 1918.

Even though there has been vast improvements in health and life expectancy in certain parts of the world in the twentieth century, it cannot be universally asserted that the modern world is invariably good for human health. Instead, the modern world created the conditions that allowed
for the deaths of up to 100 million individuals in the 1918 Spanish influenza pandemic and there is little reason to believe that it could not happen once more; it could be hypothesized that in conditions where there is overcrowding, poor nutrition, poor sanitation, and high levels of endemic infectious diseases, that epidemics, and potential pandemics will not be far behind. Our analysis demonstrates that previous experience early in life with the various forms of the influenza virus may affect the age distribution of susceptible individuals in future pandemics and should be addressed in order to guide our responses to outbreaks and to inform our preventative policies.
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Figure 1.
Figure 2.
Figure 3

![Graph showing the mortality rates in different regions of Ontario, with a comparison to Toronto. The graph depicts the number of deaths (N) against age at death, highlighting a peak in mortality in the 25-30 age group.](image-url)
Figure 4.