“SEND ONLY YOUR SERIOUS CASES.” DELIVERING FLU TO TORONTO: AN ANTHROPOLOGICAL ANALYSIS OF THE 1918-19 INFLUENZA EPIDEMIC IN TORONTO, ONTARIO, CANADA

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Doctorate of Philosophy

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by

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The undersigned, appointed by the Dean of the Graduate School, have examined the dissertation entitled

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Presented by Karen Slonim

A candidate for the degree of Doctor of Philosophy

And hereby certify that in their opinion it is worthy of acceptance.

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Professor Mark Flinn

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Professor Matthew Gompper

_____
Professor D. Ann Herring

This dissertation is dedicated to Ethan Slonim Reusch. Don't ever let anyone else define your potential. If you invest your heart, your brain will follow.

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ABSTRACT

This project looks at the 1918-19 pandemic influenza experience in Toronto, Ontario, Canada. Based on historical records (most notably death registries and archival material) this work strives to understand the social, biological, and environmental conditions that facilitated the spread of this virulent microorganism throughout the city. Grounded in the anthropological frameworks of evolutionary medicine, political economy and syndemics, this dissertation is designed to present a qualitative historical account of the pandemic in one of Canada's largest cities. This piece adds to the growing body of literature aimed at documenting one of the most catastrophic events of the 20th century. It also explores how epidemics are shaped by and in turn shape history. Many of the key findings of this work stem from the relationship between the Great War and the H1N1 strain responsible for Spanish flu. Soldiers appear to have brought the disease to Toronto and the conditions generated by the prolonged conflict in all likelihood increased individual susceptibility (via. increased stress, sustained food shortages and promotion of status incongruity). It is important to note however, that the effects of the war were not all detrimental to the population of Toronto. Sustained investment in the military effort promoted the development of informal networks of care, which were paramount in the city's effort to curtail influenza mortality. This dissertation generates as many questions

as it answers, with the main message being that an analysis of infectious disease experiences must be cognizant of the two-way linkages between culture and biology.

Chapter 1 Introduction

It is exceedingly difficult for me to write about why I study flu, for my love affair with the disease was born out of the idea that the pandemic experience had largely been forgotten. I was enthralled by the social construction of this disease. How could a malady that took the lives of so many be perceived as so benign? While in the midst of writing my MA thesis examining the pandemic experience of two Aboriginal groups in the Lake Winnipeg region of Manitoba, this attraction faded away as the experience of SARS re-conceptualized how the world perceived viral threats. Much like a train accident that results in a fear of flying, the expert and lay individual alike took this brush with SARS as a reason to be apprehensive about the looming threat of pandemic influenza[1]. Since 2004 public health agencies have created numerous pandemic plans[2], often anchoring perceptions of the disease to the experiences of 1918. Rows of hospital beds and individuals donning masks generated fear and panic among increasingly germ-conscious citizens (Tomes 1998). This newfound attention has transformed influenza from humdrum to heartless mercenary, bringing along with it an increase in fear and heightened surveillance and shifting flu from forgotten to foreboding.[3]

As is true with many relationships in life, although the initial reasons for attraction may have fallen away, passion for the subject need not be diminished. I am

[1] This was also assisted by fears of avian flu sparked in 1997.
[2] For example, Health Canada (2004), United States Department of Health and Human Services (2004), UK Health Department (2005)
[3] For a more complete discussion of this phenomenon see Herring (2008), Lockerbie and Herring (2009) and Herring and Lockerbie (2010).

left, still in love with flu, but less attracted to its shiny exterior and more enthralled with the nuances of its 'insides.' I have put away my red shirt with its black writing that read "Influenza Kills" and instead find myself telling people "you might get sick, but chances are you are not going to die." I see flu in a different light, and I think in many ways this dissertation reflects that. I am no longer interested in capturing the shock and awe associated with the disease; rather it is my desire to better understand *who* got sick and what were the contributing factors that aided in both the increase and decrease of mortality.

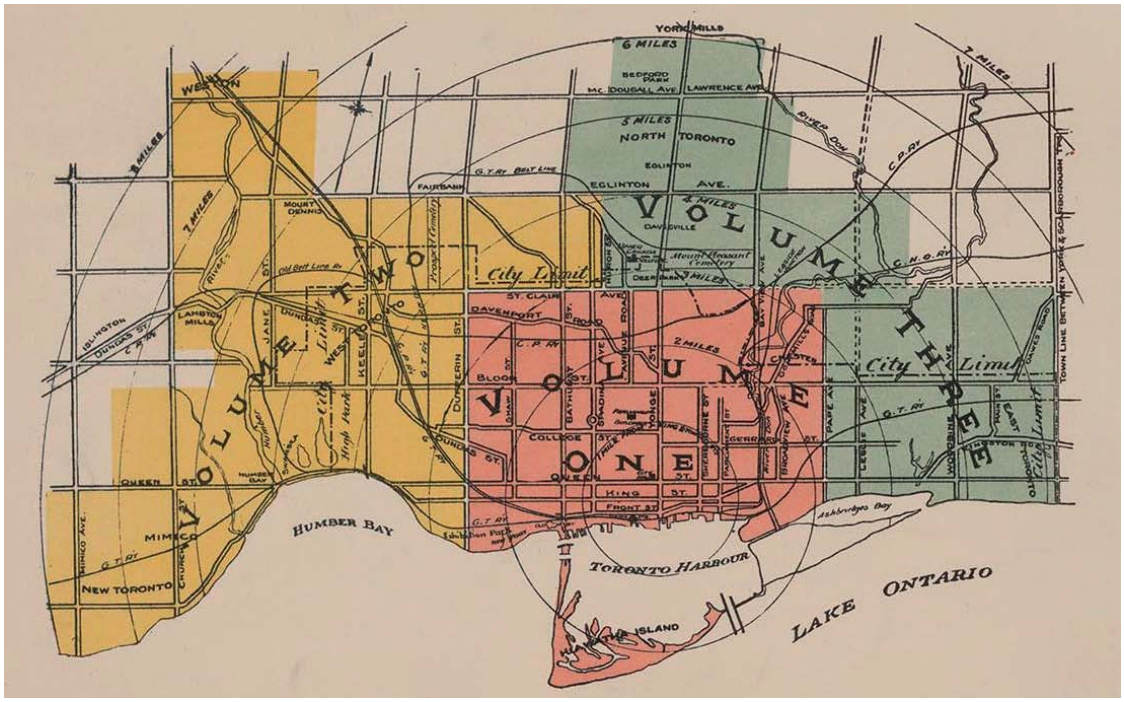

Figure 1.1 - Central Atlas Key to the City of Toronto 1910. Goad's Atlas. City of Toronto Archives OV-FLAT 368.11 0223 G53 1984.

The setting for this work is the Canadian urban city center of Toronto (Figure 1.1). The image was taken from a set of fire insurance plans drafted by Charles E. Goad in 1910. It not only demonstrates Toronto's physical layout but provides a sense

of the constant growth of the city. Toronto was chosen for its importance on the Canadian landscape, its diverse population and its rich archival resources. In 1918 the city was in the midst of a massive population boom more than doubling its size from 1901 to 1913 (Harris 1990: 388). The beginning of the 20th century was also marked by a rethinking of the public health system, which called for improved services, better regulation of foodstuffs, housing reforms, and contagionist conceptions of infectious disease transmission. Like most other major cities of the world, Toronto was hard hit by World War I (1914 – 1918), sending some 70,000 men overseas and suffering massive material shortages, which resulted in population aggregation and a 50% increase in the cost of living between 1915 and 1919 (Benn 2006).

The city of Toronto was also chosen as very little research had been carried out on the city to date. Only two authors, Eileen Pettigrew (1983) and Heather MacDougall (2006), have examined the epidemic experience in the city in any detail. Pettigrew presents a cursory overview of the impact of the epidemic over the Canadian landscape and MacDougall (2006) uses the 1918 influenza epidemic as a point of comparison with the 2003 'outbreak' of SARS in the city, focusing predominantly on the action of the Toronto Health Department.

The problem and place were easy decisions, but choosing a theory in which to ground this research was a daunting task. As I reach the end of my tenure as a student of anthropology I am still faced with the same quandary I had going in. Why do we specialize? Do other fields do it to the same degree we do? I have now taught an introductory anthropology course and despite my reservations I began by describing our four field approach. When it came time to assign myself to a category I stuttered,

for I am in full agreement with Moran (1998) that early studies of health within anthropology are not overly helpful due to their inherent separation of biology and culture. I therefore knew that this work would be grounded in biocultural anthropology, for it celebrates our discipline's ability to understand the social, environmental, and biological dimensions of problems surrounding health. Dubos (1977:34) states

> Exposure to one of several viruses is a *necessary* condition for the development of the common cold but not a *sufficient* condition. Exposure results in disease only when the exposed person is in a receptive state. This receptivity is in turn affected by the season, the weather, and almost certainly by a host of other ill-defined factors…

But the biocultural paradigm in itself is too vague and I wanted to tether my results to more grounded theory. I was looking for epistemologies that would not forsake the richness of people's lives but rather to help elucidate the "ill defined" conditions that help to shape the disease experience. It was my hope to find theories that respect the importance of history, and target "the way people *really* approach illness and cope with death" (Nations 1986: 97). I also looked for ideologies that perceived the world as a continuous feedback loop where interactions between microscopic agents and human beings are created by process in which behavioral choices are sorted out within a developing sociocultural system (Alland 1970). Further I desired principles that were not based on a Cartesian dualism (Scheper-Hughes and Lock 1987), but that viewed biology and culture as inextricably linked. To this end the theories of political economy, syndemics and evolutionary medicine were utilized to help make sense of the disease experience in Toronto.

The primary objectives of this work are three-fold. The first was to capture the details of pandemic mortality during the second wave of influenza in Toronto which encompasses 1 September to 31 December 1918. Second, I wanted to examine the relationship between war and disease; and finally I wanted to add to the literature that examines health through a political economy, syndemics or evolutionary medicine lens.

This thesis is organized into eight chapters. Chapter 2 introduces readers to the three frameworks that provide the backbone to this work. It is designed to provide a general understanding of the epistemological frameworks that were utilized as well as specific information as to how they shaped this analysis. Chapter 3 provides background information on the study area as well as a brief synopsis of details of the virus, pandemic and the epidemic within the city of Toronto. Chapter 4 describes and evaluates the documents utilized for this thesis. Chapter 5 adopts an anthropologically oriented epidemiological framework to examine the interrelationship of the virus, host and environment. Chapter 6 strives to capture a qualitative look at the social conditions in which the pandemic was framed. Chapter 7 looks directly at how World War I may have altered the disease experience in Toronto and Chapter 8 is a synthesis of information derived from the preceding three chapters, introduces rich areas for future research and applies lessons learned from the 1918-19 influenza pandemic to the 2009 H1N1 influenza experience.

I have often been told that a dissertation should not be your life's work; it is the piece that paves the way for your life's work to begin. This dissertation does not mark the end of my journey as a student but rather the beginning of my time as a scholar. In

essence it serves the same function as an archaeological survey creating a map of the potentially richest areas to dig.

Chapter 2 Frameworks

One of the beautiful elements of anthropology is the discipline's ability to incorporate elements from both the biological and social sciences. This diversity provides numerous approaches, or theories, aimed at contextualizing data. As an anthropologist, the decision of where to focus one's analysis can be daunting, as each of these theoretical paradigms brings with it new questions, challenges and ways in which to explore one's research material. In order to embrace the discipline's richness this dissertation is grounded in three frameworks: evolutionary (or Darwinian) medicine, political economy theory and syndemics. This chapter presents a brief history and description of each of these theories as well as ways in which they have helped to shape this research project.

Evolutionary Medicine

Evolutionary medicine explores the relationship between selective pressures and human health. In order to understand how this theory operates, a basic understanding of the principles of evolution through natural selection is required. An extremely simple description of this process is as follows. Environments[4] exert particular pressures on the organisms inhabiting them. Those organisms that are best able to contend with these pressures, attract mates and in turn produce viable offspring[5] will

[4] Gluckman et al. (2009) provide an excellent definition of what is meant by environment. They state "the environment of an organism is the sum of all the external conditions and stimuli that it experiences, including climate, nutrient supply, social structure resulting from cooperation with or competition from other members of its own species, and threats from other species in the form of predation, parasitism or infection" (Gluckman et al. 2009: 4).

[5] Applicable for organisms that reproduce sexually

have a greater reproductive success, thereby passing on a greater number of genes to successive generations. Working with the phenotypic expression of these genotypes, those traits that increase survivorship will be carried forward. For example, antibiotics kill bacteria by inhibiting cell-wall synthesis and protein synthesis. Bacteria are able to evade this process by four main mechanisms: barring antimicrobial agents' entry, rapid extrusion of the drug, enzymatic modification, thereby rendering the treatment inactive or alteration of the molecular target of the antimicrobial agent (Berkowitz 1995). Therefore, bacteria possessing genes that code for these traits will have a greater fitness within environments where antibiotics are heavily relied upon, thereby leading to increases in bacteria with drug resistance[6].

The term Darwinian Medicine was first used by Williams and Nesse (1991), who explored the predictive significance of natural selection and the complex evolutionary relationship between infectious and chronic disease and immune function. Their work called for a new way to conceptualize health and illness, one that was based on significant time depth and cultural context. From its inception, evolutionary medicine has achieved widespread public and academic attention (Trevathan et al. 2008). Although the moniker, evolutionary medicine, was relatively new, the ideas encapsulated within the theory were not. As early as 1796, Erasmus Darwin[7] was writing about the importance of an evolutionary perspective in medicine (Nesse 2008). Physical anthropologists, long interested in the significance of

[6] Not to confuse the point, it is important to note that the majority of bacterial species do not reproduce sexually; however, they are able to acquire genetic material from an external source in one of three ways. The first is conjugation (where a plasmid is passed from one organism to another via a pilus). This can occur between bacteria of the same or different genera or families. The second is transformation in which DNA may be assimilated from an external environment and the third is transduction where genetic material is obtained from an infecting bacteriophage (Berkowitz 1995).
[7] Grandfather of Charles Darwin

evolution, were among the first to try to understand and document this complex relationship. In the early twentieth century, working in conjunction with geneticists and biologists, anthropologists explored how humans contend with particular environments from high altitude to extreme temperatures (Trevathan et al. 2008). In 1958, the physical anthropologist Frank B. Livingstone published a groundbreaking study that outlined the complex relationship between mosquitoes, malaria, humans and genetic adaptation[8], further demonstrating the advantages of an evolutionary perspective.

This methodology for understanding health is multifaceted and has enormous implications for how fitness and disease are to be studied, treated and contextualized. Current studies include, for example, appreciating how the environment in which hominin evolution took place has shaped current health realities,[9] the adaptive significance of evolved defenses, [10] or understanding genetic 'disorders',[11] pathogen evolution, and mental illness. This list is by no means exhaustive, for the human animal is shaped by evolutionary processes and therefore every aspect of human life can be interpreted in some way as a reflection of this process. Although many of the ideas encapsulated within this dissertation have been shaped by an evolutionary perspective one concept has been paramount in guiding this research: Ewald's work on pathogen virulence (see for example Ewald 1994, 1983, 1990, 1991)

[8] Through the presence of the sickle cell trait.
[9] For example, see Wiley's (2008) work on lactose tolerance, Turner et al.'s (2008) work on food availability and choices or Eaton's (1994) exploration of menstruation and first birth and reproductive cancers.
[10] Examples include fever and iron withholding (Nesse and Williams 1994) or stress (Brunner and Marmot 1999).
[11] Work in this area covers topics such as the relationship between cystic fibrosis and cholera or sickle cell anemia and malaria.

There is a common misconception that given time pathogens will evolve toward lower virulence in the hopes of sustained transmission possibilities (Sherman 2006). Ewald challenges this notion, and instead suggests that pathogens, like most living creatures, are at their core opportunistic and therefore virulence is a byproduct of how pathogens move from an infected to a susceptible host. According to Ewald's work, pathogen success is dependent on two factors: the parasite's ability to use a host for 'food' and the greater number of progeny and transmission that might result from exploitation of the host. If a pathogen is too virulent it may impede its host's ability to introduce it to new susceptibles and when being too frugal the disease may not be abundant enough to transmit itself when the opportunity presents itself. If an immobile host is selected for, then increased virulence may be desired, whereas if opportunities for transmission are limited, a strategy of latency (or frugality) may be employed (Ewald 2002).

In order to illustrate how his theory works, Ewald has explored a number of diseases within an evolutionary perspective aimed at understanding how pathogen virulence is connected to its mode of transmission. Water-borne and vector-borne pathogens, Ewald suggests, are not dependent on host mobility for transmission and therefore may reproduce unchecked within the host[12] (Ewald 1983, 1990, 1991, 1994). Take for example malaria. Pathogen transmission is more likely to occur in cases where extensive and systemic spread of the *Plasmodium* sp. is present as the biting vector is more likely to ingest the pathogen. Furthermore, the infected host is less

[12] Fecal-oral transmission is facilitated in the cases where an attendant is present to transport fecal material away from the infected individual to others in the population, as is the case when soiled bedding is taken to be washed in a local water sources by an individual attending to the immobilised patient. In the case of vector- borne disease lowered virulence within the vector rather than the vertebrate host would be selected for.

likely to defend itself from the mosquito's bite if it is immobilized. HIV also presents an interesting case study by which to understand pathogen virulence. Analyses demonstrate two major strains of HIV: HIV-1 and HIV 2, which diverged from a common simian virus 80 years ago and subsequently split from one another.[13] Available evidence suggests HIV-2 is less virulent than HIV-1 and Ewald postulated that HIV-1 is more prevalent in areas with greater diversity of sexual practices (Ewald 1990, 1994).

According to Ewald (1990, 1994), the influenza virus that sparked the pandemic of 1918–1919 was an evolutionary abnormality, which was the product of extraordinary circumstances rather than simple biological fitness. He attributed the conditions created by trench warfare and the overcrowding of hospitals and transport vehicles as the determining factors in influenza A's success. To illustrate his point, in his 1994 work, Ewald provides an account of an ambulance driver. In it the ambulance driver recollects his transport experience of servicemen (infected and susceptible alike), stating the men would travel in crowded vehicles to arrive at crowded hospitals only to be transported via crammed trains and boats to their countries of origin. He further identifies a general populace that was immunocompromised both psychologically and physically by the ravages of war, and attributes this to the spread of the pathogen. Ewald terms this idea the 'cultural vector hypothesis,' which suggests that the influenza mortality experienced in the latter half of 1918 was an evolutionary byproduct of the war rather than being just coincidental with its end (Ewald 1994). Much like the blood meal carried by a mosquito from an incapacitated malaria victim to an unwitting new host, the sick were carried by gurney into pools of susceptibles via

[13] According to Ewald, HIV-2 is the ancestral form and HIV-1 the derived.

the cultural vectors put in place to contain sickness. One of the endeavors of this thesis is to see how Ewald's theory plays out in the disease experience in Toronto, not simply stopping at the introduction of the malady but also examining how perceptions, mobility, social circumstances and actions of the inhabitants of the city may have helped or hindered this abnormally virulent pathogen.

Political Economy

A cursory reading of modern political economic theory within anthropology would lead a reader to believe that this ideology originated within or as an outgrowth of Marxist writing. It was not, however, Marx's creation of the theory, but rather his critique of it which is utilized to construct the anthropological theory. A letter to Joseph Weydemeyer (March 5, 1852) captures this statement well; in it Marx critiques political economists for talking in universals:

> …no credit is due me for discovering the existence of classes in society nor yet the struggle between them. Long before me bourgeois historians have described the historical development of this class struggle and bourgeois economists the anatomy of the classes. [quoted in Venable 1945: 6, n.3]. (Wolf 1997: 20).

The term political economy was first used (in French) in 1613 but was not widely utilized as a theoretical concept until the second half of the eighteenth century (Burke 1979: 271). In its original inception, political economy was a theory discussed by economists who wished to study the wealth of nations (with reference to the value of labor) in contrast to the earlier ideas of Physiocrats who attributed wealth to landholdings. This new incarnation was the by-product of Adam Smith's *Wealth of Nations,* which to many marks the beginning of economics as a formalized discipline (Burke 1979: 283). The applicability of political economy theory within anthropology

as stated is a direct outgrowth of the issues brought forward by Marx's critiques of these earlier writers. Marx integrated 'Culture' into the analysis of economics with his central idea that "the mode of production of material life conditions the social, political and intellectual life processes in general" (Thomas 1999: 24).

According to Roseberry (1989) the political economy school drew its inspiration from Wallerstein's world systems theory and dependency theory made popular by Frank's work in Latin America. These ideas challenged anthropologists to begin to understand, as Marx put it in 1851, that "men make their own history, but they do not make it just as they please; they do not make it under circumstances chosen by themselves, but under circumstances… given and transmitted from the past" (Bender 1988: 33 citing *The Eighteenth Brumaire of Louis Bonaparte* 227n – 28n). In conjunction with this emphasis on the importance of understanding history, a political economy approach also challenges anthropologists to understand how global, regional and governmental decisions affect individuals' everyday lives and how these decisions restructure their access to labor, capital and agency. Although this theory is riddled with complaints about its reductionist focus (see Vayda and Walters 1999 for an example) it is still a widely adhered to and utilized paradigm employed to understand the complex human condition.

The adoption of a political economy framework is not as cut and dried as the utilization of an evolutionary paradigm, as the testability and apparent application of the latter is much more defined than the former. That is not to say that this approach is without utility; rather, it operates as a tool by which to frame and understand data. It poses that there is a "universality of human connections" (Roseberry 2001:81) that is

structured by historic processes and one's access and relation to resources. Analyses within this paradigm range from understanding the impact of subsistence strategies upon individual autonomy,[14] negotiating problems surrounding class and access to labour and resources[15] and exploring how political and economic decisions structure everyday life. There is also a large body of literature that is directly concerned with how these processes impact health.[16]

Two sets of published material have profoundly impacted this dissertation. The first is a body of literature that comes from the physician-anthropologist, Paul Farmer. Working in Haiti, Farmer has long been interested in the connections between infectious disease[17] and the political and economic climate within which people reside. Much like Ewald, he is a strong proponent of the idea that disease virulence and transmission must be viewed as a byproduct of social conditions. However, where Ewald sees a crowded ambulance, Farmer would inquire as to the social conditions that led to the population aggregation within the politically funded emergency vehicle. In order to expose the relationship between disease transmission and social circumstance Farmer might pose questions like: "… by what mechanisms and processes might social factors be transformed into personal risk? How do forces as desperate as sexism, poverty, and political violence become embodied as individual pathology?" (Farmer 2000: 413). Throughout his work, Farmer also challenges

[14] See for example Saitta's (2001) archaeological discussion of labor appropriation and biological consequences or Goodman's (2001) analysis of the health consequences associated with agricultural intensification and increased social hierarchy.
[15] Analyses in this area are plentiful, and to some extent most articles utilizing a political economy approach will touch upon this issue. Some examples include Baer et al. (2003), Rothstein (1999), Scheper-Hughes and Lock (2006), and Singer (2010).
[16] Again, examples are too numerous to list but to get an idea of the range of studies see Armelagos et al. (2005), Baer et al. (2003), Goodman and Leatherman (2001), Richmond et al. (2005), Salehi and Ali (2006), and Farmer (1992,1996,1999,2003).
[17] usually tuberculosis and HIV/AIDS

readers to understand how both microsociological and macrosociological levels of human connectedness affect an individual's health by shifting scales of analyses to looking at factors ranging from community interaction to political policy creation.

The second source is Leatherman and Thomas' (2009) paper aimed at understanding the impact of civil conflict within Peru. Although, arguably in no way comparable to the conditions in Toronto during the First World War, their analysis provides numerous ways in which to consider the impact of conflict. Most notably they use the concept of structural violence, as outlined by Farmer, which defines structural violence as "violence of poverty, social and political marginalization, racism, sexism, and other forms of structured inequalities and their effects on people's lives, health, and agency" (Leatherman and Thomas 2009: 197). They also provide a useful framework to assess the costs of conflict, which include increased morbidity and mortality, breakdowns in infrastructure, social and psychological implications, impact on public health systems and environments and food security.

This dissertation tackles the ideas encompassed within a political-economic approach in the following ways. First, it explores how political decisions impacted the city during the epidemic; examples of this include forced quarantines, prohibitions on social gatherings, and reporting regulations. Second, it explores the creation of public health programs and allocation of funds to public institutions (hospitals, public health workers, temporary facilities) and how these decisions impacted the severity of the epidemic experience. Third, it assess the cost of war on the city's inhabitants to frame how the pressures created through involvement in the First World War transformed

personal risk. [18] And lastly, it provides a cursory analysis of the question 'did the disease affect people from differing socioeconomic backgrounds differently?'

Syndemics

First introduced in 1994 by Merrill Singer in an anthropology text co-authored with Hans Baer and Ida Susser, syndemics calls for "big picture dialectical thinking in health" (Baer et al. 2003: 15). Frustrated by the limitations of existing epistemological frameworks, Singer called for a holistic analysis that took into account "the full array of the health and social problems suffered by an individual or community" (Singer 2009: xiv). The approach encompasses a number of stimulating yet challenging agendas. The first is to view infectious diseases as integrated entities that co-exist in a host and in a given environment.[19] Much like the ecological idea of synergy, this technique challenges scholars to explore and understand the interactions of disease causing entities. Second, the syndemic approach challenges proponents to view these co-infections as a consequence of health threatening conditions. For example, poverty can result in malnutrition, which has been shown to compromise the immune system (Baer et al. 2003: 17). Third, Singer stresses the importance of understanding social and environmental factors such as treatment options, social responses, climate, or economic factors. Finally, much like a political economic approach, Singer stresses the importance of incorporating history and inequality into one's analyses.

Although the theory is relatively new, numerous scholars have stepped up to the challenge of this multidimensional agenda. Van Lettow et al. (2003) have utilized

[18] Singer (2009: 140 - 154) also provides a wonderful resource to understanding the consequences of structural violence.
[19] For an excellent discussion of this topic, see Singer (2009: 54-57).

it to explore the association between HIV/AIDS and malnutrition throughout areas in Africa. Freudenberg et al. (2006) look historically at the connection between poverty, tuberculosis, HIV and homicide in New York. Herring and Sattenspiel (2007) examine the interaction of tuberculosis and influenza in their work on the pandemic experience of Aboriginal populations in Manitoba, Canada. Nichter (2008) has adopted the framework to improve global public health initiatives[20] of the Centers for Disease Control and Littleton and Park (2009) link diabetes and tuberculosis together, further demonstrating the utility of this paradigm.

Within this dissertation syndemics is viewed as the natural progression of political economy theory, and much like political economy, a syndemics lens was adopted to serve as a descriptive tool designed to probe the available historical material in order to generate a richer historical account of the epidemic and the events leading up to it. The paradigm also helped shape what types of questions were explored. This theory is most apparent in the following explorations. As syndemics analyses are interested in understanding the impact of pathogen interaction, special attention was paid to the relationship between influenza and pneumonia. The idea that pathogens impact one another's progression also played a role in trying to establish the health profile of the city's inhabitants prior to the pandemic. In order to begin to understand the impact of the total environments, the timing of the epidemic was also explored in hopes of understanding how seasonality, movement of individuals and environmental conditions[21] impacted the severity of disease. An attempt was also

[20] See also Syndemics Prevention Network (2005).
[21] For example, it was believed that Toronto's unseasonable weather worsened the disease experience.

made to establish the overall health of the population just prior to the epidemic to understand how historical context impacts disease severity.

Chapter 3 Historical Context

The aim of this chapter is to historically contextualize the two main elements of this dissertation: the city of Toronto and the influenza virus. This section begins by providing a brief history of the development of one of the largest and most influential cities on the Canadian landscape. At the time the epidemic hit, the city was undergoing significant economic and demographic change; this discussion is intended to document this shift as well as capture the public health infrastructure that existed in 1918. An in depth description of the biological characteristics of the influenza virus is then provided. The information presented is designed to expose the reader to the high mutation rate, and therefore unstable nature, of the influenza viruses. A more in depth description of the 1918-1919 influenza pandemic follows. The chapter concludes with an introduction to the work that has been carried out in Canada thus far, going into further detail on information provided by scholars who have examined the epidemic experience in Toronto.

Toronto the Good[22]

Although it currently is the most important city in Ontario, acting as the province's capital, Toronto had a very modest start. Used predominantly as a way station for Aboriginal and early French and English settlers, Toronto's early importance was negligible. Initial settlement within Ontario was confined to the fort towns of Niagara and Kingston (Glazenbrook 1971). Around 1720 a small trade post

[22] This was a nickname provided by Toronto's 25th mayor, William Holmes Howland, intended to capture the cities identity as a bastion for 19th century Victorian morality (Davidson 2007)

was established by the French-owned North West Company, but since very little emphasis was placed on the location its operations ceased in 1730 (Glazenbrook 1971). In 1750 renewed trade in the area facilitated the construction of a larger fort, Fort Rouillé, that occupied an area of some 180 ft x 180 ft and included a dwelling for the commandant as well as a separate lodging for the garrison, a store, bakery, and a blacksmith's shop. By 1754, Fort Rouillé[23] housed nine permanent residents as well as a few boatmen and laborers who lived on or near the fort. Until 1770, the location operated as a small scale fur trading site. In 1783, its importance on the landscape shifted as increased competition between the English-owned Hudson's Bay Company and the North West Company prompted a push to identify and own long-distance trade routes. This period was also defined by the American Revolution which had the effect of redefining perceptions of threats from the south as well as altering immigration patterns. These factors stimulated the purchase of land by the Crown from Aboriginal groups in the area. It also necessitated the creation of naval bases to protect against enemy attack. In 1787, Toronto was officially purchased from the Mississauga Indians by Governor General Lord Dorchester to serve as the capital of Upper Canada (Rayburn 1994). This choice was a contentious one, as the area had very little in the way of supplies. Dorchester felt that nearby lands would be suitable for sustained agricultural growth and, due to its less accessible port, the new capital would be less vulnerable to attack (Glazebrook 1971).

From 1793 to the 1840s, Toronto functioned as a colonial capital and early center for trade, transportation and agriculture. By the mid 1840s, it had developed its

[23] The site was officially named after the Minister of Marine, but it was more commonly referred to as Fort Toronto.

own industrial base serving as a center for regional trade and finance, and by the 1880s, it had firmly distinguished itself as a booming manufacturing center (Gad and Holdsworth 1984). In the early 1830s, the city's industries were limited to breweries, saw and grist mills, a few bakeries and a brickyard (Piva 1979), but by 1850 one-quarter of the city's inhabitants were employed within manufacturing (McNab 1955). Early census data (1851) indicates that 97% of the city's inhabitants claimed origins in the United Kingdom. Toronto was also marked by a heavy Protestant influence, with 73% of the population self-identifying as belonging to this religious affiliation. At the close of the century, growth within the city was thwarted by a widespread depression, which would alter migration patterns and industrial growth within Toronto in the beginning of the 20th century.

The first decades of the new century brought tremendous change within the city. Immigrants, who had previously predominantly travelled to the United States, began to pour north. Settlement provided a rapidly expanding domestic market, which in turn stimulated further industrial expansion in the east. At the center of Canada's newly invigorated industrial economy stood Toronto (Piva 1979). Capital in manufacturing increased by 618.4 per cent between 1900 and 1921. The gross value of production rose to 147.9 in 1905, 255.6 in 1910 and by 1919 stood at 847.6 (Piva 1979). Although the city's economy was booming, not all of its residents shared in the new wealth, and the inequitable distribution of resources began to be witnessed throughout the city. Between 1911 and 1921 two important changes helped to define

Toronto's workforce, an increase in unemployment and the rise of white-collar work[24], further deepening the chasm between the haves and the have-nots.

This massive influx of immigrants not only contributed to the intensification of classes within the city but also altered its ethnic, religious, and spatial characteristics. By 1911, there were over 1.5 million immigrants living in Canada. Over the next three years more than a million more flocked to the country.[25] Unlike earlier immigration waves that were predominantly comprised of immigrants of British origin, between 1904 and 1914 an estimated 28% came from continental Europe, 41% came from Britain and 31% hailed from the United States (Reynolds 1935: 299).[26] Of those aliens who migrated to Toronto, three-quarters had previously resided in the United Kingdom, causing the city composition to remain predominantly (93%) individuals claiming British origin (Piva 1979). One of the largest demographic changes within the city during this period of population growth was the influx of Jewish immigrants. Waves of Jews to Canada resulted from pogroms throughout Europe between 1899 and 1908. The first influx of European Jews occurred in 1899 when 2000 Romanian Jews immigrated to Toronto and Montreal. A second wave of eastern European Jews arrived in the wake of the Kishinev pogroms of 1903 and 1907 – 1908 witnessed the arrival of over 10, 000 Jews escaping pogroms throughout Russia and Poland (Piva 1979). According to the 1901 census, 1.4% of the population

[24] For a complete discussion see Piva (1979: 15-17).
[25] 354,237 in 1911 – 1912, 402,432 in 1912 – 1913 and 384,878 in 1913 – 1914 (The Canada Year Book 1916 – 1917).
[26] These immigrants did not distribute evenly throughout Canada. Most new immigrants from the United States settled in the West whereas the majority of British-born individuals migrated to the east. Immigrants from continental Europe took up jobs working in lumber, mining and railway camps and were likely to reside in the interior of Canada.

of Toronto self-reported as Jewish; by 1921 that number rose to 8.7% of the city's inhabitants.

The beginning of the 20th century also witnessed a rapid expansion into suburban areas. Influenced by the Chadwick Report of 1842, which lamented the immorality and overcrowding of the new industrial centers, British reformers proposed a move to utopian rural settlements. Heralded by the writings of Ebenezer Howard and his Garden City concept, the plan called for city dwellers to ease population aggregation by migrating to nearby suburban communities. This idea was quickly adopted by Toronto's Housing Commission which, in 1912, established the *Cities and Suburbs Plans Act* which insured that single family homes[27] were laid out along definite roads and diagonal streets (Solomon 2007).

The city's mushrooming population also sparked massive alterations to Toronto's public health department. In the last decades of the nineteenth century the city's 'health department' was comprised of a health committee made up of aldermen, private citizens, and, at times, a physician. Concerned predominantly with the elimination of unsanitary conditions within homes, sewers, and streets, this body operated essentially as a form of sanitary police. In 1878 a municipally funded Department of Public Health was created and in 1883 the first Medical Health Officer (MHO)[28] was appointed (Bator 1979: 85). Early work within this organization was concerned with miasmatic theories on the spread of infectious disease but the appointment of the first MHO marked a shift toward the new science of preventive medicine. This shift however, did not make preventive health measures any easier to

[27] As opposed to apartment buildings or rooming-houses
[28] The Ontario Public Health Act of 1912 renamed this position Medical Officer of Health.

enforce. The city council still wielded extraordinary power over the MHO. Council members, by-in-large, refused to abandon earlier notions of the spread of disease, which created antagonism between the MHO and the city council, resulting in insufficient funding for the newly formed department (Solomon 2007). The small and underfunded department could not keep pace with the rapid rate of industrial expansion and population growth. As a result, in the early 20th century, the department's actions were predominantly responsive, reacting to the proximate, rather than ultimate contributors to ill health.

Figure 3.1 Rally of the Anti-Vaccination League of Canada 13 November 1918. Source: Toronto Archives Fonds 1244 item 2517. Photo by William James.

One of the first concerns of the newly formed Health Department was the disposal of animal and human excrement. The original plan diverted unfiltered human waste into Lake Ontario, leading to periodic outbreaks of typhoid fever (Bator 1979). The relationship between infected human waste and disease was still a contentious one, which impeded the construction of a costly treatment plant. The city's strategy was to put the decision to carry out public works projects in the hands of voters, who were reluctant to approve costly expenditures on sanitary installations. It was not until the early months of 1910 that the construction of a water filtration system began. The limited authority of the Health Department can also be witnessed in the city's compulsory vaccination program. In 1894, the Toronto Board of Education made immunization against smallpox a prerequisite for entrance into school, but in 1906 the Anti-Vaccination League (Figure 3.1), armed with a petition signed by thousands, was successful in overturning the regulation (Bator 1979).

In order to undertake the degree of reform required to adequately safeguard the city, an individual possessing "good sound judgement, an unusual amount of common sense and excellent executive ability" (MacDougall 1990: 27) was required. In 1910, a fifty-two year old obstetrician, Charles Hastings, answered the call and was appointed to the position of MHO. A long time social activist and supporter of community health, Hastings was just what the city needed. His position was further strengthened in 1912 when the provincial Public Health Act bestowed upon him the position of chief executive officer of the local board of health and gave him the opportunity to act as the administrative head of his department, freeing him from the shackles of politicians and public opinion (MacDougall 1990).

Operating more like a politician than a public health officer, Hastings vowed to dismantle slums, safeguard the city's milk supply, exterminate the house fly, create guidelines and impose mandatory medical examination of school children, and establish an efficient diagnostic laboratory (Bator 1979). He also lobbied to increase funding for public health,[29] successfully tripling the annual appropriation by 1914. It did not take long for Hastings to put his reforms into action. In May 1911, he created the *Health Bulletin*, a monthly publication which disseminated pertinent public health information to businesses, professionals, and religious and educational institutions (Piva 1979). The relationship that Hastings nurtured with the community would serve him well during times of crisis. Between 1913 and 1918, Toronto demolished 1,600 homes that failed to meet Hastings' standards[30] (Solomon 2007) and instituted a program whereby municipal funds could be used to construct indoor plumbing in the homes of those who could not afford it. In 1914, Hastings called for the chlorination of Toronto's drinking water, which contributed to a decline of typhoid deaths from 40 per 100,000 in 1910 to 2 per 100,000 in 1914 (Piva 1979). In this same year he relied on scientific evidence to help convince the city council to require the compulsory pasteurization of milk (MacDougall 1990).

Hastings also had a significant impact on the structure of the agency. Between 1911 and 1915 he re-envisioned the city's communicable disease response. Starting with the establishment of a Division of Communicable Disease Control and Quarantine, Hastings' new plan emphasized home health care. A chief inspector, who

[29] A goal that was greatly assisted by the poor health of men enlisting to participate in the First World War.

[30] In some ways this created more problems than it solved as only 1 per cent of houses were rebuilt during this time. Multi-family residences, housing the working poor, were also disproportionately toppled, further widening the chasm between the rich and poor (Piva 1979; Solomon 2007).

supervised ten staff inspectors, operated as facilitator for in-home hospice and provisioning of the basic necessities to those who could not afford it (MacDougall 1990). In 1913, Hastings convinced the city council to approve the allocation of $1 a day to working women so that they could stay home and care for their children. In 1914, he created the Division of Public Health Nurses, who also served to reinforce this mandate (MacDougall 2007). He also used this body of women as educators, travelling from home to home, and instructing residents on the virtues of disease prevention (Bator 1979). In addition, as Minister of Health, he worked to expand the Division of Laboratories, which grew from one bacteriologist in 1910 to twelve by 1915 (MacDougall 1990).

Understanding the historical context of the city of Toronto prior to the epidemic is important for a number of reasons. At a theoretical level all of the frameworks in this thesis rely heavily on the effect of history, for as Marx reminds us, individuals do not make history in as much as they are a by-product of the historical circumstance which preceded them. On a more practical level, understanding the previous actions of Hastings as well as the shifting composition of the city helps us to contextualize the pandemic experience as not just an isolated moment in time but as a situated event embedded within a city's history.

Influenza Epidemiology

Within the context of a western biomedical paradigm an epidemic cannot be fully understood without a thorough analysis of the causative agent. What follows is a discussion of what electron microscopy and virus filtration have been able to tell

researchers about the nature of the pathogen responsible for the 1918-19 influenza pandemic.

The influenza virus genome consists of eight separate pieces of ribonucleic acid (RNA). Due to the segmented nature of these RNA strands, differing flu viruses can very easily exchange genes to produce hybrid progeny viruses with sequences of RNA from each parent virus (Laver et al. 2000). There are two glycoprotein molecules that define the virus: hemagglutinin (HA) and neuraminidase (NA). Hemagglutinin is a triangular, rod-shaped protein whose function is to attach the virus onto its host's cell. The virus fuses to cells that contain sialic acid, which allows the RNA of the virus to produce thousands of new virus particles. The second component is neuraminidase, a mushroom-shaped enzyme whose job is to remove all the sialic acid from the newly infected cell. This enables the virus to escape the primary infected cell and spread throughout the rest of the body in order to contaminate subsequent cells with which it comes into contact (Laver et al. 2000).

The influenza virus is classified into three types: A, B and C. Type C is relatively uncommon. Type B has given rise to epidemics but it only infects humans, and outbreaks have been predominantly limited to children (Beveridge 1977). Type A is perhaps the most interesting. Common and adaptable, it was responsible for the 1918-1919 pandemic. Its lethality rests in the fact that it not only infects humans, but also pigs, horses, seals, whales and many kinds of birds (Laver et al. 2000). This animal host diversity gives rise to novel strains through the process of pathogen recombination.

There are two main ways by which influenza recombines—antigenic drift and antigenic shift. Antigenic drift occurs when new strains evolve as a byproduct of point mutations on the surface of the pathogen. These are not novel strains but they are new enough to evade the immune system and cause infection. Antigenic shift on the other hand is the emergence of an entirely new virus (containing a novel HA or NA). This is only seen to occur with influenza A and is the byproduct of coinfection with human and non-human viruses. The 8 separate genes of influenza A allow for over 250 combinations during coinfection and reassortment of human and non-human viruses (Lu et al. 2007).

It is likely that influenza A will always be endemic in various ecosystems, as the virus persists in wild populations of water birds. For example, ducks found in northern Canada appear to carry most kinds of Influenza A, acting as a natural reservoir for the pathogen. Although infected with the virus, which resides in their intestinal tracts, no harm is done to the ducks (Laver et al., 2000). From this reservoir, the virus can be transmitted to other mammals through intestinal excretions. Although the duck ensures the propagation of the virus, the pig guarantees its continued virulence. In the midst of the 1918-19 influenza pandemic, Robert Shope, an American veterinarian, recognized an interesting phenomenon among Iowan pigs at the Cedar Rapids Swine Show. Shope noted that a disease similar to human influenza appeared to be making its way through swine populations (Zimmer and Burke 2009).

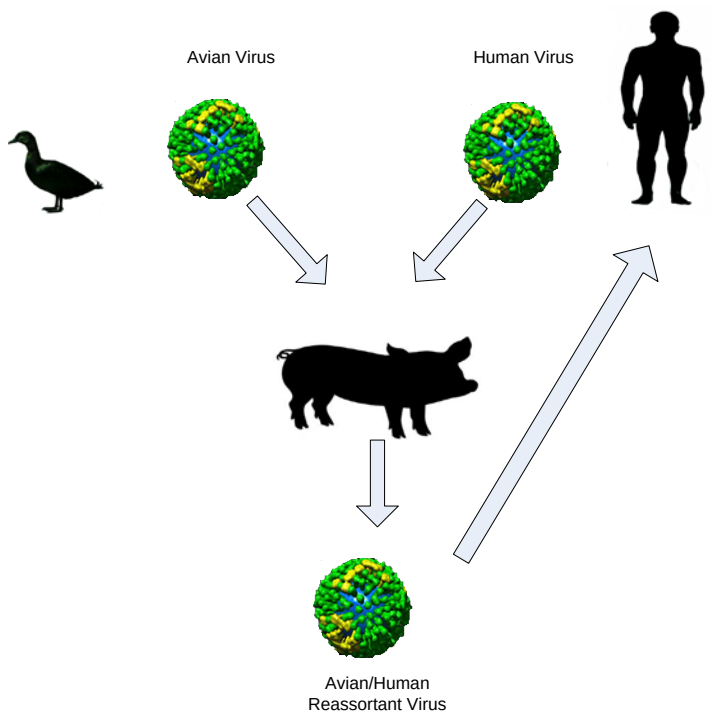

Figure 3.2 Influenza A Genetic Reassortment Schematic

After 13 years of study, Shope was successful in transmitting the infectious agent of swine influenza into healthy animals. His further work demonstrated that, in mice, inoculation with human adult serum taken from pandemic survivors could neutralize the swine flu virus (Zimmer and Burke 2009). It is now understood that the pig can act as a mixing vessel for the virus. Pigs can become infected with avian influenza virus after the consumption of bird feces; simultaneously they can catch the human virus from a farmer's cough or sneeze. If the pig becomes infected with both viruses at the same time, the genetic material can mix, which has the potential to create a new strain that may find its way back to the respiratory tract of a farmer and thus spread a novel influenza to the human population (Figure 3.2) (Scholtissek 1992). It

had long been believed that influenza required an intermediary (such as the pig) to cross the species barrier from birds to humans as, by-and-large, influenza viruses from one species do not replicate well in another (Baigent and McCauley 2003). Although this holds true for most cross-species infection, avian influenza viruses have been documented to be transmitted directly to human hosts without any recombination or apparent mutation (Baigent and McCauley 2003).

Influenza is transmitted person-to-person when an uninfected person inhales the aerosolized droplets containing the viruses that have been expelled via a cough, sneeze or any other method that could effectively launch the virus out of the infected person's respiratory tract (Herring 1994). Once the virus particles enter the host they attach to red blood cells, aided by hemagglutinin, and take over the host cells to facilitate in reproduction, utilizing neuraminidase. This process takes about six hours to commence. Once entrenched within the host, the virus replicates, causing the host to enter into an asymptomatic (carrier or latent state) or symptomatic phase. During this time, the infected individual sheds virus for a period of one to ten days (Ohadike 1991). In the end the virus will either be defeated by the immune response, it will kill the host or it will weaken the host leaving it susceptible to attack from another pathogen (i.e., pneumonia). If the virus is defeated by the immune response, memory B cells (and antibodies) will be created against the surface proteins HA which will help the immune system evade a subsequent infection by the same strain of flu.

Symptoms of influenza most commonly include headaches, fever, hacking cough, chills, malaise, leg and kidney pain, muscular pain, vomiting, dizziness, profuse sweating, sneezing, and labored breathing (Herring 1994; Herring 2000;

Ohadike 1991; Laver et al., 2000). Without complications an average bout of influenza lasts about ten days after the onset of illness. However in some instances persistent weakness or mental depression may necessitate longer convalescence. Highest rates of hospitalization usually occur in very young and very old individuals. Although influenza itself can lead to complications, most influenza mortality is associated with sequelae. These sequelae fall into two major types: cardio-pulmonary conditions, which include other respiratory diseases, and neurologic conditions (i.e. schizophrenia). Pneumonic complications are widely described in association with the 1918-19 influenza pandemic (Johnson 2006).

1918 – 1919

The influenza pandemic of 1918–1919 was possibly the greatest infectious disease-related catastrophe that the world has ever seen. The global death toll lies somewhere between twenty and possibly 100 million people (Crosby 1989; Phillips and Killingray 2003). It is estimated that over a billion people, equivalent to half the entire human population at the time, were infected with the virus during this twelve month period (Collier 1974). At the height of the pandemic, morbidity rates hovered around 50% (Reid et al 2001), with an average mortality rate of 2.5%. Within some isolated regions mortality rates were reported as high as 70% (Taubenberger 2003).

There are many different hypotheses regarding the origins of the virus that caused this massive outbreak. The most widely accepted view is that the influenza A strain associated with the 1918 – 1919 pandemic originated somewhere in the United States. Many scholars see Fort Riley, Kansas (also known as Camp Funston) as the starting point for this outbreak (see Crosby 1989). There are reports from 4 March

1918 that the hospital at Camp Funston was swamped with soldiers complaining of flu-like symptoms (Herring 2000). This predates the first wave of illness that swept across the world. Jeffery Taubenberger's team at the US Armed Forces Institute of Pathology sequenced tissue samples from three exhumed bodies, two soldiers and an Inuit woman. The entire genome was sequenced for both the hemagglutinin (HA) and neuraminidase (NA) components of the 1918 strain. Their analysis showed some very interesting results: the 1918 HA shares characteristics with both the human clade and swine clade, suggesting that it derived from a combination of the two. On the other hand, the NA gene is most closely related to avian isolates. They report further that the 1918 sequences were distinguishable from avian clade viruses, as a number of mammalian isolates were present (Reid et al 2001)[31]. It is believed the new influenza strain traveled across the Atlantic with American troops on the *Leviathan* and arrived in France on 7 October 1918 to start the most virulent of three epidemic waves that would rock the world (Crosby 1989).

According to Herring and Padiak (2008), the idea of a European epicenter was first postulated by Jordan (1927) and MacFarlane Burnet (1942). More recently Oxford et al. (2001, 2002) argued that the virus responsible for the pandemic was circulating around Europe as early as 1915. They pointed to outbreaks of respiratory disease in France and the UK during the years 1915 – 1917 and observed that in 1916 – 1917 there were outbreaks at army camps at Etaples (France) and Aldershot (United Kingdom) that caused very high mortality among 25 – 35 year olds (Oxford et al. 2002). They suggested that this new interpretation of the delayed dispersal of Spanish

[31] This is an oversimplified summary of their findings, for a more detailed discussion see Reid et al. (2000, 2001), Taubenberger et al. (2000), Worobey (2002), and Phillips and Killingray (2003).

Flu is indicative of the characteristics displayed by pandemic influenza. They arrived at this conclusion by looking at the behavior displayed by the 1957 and 1969 pandemics. The H_2N_2 pandemic of 1957 was isolated in China in February yet did not spread to the rest of the world until July through September, indicating that a period of 10 months was required for global spread. The influenza A (H_3N_2) pandemic of 1968 spread throughout China in July yet did not reach the UK and Europe for 12 – 14 months (Oxford et al. 2001; Oxford et al. 2002). Oxford et al. claim that the two year period between the appearance and subsequent dispersal of the virulent virus was a by-product of the absence of air travel in conjunction with distortion and restriction of other forms of travel during the Great War (Oxford et al. 2001).

Although Oxford et al.'s work is provocative; there are a number of issues with their interpretation of the pandemic. In the 2001 paper they cite Spain as the 'accepted' country of origin for modern day understandings of influenza dissemination. However, in general little credence is given to the idea that Spain was the country of origin for the pandemic. Again, the widely accepted hypothesis is that the virus originated in North America or China, with the former being the most likely candidate. It is believed that the misconception of a Spanish flu was a by-product of Spain's involvement, or rather neutrality, throughout the Great War. It has been noted that countries involved in warfare censor information about epidemics occurring on the front as well as at home. Spain's lack of participation in WWI provided a climate for accurate reporting and the subsequent placement of proactive measures, such as quarantine and dissuasion against public gatherings (Echeverri 2003). This point is noteworthy because flu was not reported in Spain until the 29th of June 1918

(Echeverri 2003), some four months after it was recorded in Kansas. In addition, Oxford et al.'s (2001; 2002) claim that travel was restricted as a result of the war is a little presentist. Although air travel was not used, boats, automobiles and trains were heavily utilized. Many authors attribute the conditions created by these forms of military travel as aiding in the dissemination of the virus rather than acting as a hindrance (see Ewald 1994 and Crosby 1989 for examples). Recently, Herring and Padiak (2008) have re-examined the British War Diaries to test Oxford et al.'s hypothesis and found that prior to 1918, influenza was a relatively minor illness. In conjunction with the severity with which the troops were infected, this suggests that the autumn 1918 outbreak was a new rather than pre-seeded disease among the British Expeditionary Forces, further weakening the Oxford hypothesis.

A third hypothesis for the origin of this new strain of influenza is presented in Humphries (2008), who fully and convincingly details a new journey for the virus--one that begins in China, spans Canada, then is conveyed to France and England. According to Humphries, in 1916 the British Army found itself lacking in manpower. Adopting a strategy already employed by the French, the decision was made to recruit Chinese laborers to work behind the lines, thereby freeing up soldiers to return to battle. Between 1916 and 1918, 94,000 Chinese workers were shipped by the British from China. Initially the transport took the men from Weihaiwei to Europe via Singapore, Durban, and Cape Town, or through the Mediterranean via the Suez Canal (Humphries 2008), but a lack of ships and an overwhelming Chinese response forced officials to seek an alternate route of travel. In February 1917, it was suggested that Chinese laborers be shipped from China to Vancouver where they would board the

Canadian Pacific Railroad to Halifax and then be transported on to England and France.

Borden, the Prime Minister of Canada, agreed to the transport of the men in an effort to further the war effort, but fears of foreigners not allied with Britain, represented in this case by fears of Chinese escaping into Canada and an inadequate public health system, made containment of disease a profound problem. The first transport of Chinese laborers into Canada left Weihaiwei on the *Empress of Russia* on 19 March 1917 and arrived at the William Head Quarantine station in British Columbia on 2 April 1917 (Humphries 2008). The site, which had been established at the end of the nineteenth century to deal with plague and other diseases, was in no way equipped to handle this huge influx of Chinese laborers. As such, thousands of Chinese laborers crammed into tents and poorly constructed shelters that lacked basic sanitation or provisions. This environment created ideal breeding grounds for many pathogens. This can be witnessed by a report from the superintendent at William Head from the end of 1917 in which he stated, "almost all of the Coolie transports were infected 'more or less with mumps'" (Humphries 2008).

In the early months of 1918 a disease identified as pneumonic plague began to circulate throughout China, as well as at a Chinese Labour Corps just outside of Étaples. The presence of disease did not prevent the transport of workers. On 1 March 1918, despite the presence of plague in the port of Weihaiwei, the *Conconada* picked up 1,899 Chinese laborers and set sail for Vancouver. When it arrived on 23 March, two men had succumbed to the disease at sea. By 2 April, when the men were to journey on to Halifax, between two and three hundred men were unable make the

trip due to illness (Humphries 2008). At this time, Humphries also notes an increase in the number of hospitalizations and deaths due to pneumonia among the civilian population in British Columbia. Between January and June 1918, he reports that the illness was responsible for 13.5 percent of all deaths, an increase from 10.24 percent in the preceding five years (Humphries 2008). Humphries contends that what had been identified as pneumonic plague was indeed a new virulent strain of influenza, nurtured by crowded transport conditions, a lack of public health infrastructure, and a desire to sustain the war effort at any cost.

Regardless of its origins, there is agreement that the influenza pandemic of 1918-1919 was characterized by three successive waves, although it was common for communities to experience fewer than three disease episodes. The first wave hit in the spring of 1918. Although it boasted the strain's high morbidity rates, which ranged from 20 – 50% (Reid et al 2001), it produced nothing terribly out of the ordinary in terms of its death toll (less than 0.1%) (Taubenberger 2000). Its only odd characteristic was that rather than attacking the very young and the very old, as influenza is known to do, it instead was striking down individuals in their prime (between the ages of twenty to forty) (Osborn 1977). Still, due to the all-consuming nature of war, it was able to sweep by barely noticed. This wave waned by July and the bulk of the deaths were "blamed largely on malnutrition and the general weakness of nerve power known as war-weariness" (Crosby 1989).

The second wave occurred in the autumn of 1918 when the virus is believed to have mutated. There is some debate over the succession of these waves with some scholars believing that this second strain involved a novel recombination of the

influenza virus and not a mutated continuation of the virus associated with the first wave (Reid et al. 2001; Crosby 1989). Students of the pandemic are quick to note however that in places where both the spring and fall wave hit individuals who fell ill during the first wave escaped or experienced mild illness when the fall wave hit (Reid et al 2001), indicating the re-emergence of a remarkably similar virus. This second wave was characterized by unprecedented mortality. It was the most lethal of the three waves, killing ninety people in Toronto in one day alone. Another incredible aspect of the second wave was that it arrived at the ports of Freetown, Sierra Leone; Brest, France; and Boston, Massachusetts within the span of seven days (Crosby 1989), causing chaos worldwide and proving that this strain was not only extremely lethal but also highly communicable.

The third wave came in the winter of 1919. It was much less severe due to the fact that much of the world's population had already been exposed to the virulent virus or a related strain. It was, however, evident that this epidemic was tied to the two that came before it because the age at death distribution was similar to the previous two outbreaks (Johnson and Mueller 2002). Although the epidemic is believed to have waned by 1919, it is widely believed that pandemic activity continued well into 1920.

It is interesting to note that while all three waves showed uncharacteristic mortality within the 20 – 40 age group[32], Luk et al (2001) show that there was also negative excess mortality[33] displayed among elderly individuals. They conclude that

[32] Although the most commonly expected definition of this age group is 20 – 40 (i.e. Phillips and Killingray 2003; Luk et al 2001; Crosby 1989) some scholars define the group displaying the highest mortality as individuals 15 – 45 (Reid et al 2001; Taubenberger 2003), 25 – 39 (McCracken and Curson 2003) or those 20 to under 65.

[33] This means that there were fewer deaths in this age category than is usually experienced during a typical experience with this virus.

this negative excess mortality was likely the result of previous exposure to an influenza virus that was similar to the 1918 strain, possibly a pandemic of influenza that began in late spring of 1889 and subsided in early 1892 (Reid et al 2001).

The Epidemic in Canada

The summer wave is thought to have been brought to Canada aboard the *Araguayan* and *Somali*, which, starting 26 June 1918, transported infected soldiers from England. On 11 July, crew members were reported ill at Grosse Isle, a quarantine station in the St. Lawrence River. On this same date, the *Nagoya* arrived in Montreal and the *Med 1099* docked in Halifax. Both were assumed to be unwitting transporters of this new strain of influenza (McGinnis 1981). By all accounts, the summer wave of influenza went unnoticed throughout all of Canada (Humphries 2008).[34] Early accounts by Canadian scholars (see for example McGinnis 1981 and Pettigrew 1983) claim that the end of the war was the primary factor for the intensification of the influenza pandemic in Canada and that the return of infected soldiers from the front was the portal of entry for disease throughout the country. Humphries (2005) challenged this notion, stating that the majority of Canadian soldiers did not return from the trenches until late spring of 1919. While he still recognized the war as the major contributing factors for the dissemination of influenza into Canada, he postulated that it was an intensification of the war effort, not its decline that was responsible for the movement of the pathogen (Humphries 2005). He

[34] Humphries (2005) suggests this was due to previous Canadian exposure to the virus in 1917 via contact with infected Chinese labourers. He also believes that the *Araguayan*, *Somali* and *Med 1099* arrived empty, with the intention of transporting troops to the front and therefore were not responsible for the sparking a summer wave in Canada.

suggested that the transport of American soldiers through Canada to European destinations was responsible for the virus entering Canada.

In any event, according to Pettigrew (1983), the first major civilian outbreak began in Canada on 8 September 1918 when two students attending Victoriaville College in Quebec came down with a new virulent strain of influenza. By 23 September, 200 college students were ill and the disease was also firmly entrenched at the Polish Camp located at Niagara (McGinnis 1981). Epidemics raging in New York and Massachusetts ensured further transmission from the south as Canada was unable to impose quarantine against the United States.[35] The disease moved quickly across the country, spreading East to the Maritimes by the end of September. By October it was making its way west along railway lines and highways (McGinnis 1981). From the middle of September to the middle of December 1918, 19% of troops in Canada were reported to be infected (Zhang et al. 2009). At a national level, approximately 50,000 Canadians lost their lives in the winter of 1918 -19 (Humphries 2005; Jones 2005; Johnson and Mueller 2002).

The impact of the influenza epidemic in various parts of Canada has been addressed by a number of researchers. At the national level the epidemic has been described by Pettigrew (1983) and McGinnis (1981). In addition, Humphries (2005, 2008) has looked at the Canadian military's involvement in transmitting the virus across the Canadian landscape. Palmer et al. (2007) are the first to explore the impact of the epidemic in Newfoundland, and focus predominantly on the role of transportation and the differential mortality experienced on the island. Quiney (2002)

[35] The United States had objections over the expense of quarantine and even after Canadian officials agreed to incur half the costs in mid-January 1919, they were still given no say as to how American inspections were to be carried out (McGinnis 1981: 462).

has looked at the role of voluntary nursing staff in both the pandemic and the Great Explosion of 1917 in Halifax, Nova Scotia. Jenkins (2007) examined the public health movement's impact on influenza in New Brunswick. In Quebec, Fahrni (2004) has explored how the consequences of the epidemic were felt by women.[36] Using mostly newspaper accounts of the epidemic, MacDougall (2006) attempted to recreate the epidemic experience in Toronto, Ontario, and Herring (2006) has recently shifted her focus to Hamilton, Ontario. The disease in Canada's capital, Ottawa, has been detailed by Bacic (1999) and Belyk and Belyk (1988). In a series of papers, Herring and colleagues[37] provide an in-depth analysis of the pandemic's effect on several communities in central Manitoba. Through information collected from Anglican and Methodist parish records and Hudson's Bay Company records and with the aid of computer simulations, they have been able to significantly outline the experience of Aboriginal groups engaged in the fur trade. Further work has been carried out on Manitoba by Jones (2005, 2008), who examined how gender and class standing impacted the family. Jones also explored the relative success of public health initiatives in Manitoba. Lux (1997, 2001) examined the epidemic in Saskatchewan and Kelm (1999) has tried to reconstruct the experiences of Aboriginal Populations in British Columbia. Currently, the dynamics and impact of influenza in the Yukon Territory, North West Territories and Nunavut Territory have gone undocumented.

Very little is known at present about the influenza epidemic within the entire population of Toronto. Hallman's 2009 master's thesis examined infant mortality

[36] There is also a MA thesis by Francis Dubois (2007) that looks at flu in Quebec. Both texts are only available in French and therefore content had to be gleaned from abstracts.
[37] See Herring (1993); Herring (1994); Moffat (1992); Moffat and Herring (1999); Sattenspiel and Herring (1998); Sattenspiel et al. (2000); Herring and Sattenspiel (2003); Sattenspiel and Herring (2003); Waldram et al (2006); Herring and Sattenspiel (2007).

associated with the epidemic. Pettigrew (1983) and MacDougall (2006) have documented the pandemic experience in Toronto in some detail. Pettigrew presented a cursory overview of the impact of the epidemic on the Canadian landscape; in her manuscript the experience of the city is limited to ten pages of text. According to Pettigrew, the disease was considered to have entered the city officially on 3 October 1918, as marked by the quarantine of fifty soldiers[38] (Pettigrew 1983). By 9 October, there were believed to be over 1000 cases plaguing the city and by 21 October mortality had reached 502 (Pettigrew 1983). She further contends that by 31 October the influenza virus had almost fully retreated from the city. Her remaining work provides snippets about how the city responded and experienced the epidemic. MacDougall (2006) uses the 1918 influenza epidemic as a point of comparison with the 2003 outbreak of SARS in Toronto. She focuses predominantly on the role of the Toronto Health Department in order to gauge its success in communication, confinement and coordination during the pandemic. Overall, neither work has looked at the epidemic in Toronto in great detail. This dissertation picks up where these two scholars left off.

[38] This date has already been challenged by information collected from death registries.

Chapter 4 Methods and Materials

The aim of this chapter is to detail the methods that were employed during data collection as well as to provide readers with a critical examination of the material upon which this dissertation is based. The analysis begins by outlining the stages of this project. From there a discussion on the value of information derived from death registries is presented. Following this section is a description of the inclusion parameters used to define the data used in this project as well as a discussion of the difficulties of finding Spanish influenza in the archival record. Reconstruction of the pandemic in Toronto is largely based on newspaper accounts and as such this chapter delves into their history in order to help interpret the material presented.

A Day in the Life: Reconstructing the Spanish Flu in Toronto

The launch point for this project is death registries collected from the Archives of Ontario for Toronto for the years 1917 to 1920 (MS 935 Reels 227, 228, 229, 238, 239, 240, 251, 252, 261, 262, 263, 273, 274, and 275). These records are publicly available on microfilm, and for ease of transcription were digitized by Progressive Technology Federal Systems, Inc. Starting on 1 January 1918 and ending in 31 December 1919 the records were viewed to identify deaths attributable to the pandemic. All the relevant data were extracted and transcribed into an Excel spreadsheet. To minimize secondary transcription errors the data were entered by a single individual and transcription sessions were limited to no more than four hours.

For each individual identified all of the available information was recorded, which included the following: surname of the deceased, Christian name, sex, age, date of death, place of birth, place of death, place of burial, occupation, marital status, name of father, maiden name of mother, name of physician, name of informant, address, date of return, disease causing death, duration of that condition, immediate cause of death, duration of that condition, physician's address and remarks. The entries consist of two parts, an original entry and the physician's return. A byproduct of this format is that cause of death (when listed in the original entry), first and last name, date of death and date of return are recorded twice.

Table 4.1 Pneumonia or Pneumonia Derivatives Listed More Than 5 Times in Toronto Death Registries Examined from 1 September to 31 December 1918

Cause of Death (ICD and DCD)	Number of Cases Recorded
pneumonia	616
bronchopneumonia	294
lobar pneumonia	81
pleura pneumonia	15
double pneumonia	11
acute lobar pneumonia	7
pneumonia following influenza	7
double lobar pneumonia	6
tuberculosis bronchopneumonia	6

In order to create a comprehensive and unrestrictive database all deaths with pneumonia (n=616) or pneumonia derivative (n=482) or influenza (n=1192) or an influenza derivative (n=442) listed as either disease causing death (DCD) or immediate cause of death (ICD) were transcribed. Tables 4.1 and 4.2 and are provided to illustrate which conditions were listed in more than five instances. For a complete list of conditions captured in this analysis, see Appendix 1.

Table 4.2 Influenza or influenza Derivative Listed More Than 5 Times in Toronto Death Registries Examined from 1 September to 31 December 1918

Cause of Death ICD and DCD	Number of Cases Recorded
Influenza	1192
influenza and pneumonia	142
Spanish influenza	92
influenza and broncho pneumonia	40
epidemic influenza	15
influenzal pneumonia	14
influenza followed by pneumonia	9
influenzal broncho pneumonia	8
influenza with bronchopneumonia	6
Flu	5
probably influenza	5

Archival material relevant to the epidemic was also gathered. Monthly reports to the Department of Public Health were collected at the City of Toronto Archives. The years 1918-21 were searched for reference made to the pandemic. Seven of the 48 months that were examined contained information regarding the epidemic. The September 1918 report is filled with information regarding the epidemic experience in the city, as is the report for October of the same year. November of 1918 is an account of the 49th Annual Convention of the American Public Health Association. It deals more directly with the epidemic experience in major American cities. Brief mention is made in February 1919. The January 1920 and February 1920 reports provided a synopsis of the public health response to the influenza epidemic (January) and a statement regarding the influenza epidemic (February), while the report for March 1920 makes a brief reference to the continued excess mortality from influenza and pneumonia experienced within the city. The annual reports for the Provincial Board of Health were also downloaded for 1919 and 1920 from The Internet Archive

(http://www.archive.org).[39] The Library and Archives Canada was contacted in an effort to gather information pertaining to postcards that were distributed by postal workers during the height of the epidemic; sadly it appears that these cards have been destroyed.[40] Census data were collected from the Sixth Census of Canada (1921), Volumes 1-4 and the Fifth Census of Canada (1911), Volumes 1 and 2, available at McMaster University library. Information regarding the number of families, average earnings per male head of household, number of wage earners (listed by profession), population (for the city as a whole, by principal country of origin, and by religious affiliation), occupations for the population (divided by sex and age), literacy rates and languages spoken, were collected from both censuses (whenever possible).

In an attempt to gain further insights into the contribution of charitable organizations select groups were contacted. Of these The Rotary Club of Toronto responded and granted access to their monthly newsletter.

Newspapers were also examined to capture the day-to-day experiences of the inhabitants of Toronto. At the time of the epidemic there were two major publications in press *The Globe*[41] (established in 1836) and *The Toronto Star* (established in 1894). The *Globe and Mail* was accessed through the McMaster University library system. A keyword search was carried out for both publications from 1 September 1918 to 31 December 1919. For *The Globe* the words influenza, flu, and grippe were searched. All relevant articles were transcribed verbatim and saved as PDFs. For *The Star*, due

[39] The annual report was not available online for 1918.
[40] The cards were created by the Rotary Club (Rotary Committee on Federation for Community Service) and distribution began on Thursday October 24, 1918. Approximately 30,000 cards were put into circulation. They may never have been stored. The Archives has no record of them ever being kept (or created for that matter) nor does the Toronto Rotary Club.
[41] Became *The Globe and Mail* in 1936

to time constraints, all articles that mentioned influenza, flu, grippe, and Spanish flu, were saved and then transcribed verbatim. Articles included obituaries, opinion pieces, public health notices, transcribed lectures, advertisements, calls for volunteers, government mandated closures and charts that tracked the progression of the epidemic within schools, hospitals and cases reported to the city clerk's office.

Death Registries

Previous work carried out by this researcher on the 1918-19 influenza epidemic concentrated on the experience of two Aboriginal communities from the Lake Winnipeg region of Manitoba. Information captured for these smaller populations was compiled from parish registries penned by local clergy. While conducting this earlier research there was never a thought of reminiscing of the days of deciphering bias inherent within the parish records. When facing the task of fully exploring the pitfalls of government mandated death registries a longing arises for the problems associated with parish documentation. There is a litany of sources that aid in the evaluation of statistics pulled from the parish registry, and, in many instances, information is available about the individual who created the lists. As a result of this, the bias interlaced within this data set can be somewhat easily inferred. The same critical analysis is not available for death registries, which are often presented as less controversial sources of information. And while it is true that may have fewer problems, to gauge them as unbiased or error free is somewhat problematic.

Death registries, much like parish registries, have the potential to be littered with clerical issues. To minimize problems arising with secondary transcription, as has already been mentioned, the data were entered by a single individual (myself) and

periods of transcription never exceeded four hours without a lengthy break. Yet even with the utmost care, as the records were handwritten, difficulties with character recognition (especially in the early stages of transcription) were not uncommon. To allow for ease of retrieval, reentry and data conformation all entries included a reel number, JPEG (image) number and original registry number. The fact that the microfilm was scanned into PDF format also aided immensely in minimizing the instances of secondary transcription error as Acrobat Reader allows a researcher to zoom in on the offending characters in order to get a clearer look.

There is also information within the registries that suggests that errors may have occurred when the city clerk's office entered the data from the original report of death or that physicians themselves may have provided incorrect information about the deceased. The date of return, date of death and name of the individual are listed twice on each registration entry, once as a part of the original entry and again in the physician's return. This provides some proxy by which to measure error. Of the 3012 entries included in this portion of the analysis (all deaths listed from pneumonia or pneumonia derivative or influenza or influenza derivative from 1 September 1918 to 31 August 1919), 14 entries contained discrepancies.[42] Four entries have information

[42] On reel 240, jpeg 519, page 363 (Pirtiopertosa, Rosina) the date of return predates the date of death, and for Harry Pickering (240, 520, 364) there are two different recorded dates of return. Entry 240, 679, 523 has an inconsistent middle name; 240, 716, 14 (Murrell, George F.) is recorded with two different dates of death. The entry for Neil Robinson (240,746, 44) has an arrow pointing to the name of his father (Wm Robson) yet there is no further indication as to which is the correct spelling. Entry 240, 775, 73 is recorded with differing surnames, entry 240, 899, 196 (Leabovitch, Issac) original date of death listed as October 9, 1918 yet in the physician's return it is recorded as November 7, 1918. The entry found on page 226 (reel 240 jpeg 929) for Irish Nuiman also contains month confusion. John Skain (240, 1997, 393) is listed as a female which is most likely incorrect. For entry the entry on reel 240, jpeg 1168 page 465 the date of return is listed as December 13, 1918 and then as October 13, 1918. On the entry listed on reel 251, jpeg 855 page 251 the individuals surname appears as Coward on the original entry then as Edward on the physicians return (his given name is consistently listed as William Spiner). Entry 251, 1207, 598 also is comprised of alternative surnames (Sackville and Sackfield). Entry 252, 10, 268 is the second instance in which the date of return predates the date of death. Within

recorded in the wrong location (confusion with names of parents or simply off a column) and there are ten instances in which records have been changed at the request of a relative. It was clear when requests were carried out to update entries because information within the entry has been altered by hand and the registration is stamped Corrected by (name of clerk) and declaration dated (date of correction provided) by (name of individual who requested the change, and often the relationship to the deceased is listed). Dates of declaration fluctuate from months after death (May 31, 1919) to decades later (August 10, 1941). It is safe to assume that information which was not duplicated was recorded with as much diligence as information which was. With the error exposed above it can therefore be determined that the information was transcribed with a high degree of fidelity.

Problems with the data set are not restricted to what information is available; just as problematic is the absence of information. Table 4.3 outlines the data missing from the registries. Column one lists the variables recorded within the death registries. Column two represents the number of cells in which there was no recorded data available and column three is the calculation of the percentage absent for each given value. The dashed line represents the transition from the original return to the physicians return. Surname, Christian name, sex, age, date of death, place of death, name of informant, informant's address, date of return, disease causing death, and physician's name and address were recorded in almost every instance.

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entry 252, 357, 90, both the dates of death and date of return are inconsistent within the original entry and the physician's return.

Table 4.3 Completeness of Death Registries for Influenza and Pneumonia Deaths Entered From 1 September 1918 to 31 August 1919

Value	Empty Cells	Percentage
Surname of Deceased	0	0.00
Christian Name	1	0.00
Sex	0	0.00
Age	0	0.00
Date of Death	0	0.00
Place of Birth	67	0.02
Place of Death	0	0.00
Place of Burial	234	0.08
Occupation	1751	0.58
Marital Status	84	0.03
Name of Father	1044	0.35
Maiden Name of Mother	1413	0.47
Cause of Death	1567	0.52
Name of Physician	444	0.15
Name of Informant	1	0.00
Address	1	0.00
Date of Return	0	0.00
Surname of Deceased	0	0.00
Christian Name	1	0.00
Date of Death	0	0.00
Disease Causing Death	0	0.00
Duration	144	0.05
Immediate Cause of Death	382	0.13
Duration	833	0.28
Physician's Name	0	0.00
Address	0	0.00
Date of Return	0	0.00

Sixty-seven (2%) entries failed to list an individual's place of birth, 144 (5%) failed to include the duration of the disease causing death, where as duration of the immediate cause of death went unrecorded on 833 (28%) entries . Eighty-four (3%) individuals have no information regarding marital status (although 40 are under the age of 6, and could therefore be ascribed to the single status). 234 (8%) entries provide no information with regard location of interment. Thirteen per cent (382) of the entries were listed without an immediate cause of death and 15% (444) failed to list a physician's name within the initial return. Parents' names were often omitted from the registry, with mothers absent 47% (1413) of the time and fathers missing at a

rate of 35% (1044). Although listed within the physician's return it was not uncommon to see the cause of death absent on the original record (1567 or 52%). Finally a staggering 58% (1751) of entries failed to record the deceased's occupation, a true stumbling block when attempting to ascertain the pandemic's relationship to social status and subsistence.

There is also the concern about under registration, omitted entries and defining a population at risk. In 1918 doctors practicing within the province were not required to report influenza to the Provincial Health Department (*The Globe* News, October 5, 1918 page 07) and therefore during the early stages of the epidemic the disease may have gone unnoticed. Once the epidemic was raging this would no longer be the case but this fact could help explain why it would appear that Toronto[43] was not impacted by the first wave of the pandemic. It may also affect later stages of the outbreak, when the population was less inclined to continue to deal with the disease. As will be discussed in Chapter 6, Rosenberg describes a series of stages that a population goes through when encountering an epidemic. When a disease goes out of view does not always coincide with the time at which a pathogen is no longer responsible for excess mortality. It is also probable that influenza interacted with other health conditions (for example tuberculosis and pertussis) and, especially during the first and third waves, deaths attributable to influenza may have been listed under other immediate cause of death (ICD) and disease causing death (DCD). It is just as important to note, however,

[43] For that matter Canada as a whole (Humphries 2008).

that during the second wave physicians may have been over eager in attributing deaths to influenza and pneumonia.[44]

Other infectious diseases are not the only way in which deaths relating to influenza may be masked or amplified. Inclusion of infants can also be a difficult problem to solve. Physicians are often inconsistent in how they classify infant mortality if a mother falls ill or dies of influenza while pregnant. For example, Isabella Lumsden Imbash's DCD is listed as influenza and her ICD is listed as pneumonia (Reel 204, Jpeg 418 page 262); the next entry is for her baby born prematurely,[45] but flu is not mentioned for her infant. On the other hand there are three children[46] listed as having died one day after birth of influenza, two of the entries note that the infection was acquired from the mother. In the twelve months (1 September 1918 to 31 August 1919) scrutinized for this project there were 658 deaths attributable to early labor and stillbirth.[47] Beveridge (1977) demonstrates a clear association between increased infant and perinatal mortality and Spanish flu. Deciphering which of these deaths are related to the influenza pandemic, however, would be a futile task involving guesswork.

Epidemics are often periods defined by social upheaval as a large portion of the population has fallen ill and city clerks are left to record a higher number of deaths

[44] There were 7 cases of bronchitis, 21 instances of whooping cough, 21 tuberculosis fatalities and 3 meningitis deaths that were listed in conjunction with influenza, pneumonia and/or bronchopneumonia.
[45] This is not the only case where an infant's cause of death is listed as prematurity and influenza has clearly played a role the circumstances that led to the baby's death. On Reel 240, Jepg 1235, Page 531, the physician notes in relation to the still birth of Baby Raycroft "premature labor due to high temperature of mother from Spanish Influenza." 10-hour-old Roy Hued (251, 666, 65) was born prematurely because "Mother afflicted with flue [which] caused premature birth and rupture of membranes [leaving the] child weak when born." Influenza in parent is the listed cause of death in the case of stillborn Louis Marks (251, 1069, 461) and influenza of mother is listed in relation to the death of the McGowan twins (240, 229, 75).
[46] Walter Brydon (251, 764, 191), Baby Hensen (240, 691, 535) and Teresa Kempffer (541, 970, 364).
[47] These are not included in the study sample for this project

than usual. As a result it is possible that a few may have inadvertently 'fallen through the cracks.' Palmer et al. (2007) compared provincial registries from Newfoundland to hospital records collected in the community of St. Anthony. During their study period, 15 patients were admitted to the hospital with influenza, four of whom succumbed to the disease. When they compared the hospital records to the records in the Provincial Archives they discovered one death was not recorded and two individuals were listed as having died from other causes.

Finally there is the problem of establishing a population at risk. Death registries simply record all of the deaths that occurred within a given population during a set time period. Although information, such as place or birth and place of burial (when provided), may afford some proxy by which to gauge citizenship, it is by no means a robust measure by which to include or exclude members within a study sample. A long time resident of Toronto may choose to be buried in New York next to a loved one or a recent immigrant may have contracted the disease shortly after settling in the city. The problem is further confounded when we acknowledge that Toronto housed a temporary army base at Exhibition Stadium. Due to a lack of hospitals in smaller communities, larger Ontario towns (including Toronto) played host to many of the sick and suffering (McGinnis 1981). Again there is no means by which to extract these individuals but the presence of people from other counties, cities, provinces, and countries reminds us that there were, in all probability, a number of Torontonians who perished elsewhere.

Although there are potential ways of assessing the quality of the death registry (for example, cross referencing other historical documentation such as hospital

records) there is no real way to correct this problem. If a sample is altered then the information derived from it becomes inherently flawed. The only true way to overcome this shortcoming is to define one's study population when running statistical tests or qualitative analyses. To this end this dissertation's focus in on influenza mortality recorded in the provincial archive for the city of Toronto, not influenza mortality for Toronto as a whole.

Establishing Inclusion Parameters – Flu vs. Pneumonia

One of the largest aspects to defend in this dissertation is the question of who/what to include within the study sample. Initial data collection began with the transcription of any conditions attributable to death that were listed as pneumonia (or a pneumonia derivative) and/or influenza (or an influenza derivative) (See Tables 4.1 and 4.2 and Appendix 1). Although a logical decision in the early stages of data collection,[48] the question remains if this is it justifiable at the level of analysis. Of all of the conditions listed the four most common were; influenza (n=1192), pneumonia (n=616), bronchopneumonia (n=294) and lobar pneumonia (n=81). One hundred and forty-two entries were listed as both influenza and pneumonia. The choice to include both conditions came from the January and February (1920) monthly reports for the Department of Public Health of the City of Toronto as well as observations from newspaper accounts during the epidemic. The two public health reports are designed to examine the impact of the epidemic waves of 1918 and 1920 within the city. Public health officials make no distinction between the two conditions. In the Monthly Report of the Department of Public Health (October 1918:2), Dr. Hastings states that

[48] It is always easier to remove data then it would be to go back and try to retrieve it. It also aids in the consistency of data entry if all data points are transcribed at or around the same time.

the causal agent of the pandemic has yet to be identified, and that "the majority [of scientists] favor the Influenza Bacillus, which was discovered in 1892, but it is yet to be determined whether the epidemic, which we have just passed through is one of influenza or one of broncho-pneumonia, or one of general blood infection."

Newspaper accounts provide a much more in depth understanding of how both the lay individual and professional practitioner viewed this disease. Numerous charts track the progression of the epidemic, and although the two conditions were commonly separated, reference made to the overall mortality rate for the epidemic combined them. It was often stressed that if bed rest was not adhered to the natural progression of the disease was to increase in severity from influenza to pneumonia.

> As had been impressed upon the people time and again, the flu would be far less dangerous than it is if patients went to bed as soon as they are taken ill and stayed there until better. But the man whose wages cease when he is ill hesitates to take to his bed. He sticks to his work as long as he can, thereby prejudicing his chances of recovery. Furthermore, he is tempted to get up and back to work too soon, a course which invites pneumonia and other complications. (The Star 18 October 1918 page 11)

Finally one of the best justifications for the inclusion of both conditions comes from a widely distributed report from Surgeon-General Blue (reproduced in both the Star (5 October 1918 page 23) and within the Department of Public Health's Monthly Reports (September 1918)). Blue's report reads as follows:

> Bacteriologists who have studied influenza epidemics in the past have found in many of the cases a very small rod-shaped germ called, after its discoverer, Pfeiffer's bacillus. In other cases of apparently the same kind of disease there were found pneumococci, the germs of lobar pneumonia. Still others have been caused by streptococci, and by other germs with long names (Star 5 October 1918: 23).

The presentation was so similar for these conditions that when researchers attempted to isolate the causal agent they in fact included a number of different

pathogens. Whether this is a byproduct of multiple infections within a single host or an array of conditions which present the same symptoms is difficult to ascertain. In order to get at this question one of the aims of this dissertation is to examine how these two pathogens may have worked synergistically with one another to increase the severity of the pandemic, and as such the inclusion of both disease states becomes even more essential.

America's Forgotten Pandemic – Finding Flu in the Archive

Most government documents are written for a particular audience, which in turn causes them to be shaped by an inherent bias. The monthly health reports make this bias apparent. The monthly reports depict the pandemic response and experience in Toronto to be superior to that of the United States. Much of the focus of these reports are to examine the situation in United States and they stress how mortality in Toronto can be positively compared to the situation in Boston, Chicago and New York. The monthly reports were written by Minister of Health, Dr. C.J.O. Hastings. He appears to have been competent at his job, and with the exception of a trip he took to New York at the beginning of the epidemic and his decision to restrict attendance at church services, he faced very little public criticism with regard to his treatment of the epidemic. He first mentions the pandemic in the September 1918 report. In this account Hastings details his trip to New York, which was undertaken to acquire advice on how best to safeguard against the disease. He also provides a reproduction of Surgeon-General Blue's published brief on how best to deal with the microorganism. In this report Hastings asserts his belief that the epidemic in Toronto will not be severe and likens it to epidemics experienced in Toronto in 1889 and 1890.

His report the following October is devoted entirely to the epidemic. In it Hastings pays special attention to the etiological agent, Toronto's success in battling the disease through preparedness, the sacrifice of physicians and nurses and diligent educational efforts. The November report details the proceedings of the 46th Annual Convention of the American Public Health Association, which were held in Chicago. The focus of this report is again on isolating the characteristics of the epidemic and the situation throughout the rest of North America. Three reports exist for 1920 (January, February and March) which appear to have been in response to a scare over an echo wave of the virus.

The other major source of government mandated information is the national census, which is used to estimate the population at risk.[49] Traditionally Canadian censuses are conducted every five years. A stumbling block for this dissertation, the First World War caused Canada to skip the 1916 census, leaving estimates of population reliant on the 1921 census. The 1911 census is examined to help measure the population parameters of Toronto. By-and-large the two documents present information guided by a common format. The only notable difference is that the 1911 census records religion divided up by region,[50] whereas the 1921 census records the data for the city as a whole.

The remainder of the story is one of the absence of information. Although it has been well known to cause pandemics at somewhat regular intervals (Beveridge

[49] As has already been established this dissertation is not looking at the number of Torontonians that died during the epidemic, rather it represents the number of people that died in Toronto. Death registries are not designed to collect or dispatch information from/to other regions and therefore will omit and include individuals outside the study area. As well population figures are not available for every given year and therefore the closest census year must be utilized.
[50] Toronto E, Toronto N, Toronto S, and Toronto W-O

1977) influenza traditionally is not a disease that elicits much fear and panic. Perhaps overshadowed by the war or fed by conceptions of the banality of flu, Spanish flu largely goes unmentioned throughout the pages of history. The board minute meetings of Toronto's The Hospital For Sick Children make no mention of flu nor does it appear within the pages of The Rotary Voice, the newsletter for the Toronto Rotary Club, a social group responsible for organizing a good portion of the civic action carried out during the epidemic. In fact it would be difficult to grasp that anything out of the ordinary was happening from September to November of 1918 in Toronto, as demonstrated by the following quote:

> Fourteen members have not attended a meeting for the past four weeks. A few members have not attended a meeting since the present session commenced in September. Neither have they sent any excuse for absence. What about it? We do not wish to publish the names of these members, but if we are so instructed by the Directors, and perhaps we may be, to do so, then it will be so. Several members dues are outstanding. We would like to see the colour of their cheques - promptly please" (Rotary Voice Vol.1 No. 19 October 29, 1918).

This absence of information ranges from the city and provincial archives to the Jewish archives of Ontario. It is difficult to interpret this lack of information. Can it be inferred that the pandemic's impact was not severe or disruptive or did it occupy too short a time to be collectively recalled? This dissertation struggles to adequately respond to this question, as it will be demonstrated that the city of Toronto was preoccupied with the war, but the pandemic, however short-lived, significantly altered the composition and constitution of its people.

Reporting Bias – Assessing Toronto Newspapers

This dissertation retells the tale of influenza in Toronto through the examination of two prominent newspapers: *The Toronto Star* and *The Globe*. In order to interpret the information contained within the pages of these paper 'time machines,' a thorough understanding of their history, mandate and political bias must be explored. The daily newspaper came to Canada in 1840, facilitated by forms of transportation that aided in dissemination of information. These included stagecoaches (1830s), steamships (1840s), the railway (1850s), telegraph lines (mid-1840s) and the laying of the Atlantic cable (1866) (Rutherford 1978). Touted first as purveyors of education and social guardians, newspapers quickly fell victim to the capitalist machine and by the 1890s they were consumed with boosting circulation and enticing advertisers. This shift caused an increase in sensationalism, the inclusion of trivial news, entertainment pieces, social analysis, and serious commentary and gave companies the ability to edit the news through the threat of monetary losses (Sotiron 2004; Rutherford 1978)

The Globe, first published in 1844, was born from the merger of the two moderate Liberal newspapers, the *Examiner* (est. in 1838 by Sir Francis Hincks) and the *Banner* (est. in 1843 by Peter Brown) (Wallis 1908). It was spearheaded by Brown's two sons, Reform politician George Brown and J. Gordon Brown. Known for excellence as a purveyor of news as well as fair and accurate reporting, it was read not only by those who ascribed to its Liberal standing, but also by Conservative constituents as well (Sellar 1908). In his eulogy to Gordon Brown (assassinated by an employee in 1890), president of the Canadian Press Association A. Matheson said of Brown "Like all good journalists, he loved his profession, and he believed in the

newspaper as a public educator and as a power to defend the rights and privileges of the people" (Colquhoun 1908: 91). The paper also touted the lofty ambition of reaching a provincial audience; for example, George Brown used the *Weekly Globe* to appeal to residents of rural Ontario by including extra matter that was more suitable to country tastes (Rutherford 1978). *The Globe* continued to ascribe to the high Liberal standards set out by Brown under the editorship of Mr. John Cameron, Mr. E.W. Thompson, Mr. J.S. Willson and Rev. James A Macdonald. Due to its prominent standing, and therefore secure financial status, *The Globe* never fell prey to sensationalism (Robinson 2004) or other tactics used to lure readers but rather set the standard for excellence in reporting (Sellar 1908). It maintained its Liberal leanings and zeal for responsible reporting until it was merged with *The Mail* in 1936.

The Globe offers a 'just the facts' approach to reporting the circumstances of the epidemic. Spanish Flu only appears on page one twice throughout the four months that were viewed. On Tuesday 15 October 15 1918 it reports the closing of Toronto city schools and on Monday 21 October 1918 it informs readers of the University of Toronto's announcement of a discovery of a vaccine for the disease. In total the epidemic is mentioned in 113 articles from 1 September 1918 to 31 December 1919. In line with its provincial agenda many of the stories are concerned with the epidemic experience throughout Ontario (and to a limited degree Canada) and information pertaining to the situation in Toronto is predominantly restricted to the Births, Deaths and Marriages and Lifestyle sections of the newspaper. With regard to information about the city, emphasis is placed on reporting the morbidity and mortality associated with the pandemic, the advice of Dr. Hastings (Minster of Health), the actions of

public officials, the volunteer efforts of various women's groups and the situation among military men at Exhibition Camp.

The *Evening Star* was founded in 1893 (Wallis 1908) by a group of printers who supported organized labor, but the printers soon ran out of money and members of organized labor lost interest. Over the next six years the *Evening Star* changed owners four times, so much so that by 1899 it was "unrespected, uninfluential and almost unread" (Harkness 1963:21). It was also in this year that up and coming journalist Joseph E. Atkinson was courted by Hugh Graham, owner and publisher of the *Montreal Star*, the largest English-language newspaper in Canada, to come aboard as managing editor. Turned off by the paper's conservative views and capitalist leanings, Atkinson sought the advice of long time friend and *Globe* editor J.S. Willson. Willson informed Atkinson, that at the request of Sir Wilfrid Laurier, the current Prime Minister of Canada, a number of wealthy Liberal Toronto men were considering the purchase of the *Toronto Evening Star*. As only one Toronto newspaper, the *Globe*, had supported his candidacy, it was felt that another should arise in support of his Liberal agenda (Harkness 1963). On December 31, 1899 Atkinson took on the task of reinventing the new *Star*. In doing so "*The Star* had become Canada's first "popular newspaper," the first to appeal to the "mass circulation" market" (Harkness 1963:40).

The paper was guided by his highly personal style of journalism stressing colourful human interest stories and local coverage over broader regional issues (Harkness 1963:11), and as a reflection of this new way of conceptualizing the news the format of *The Star* was drastically altered. It was augmented by an expanded sports news section, an insightful editorial page, and an extended women's section

(revamped by Mrs. Atkinson) which included gossip columns, advice to the lovelorn and light comment on topical subjects. The paper was also not above sensationalism to sell papers. Atkinson purposefully tailored his paper to "the industrial workers and the 'little people' who had no other spokesmen" (Harkness 1963:41). In 1913, Atkinson became the paper's majority shareholder and by early 1916 he began his campaign calling for a Liberal welfare program, petitioning for old age and widow pensions, unemployment insurance, a minimum wage and maximum hours act for women and children and welfare legislation. By January of 1917 this position had found its way into the pages of *The Star*. Dubbed "Whispering Joe" by the *Telegram,* Atkinson was believed to have the power to sway the populace and alienate the elite (Harkness 1963:117). Atkinson represented a polemic mixture of staunch liberalism and old-time Methodist upbringing that caused him to question the church's theology and support its ethical teachings and as such he led *The Star* to support the church's good works and bring to the forefront doctrinal disputes (Harkness 1963:150).

This emphasis on public interest stories and local coverage is paramount in *The Toronto Star's* reporting of the 1918 – 1919 influenza pandemic. Two hundred and ninety-six articles about the epidemic can be in *The Star* within numerous sections ranging from life, fashion and family to editorials and opinions. Journalists utilize heavy narrative aimed at not only reporting the news but interpreting it as well. Atkinson's *Star* is clearly concerned with unique problems of the poor, including many stories about insufficient resources, the necessity of health insurance and the difficulties faced by wage laborers. One of the most useful features of *The Star* is its Local City News section which details unique stories from around Toronto. The city

is divided up by the following districts: Danforth, Earlscourt, North Toronto, Todmorden, West Toronto, Birchcliff, The Beaches, Silverthorne, and East Toronto and a local reporter outlines the experiences of each of these neighborhoods almost daily. The paper explores the advancement of the epidemic, the contribution of Dr. Hastings, the experiences of different cultural groups, the work of women's groups, the conflict between the church and public health, the situation at soldier encampments, prohibitions on sporting events and public gatherings and to a lesser extent the pandemic progress throughout the rest of Canada. The pandemic is mentioned 11 times on the *Star's* crowded front page, four of these times in conjunction with its impact on the Liberty Drive, once in reference to the opening of theaters and in the remaining instances in regard to noteworthy deaths, the situation overseas or throughout the rest of Canada.

Rice's (2003) analysis of the epidemic experience in New Zealand discusses the role of newspapers in curtailing influenza mortality. He describes how they became tools of the government, which aided in informing the public and as such they may have represented not the actual proceedings of the epidemic, but how officials wanted citizens to perceive the pandemic.[51] He also states that the government prohibited reporting death rates so as not to induce public panic.[52] With the exception of government officials chastising newspaper men for creating unnecessary panic among the citizenry during the early part of the epidemic, officials in Canada did

[51] This is my interpretation of his work. Rice does not state that there is any bias in reporting.
[52] This omission actually had the opposite impact as residents assumed mortality was higher than it actually was.

nothing to thwart the reporting efforts of Ontario newspapers.[53] They did utilize them as a means to disseminate information about closures, prevention procedures and their ongoing efforts to contain the disease.

Armed with the tools to interpret the data (both analytically and historically) this dissertation aims to outline the myriad of factors that contributed to the pandemic experience between 1 September and 31 December 1918 throughout the city of Toronto. It is important to keep in mind that the population explored is not the city itself, but individuals captured within its death registries.

[53] Of course this statement cannot be made with absolute certainty but based on *The Star's* proclivity to openly question public officials, censorship would have surely been met with some public discourse.

Chapter 5 An Epidemiological Look at Flu

The relationship between anthropology and epidemiology dates back to the late 1950s, when medical anthropologists worked alongside epidemiologists to better understand the nature of psychiatric disorders (Dunn and Janes 1986). Since then anthropologists have employed epidemiological methodology to explore the behavioral components of the incidence and spread of various diseases (Johnson and Sargent 1990). This chapter aims to take an anthropologically oriented epidemiological look at the second wave of the influenza epidemic in Toronto, Ontario, Canada. In order to utilize this framework a researcher much focus on the interactions between a given host, etiological agent and the environment (both social and physical) in which they reside. Key demographic data, such as age, sex, ethnicity, and economic status, must also be considered to help establish factors that may affected the introduction and persistence of a disease. This chapter will utilize a traditional objectivist/positivist approach wherein the death registries and historical documentation will be scrutinized to present an aggregate analysis[54] of the disease experience. It strives to address some fundamental questions about the dynamics of the epidemic by examining some basic questions:

- How did the disease enter the city?
- How many people died?
- How did physicians classify the disease (pneumonia vs. influenza)?

[54] Aggregate methods serve most commonly as counting techniques that provide information on the flow of events (Knodel 1988).

- What was the age and sex distribution of mortality?
- Who died (an analysis of occupation, marital status and country of origin)?

These questions help expose how the virus traveled though the community. It also provides a basis upon which the experience in Toronto can be compared to the impact of the epidemic in other major cities throughout the world. Finally, an exploration into the disease causing death and the immediate cause of death can help contribute to the ongoing debate concerning which conditions to include when recreating mortality attributable to Spanish flu.

Timing of the Epidemic

Popular scholarship attempting to understand how the first wave of influenza entered Canada in late spring and early summer usually turns on the arrival of troop ships from Europe, most notably the *Araguaya* (which docked at Grosse Isle on 26 June 1918), the *Somali* (which was refused port in Quebec in late June due to sickness on board), the *Med 1099* (which docked in Halifax on 9 July 1918) and the *Nagoya* (which may have brought flu to Montreal on 9 June 1918) (McGinnis1992; Patterson and Pyle 2004; Pettigrew 1983). Humphries' (2008) alternative hypothesis that these ships arrived empty to transport troops to the front resulted from his demonstration that scholars have misinterpreted ship logs. He therefore contends that it was the aggregation of American troops preparing for deployment to the front that brought flu to Canada. In any event it would appear that the first wave in Ontario went unnoticed or perhaps never occurred. There is little doubt, however, regarding the introduction of the second wave into Central Canada. At the beginning of September 1918, epidemic activity had already been noted in the ports (and cities) of the northeastern

United States. According to Humphries (2008: 221) the majority of cases spread across the "border to Niagara-on-the-Lake, Ontario, Victoriaville, Montreal, and Quebec City, Quebec, and to Sydney, Nova Scotia." Patterson and Pyle (2004: 10) on the other hand postulate, working off the information provided in McGinnis (1977), that "Canada was attacked along the Atlantic coast, by shipping down the Saint Lawrence, along the route of the Trans-Canada railway, and overland from the United States, especially in the west."

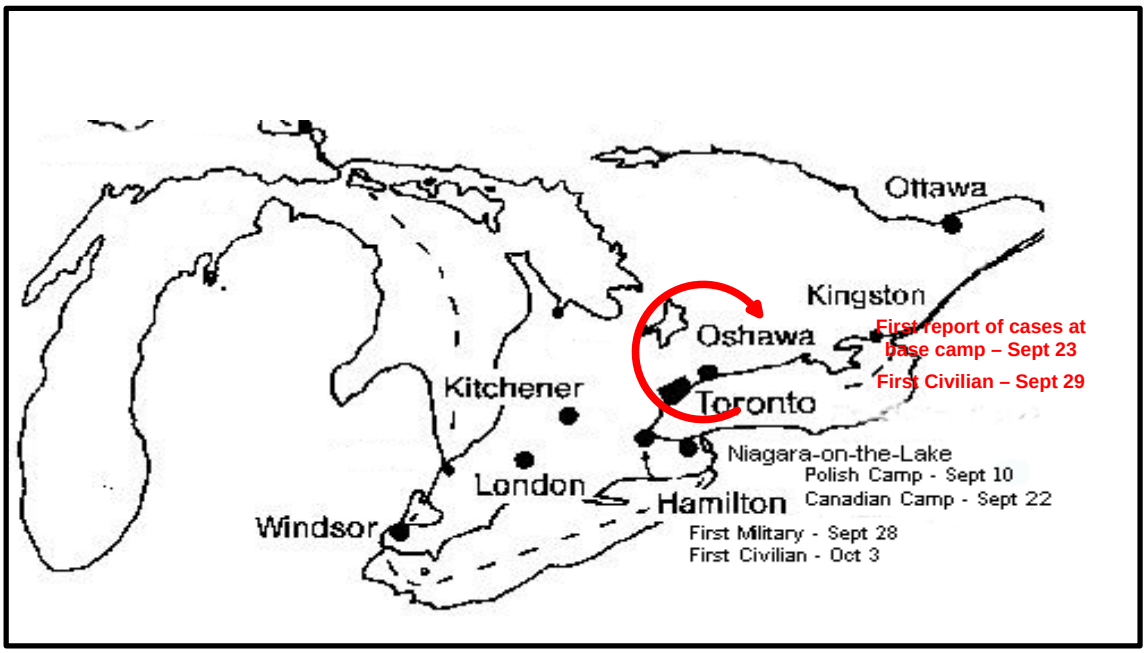


Figure 5.1 Introduction of the Virus From Niagara-on-the-Lake to Toronto via Troop Movements. Dates for Niagara-on-the-Lake gathered from LAC RG 24 Vol 4270 File 15-2-72 vol.2, Dates for Hamilton (Herring and Korol 2010)

"Send Only the Very Serious Cases" - Delivering Flu to Toronto

Records indicate that the virus first hit the Polish Army Camp, which was populated by Polish immigrant soldiers recruited for the French Army in the United States (Humphries 2006), stationed at Niagara-on-the-Lake on or around 10 September 1918 (RG 24 Vol 4270 File 15-2-72 vol.2 Polish Camp Hospital Niagara-on-the-Lake

Consolidated Report on the Epidemic of Influenza in M.D. # 2). The Canadian military placed the Polish Army Camp under strict quarantine on 18 September 1918 (RG 24 Vol 4270 File 15-2-73 vol.1 Influenza Quarantine J.L. Robinson Nov 1, 1918) but not before the disease had already spread to the nearby C.O.R. Camp (RG 24 Vol 4270 File 15-2-73 vol.1 Influenza situation at Niagara Camp 10 October 1918).

In 1914, for administrative purposes, the country was divided into nine military districts, with each run by an independent district headquarters. Niagara-on-the-Lake was part of Military District Number 2, which encompassed most of Central Ontario including Lincoln, Welland, Haldimend, Norfolk, Brant, Wentworth, Halton, Peel, York, Ontario, Grey, Dufferin, Simcoe, the districts of Muskoka, Parry Sound, Algoma and Nippissing north of the Mattawa and French Rivers (Love 1999: 50). Toronto served as its district headquarters. Although no specific mention is made of the transport of men to Base Hospital[55], a memo dated 7 October 1918 orders very serious and complicated cases to be transported from army camps to existing military hospital accommodations (RG 24 Vol 4270 file 15-2-73 vol. 1 Possible Epidemic of Influenza). From the *Nominal Roll of Deaths Occurring in Military Hospitals M.D. 2 From Outbreak of Spanish Influenza* (RG 24 Vol 4270 file 15-2-73 vol. 1) it is apparent that hospitals were established in Brant, Whitby, Hamilton, Kapuskansing and Toronto. Examination the number of cases at each hospital (Table 5.1) shows that the vast majority of severe cases were transported to Base Hospital, in Toronto.

[55] Base hospital was a facility set up in Toronto to house and treat sick and wounded soldiers.

Table 5.1 Cases and Deaths Occurring in Military Hospitals From Outbreak of Spanish Flu

Hospital	Total Cases	Total Deaths
Base Hospital	1761	91
Brant Military Hospital	425	18
Hamilton	115	13
Kapiskasing Hospital	91	4
Whitby Military Hospital	67	2

Source LAC RG 24 Vol. 4270 File 15-2-73(1)

Base Hospital was created in the spring of 1916 to deal with the shortage of hospital accommodations for members of Military District no 2 (RG. 24 Vol. 4385 File 34-7-136 vol. 1 Hospital Accommodation 2nd Division 4 February 1916). After months of searching officials agreed to locate the new Base Hospital in the old General Hospital facility, which was located on Gerrard Street East (RG. 24 Vol. 4385 File 34-7-136 vol. 1 White Hospital Beds). The location was operated by the General Hospital from 1865 to 1913, at which time the site was abandoned partly out of the need for improved facilities and partly due to the influx of monetary resources received through an amalgamation of the Hospital Board and the University of Toronto (Crosbie 1975). Base Hospital was initially established as a temporary facility that was projected to house 1040 patients. Throughout 1916 and 1917 there were numerous complaints regarding the inadequate staffing of the facility. On 27 August 1916, Lieut. Colonel T.R. Richardson, commanding officer of the Base Hospital reports

> If it requires a total staff of 166 to care for 400 people, it will be obvious that 208 are required to care for 500: in other words, this unit is at present about 45 short of its proper strength. A great many complaints are coming in to my office from the patients and the friends of the patients, to the effect that there are not sufficient orderlies to care for the sick, a circumstance the truth of which I fully realize. (RG. 24 Vol. 4385 File 34-7-136 vol. 1. Personnel Base Hospital)

It is unclear to what extent the problem of staffing was addressed by the time of the Spanish flu in 1918. A report dated 3 May 1919 states that during the epidemic there were 3 consultants, 15 medical officers, 40 nursing sisters, 20 V.A.D's[56] and 12 civilian nurses supported by 193 additional support staff members. From 1 October to 28 October, 800 R.A.F. members were also called upon to act as additional support staff (RG. 24 Vol 4270 File 15-2-73 vol. 2 Influenza Epidemic October 1918).

It is difficult to ascertain when men with the disease were moved from Niagara-on-the-Lake to Toronto. A circular letter, drafted 7 October 1918, indicates the military's desire to not call attention to the epidemic. Sent out to all medical officers, it states that "the disease, although extremely contagious, is not a serious one and every effort must be made to control alarm, not only among the troops but among the Public and the Press. The daily publication of statistics is very undesirable" (RG 24 Vol. 4270 File 15-2-73 Vol.1 7 October 1918 Possible Epidemic of Influenza). As such, very few records exist as to the transport, housing or other particulars regarding military personnel suffering from influenza. A nominal roll of deaths occurring in military hospitals for Military District no. 2 does exist but it only begins on 26 September with five deaths listed at the Polish Army Camp between 26 and 29 September. This information would appear to be incomplete as a newspaper report from the *Star* dated 23 September 1918 indicates that five deaths had already occurred at the Polish camp as of this date. According to this document the first death registered at Base Hospital was on 10 October. Death registries for the city of Toronto list the first death at Base Hospital as occurring on 5 October. According to a report

[56] V.A.D.s (or voluntary Aid Detachments) were comprised of women, usually from the middle or upper middle classes, who volunteered their services and were partially trained to act as nurses and aids during the First World War (Jones 2007)

generated by the commanding officer of St. Andrews Military Hospital, North Rosedale, Toronto dated 3 May 1919 (RG 24 Vol. 4270 File 15-2-73 vol. 2), Base Hospital housed 173 influenza patients as of 23 September. In light of the fact that the first civilian death did not occur in the city until 29 September, it is reasonable to assume that the virus was transported to the city via infected military personnel.

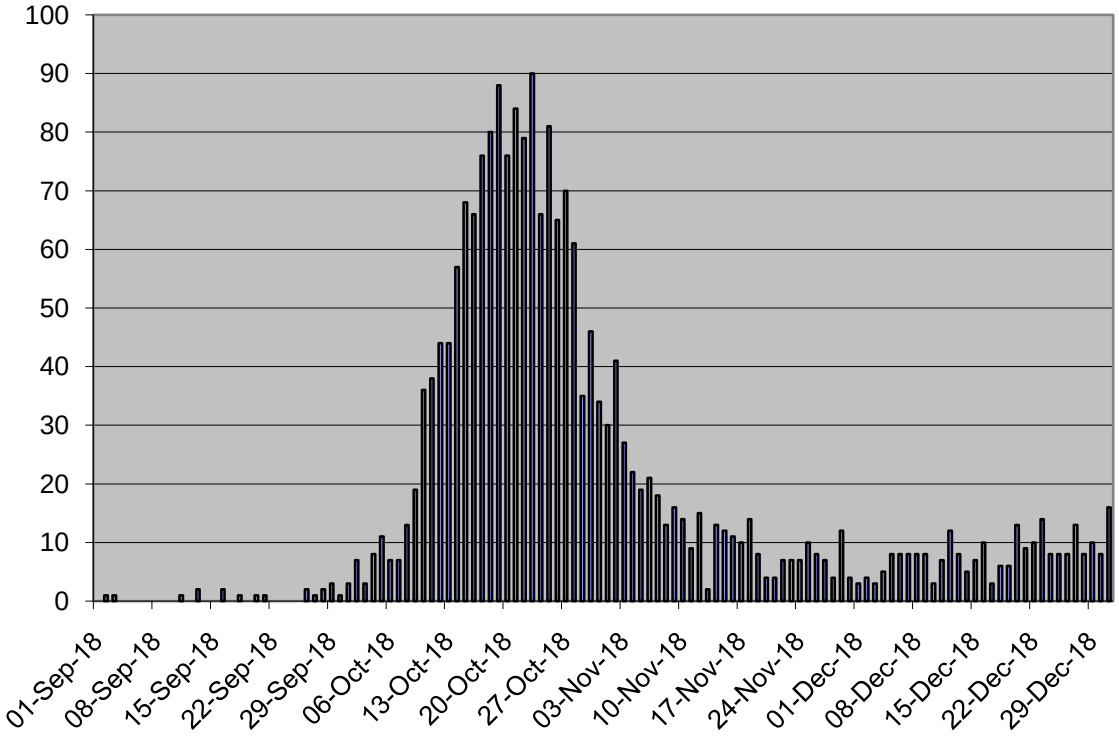


Figure 5.2 Deaths Attributable to Spanish influenza, Toronto, 1 September 1 to 31 December 1918

Figure 5.2 depicts the mortality experienced during the second wave of the pandemic in Toronto. The figure encompasses deaths attributable to influenza (or an influenza derivative) and/or pneumonia (or a pneumonia derivative) from 1 September through to 31 December 1918 gathered from the public death registries. This period was chosen as it utilizes the time parameters established for the second wave of the

epidemic[57] as well as the accepted time period in which flu entered Canada[58]. According to the monthly health report for October 1918 published by C.J.O. Hastings, Toronto's Minister of Health, "the epidemic struck the city almost like a cyclone, assuming epidemic proportions on or about 9 October, and continued until 2 November, when it subsided quite as rapidly as it began (Monthly Health Report October 1918: 4)." Although Figure 5.2 roughly corroborates the start date suggested by Hastings it challenges the notion that the epidemic had fully subsided by 2 November[59]. There is no question that deaths begin to taper off by 2 November deaths remained in the mid twenties and high teens from 3 November until 12 November of November, and then fluctuated between 6 and 12 for the remainder of the month.[60] The peak of the epidemic was experienced on 23 October with 90 deaths recorded in one day. The crest lasted nine days beginning with 76 deaths recorded on 17 October and ending with 81 deaths recorded on 25 October.

The overall mortality rate for the second wave of the pandemic in Toronto was 4.1 per 1000 (Table 5.2). This table includes all deaths documented in the death registries for the city of Toronto as influenza (all types) and/or pneumonia (all types) for the period of 1 September to 31 December 1918 (n= 2117). The population at risk was obtained using the 1921 census (which recorded the population of Toronto as 521,893). Hastings estimated the case fatality rate to be around one percent (based on observations in other cities and Toronto); if this table is correct it is possible that in the

[57] Crosby 1989; Johnson and Mueller (2002); Phillips and Killingray (2003).
[58] McGinnis (1981); Herring (1993); Jones (2005); Kelm (1999); Lux (1992); Palmer et al. (2007);Pettigrew (1983).
[59] Deaths from influenza and pneumonia already started to rise by the 2 October (with seven deaths on the 2 October, eight on 4 October, eleven on 5 October , seven on 6 and 7 October , and thirteen on 8 October
[60] It is interesting to note that deaths pick back up on December 31 and remained higher through the first part of January. For a complete discussion see pp.122-125

four month period encapsulated by this analysis as many as 209,583[61] Torontonians may have been sick with flu[62].

Table 5.2 Death Rate per 1000 Population for Mortality Attributable to Spanish Influenza for Canadian Cities and Provinces 1918 - 1919

City/Province	Population	Influenza Pneumonia Deaths	Death Rate per 1000*	Updated Mortality Figures
Windsor, ON	30000	32	1.07	
Winnipeg, MB	183595	388	2.11	6.6++++
Hamilton, ON	104491	244	2.34	6.1**
St. John, NB	42511	126	2.96	
Sault Ste. Marie, ON	12829	41	3.20	
London, ON	57301	187	3.26	
Toronto, ON	490000	1600	**3.27**	**4.1**
Halifax, NS	46610	153	3.28	
New Brunswick				4***
Montreal, Que	640000	3128	4.89	
Newfoundland	238670	1179		7.8****
Ottawa, ON	104000	570	5.48	
British Columbia				6.2+++
Kingston, ON	22265	145	6.51	
Saskatchewan (major cities)				6.6+
Ontario				3.6***
Canada				6.1++

Source: ++++Humphries 2008: 215; ***Jenkins 2007: 318; ++Johnson and Mueller 2002: 112; ***Jones 2007: 18; +++Kelm 1999: 25; +Lux 1989: 48; *McCullough 1918: 1086; ****Sattenspiel, personal communication 2010.

[61] This figure was calculated assuming a1% case fatality rate (for every individual that died 99 people survived or served as asymptomatic carriers). If Hastings calculated fatality rate of 3.3 is utilized, 204, 714 may have fallen ill.

[62] It is important to note that on Friday 15 November 1918 in his report on the pandemic(provided in *the Globe*, page 9), Dr. Hastings stated that the city had experienced 1614 deaths and that the population of Toronto at the time was 490,000, which would therefore return a death rate of 3.3 per 1000. The large discrepancy between the mortality figures reported in this analysis and those provided by Dr. Hastings are a by product of the time period captured by the analysis. It is not clear when Dr. Hastings identifies the start of the pandemic (although from his reports it is somewhat safe to assume 9 October) and as this article is dated 15 November, therefore it is probable that all deaths up until the 14 November were included in his figure. Between 1 September and 8 October , 78 deaths were recorded (from flu [all types] and pneumonia [all types]) and as of 14 November, 1714 deaths had been transcribed into the database for this dissertation, leaving a difference of 22 individuals between Hastings figures and work carried out by this researcher. The additional 22 deaths could be a result of inclusion parameters for this dissertation, for example tuberculosis and scarlet fever deaths listed in conjunction with influenza or pneumonia were included as deaths attributable to Spanish flu. When Hastings compiled data for his report, these deaths may have been omitted. They could also be the by product of a delay in the receipt of death registries to City Hall.

According to the October Monthly Report for the Department of Public Health of the City of Toronto "The duration of the epidemic in Toronto was at least one week less than that of any other major cities of the same size on the Continent, and the mortality was decidedly lower" (1918: 3-4). To assess the validity of this statement the mortality rates for other parts of Canada (Table 5.2), major cities in the United States (Table 5.3) and countries (Table 5.4) were compared to the mortality rate calculated for Toronto. Table 5.2 was compiled using data available during the pandemic as well as current studies that investigate influenza mortality. All of the mortality rates in the death rates per 1000 column were derived from a report by John W.S. McCullough, Chief Officer of the Ontario Provincial Board of Health, dated December 1918.[63] Updated mortality figures for Winnipeg were provided by Jones (2007),[64] an updated mortality estimate for Hamilton was published in Humphries (2008),[65] data for New Brunswick and Ontario was described in Jenkins (2007), Newfoundland mortality has been explored by Palmer et al. (2007),[66] figures for British Columbia were calculated by Kelm (1999),[67] the data for Saskatchewan was

[63] This is important to note as the pandemic is thought to have swept from East to West and it is therefore likely that mortality estimates for Western provinces may not be accurate as pandemic activity had not yet ceased. For example influenza did not strike Winnipeg until September 30 (Pettigrew 1983:56).

[64] It is important to note that this figure represents calculated mortality within the city. Work done on aboriginal populations displays a much higher mortality rate ranging from 183 per 1000 among indigenous residents of Norway House (Herring 1994) to 102 per thousand for Fisher River (Slonim 2010).

[65] A detailed description of influenza in Hamilton can be found in Herring (2007) and Herring (2010). This work was carried out using deaths attributable to influenza only (and not pneumonia) and therefore was not comparable to this dissertation.

[66] At the time of the epidemic Newfoundland was a self-governing Dominion within the British empire and did not become a province of Canada until 1949. Sattenspiel (personal communication) suggests that the actual death rates were somewhat higher than calculated using deaths reported in the death registers, which severely underestimate deaths in among aboriginal groups. These underreported deaths are heavily weighted towards aboriginal peoples. Mortality in the village of Hebron was 68% and at Okak a staggering 78%, with deaths occurring almost entirely Aboriginal populations (Markham 1986).

[67] This figure represents non-Native mortality. The mortality rate among First Nations of British Columbia was 46 per 1000 (Kelm 1999: 25).

provided by Lux (1989)[68] and the overall rate for Canada was provided by Johnson and Mueller (2002). These data indicate that the mortality captured by the death registries for the city of Toronto was in line with and at times, considerably lower than the mortality experienced in other large Canadian cities (as well as a marginally lower rate than other provinces throughout the country). Low mortality rates in small towns like Windsor may highlight less severe disease experiences in these communities or a trend to send patients to major cities to receive treatment. According to McGinnis (1981: 464) "larger Ontario towns provided accommodations and help for sufferers from surrounding communities which had not had the foresight to build hospitals."

Table 5.3 Epidemic Mortality Rates per 1000 in Major American Cities and Toronto.

City	Population	Influenza Pneumonia Deaths	Death Rate per 1000	Updated Mortality Figures
Boston	670585	2084	3.11	16.6
Toronto, ON	490000	1600	**3.27**	**4.1**
New York	5737492	22950	4.00	
Washington	331069	1564	4.72	
Pittsburgh	553905	3894	7.03	
Philadelphia	1549008	12687	8.19	37*
Indiana				20.2*
Kansas				17.5*
Canada				6.1
United States				6.5

Source: Johnson and Mueller 2002; McCullough 1918; Rogers 1920. * Data represents mortality from all causes 1 September1918 to 31 December 1918.

Table 5.3 compares mortality in Toronto to major American cities. Population and mortality data for Boston, Toronto, New York, Washington, Pittsburgh and Philadelphia were reported in McCullough (1918). Torontonian health officials strove to demonstrate Ontario's superior handling of the pandemic; this, in conjunction with the overt desire to present the country as 'healthier' than others may have prompted

[68] This is the calculated mortality rate within the city. According to Lux (1989: 48) individuals residing in rural municipalities and villages experienced a mortality rate of 10.5.

the Ontario Provincial Board of Health's Chief Officer to purposefully choose American cities with the highest mortality rates, thereby over representing mortality in the United States. The updated mortality figures for Indiana, Kansas and Philadelphia were provided from special report compiled by the United States Bureau of the Census on pandemic mortality.[69] It would appear, utilizing the information present in Table 5.3 that Toronto also fared better than these selected major American cities.

Table 5.4 - Spanish Influenza Global Mortality

Location	Death Rate (per 1000)
Australia	2.7
Germany	3.8
Norway	5.7
England and Wales	5.8
Finland	5.8
Canada (**Toronto**)	6.1 (**4.1)**
India	6.1
United States	6.5
France	7.3
Italy	10.7
Egypt	10.7
Spain	12.3
New Zealand	<20.0
Mexico	20.6
Nigeria	24.2
Indonesia	30.4
South Africa	44.3
Western Samoa	236.1
Cameroon	445

Source: Johnson and Mueller 2002: 110 -14

Table 5.4 examines recalculated global mortality figures provided by Johnson and Mueller (2002). Their paper tackles the obstacles of underreporting and conveys recalculated rates postulated by a number of scholars working to understand Spanish influenza mortality. When the death rate for Toronto is compared to these available

[69] These figures include deaths from all causes for the four month period that comprises the second wave of the epidemic (September through December). The report also states that for the four months represented influenza and pneumonia (all forms) accounted for 77.8% of all deaths (Rogers 1920: 7).

data on global mortality it becomes apparent that the death rate per 1000 was much lower in Toronto than was the norm across the globe.

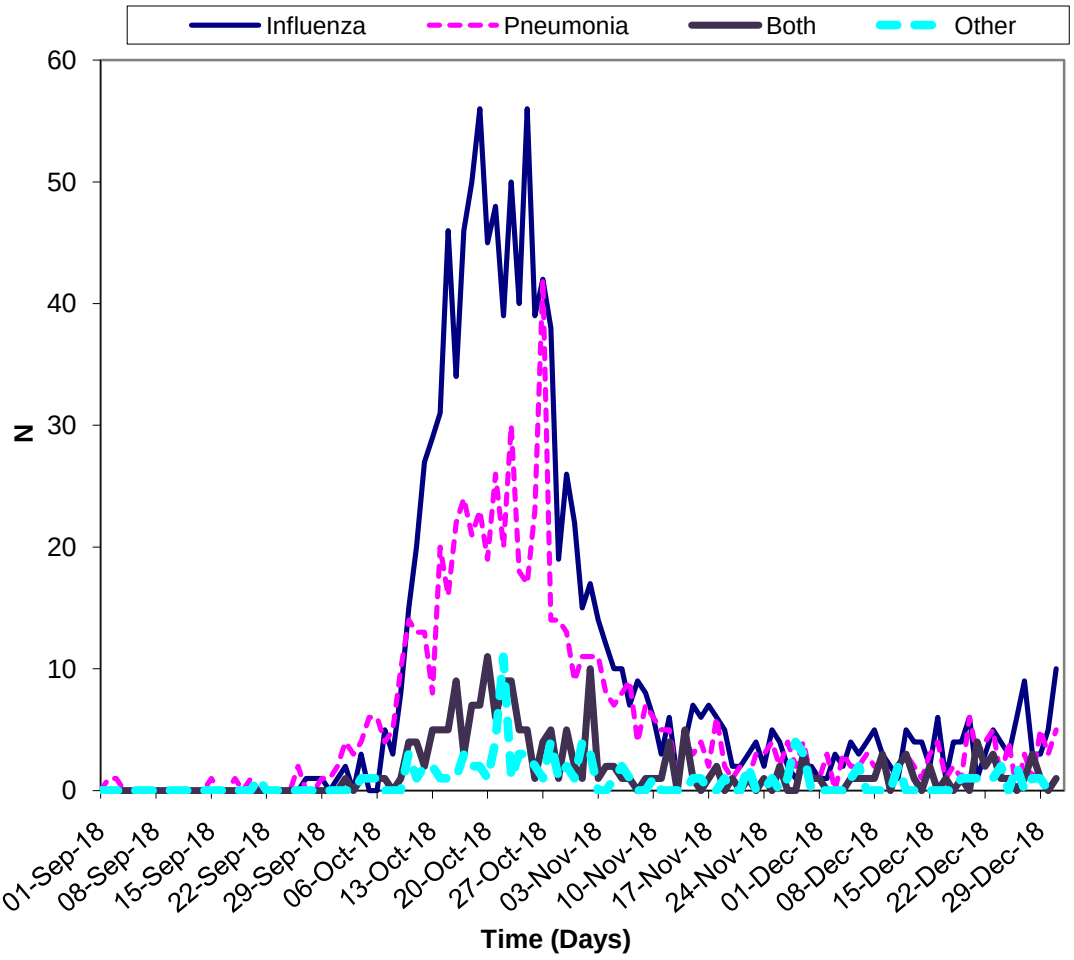


Figure 5.3 Disease Causing Death for Compiled Toronto Data, 1 September 1 to 31 December 1918

Figure 5.3 represents the listed disease causing death (DCD) for the study period of 1 September to 31 December 1918[70]. This graph demonstrates that the majority of deaths listed between 11 October and 26 October are attributed to influenza.

[70] Pandemic deaths or mortality associated with the pandemic refers to deaths attributable to influenza (or an influenza derivative) and or pneumonia (or a pneumonia derivative) extracted from the Toronto death registry. For a complete discussion see chapter 4.

Figure 5.4 examines physicians' listed immediate cause of death (ICD)[71] by day for 1 September to 31 December, 1918. The striking feature here is that that the majority of physicians listed pneumonia as the immediate cause of death when reporting pandemic mortality. Individually, these two graphs tell a very interesting story about influenza mortality during the second wave of the epidemic, but when the two leading causes of death (pneumonia and influenza) are compared a very interesting pattern emerges, as is illustrated in Figure 5.5.

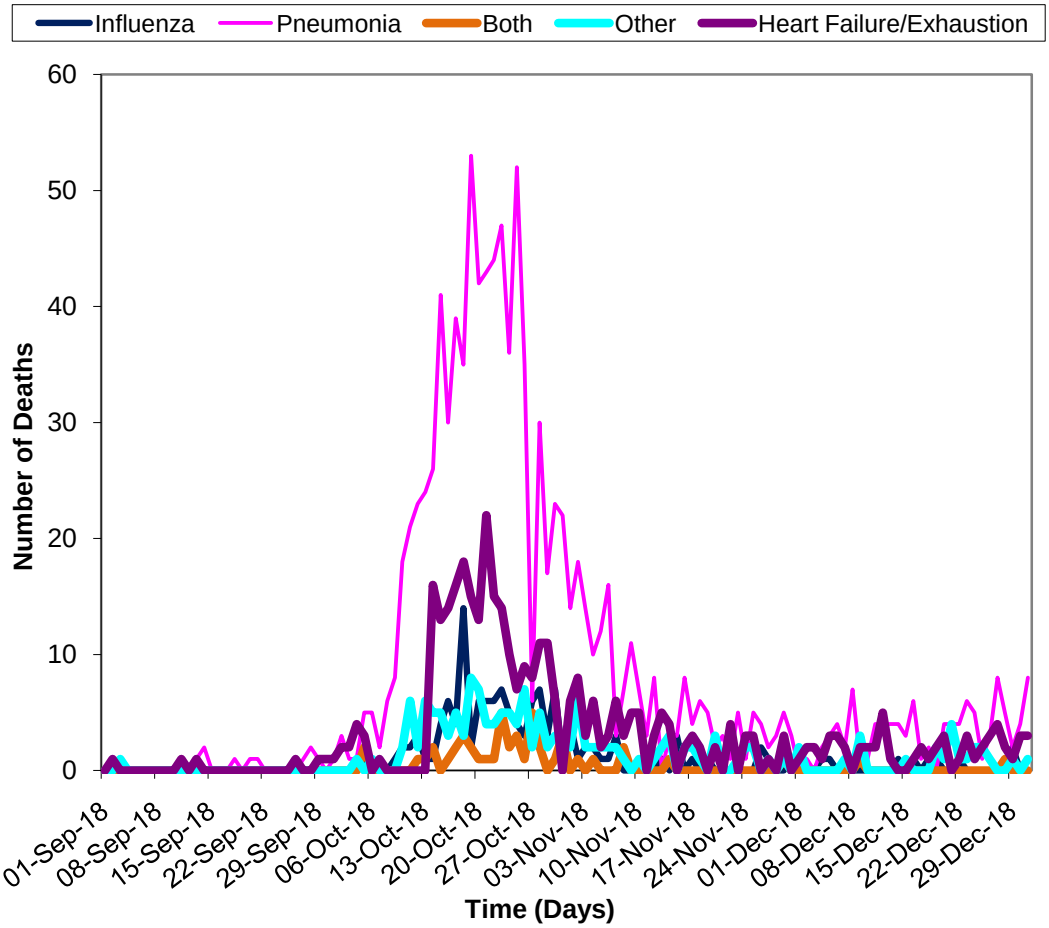


Figure 5.4 Immediate Cause of Death for Compiled Toronto Data, 1 September 1918 to 31 December 1918

[71] Immediate cause of death is in reference to the malady that is responsible for mortality, where as disease causing death often denotes the underlying condition that may have contributed to the passing of an individual. For example in an HIV positive individual, AIDS would represent the disease causing death and pneumonia may be listed as the immediate cause of death.

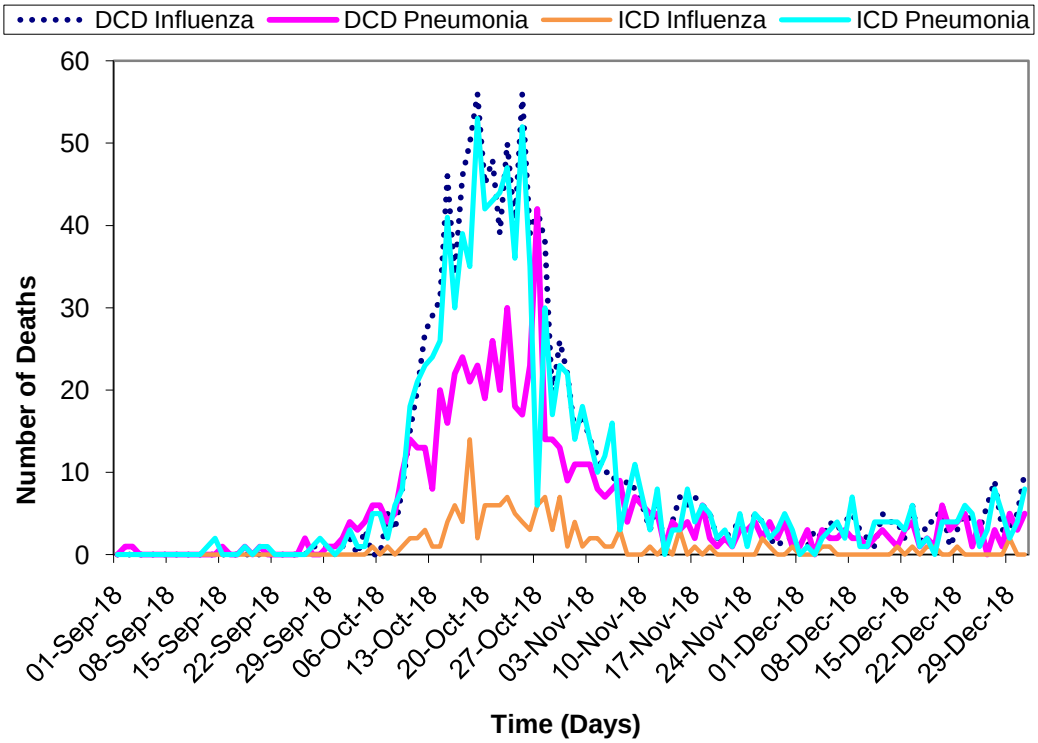


Figure 5.5 Comparison of Disease Causing Death and Immediate Cause of Death for Compiled Data, Toronto, 1 September to 31 December 1918

Figure 5.5 compares physician reported disease causing death (DCD) to immediate cause of death (ICD) for pandemic mortality extracted from the registry from 1 September to 31 December 1918. This graph demonstrates a relationship between physicians recognizing pneumonia (or a pneumonia derivative) as the disease causing death and influenza (or an influenza derivative) as the immediate cause of death suggesting that medical professionals viewed the two conditions as related. Moreover, it suggests that infection with influenza increased one's chances of contracting and succumbing to pneumonia infection.

There are several explanations for this trend. The first is that it was a construction of how medical personnel conceptualized the epidemic experience. According to the ethnomedical approach within anthropology detailed by Brown et al.

(2010), all medical systems are comprised by three integrated elements. The first is that there is a theory which underlies the causation of disease, the second factor is that there is a method of diagnosing a patient which is reliant on this etiological framework and the third notion is that a course of treatment will be developed that is in line with the resulting diagnosis. In the case of the Spanish flu epidemic the prevailing medical opinion was that the initial disease was caused by the influenza bacillus and if the appropriate measures were not taken the disease would advance to pneumonia (which was considered much more lethal). Patients would be diagnosed accordingly (newly infected individuals would be identified as suffering from influenza and individuals battling the pathogen would be listed as infected with pneumonia) and treatment would be contingent on immediate bed rest to halt the progression of the disease.

There is evidence in the historical record that suggests this was how medical authorities culturally constructed the disease experience. Dr. Charles Hastings, Minister of Health, made no effort to hide his belief that the natural progression of the pathogen was from influenza to pneumonia. Placing much of the onus on the infected individual, Hastings informed the public, "The great danger is from pneumonia. Avoid it by staying in bed while actually ill and until convalescence is fully established. The after effects of influenza are worse that the disease" (*The Globe* October 8, 1918: 6). One interpretation of this attitude is that the actual biological causative agent is less important than the social tool it represents. By linking influenza and pneumonia, physicians could persuade patients to stay in bed and thereby restrict the spread of the pathogen.

It is equally possible that this natural progression of the virus was not a cultural construct but in fact a biological reality. Syndemics theory challenges researchers to observe pathogen interaction as a component of the disease experience. The relationship between influenza and subsequent pneumonia infection is well documented (see for example Klugman et al. 2009, Sun and Metzger 2008, and Palacios et al. 2009). It is believed that influenza A virus infection promotes the growth of other bacterial infections that enter through the respiratory route (Hartshorn 2010). Numerous studies suggest a variety of ways in which influenza facilitates this apparent secondary infection by pneumonia bacteria.

The most common of these ideas is that the influenza virus has the ability to cause a change in the respiratory system, increasing individual vulnerability to subsequent infection[72]. This idea is not new and had been discussed in detail long before the epidemic. In 1803, following an influenza epidemic, Laennec observed an increase in the prevalence of pneumonia and postulated that the two conditions were somehow related (McCullers 2004: 519). In his official report on the influenza epidemic, Parsons (1919) stated that influenza was a rather benign acute infection in which the respiratory tract was the main point of attack. He further stated that "as a result of the infection, the other organs, kidneys, spleen and heart muscles show great degenerative or inflammatory changes" (Parsons 1919: 354) leaving victims susceptible to much more dangerous secondary infections. Jordan (1927) outlined the

[72] It is important to note that his relationship does not simply relate to pneumonia co-infection. Studies have also demonstrated correlations between *Streptococcus pneumonia and Staphylococcus aureus* (Karlstrőm et al. 2009), *Haemophilus influenzae* (Lee et al. 2010) and tuberculosis (Noymer 2010) susceptibility.

popular academic belief of the time regarding how influenza-related pneumonia was produced. According to his study,

> (1) The influenza virus weakens the resistant power of the pulmonary tissues so that various bacteria are able to play the role of secondary invaders; (2) the precise nature of the secondary – and tertiary – invaders is largely a matter of accident, dependent on the occurrence of particular bacteria in the respiratory tract of persons at the time of infection, and in the case of group outbreaks, on their occurrence in contacts; (3) the character of the resulting pneumonia, clinical and pathologic, is largely determined by the nature of the secondary invaders, whether Pfeiffer bacillus, streptococcus, pneumococcus, or other organisms; (4) there seems little doubt that the influenza virus, besides depressing the general pulmonary resistance, also acts directly on the pulmonary tissues, causing capillary necrosis, edema, and hemorrhage; (5) it seems to be true, therefore, that the fatal outcome of influenza pneumonia is determined partly by the degree to which the influenza virus[73] depresses local and general pulmonary resistance, and partly by the virulence and nature of the bacteria which invade the tissues in the wake of the specific virus (Jordan 1927: 271).

Studies from the 1980s and 1990s demonstrated that influenza infection increases the adherence of bacteria to epithelial cells within the respiratory system (Fainstein et al. 1980; Wadowsky 1995). This hypothesis is supported by analyses of 58 tissue specimens collected during autopsy from Spanish flu victims and 8398 postmortem examinations (Morens et al. 2008). Morens et al. suggest that influenza A infection working in concert with other bacterial infections led to the majority of deaths during the 1918-1919 pandemic (Morens et al. 2008). This lethal synergy clinically manifested itself in the following ways:

> (1) the high incidence of secondary pneumonia associated with standard bacterial pneumopathogens; (2) the frequency of pneumonia caused by both mixed pneumopathogens (particularly pneumococci and streptococci) and by other mixed upper respiratory-tract bacteria; (3) the aggressiveness of bacterial invasion of the lung, often resulting in "phenomenal" numbers of bacteria and polymorphonuclear neutrophils, as well as extensive necrosis, vasculitis, and hemorrhage; and (4) the

[73] The causative agent of influenza was not identified as a virus until 1933.

> predominance of bronchopneumonia and lobular pneumonia, as opposed to lobar pneumonia, consistent with diffuse predisposing bronchiolar damage. (Morens et al. 2008: 963)

These pathological findings demonstrate that the influenza virus responsible for the 1918 epidemic led to higher pneumonia morbidity and increased mortality due to its ability to damage bronchial and/or bronchiolar epithelial cells.

One of the most recent areas of research attributes this damage to the study of an aberrant innate immune response. Animal studies on mice (Kash et al. 2006) and monkeys (Kobasa et al. 2007) infected with the 1918 influenza strain have shown excessive stimulation of the innate immune system, resulting in severe damage to lung tissue. It is postulated that damage increases both influenza mortality as well as subsequent bacterial infections. These findings are further supported by the increased mortality in individuals aged 20 – 40 during the pandemic, as individuals in this age range were more likely to be in good health and therefore to have a more robust immune system.

These findings have tremendous clinical significance. According to McCullers (2004: 519) after the advent of antimicrobials the case fatality ratio for *Streptococcus pneumoniae* fell from 30% to 5% but when bacterial pneumonia followed influenza infection the effectiveness of antimicrobials was negligible and the case mortality rate was increased (37% - 50%). Demographically these findings also aid researchers interested in recreating the pandemic experience as they provide further evidence for the aggregation of influenza and pneumonia deaths in the reconstruction of Spanish flu mortality.

Age Distribution of Pandemic Mortality in Toronto

Figure 5.6 and Table 5.5 examine the age and sex distribution of mortality for the second wave of the pandemic in Toronto (1 September to 31 December 1918). Overall the mortality was higher in males than females, with males experiencing 4.42 deaths per thousand as opposed to the female rate of 3.71 per thousand.[74] For both sexes the highest mortality rate was experienced in individuals between the ages of 20 to 39 years of age (see Table 5.5). This over representation of mortality in individuals in the prime of life is one of the defining characteristics of the strain that was responsible for the 1918 – 1919 pandemic (see, for example, Osborne 1977, Crosby 1989, Herring 1994, Phillips and Killingray 2003), resulting in epidemic curves that displayed a characteristic 'W' shaped mortality profile.[75]

There also appeared to be a slight clustering of deaths in persons aged one to four. As had been demonstrated in other studies (for example, see Beveridge 1977) mortality from the pandemic also sometimes resulted in an increase in infant and perinatal mortality. Beveridge states that in 1918-19 a study was conducted that looked at 1350 pregnant women who suffered pandemic influenza: abortion, stillbirth or premature labor occurred in 26 per cent of women without pneumonia and 52 per cent of those with pneumonia sequella. This was not a feature that was expressed

[74] The mortality rate was calculated using the 1921 Canadian census. In 1921, there were 250,941 males and 270,949 females in Toronto. During the four months captured in this study 1100 males and 1016 females died of pneumonia and/or influenza according to the information collected from the death registry.

[75] The normal distribution of seasonal influenza mortality is a 'U' shape with deaths clustering in the very young and the very old. The excess mortality in 20-40 year-olds created an additional peak, thereby transforming the traditional 'U' into a 'W'.

either in the 1957 – 58 or 1968- 69 pandemics (Beveridge 1977). According to Hallman (2009: 94), although the epidemic was responsible for an increase in the infant mortality rate in Toronto this "increase was not statistically significant [as it] fell within the range of the random yearly variation."

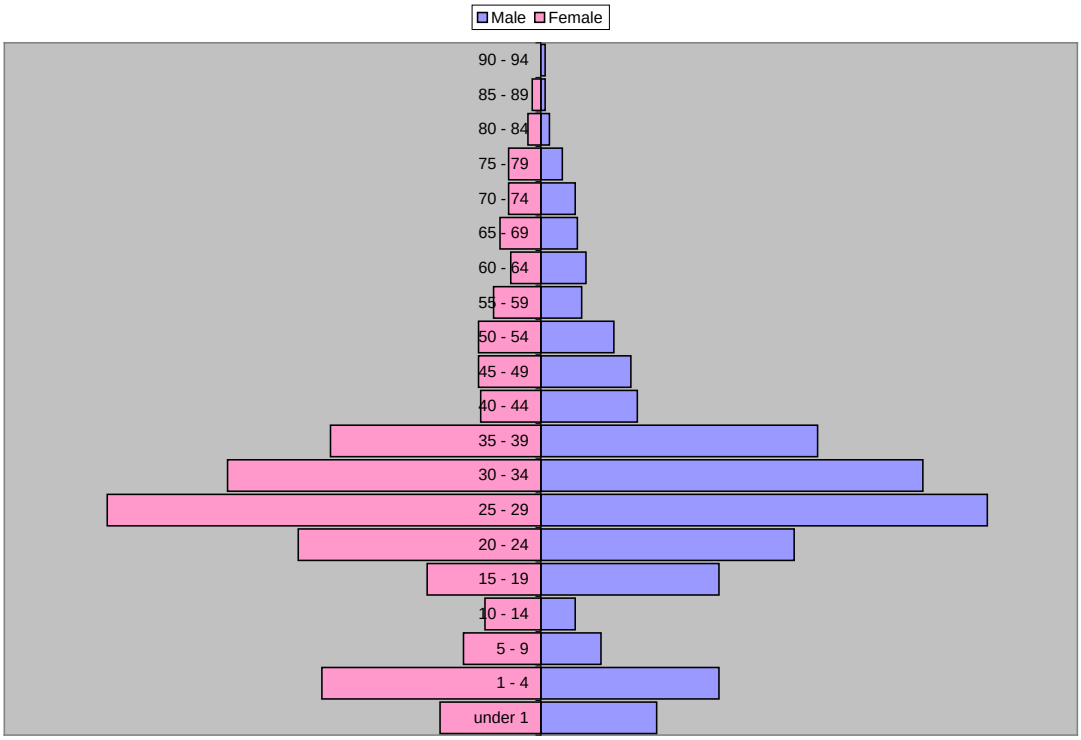


Figure 5.6 Population Pyramid Displaying Mortality in Toronto from 1 September to 31 December 1918

Finally, individuals aged 55 – 94 appear to demonstrate negative excess mortality[76] comprising a mere 91 deaths (8%) among males and 95 deaths (9%) among females. Annual seasonal influenza mortality data collected in Canada between the years of 1990-1999 demonstrated that for children and young adults the number of influenza deaths was quite small. Schanzer et al. (2007) further report that

[76]The negative excess mortality rate indicates that there were fewer deaths in this age category than is usually experienced during a typical experience with this virus.

over 90% of influenza deaths examined in this ten-year period occurred in individuals over the age of 50.

Table 5.5 Male and Female Proportionate Mortality for Spanish Influenza in Toronto 1 September to 31 December 1918 in 5-year age intervals

Age	Male	Male Proportionate Mortality	Female	Female Proportionate mortality
under 1	54	5%	47	5%
1 – 4	83	7%	102	10%
5 – 9	28	3%	36	4%
10 – 14	16	1%	26	3%
15 – 19	83	7%	53	5%
20 – 24	118	**11%**	113	**11%**
25 – 29	208	**19%**	202	**20%**
30 – 34	178	**16%**	146	**15%**
35 – 39	129	**12%**	98	**10%**
40 – 44	45	4%	28	3%
45 – 49	42	4%	29	3%
50 – 54	34	3%	29	3%
55 – 59	19	2%	22	2%
60 – 64	21	2%	14	1%
65 – 69	17	2%	19	2%
70 – 74	16	1%	15	1%
75 – 79	10	1%	15	1%
80 – 84	4	0%	6	1%
85 – 89	2	0%	4	0%
90 – 94	2	0%	0	0%
TOTAL	1109	1	1004	1

Sex Distribution of Pandemic Mortality in Toronto

According to Jordan (1927) the case incidence rate for Spanish flu was slightly higher among females than males. Using data from the United States Public Health Service and published reports from England and Wales, Jordan demonstrated that this difference ranged from attack rates of 230 per thousand for boys and 231 per thousand for girls[77] to 155 per thousand for males and 274 per thousand for females.[78] Death

[77] Data from a study by Jordan, Reid and Fink (1919) examining pupils of a Chicago school.
[78] These data were derived from a study by Billings and Wynne (1920) looking at sex differentials among employees of the Eastern Group Telephone Company located in and around New York and Philadelphia.

rates appear to be higher in males than among females, however Jordan (1927) reported that males had a higher death rate at the age groups of 15-19 and 29-44 than did females of the corresponding ages. On the other hand, death rates of females were higher than those of males in the age groups 4 – 14 and 20 – 28. A chi square analysis was performed in order to determine if there was a statistically significant difference in the distribution of deaths by age between males and females in the Toronto data (Table 5.6). For ease of calculation age groups were placed into 10 year age intervals.

Table 5.6 Chi Square Results Examining Differences in Spanish Influenza Mortality in Toronto Based on Age (in Ten-year Intervals) and Sex

Sex	Age	Observed	Expected	Sex	Age	Observed	Expected
Male	Under 1	54	53.01	Female	Under 1	47	47.99
	1 – 9	111	130.69		1 – 9	138	118.31
	10 - 19	99	93.42		10 - 19	79	84.58
	20 - 29	326	336.43		20 - 29	315	304.57
	30 - 39	307	289.19		30 - 39	244	261.81
	40 - 49	87	75.58		40 - 49	57	68.42
	50 - 59	53	54.58		50 - 59	51	49.42
	60 - 69	38	37.26		60 - 69	33	33.74
	70 - 79	26	29.39		70 - 79	30	26.61
	80 - 89	6	8.40		80 - 89	10	7.60
	90 - 100	2	1.05		90 - 100	0	0.95
			Chi-Square Value = 17.8				
			Critical Value (0.05, 10) = 18.3				

Results from the chi square analysis (Table 5.6) show that there is no difference in the age distribution of influenza deaths by sex ($x^2 = 17.8$, df = 10).

Table 5.7 Sex Specific Death Rate for Toronto Spanish Influenza Mortality, 1 September to 31 December 1918

	Deaths	Population*	Death Rate
Male	1110	250941	0.44
Female	1007	270949	0.37

***Population established from 1921 census**

Finally, Noymer and Garenne (2000) demonstrate a male bias in the age and sex-standardized death rate in the United States of 174 per 100, 000.[79] Although the difference is not as marked in Toronto (experiencing 71 per 100,000) this overrepresentation of male mortality holds true for the city. It is however important to note that if soldiers are removed from the male mortality figures (Table 5.8) females and males experienced similar proportionate mortality.

Table 5.8 Sex specific death rate for Toronto Spanish flu mortality, 1 September to 31 December 1918. Soldiers removed.

	Deaths	Population*	Death Rate
Male (soldiers removed)	924	250941	0.37
Female	1007	270949	0.37

***Population established from 1912 census**

Oertel (1918), studying the pandemic experience in Montreal, suggested males were the first to be admitted to hospitals and subsequently perish from the disease. He states

> it is of some interest to observe that there is a marked change in the type of admission, for from the 27th of September to the 15th of October, autopsies were performed on twenty-two males and one female only, the twenty-two males being mostly soldiers; while from October 15th to November 21st, there were autopsies on thirteen females and six males. (Oertel 1918: 399).

To test if this dataset provides support for this observation, i.e., that there was a difference in the clustering of male and female deaths, mortality was plotted by day and by sex (Figure 5.7). The blue line represents male deaths and the pink female mortality. This graph demonstrates that although male mortality was

[79] This statistic was calculated by using the male minus female difference in the age-standardized death rate for deaths attributable to flu or pneumonia.

somewhat higher at the onset of the pandemic in Toronto this difference was negligible and short in duration.

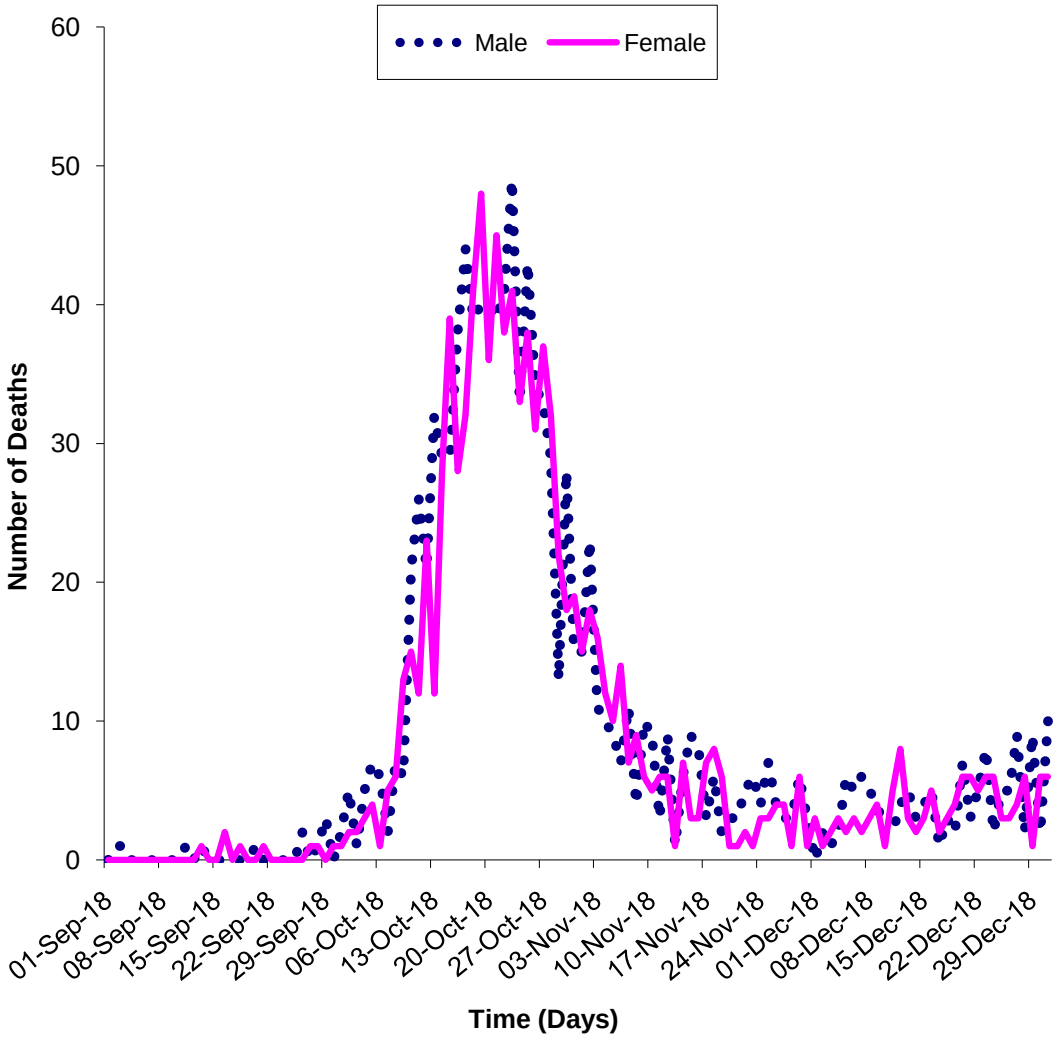


Figure 5.7 Influenza and Pneumonia Deaths for the City of Toronto 1 September to 31 December 1918 by Sex

During death registry transcription it was apparent that individuals appeared to perish in age clusters. To test this hypothesis, the age specific mortality was analyzed by day for the crest of the second wave of the epidemic in Toronto (9 October to 2 November 1918). The proportionate mortality was calculated for each 10 year age

category for each day (Figure 5.8). This figure clearly demonstrates that for the height of the epidemic individuals in the 20-29 and 30-39 constituted the majority of the mortality, with very few individuals over the age of 50 dying as a result of recognized influenza/pneumonia infection.

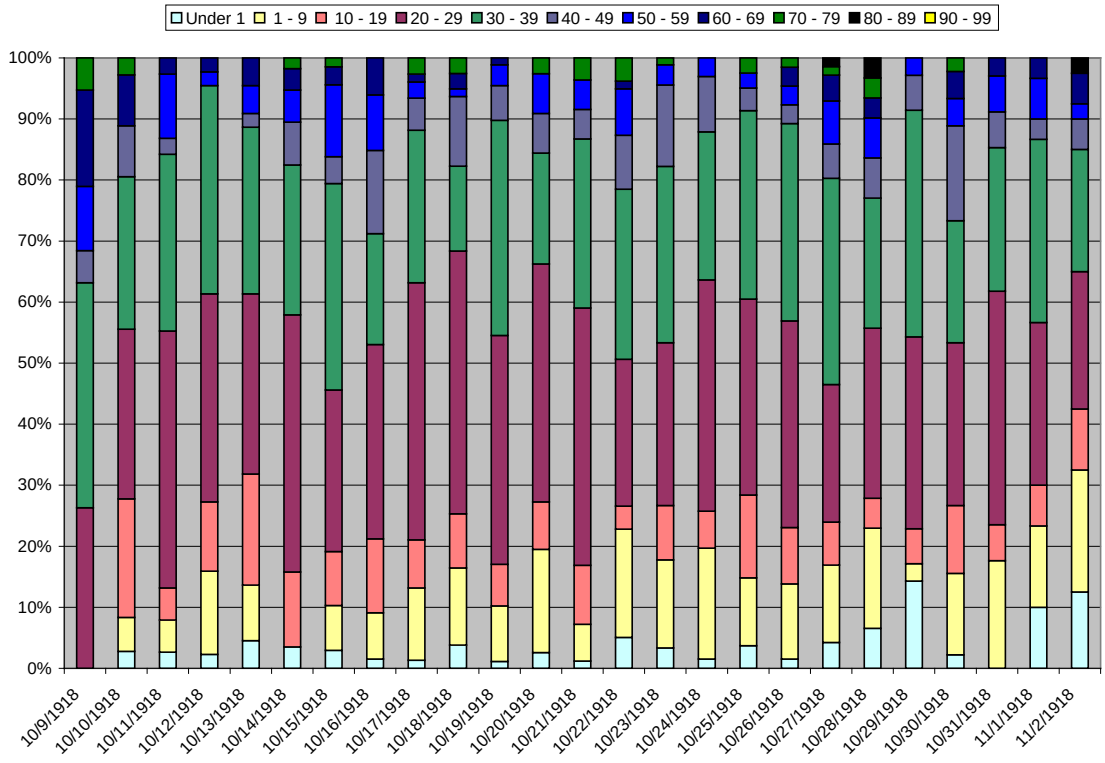


Figure 5.8 Proportionate Mortality by Age in Ten-year Intervals for the Crest of the Second Wave of the Spanish Flu Epidemic, Toronto, 9 October to 2 November 1918

Occupation and Ethnicity

There is quite a bit of debate as to the social neutrality of influenza. Many early authors postulated that the virus was the great equalizer, selecting its victims at random, whereas more recent work suggests that social class and ethnicity should be considered as contributing factors when examining influenza mortality. Mamelund

(2006) and Herring and Korol (2010) examined the relationship between mortality and social class. Mamelund (2006) concentrated his study on two socially contrasting parishes from the Norwegian capital of Kristiania (now Oslo). Utilizing census and registry data Mamelund demonstrated that influenza mortality was 19-25 per cent higher among the working class, a finding which was not statistically significant, and on average the mortality of individuals residing in apartments with four to six rooms was 50 per cent lower than those occupying one room apartments, a finding which was statistically significant. His contention was that this represented an effect of increased wealth as people living in larger apartments were presumably wealthier. Herring and Korol (2010) utilized public records available for Hamilton, Ontario in conjunction with GIS to plot the spatial distribution of influenza mortality. Their analysis demonstrated that people living within lower socioeconomic areas (linked to industrial/working-class areas) in the northern wards of Hamilton had higher mortality rates then individuals residing in the more prosperous southern wards.

The registry data available for Toronto were not conducive to spatial analysis. Although informant information was recorded, in the majority of instances this entry provided details pertaining to the choice of undertaker. Where individual contact information was provided establishing the relationship between the informant and the deceased was not always clear, making inference to cohabitation exceedingly difficult. Although other nominative sources could be searched (i.e. city directories) to ascertain individuals' residences, the number of deaths (2117) included within this analysis is prohibitive at this time. The closest measure to socioeconomic status therefore becomes occupation.

Table 5.9 Pandemic Mortality by Occupation, Toronto, 1 September to 31 December 1918.

Occupation	Recorded Deaths	Population at Risk	Mortality Rate*
Agriculture	15	1292	11.61
Manufacturers	131	66709	1.96
Construction	159	16886	9.42
Transportation	71	21272	3.34
Trade	36	42009	0.86
Finance	25	9965	2.51
Service	293	52059	5.63

Table 5.9 examines the pandemic mortality by occupation. It is important to note that of the 2117 individuals included in this portion of the study only 891 (43%) had an occupation listed. Of those, only 730 could be included in this analysis as 161 had occupations listed that were too general for inclusion or did not fit within categories provided within the census data.[80] Specific occupations were listed on the death registry and placed into categories utilizing the 1921 Census for Toronto (4: 532 - 554). From the listed occupations the highest mortality appears to have been experienced within construction workers and agriculturalists. It is very difficult to ascertain why these two groups may have had a higher mortality rate, although an earlier quote from Hastings may provide some clues. He associates fully establishing convalescence with a reduced likelihood of negative disease progression. Agriculturalists and manufacturers, due to the demands of war and the general population, would have been less able to fully convalesce prior to returning to work.

Census data were also used to calculate the average weekly income for each employment group.[81] Table 40 (Sixth Census of Canada 1921 Volume 3) reports

[80] 50 were listed as clerks, 30 as students, 27 as housewives, 22 as retired, 15 as managers, 10 as travelers, 9 as lady, 5 as gentlemen, 3 as inmates and 3 which could not be identified.
[81] It is essential to note that this is a vague approximation of individual income as occupations and salaries within these groups are highly variable. For example the service group ranges from professors and lecturers ($66.29 a week) to waiters ($14.11 a week).

wage earners 10 years of age and over by age groups, weeks employed and earnings for the census year in cities of 30,000 and over. For each occupation and group (Agriculture, manufacturers, construction, transportation, trade, finance and service) the total number of persons employed and their collective earnings are reported.

Table 5.10 Weekly earnings by occupation, Toronto 1921

Occupation	Persons	Earnings	Weekly Income
Agriculture	860	655,111	14.65
Manufactures	39,323	46,688,013	22.83
Construction	12,366	13,073,035	20.33
Transportation	13,376	17,373,489	24.98
Trade	20,344	29,569,566	27.95
Finance	11,321	15,765,172	26.78
Service	23,211	32,025,892	26.53

Source: Sixth Census of Canada Volume 3 1921

The earnings were divided by the number of persons employed and then divided by 52 to establish a weekly income (Table 5.10). These data suggest that socioeconomic status may have been a contributing factor to mortality as overall agriculturalist and individuals employed in the area of construction appear to have the lowest weekly income. Further analysis is beyond the scope of this dissertation and will have to await future research.

Available archival material suggests a correlation between occupation and risk of infection. In the October report for the Department of Public Health (1918:3), Hastings states:

> A large number of physicians and nurses, in this as in all epidemics, by reason of their more or less constant exposure to infection and their lowered vitality in their efforts to meet the innumerable demands made upon them fall victims. During the epidemic, in the United States, over one hundred physicians a week have fallen victims to the disease, and probably a corresponding number of nurses. The layman may, if he profits by the information given him, keep from exposing himself to infection, but the physicians and nurses must go when called, without thought of the consequences to themselves.

Articles in the Star and Globe during the early stages of the epidemic also single out soldiers and teachers as being at an increased risk of infection. A Star article dated 7 October 1918 carries with it the header "Toronto Teachers Attacked and Schoolrooms Have to Close" the article goes on to state that "indications point to a serious outbreak of influenza among Toronto teachers, more particularly in the east end of the city" (The Star 7 October 1918: 2). Numerous early reports[82] also discuss the severity of the epidemic at army camps. In an attempt identify if any individual occupations presented a greater risk for mortality death registries which listed occupation were searched and the top ten occupations which suffered the highest number of deaths were extracted (Table 5.11).

Table 5.11 Top Ten Occupations for Influenza/Pneumonia Mortality, Toronto, 1 September to 31 December 1918

Occupation	Deaths	Occupation	Deaths
Soldier	131	Nurse	18
Clerk	50	Carpenter	17
Student	30	Machinist	16
Housewife	27	Merchant	16
Retired	22	Manager	15

This list is suggestive that population aggregation or contact with sick individuals may have increased the likelihood of mortality (and in all probability morbidity). Soldiers, clerks and school children appear to have been at higher risk (most likely due to the conditions of the 'workplace'). One of the salient features of person-to-person transmitted disease is the risk they pose to care givers. Women of all walks of life were called upon to volunteer their time and efforts to care for the sick. It is not unlikely that a large number of housewives stepped up to this challenge, putting

[82] See for example The Globe 3 October 1918:6; The Star 4 October 1918:9; The Star 28 September 1918:2.

them at the same risk for infection as trained nurses.[83] However it is also possible that these occupations may have suffered higher mortalities due to increased population size[84] or as a byproduct of belonging to a high risk group.[85]

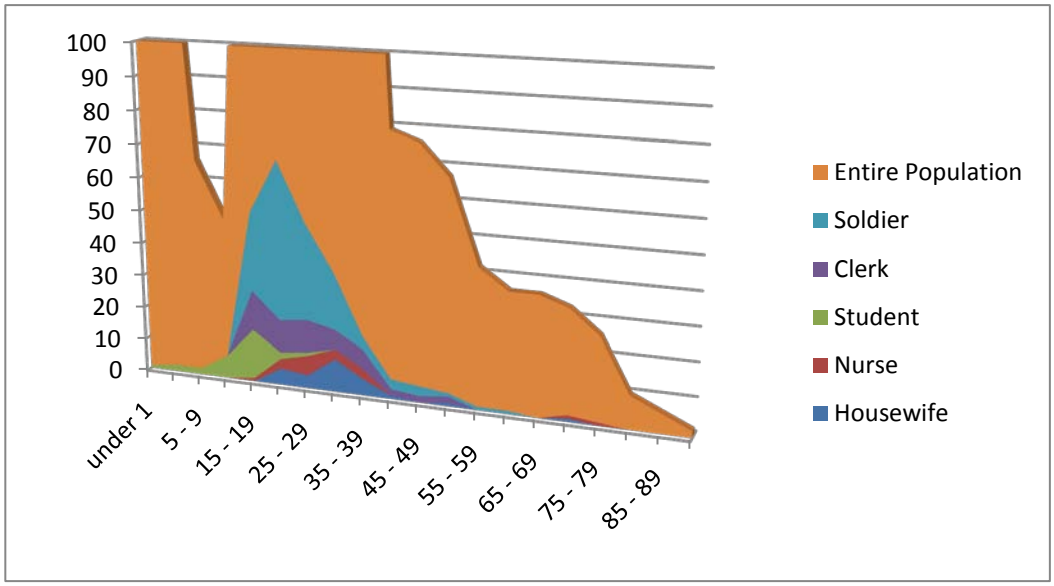


Figure 5.9 Six Occupations With the Highest Mortality, by Age

Figure 5.9 examines the age distribution for the six occupations that had the highest rates of mortality. Although not conclusive (as populations at risk cannot be established), it is possible that these occupations may be overrepresented due to the age of individuals and not their means of subsistence.

[83] The choice to include nurses and not doctors is quite purposeful. This decision is not intended to downplay the significant contributions of doctors, but rather to highlight the hands on, and inherently more dangerous work of nurses. According to Helman (2007: 162) a nurse's job involves intimate contact with the surface of the patient's body and its various waste products whereas a physician's focus is usually associated with an intimate knowledge of the inner workings of the body and therefore lessens the amount and duration of physical contact a physician has with his or her patients.

[84] For example there were many more soldiers in Toronto as a result of large centralized army camps located in the vicinity of the city. As has already been indicated large numbers of military men were brought to the city for care.

[85] Individuals between the ages of 20-40 were at increased risk of dying from infection with influenza.

Table 5.12 Pandemic Deaths by Country of Origin, Toronto, 1 September to 31 December 31 1918

Country	Number of Fatalities	Population at Risk	Deaths Per 1000
Austria	13	1175	11.06
Canada	1175	226365	5.19
China/Japan	16	2176	7.35
England	360	260860	1.38
Ireland	69	97361	0.71
Italy	44	8217	5.35
Russia	67	1332	50.30
Scotland	113	83620	1.35
USA	61	N/A	

Table 5.12 explores influenza and pneumonia deaths that occurred in Toronto during the second wave of the pandemic by nationality. Population at risk estimates were derived from the 1921 census (Table 15 – Population, Canadian, British and Foreign Born, Classified by Sex for Dominion Electoral Districts [Canada] and Table 27 – Population Classified according to Principal Origins of the People by Countries and their subdivisions). Only countries in which more than 10 individuals were born were included in this analysis. Russia (50.3 per 1000) and Austria (11.1 per 1000) demonstrated the highest proportionate mortality, followed by China/Japan[86] (7.4 per 1000), and Italy (5.4 per 1000). There were no available statistics for the number of individuals born in the United States so a death rate could not be calculated. The overall mortality of Canadian born individuals was 5.2 per 1000. The majority were born in Ontario (n = 822; 527 were native Torontonians). Three hundred thirty of the remaining 389 were listed as having been born in Canada, 24 individuals hailed from Quebec, 16 from Newfoundland, 7 from Manitoba, 6 from Nova Scotia, and 2 each from Prince Edward Island, Saskatchewan and British Columbia.

[86] 15 individuals were listed from China and 1 from Japan but the 1921 census lumped the two countries so in order to obtain an accurate population at risk the countries were joined for this analysis.

This chapter suggests that influenza mortality was not randomly distributed throughout the city. Deaths appeared to cluster in individuals in the prime of their lives, and socioeconomic status and ethnicity may have contributed to risk of infection and death. There also may have been a slight increase in male mortality within the city[87]. Population movement and aggregation seem to be contributing factors in loss of life as well. Finally, this chapter also exposes how cultural interpretations of a biological phenomenon can alter its reconstruction. Whether influenza and pneumonia are linked at a microscopic or macroscopic level, work in Toronto demonstrates that the only way to accurately describe the pandemic experience within the city is to include both influenza and pneumonia deaths within reconstructions. The next chapter takes the importance of cultural construction to the next level by examine how the disease experience was framed within the city through existing ideologies, social structure, individual identities and institutional structures.

[87] It is important to note that there is no difference if soldiers are removed from the male population.

Chapter 6 Framing Flu

The previous chapter presented the epidemiological features of the experience of the second wave of the epidemic in Toronto. This next chapter aims to frame the pandemic experience of the city by shifting from a quantitative structure to a qualitative one. It begins with a brief narrative of the course of the Spanish flu in Toronto; from there aspects of the pandemic experience are highlighted using the framework provided by Rosenberg (1986, 1989,1989b; Rosenberg and Golden 1992) who states that there is a universality by which humans make sense and respond to epidemics.

Just the Facts: Using the Printed Word to Reconstruct the Pandemic Experience in Toronto

When Spanish flu arrived in Toronto it came as no surprise. Outbreaks in the United States were well publicized and the pathogen was already circulating close to home. In the last few weeks of September Spanish flu was reported among military men at the Polish military camp at Niagara on the Lake (The Globe News September 28:7) and on 23 September five deaths had already been recorded at this camp (*The Star* September 23: 4). Although at this time there are no cases of influenza reported at the Canadian camp at Niagara, officials were calling for the relocation of troops to Exhibition Camp (located on the south east boundary of the city of Toronto) to inhibit the spread of the disease. By 28 September flu cases were beginning to be reported among Canadian troops and by 4 October, 610 soldiers at Base Camp at Niagara-on-the-Lake were infected with the virus. It was also on this date that 50 soldiers were

transported from this camp to Military Ward M of the Toronto General Hospital (or Base Hospital) to be kept in quarantine and receive treatment (*The Star* October 4: 9). By 7 October cases at Base Hospital appeared to be abating as military authorities reported that their influenza cases had dropped from 640 to 500 (*The Star* October 7: 2).

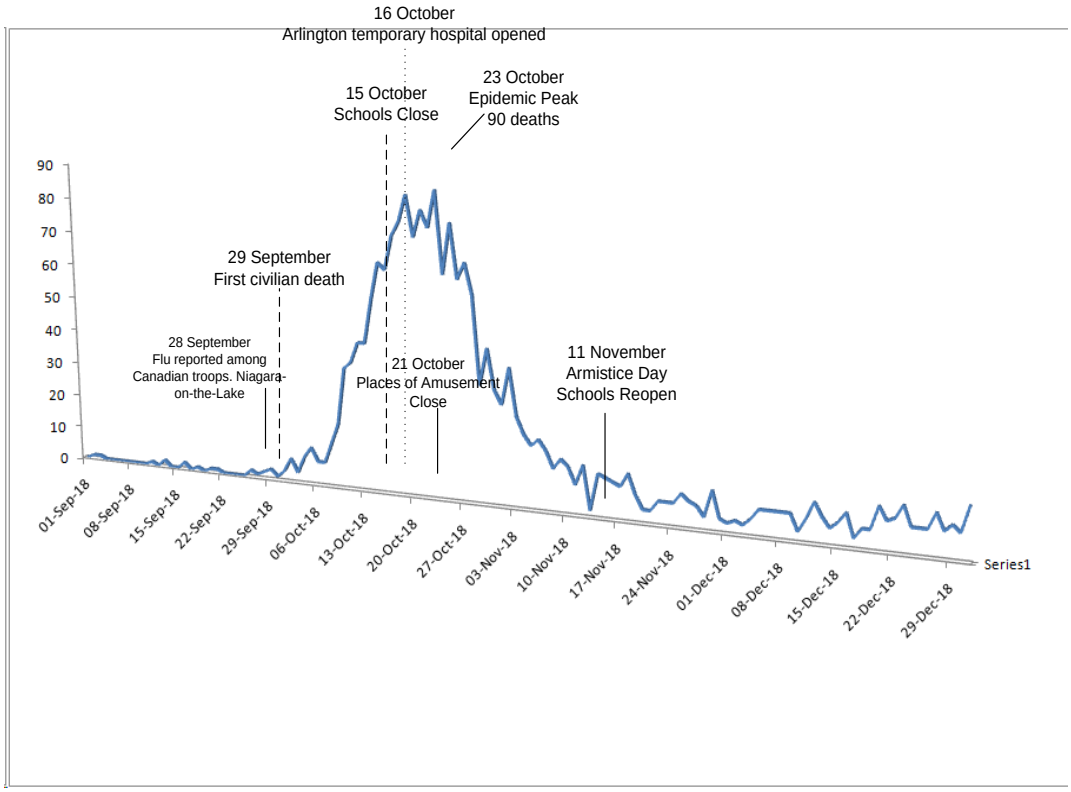


Figure 6.1 Spanish Influenza Time-Line, Toronto 1 September to 31 December 1918

Early reports (from 26 September to 5 October) of the pandemic's activity from around the city suggest that the city's residents were dealing with nothing more than the 'normal flu'. Although hundreds were reported sick, the symptoms appeared to be mild in nature and the increase in virulence was attributed to unseasonably cold weather conditions. The first civilian death, according to *The Star* (Sept 30:2), was a

young girl who died at Toronto General Hospital on 29 September[88]. The article goes on to state that as a precaution, the child's school was quarantined. A detailed article in *The Toronto Star* from 7 October 1918 demonstrated the variability in opinion regarding the severity of the epidemic in Toronto as perceived by its medical community. Dr. Hastings (Medical Officer of Health) was adamant that the city was not dealing with an unusually virulent strain of influenza. He stated that

> There is only the ordinary sprinkling of cases throughout the city, and no need for alarm. There is no necessity for hysterics on the part of the public; it is that which helps to sap their vitality and exposes them to such epidemics. It is just ordinary grippe and no worse than we have had in previous years. (*The Star* October 5, 1918:23)

Dr. Hastings was so confident that the situation would not worsen in the coming days that on 5 October he traveled to New York and other major American cities to discuss which tactics these locations had employed to deal with the disease. Dr. M.B. Whyte, director of the Isolation Hospital, challenged Hastings' notions of benignantly, calling for the disease to be taken seriously and further attested that cases of influenza should be reported and diligently monitored. Mr. R.A. Shutt, in charge of the Division of Quarantine, reports that at this time no action had been taken to quarantine homes in which the virus was present.

By 8 October, these discussions became moot, for almost overnight the situation in Toronto had severely worsened. Within this sixty-eight hour period hospitals went from low occupancy to near full capacity with people suffering from

[88] According to information collected from the death registry, Vera Isabel Robinson, aged 12, died on September 28 at Toronto General Hospital. She was the first case in which the disease causing death was listed as influenza. Her immediate cause of death was listed as bronchopneumonia (Reel 240 jpeg 135 page number 458)

the disease. Early mortality associated with the epidemic displayed the odd characteristic of hopscotching through neighborhoods and communities leaving some families untouched while others were left virtually decimated.[89] By 11 October it is reported that Toronto hospitals were full beyond capacity and unable to take new patients (*The Globe* October 11:6). The situation appeared to have worsened within schools as well. Reports from *The Star* (October 11: 2-5) indicate that few schools were free from the disease, with many of the larger schools missing half their student body and teaching staff. According to the paper in the previous "year there were 7260 children absent while at present there are 13,172 or 5,912 more than last year" (*The Star* October 11, 1918: 5). In an attempt to contain the spread of the pathogen, on 12 October Dr. Hastings called for the cessation of all conventions and other large gatherings until the virus abated. He also issued an order that only immediate family members were permitted to attend the funeral of an individual who died as a result of the virus.

On 14 October, the Minister of Health announced his plans to equip two hotels, the Arlington and the Mossop, so that they could serve as temporary hospitals. The Arlington was to open for up to 100 patients by Wednesday, 16 October and the Mossop was to be ready by the week's end.[90] On Tuesday, 15 October all city schools were closed and talk began to surface about the closures of theaters, churches and other places of entertainment. By 21 October, theaters, picture shows, billiard rooms,

[89] On October 4, the flu claimed the lives of three family members (a mother and her two children). On October 8, sixteen deaths were reported, eight of which were reported from one family. Reporting from local schools also demonstrates this differential distribution. "Both Harbord and Parkdale Collegiate report about one hundred students absent yesterday. Humberside Collegiate is so far almost immune. Oakwood Collegiate has about sixty children absent with colds or ordinary grippe. The Technical High School noticed nothing abnormal in conditions yesterday." (*The Globe* October 10: 6)

[90] Public health officials were able to open the Arlington according to their time line but did not complete preparations on the Mossop until after the virus had abated.

and bowling alleys were ordered to close their doors and to guard against under registration of deaths. Dr. Hastings ordered that "no cemetery has authority to open a grave unless the death is registered" (*The Globe* October 19:8). Dr. Hastings also prohibited participation in outdoor sports (*The Star* October 18: 2). Although churches were never officially closed, citizens were urged not to attend public gatherings; consequently, Sunday services on October 20 were very poorly attended.

By 24 October, Dr. Hastings began to report to the populace that he believed Toronto had seen the worst of the epidemic. He cautioned citizens not to be dismayed at the fact that mortality may remain high and assured them that morbidity due to the disease was waning. On the 28 October, Dr. Hastings began to talk of reopening schools and amusement houses. On Friday, 1 November he issued a decree that public schools and places of amusement were to reopen on the following Monday (4 November) and that Sunday school and private boarding schools were to resume on 11 November. However, due to the number of teachers still recovering from the virus and engaged in care of others, public schools did not reopen until 11 November. Bans on public gatherings were also lifted on the 11 November as the city celebrated Germany's defeat. By 15 November Dr. Hastings had turned his attention to declaring Toronto's apparent success in defeating the virus, boasting fewer deaths than Buffalo, Boston and Montreal.

The day-to-day workings of the city were in no doubt impacted by the severity of the disease. The majority of civil servants were down sick. On the 23 October, 54 of the 319 employees of the Health Department were off sick, as were 22 of the 218 Parks Department staff, two of the five employees of the Mayor's Office, 60 of the

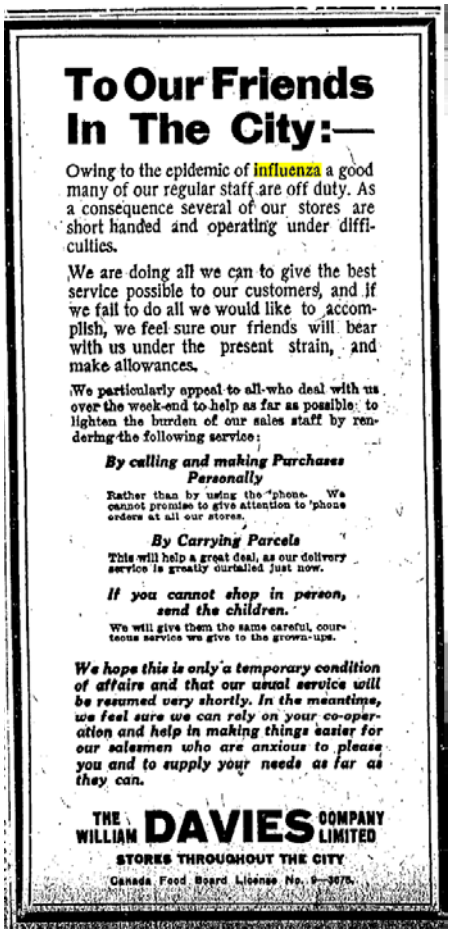
600 Street Cleaning Department employees and over 100 of the city's Works Department employees (*The Globe* Oct 23:6). Early reports from the police force indicated they were short fifteen officers (*The Star* October 7, 1918: 2), while local fire departments were down 30 men by 12 October and postal services were slowed by the absence of around 50 workers (The Globe October 12, 1918: 8). On 22 October the Grand Trunk terminal was forced to place an embargo on all freight going east, as 219 staff members were absent from work (McGinnis 1981: 464).

Keeping up with burials also appeared to be a challenge. Funerals took place seven days a week and extra men were hired to dig graves. According to *The Star* (Oct 28:2) "So acute is the shortage of grave diggers in Toronto cemeteries, due to the demand for graves as the result of the influenza epidemic, that Torontonians have been forced to dig graves for their relatives." Another article goes on to state that:

> As an evidence of the exceptionally large number of deaths during the epidemic there were on an average 30 burials a day this week in Prospect Cemetery or about 200 for the week. The usual average is about six a day or less than 40 for the week. One undertaker in the vicinity of Earlscourt has a burial every hour on Saturday, while on Friday morning another undertaker conducted five funerals before ten o'clock. On the same day a local minister officiated at five funerals. In normal times there are two grave diggers at Prospect Cemetery, but since the epidemic a number of extra men have been engaged, until now there are no less than 35 grave diggers at this burial ground, and they are all kept busy. (*The Star* Oct 28:11)

Not surprisingly nurses were hard hit by the pandemic. A report from *The Star* (October 12, 1918: 23) informed readers that 46 of the 211 nurses at General Hospital were down sick with the virus. Twenty-three nurses at Grace Hospital were also away from their duties due to influenza infection (*The Star* October 10, 1918: 21). The Department of Public Health put out a call for volunteers on 18 October, reporting

thirty-five of its nurses laid up with the malady (*The Star* October 18, 1918: 2). In his monthly report for the Department of Public Health for October, Dr. Hastings lamented the hardships faced by the Division of Public Health Nursing. He stated that the "staff averaged about thirty-five per cent. below its normal strength" (Monthly Report October 1918: 6) throughout the epidemic.



To Our Friends In The City:—

Owing to the epidemic of influenza a good many of our regular staff are off duty. As a consequence several of our stores are short handed and operating under difficulties.

We are doing all we can to give the best service possible to our customers, and if we fail to do all we would like to accomplish, we feel sure our friends will bear with us under the present strain, and make allowances.

We particularly appeal to all who deal with us over the week-end to help as far as possible to lighten the burden of our sales staff by rendering the following service:

By calling and making Purchases Personally

Rather than by using the 'phone. We cannot promise to give attention to 'phone orders at all our stores.

By Carrying Parcels

This will help a great deal, as our delivery service is greatly curtailed just now.

If you cannot shop in person, send the children.

We will give them the same careful, courteous service we give to the grown-ups.

We hope this is only a temporary condition of affairs and that our usual service will be resumed very shortly. In the meantime, we feel sure we can rely on your co-operation and help in making things easier for our salesmen who are anxious to please you and to supply your needs as far as they can.

THE WILLIAM **DAVIES** COMPANY LIMITED

STORES THROUGHOUT THE CITY

Canada Food Board License No. 8—3075.

Figure 6.2 Davies Department Store Ad
Source: The Star 25 October 1918 (4)

Individuals engaged within the private sector were also significantly impacted by the epidemic. In response to Dr. Hastings plea that large employers insist that their sick employees convalesce, 400 of the 1900 workers of the Bell Telephone Company were confined to their beds (*The Globe* October 17, 1918: 6) and department stores

scrambled to secure enough employees to run shops (*The* Star October 25, 1918: 4) (see figure 6.1). The conditions of industry were no better. According to *The Globe* (October 29, 1918: 13) productivity in certain manufacturing centers had fallen off as much as 15% in some sectors.

Framing Influenza

Rosenberg (1989) states that epidemics are events that become imbued with an episodic quality. They represent a narrative bounded in time and space which if read correctly exposes a community's ideology, social structure, construction of self and institutional practice. To Rosenberg all epidemics can be distilled down into four acts: Act I, the prologue or progressive revelation; Act II, which he terms managing randomness; Act III, negotiating public response; and Act IV, the epilogue. The following section will use these acts to recreate the disease experience in Toronto in order to frame Spanish influenza and display the beauty pageantry of the city.

Act I: Prologue

Rosenberg (1989) states that communities and public health officials are often slow to accept or acknowledge an epidemic. The desire to "turn a blind eye" can be born from factors ranging from a lack of imagination to overt decisions to stay public fears and maintain the status quo. Public recognition has little to do with desires to quell epidemic spread, but rather is contingent on the point at which an epidemic can no longer be ignored. This scenario fits the situation in Toronto. As is highlighted in Rosenberg's narrative, although the public as well as health officials were acutely aware of the pandemic activity in the northeastern United States they were reluctant to

acknowledge its arrival within Toronto. Even after cases began to mount the overwhelming desire to play down the severity of the pandemic won out. Rosenberg (1989) cautions, however, that to accept the presence of an epidemic is to risk social dissolution. Those who are in a position to do so may flee the city and breakdowns in trade and communication are unavoidable. There is no evidence of an exodus from Toronto but it is clear that the day-to-day functioning of the city was hindered. This not only affected the city's inhabitants but also impeded their ability to support the war effort.

Act II: Managing Randomness

Rosenberg (1989) terms his second act "managing randomness". It is this portion of the epidemic narrative in which a society struggles to define the parameters around infection. This is the phase in which people try to make sense of the randomness that is usually encapsulated within an epidemic experience. People strive to develop an explanatory framework by which to comprehend the pathogen to minimize inherent feelings of vulnerability. During this stage people assign blame to individual behaviors or disenfranchised groups.

In some regard the influenza pandemic in Toronto is unusual in that no overt scapegoating or shaming of victims or vulnerable populations was evident. Johnson (2006: 159) supports this notion, with his work in Britain, stating that Spanish influenza is an "epidemic that is largely downplayed, in all senses, and that includes blaming."

The process of assigning blame usually involves some form of 'othering', in which diseases are depicted as being imported by foreigners who have the potential to

hurt us (Johnson 2006).[91] The route the disease took into the city, via military personnel, did not lend itself to this framework, for the act of devaluing or ostracizing the country's military heroes would have also carried with it a high social burden. This practice of safeguarding the image of soldiers is not unique to the 1918 influenza pandemic. During the Second World War the problem of venereal disease was significant. Rather than target men and women to an equal extent for sexual transgressions, women were disproportionally 'shamed' in an attempt to quell transmission. Not only were women chastised for their sexual immorality, but they were held accountable for men's indiscretions serving, much like Eve with the apple, as irresistible temptresses (Hegarty 1998).

Although no witch hunt was ever carried out to identify a portal of entry into pious city, public health officials did use language that had the potential to ostracize or place blame on infected individuals. McCullough, the provincial officer of Health, informed citizens that "It is the patriotic duty of every citizen to avoid influenza and keep in good health" (*The Globe* September 8, 1918: 6). In addition, it was stressed by public health officials that the progression from influenza to pneumonia was the byproduct of individuals not adhering to established protocols after infection. Dr. Hastings is reported to have told a *Star* reporter "it is not the influenza itself that kills, but, in the great majority of cases complications which occur through neglect of the proper precautions" (October 11, 1918: 2). Although these recommendations were made with the populace's health in mind an unintended consequence could have been the shaming and blaming of those hard hit by the disease. Within the sources

[91] For an interesting discussion of this concept see Farmer (1992) discussion of 'the geography of blame' or Sontag's (1988) discussion of linking unnatural and 'other'.

reviewed for this work there is no evidence of public shunning, but this use of language could have resulted in reduced public relief efforts within a different cultural context.[92]

Act III: Negotiating Public Response

Within Rosenberg's dramaturgy, this is the point at which communities come together to confront a pathogen and comply with a common action. According to evidence captured within the written record, this response was orchestrated by government action. Public health officials credit Toronto's diminished mortality rate to preparedness and public health intervention. In the October Monthly Report of the Department of Public Health of the City of Toronto, Dr. Hastings states

> It is… extremely gratifying to know that the measures of control instituted in Toronto were effective in lessening considerably the duration of the epidemic, as compared with large cities on the other side [the United States]. Not only is our mortality decidedly lower but the duration of the epidemic was from a week to ten days shorter, and this was accomplished without recourse to any of the hysterical extremes that were in evidence in some cities, where street-car traffic was suspended and people were advised to wear masks on the street, etc. (1918:5)

The following section attempts to describe and quantify the actions implemented by Toronto's chief medical officials to ascertain how these institutional actions impacted influenza morbidity and mortality within the city of Toronto.

The Ministry of Health for England and Wales published a Memorandum on Influenza (1927:11) that outlined "actions by sanitary authorities to combat influenza outbreaks" based on actions undertaken during the pandemic to aid in the preparation for future disease outbreaks. This report systematically examined different approaches

[92] It is the contention of this author that participation in the First World War led to higher patriotism and an overall desire to assist members of ones community. For a complete discussion see Chapter 7

to the containment, confinement and treatment of the affected areas. The following sections use the categories and framework of this report to explore the public health measures carried out in Toronto.

Quarantine. The city of Toronto took no measures to implement quarantine of travelers or of local infected individuals. In this instance a lack of action may have actually helped safeguard the city from further infection. As was demonstrated in the previous chapter, many of the city's public health workers were sick with the flu. The few that were left on the job were charged with the task of going door-to-door to identify stricken individuals and provide them with the basic necessities that they required. The process of implementing and enforcing quarantine would have restricted already taxed resources to no avail. Dr. T.H. Whitelaw, Medical Officer of Health for Edmonton, Alberta, highlights his frustration with the apparent failure of quarantine in his jurisdiction. He states

> In spite of the energetic work of the officials of the health department, in promptly following up all reports of physicians, and information derived from all other sources, by placarding premises and establishing quarantine, it is apparent that the number of cases above reported[93] and quarantined did not at any time represent more than 60 per cent. of the actual number of cases in the community… Many citizens regarded the placard as an injustice, either because they did not believe the diagnosis justified, or because their neighbours were alleged by them to be avoiding quarantine by concealment or evasion. Some physicians began to be careless or indifferent in reporting their cases, because they alleged that other physicians were not reporting their cases, and charges of discrimination were frequently made against the officials of the health department. (Whitelaw 1919: 1071)

Authors examining the effectiveness of quarantine on Spanish influenza mortality also suggest the futility of these efforts. Sattenspiel and Herring (2003)

[93] The cases reported above were: 2208 for October, 2323 in November and, 1258 for the month of December.

utilized mathematical models to assess the efficacy of quarantine measures among Aboriginal populations in Central Manitoba. They demonstrated that although the practice of quarantine had the potential to delay epidemic peaks "there was no significant change in the peak number and total number of cases" (Sattenspiel and Herring 2003: 15). McCracken and Curson (2003) were reliant on historical documentation to recreate the disease experience in Australia. Although significant precautions were undertaken to safeguard the continent, officials were unable to effectively quarantine the country. As historical explorations have failed to identify the index case there is some debate as to how the pathogen entered the country. The Commonwealth Director of Quarantine was adamant that flu, already present in Australia, mutated and caused excess mortality; a more parsimonious explanation is that an infected individual(s) brought the virus into the geographically isolated population.

Education. The Toronto health authorities worked diligently to inform the public as to how best to combat the disease. Dr. Hastings worked in tandem with local newspapers to disseminate information regarding the "nature of the epidemic, the ways and means by which they might protect themselves against it, and their best procedure, should they fall victim to it, in order to minimize the dangers" (Monthly Report October 1918: 5). The two most common tools were a public service announcement created by John Dill Robertson, Chicago's Commissioner of Health and the advice of Lieutenant-Colonel J.W.S. McCullough, Provincial Officer of Health. In the later stages of the epidemic a pamphlet of instructions on how to avoid the flu or convalesce if the disease was contracted was created by the Department of Public

Health and distributed by the Boy Scouts Association (The Globe 22 Oct 1918). The training of able bodied women to act as nurses was also taken-up by public health officials[94] in an attempt to help provide relief to sufferers at home. Finally Hastings stressed the importance of crowd avoidance suggesting that individuals who resided within a mile of their workplace walk (*The Globe* October 14, 1918) and that individuals avoid public gatherings.

Notification. When Spanish flu appeared in Toronto, influenza was not a reportable disease. Many physicians disputed Dr. McCullough's decision not to institute mandatory reporting at the onset of the epidemic. Dr. M.B. Whyte, who was responsible for the Isolation Hospital, publicly chastised McCullough for his decision, stating that without proper reporting "how can Dr. Hastings know where the cases are, and how serious the outbreak is or what steps are necessary to fight it" (*The Star* October 7, 1918: 2). At no time throughout the epidemic did Dr. McCullough alter his stance on the mandatory reporting of influenza, although many physicians self appointed themselves to the task. Although this measure most likely had very little impact on the course of the disease throughout the city, from a statistical standpoint it becomes difficult to ascertain the prevalence of influenza or its mortality rate was. The only approximation of the number of cases in the city was made by Dr. Hastings, who estimated on the 15 October 1918 that there were between 5000 and 10,000 cases of the disease to date (*The Star* October 15, 1918: 8).

Efforts to lessen the opportunities for infection. Very few measures were taken to lessen the opportunities for infection. For the city, aside from education

[94] The government provided meeting spaces and funds for advertisement but the lion's share of the work was undertaken by women's groups in the area.

campaigns and the deterrence of public gatherings, the only significant measure undertaken was the provision of additional streetcars. On the 8 October, 1918, Mr. C.M. Colquhoun, Civic Solicitor, helped facilitate the appropriation of new streetcars to lessen overcrowding on residents' commutes to work (*The Globe* October 8, 1918: 6). Within the hospital (and home health care workers) numerous measures were pursued to help prevent transmission of the virus from patient to care giver, although, for the most part, these measures were largely ineffectual. These measures included vaccination, the wearing of masks (doctors donned regular surgeons' masks, while nurses were reliant on "layers of cambric linen or other fine material over the mouth, nose and ears to prevent inhalation of the pathogen" (*The Globe* October 16, 1918: 8)), and disinfection (the most commonly used agents were carbolic acid and Lysol (*The Globe* October 16: 9)).

Closings of schools. On Tuesday, October 15 city schools were closed with some trepidation on the part of Dr. Hastings who went against the recommendation of Lieut. Col. McCullough. The latter stated

> Where a well-equipped medical inspection of schools is in operation as in Toronto, there seems to be no doubt that the children would be better at school than running the streets and spending their time (as they have in large numbers been doing in Toronto) in the shops where the warmth and attractions are better than many of them have at home.
> (McCullough 1918: 1084)

The schools appear to have been one of the first civilian areas in which the impact of the epidemic was felt. By 7 October 1918 (two days before Dr. Hastings recognized the start of the epidemic within the city) schools were already experiencing difficulty operating, as a large number of teachers were off sick and occasional teachers were unable or unwilling to take up the task. "One principal stated that as

many as 27 calls from occasional teachers had been put in by No. 1 District, and only one supplied" (*The Star* October 2, 1918: 2). At Frankland School, short seven teachers, three classes had to be sent home; at Pape Avenue and Duke of Connaught Schools eight teachers were absent, as were a large portion of the student body. Also feeling the pressures of the disease were Rose Avenue, Perth Avenue and Jesse Ketchum, which had a number of children away suffering from 'colds.'

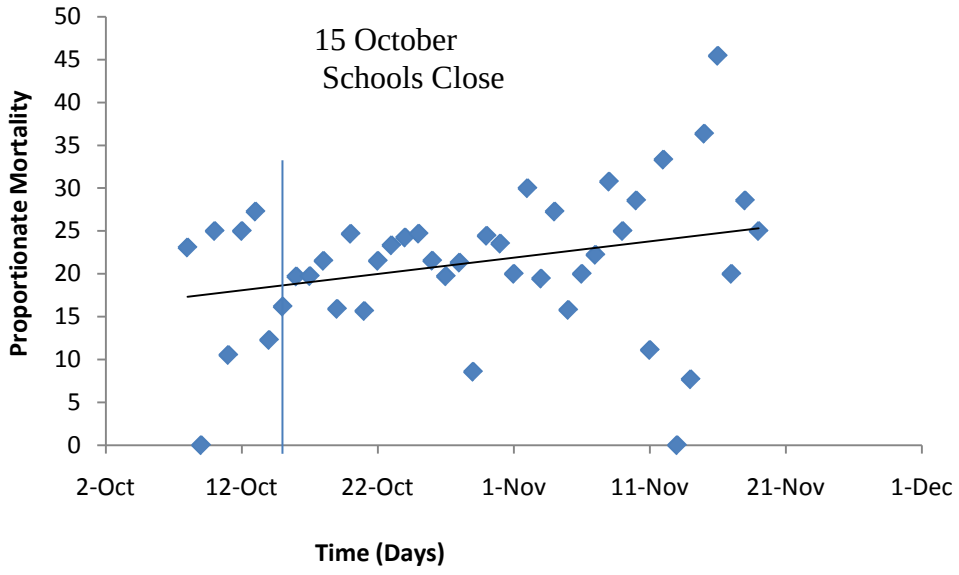


Figure 6.3 Proportionate mortality of Individuals Aged 1 – 19, 8 October to 18 November 1918

Figure 6.3 represents the proportionate mortality for individuals aged 1 – 19[95] from 8 October 1918 to 18 November 1918. This time period captures a period prior to the closing of schools and their subsequent reopening. Proportions were calculated using the number of deaths in the 1 – 9 and 10 – 19 age clusters for a given day and divided by the total number of deaths for that same day. The results were multiplied

[95] Because the data were already aggregated into ten year intervals children who would not be attending school (those under five-years of age and perhaps those over the age of 18) were included in this analysis.

by 100 to achieve a percentage. A trend line was plotted to estimate the impact of the epidemic. The null hypothesis for this test is that the closing of schools had no impact on the incidence (and subsequent mortality) of Spanish influenza in school aged children (for the purposes of analysis in this study individuals aged 1 – 19), which would be demonstrated by a horizontal line. The trend line shows a slight incline, suggesting a marginal increase in the number of deaths in school-aged children following the decision to close schools.[96] If mortality is a reasonable proxy for morbidity, it can be concluded that the closing of schools had limited success in curtailing the spread of the pathogen within Toronto youth.[97]

Although the closing of schools had little direct impact on the incidence rate of influenza among school aged children it does not mean that this action was without benefit. The freeing up of able-bodied women[98] and establishment of assistance headquarters would unquestionably have had an effect on pandemic mortality. According to recent literature that examined the impact of school closures as a result of pandemic H1N1 influenza only 22% of households reported missing work to watch children (Gift et al. 2009). Human capital was not the only benefit; home economics

[96] This analysis is problematic as we are dealing with mortality rates rather than an estimation of morbidity. This fact raises two significant problems. The first is that it is possible that there was an increase in incidence rates and the subsequent mortality was the by-product of a higher overall mortality rate or longer illness duration. The second problem is establishing a reliable time line from infection to death. The accepted incubation period of the disease is between 24 – 48 hours (Jordan 1927: 257). For the data collected for this work the average duration for DCD was 7 days (conditions in which the disease has been experienced for a year or more, i.e. tuberculosis, were omitted) and ICD was 5.8 days. It is difficult to ascertain if these two time periods were consecutive or concurrent, placing time from exposure to death on average from 8 – 14.8 days. The calculated regression coefficient was 0.98.

[97] The increase in mortality is not the result of an increase in the number of cases. Proportionate mortality was calculated in 10-year intervals (see Appendix 2) and the trend-line suggests a decrease in mortality among other age groups.

[98] Teachers were of great assistance in caring for the sick.

classrooms were of great use in the preparation of hot meals and many schools served as meeting grounds to centralize volunteers and supplies.[99]

The predominant role of teachers as support workers became most apparent when Dr. Hastings began to talk about reopening schools on October 28, 1918. When asked to reflect on the reopening of the schools, teachers interviewed by *The Globe* (October 31, 1918: 10) made the following statements:

> "I know home after home," said one teacher yesterday, "that will be absolutely without help of any kind if the teacher who is at present acting as housekeeper, cook and nurse is taken away."
> "It will involve dreadful hardship, and possibly death, if the teachers are taken from their V.A.D. work," said another [teacher] who had three families under her charge, all the older members of which are down with influenza or just recovering from pneumonia. "There seems to be an extraordinary lack of realization of the extent of the disease and of how badly off for help so many hundreds of even well-to-do people are. As for the conditions among the poor they are still appalling. I am speaking for several of my fellow-teachers, and they all say that they feel if they are withdrawn from the cases they are now nursing the consequences will be disastrous"

The impact of the removal of these women, as well as the closing of school kitchens for nourishment provision could have been the true reason that Dr. Hastings chose to delay the reopening of schools from the 5 November to the 11 November.[100]

Public places of entertainment. The closing of places of entertainment was met with a high degree of opposition from establishment owners who felt that they were being cut off from their livelihood. Upon hearing of the closing of schools "Ald. Cowan presented a petition from the Association of Toronto Theater magnets urging that their places not be closed" (*The Globe* October 15, 1918: 8). Their request was

[99] For a more lengthy discussion on the work carried out by these volunteer groups see Chapter 7
[100] According to *The Globe* (November 1: 5) the official listed reason for the delay in reopening is cited as a disproportionate number of sick teachers.

supported by Mayor Church and Controller McBride, both of whom possessed significant political clout. Regardless of this resistance, all theaters, picture shows, billiard rooms, and bowling allies were ordered to close their doors as of the 21 October 1918 and were not allowed to reopen until the second week of November. It is highly unlikely that this measure had a significant impact on lessening the spread/duration of the pandemic as the city was already nearing its peak by the time this precaution was instituted.[101] In fact it is possible that the power demonstrated by these influential business owners (and the subsequent delay of the closing of their businesses) may have assisted in the transmission of the virus. It was feared that due to school closings children would seek refuge within these places of amusement and therefore increase their likelihood of contracting influenza[102]. Although the local Board of Health issued instructions to the proprietors of theaters and moving picture shows which ordered no admittance to children under the age of seventeen (*The Globe* 17 October 1918), there was some debate over how strictly these measures were adhered to.

[101] The epidemic reached its peak on October 23 and by October 25 deaths from influenza and pneumonia were already beginning to peter off. Given the 24 – 48 hour incubation time and the subsequent period from infection to death it is highly unlikely that this decline was the by-product of these closings.

[102] Modern literature on school closings (see for example Cauchemez et al. 2009; Davey et al. 2008; Glass et al. 2006) suggests that the act of closing schools is not sufficient to quell epidemic spread; rather, population movement must also be restricted to achieve desired results.

Figure 6.4 Northway Ad for Department Store Disinfection. Source: The Star 19 October 1918:19

Other centers of overcrowding. As has already been noted, individuals were encouraged to refrain from utilizing public transit if at all possible, and additional street cars were allocated to help combat overcrowding. All conferences, conventions and other large gatherings were to be postponed on order of Dr. Hastings from 12 October 1918 until the epidemic abated. All local sporting events were also cancelled to help reduce the congregation of individuals.

Disinfection. Spoiled linens and contaminated utensils were of primary concern during the epidemic. Early efforts by the Department of Health included increased vigilance over dishwashing practices for restaurants and the removal of communal water drinking cups (October 10, 1918). People were encouraged to wash handkerchiefs often as well as linens and other items that came into contact with flu

sufferers, and they were also urged to wash their hands in a disinfection solution. Many store owners opted to disinfect stores daily (Figure 6.4).[103]

It is important to note however that this practice of may have been counterproductive, for according to the Memorandum on Influenza (1927: 13) "The practice of spraying halls and places of public resort with a disinfectant fluid is of doubtful utility, and only tends to create a false sense of security." As such citizens may have not felt the need to safeguard themselves against the pathogen within sanitized stores and therefore may have 'relaxed' recommended precautionary measures.

Organization of domiciliary medical and nursing service. Due to the war efforts the number of available nurses and doctors was rather limited. Every effort was made to make these limited personnel more accessible – from the provision of cars (and in some cases drivers, who in many instances were local community volunteers) to the removal of prospective physicians and nurses from academic institutions so that they could provide care to the sick (*The Globe* October 15, 1918: 1). In addition, the training of lay women, who would be supervised subsequently by trained professionals, also increased the potential for care available to flu sufferers. Although individuals within the professional sector held nothing back it was the service of countless volunteers that made the survival of many flu victims possible.

Provision of institutional treatment. Keeping up with the number of individuals who required institutional care was a daunting task. Table 6.1 was recreated from *the Toronto Star* (October 28, 1918: 2) and gives the number of individuals occupying beds within the hospital as of noon on October 28. "Cases"

[103] For a good discussion of common disinfection practices of the time, see Anderson (1917).

data show the prevalence rate within the hospital; "New" is a report of the incidence rate within each given hospital; "Nurses" records how many nurses were on staff; and "Deaths" indicates the number of deaths that occurred in the institution on 28 October.

Table 6.1 Number of Influenza and Pneumonia Cases in Toronto Hospitals, Noon 28 October 1918.

Hospital	Cases	New	Nurses	Deaths
General…….	321	15	72	8
Western…….	130	26	17	5
St. Michael's…	65	3	6	3
Wellesley…….	46	3	18	1
Grace…….	46	1	10	2
Children's…….	87	15	14	3
Arlington…….	60	3	2	3
Totals…….	755	76	139	25

Source: *The Star* October 28, 1919:2

One of the largest undertakings during the government's efforts to combat the epidemic was the opening of two temporary hospitals. Both the Arlington and the Mossop served as hotels for the city. On Monday, 14 October it was reported that both hotel properties were being prepared to serve as temporary care facilities. By Wednesday, 16 October the doors of the Arlington opened and soon after it began to receive patients. Although resources, time and effort were put into opening the Mossop, by the time the space was adequately prepared the necessity for increased hospital space was no longer felt. According to newspaper reports the Arlington boasted one of the highest institutional death rates, although civic officials were quick to point out that the emergency hospital was solely being utilized for pneumonia cases (*The Star* 28 October 1918: 8) and therefore the increased number of deaths was to be expected. Although no statement was issued in response to the cost incurred to curtail the epidemic it was believed that the amount probably exceed $10,000, with the largest amounts attributable to the rent of the Arlington and the Mossop Hotels and the costs

of extra equipment, service and provisioning (*The Globe* 23 November 1918: 10). To put this figure into perspective, the yearly budget for Toronto's Health Department in 1910 was $85,000 (MacDougall 1990).[104]

It is difficult to gauge the effectiveness of the opening of an additional patient care facility. Unquestionably the hospital system was overburdened. Reports in *The Globe* indicate that patients were already being turned away from Toronto's largest hospitals (General Hospital, Western Hospital and St. Michaels Hospital) by 7 October. According to current work carried out to prepare for a modern influenza epidemic, based on a modeled 35% attack rate, it is estimated that during the peak of an influenza epidemic patients would require 170% of available intensive care unit (ICU) beds and 117% of the ventilators in Ontario (Frolic et al. 2009: 56). The authors further report that the current average occupancy rate for Ontario ICU's is 85%. If these findings are representative of the situation faced in 1918 (where the attack rate was reported to have been as high as 95%[105] (Jordan 1927:196)) it stands to reason that there were two to three times as many flu sufferers as there were available beds. The only question that remains is how necessary hospital care was for the treatment of influenza when without fail the best course of action to combat influenza mortality at the time of the epidemic was absolute bed rest and good nursing (Wetmore 1919).

[104] On an interesting side note, in 2009 the Canadian Federal government spent $400 million dollars for 50 million doses of H1N1 vaccine. Ontario received 19.6 million doses at a provincial cost of $62.7 million dollars (the federal government covered the cost of 60% of the vaccine) (Sander 2010).
[105] Jordan (1927) is quick to point out that establishing a global attack rate is an impossible task as morbidity rates varied from 10% - 95% (often times population dependent). According to Reid et al. (2001:82), morbidity hovered around 50% worldwide.

Drugs, etc. – According to the Memorandum on Influenza "Attempts to induce an increased resistance to infection by administration of commercially advertised medicinal remedies appear to have been justly depreciated or actually discounted as likely to do more harm than good" (1927:13). The limited effectiveness of many folk cures and preventative medical measures employed in the city of Toronto was also discussed. Many citizens took it upon themselves to guard against the virus by self administering prophylactic agents, so much so that several wholesale drug companies were out of many medicines used for treatment of Spanish influenza. Out of the stock treatments included cough drops, phenacetin (which was used in prescriptions to keep fever down), camphor, throat eases of different prescriptions and oil of eucalyptus (The Star October 21, 1918: 7). The practice of self proscribing treatments was strongly discouraged by both the Minister of Health and the Provincial Medical Health Officer. Dr. Hastings was quoted as having told *the Star* (October 11, 1918: 2) "It is most regretable (sic) the way people are spending their money on so-called preventatives (sic), such as menthol, camphor, or the like. If people get any comfort through carrying them, all well and good, but they will not ward off the disease, and all cost money."

A tremendous amount of energy was also expended in the manufacture of a vaccine. On Monday October 21, 1918 Connaught Laboratories, associated with the University of Toronto, announced the production of a preventive vaccine of influenza. Working with "germs" provided by Boston and New York as well as samples from Toronto, scientists cultured enough material to create 7000 doses, which were subsequently administered to doctors and nurses throughout Ontario (*The Star* 21

October 1918: 5). According to researchers the vaccine consisted of eighteen strains of "dead influenza germs" (The Globe 21 October 1918:1). By October 22 *The Globe* (23 October 1918: 6) reported that 2600 doses of the preventive vaccine had been applied for by various Ontario municipalities, and that it was being sent out as fast as it could be manufactured.

Although no record can be found as to the exact composition of the vaccine utilized in Toronto, Cadham (1919) explains in great detail the aspects of the material used for members of the Canadian Expeditionary Force as well as inoculations utilized for the civilian population within Manitoba. The serum contained "approximately 300 million streptococci, 200 million influenza bacilli and 150 pneumococci" (Cadham 1919: 522). It was suggested that two doses be administered to ensure efficacy.

It is highly unlikely that this serum had tremendous clinical success. At a most basic level researchers were unclear as to the causative agent they were attempting to isolate. Although most scientists working on the problem were upfront about this uncertainty the prevailing belief at the time was that influenza was a bacterial infection caused by the "influenza bacillus" (which later came to be known as Pfeiffer's bacillus and is currently referred to as *Haemophilus influenzae*). Provided that researchers were successful in culturing and subsequently inoculating individuals inadvertently with the influenza virus, even within a modern setting the efficacy of influenza vaccines is limited. Due to the changing character of the virus over the long term, immunity is often difficult to accomplish (Minor 2010). In instances where there is some degree of viral stability, inactivated vaccines have an efficacy of 68% (95% confidence interval CI 49%–79%) in reducing laboratory (or asymptomatic) confirmed

cases but only of 24% (95% CI 14%–33%) efficacy in illnesses with clinical presentation. Simply stated, only 1 in 4 vaccinated adults acquire protection against the clinical illness (Demicheli 2001).

By taking into consideration the efficacy of the above public health measures it becomes clear that very few of the methods implemented by public health officials effectively curtailed influenza mortality. However it is also evident that the mortality witnessed in Toronto was lower than that experienced in cities of a similar size. It stands to reason that although public health measures most likely had some impact on influenza mortality, they were likely not the sole driving force behind this difference. When the archival documentation is examined for another potential source of mortality buffering there is one factor that is omnipresent and that is the significance of volunteerism. According to Dr. Hastings, 3514 influenza houses were visited.[106] He states that "[t]he large number of calls that the Department's staff were able to make was due, in no small measure, to the co-operation of the various civic departments, the Rotary Club, Wimodausis and other clubs, as well as private citizens, who made available motor cars for the use of nurses (October Monthly Report, Department of Public Health 1918, 6). These volunteer measures will be detailed in Chapter 7 and although the impact of these actions are difficult to quantify given the limited success of public health interventions, is highly likely that this outpouring of support enabled Toronto's continued success in curtailing influenza mortality.

[106] He is also quick to point out that these were just the homes that were registered and "in the press or work [of public health officials], a considerable number had to go unrecorded" (October Monthly Report Department of Public Health, 1918: 6).

Act IV: Epilogue

According to Rosenberg (1989b: 8) “Epidemics ordinarily end with a whimper, not a bang”, but in the case of the second wave of influenza in Toronto the opposite is true. Armistice was announced on 11 November 1918 at which time residents of the city put aside fears of infection and took to the streets. Miller (2002:190) provides a riveting account of the events of that morning

> At 2:30 a.m., wire services flashed the news in Toronto of war’s end. In response, church bells peeled, factory whistles wailed, and Extras were printed. Fire trucks drove through the streets sounding their sirens. Awakened from sleep, tens of thousands of Torontonians headed downtown. Many traveled in nightclothes and slippers; propriety demanded that only a winter coat be thrown overtop. Hanging off streetcars, or piling on the running boards of automobiles, people flooded to the core. Groaning under the weight, vehicles carried their screaming and singing cargo to the celebration. Young Street was packed with revelers. The crowds were so thick that dozens of people walked over roofs to get to the party. Coal scuttles, tin cans, and garbage lids were grabbed – anything to add to the din. Bonfires were set to ward off the predawn chill. Cheering, patriotic songs, and skirling bagpipes filled the air. Fireworks lit up the night sky. Conversation was impossible – and unnecessary. A twenty-four-hour victory party, 1,567 days in the making, had begun.

On 10 November 1918 the majority of Torontonians were secure in the idea that the worst of the epidemic had passed, yet they seemed reluctant to give up precautionary measures and resume life as usual. The reopening of schools had been delayed a week so that teachers could continue to provide care to convalescents. On 2 November, although most parts of the city were witnessing a decline in the number of cases, the epidemic in West Toronto appeared to be worsening. In addition, reports from the Neighborhood Workers Association indicated that demands for aid were beginning to outweigh supply as fewer Torontonians were donating money and

supplies (The Globe 2 November 1918: 10). On the days leading up to 11 November flu deaths remained in the mid-teens and numerous households were in need of care.

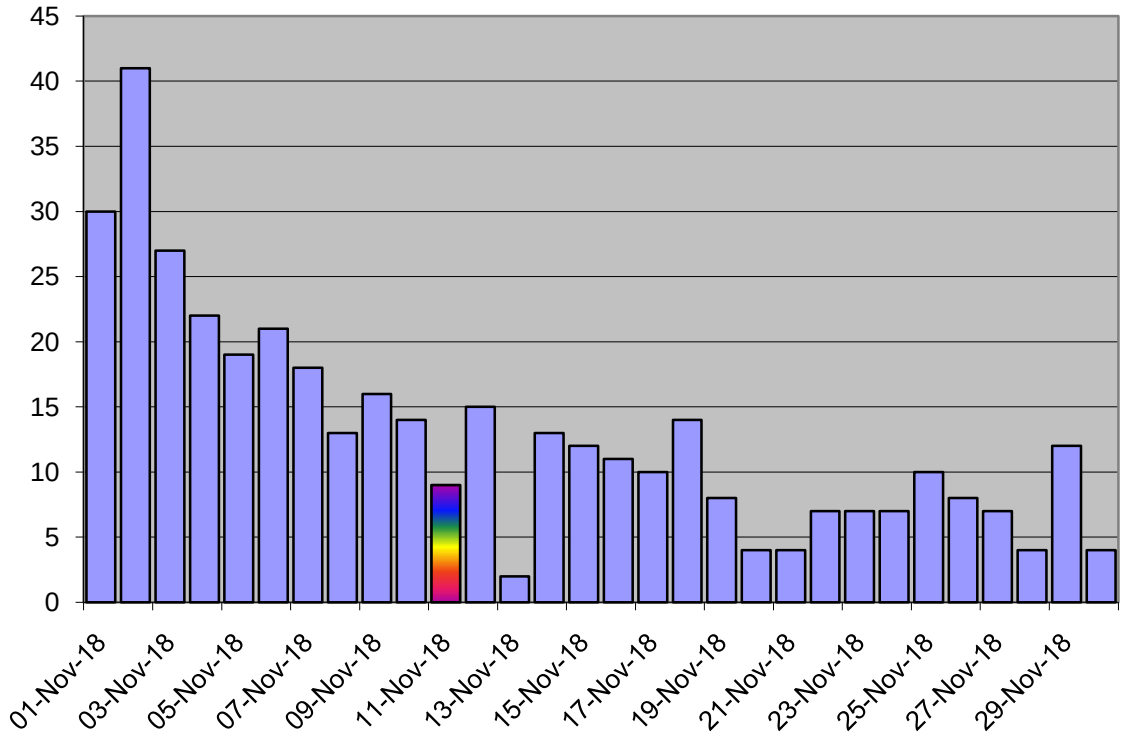


Figure 6.5 Toronto Influenza and Pneumonia Mortality for November 1918. Darker bar represents Armistice.

Figure 6.5 depicts recorded influenza mortality for the month of November, with the multicolor bar highlighting Armistice Day. This graph clearly indicates that influenza mortality was not impacted by the events of 11 November. It is also difficult to ascertain if community interest and subsequent involvement in the epidemic after armistice died off due to a lack of demand or a public's desire to move past the difficulties faced over the last four and a half years. The aim of this discussion is simply to illustrate that however severe the pandemic experience was in the city of Toronto the systemic strain felt by sustained involvement in a brutal war was far more

powerful. The suggestion put forth here is, perhaps, whenever the end of the war came, the desire to celebrate would in all probability not have been stayed. This situation is similar to that observed in Winnipeg and described by Jones (2007), who noted that end of war celebrations took place in spite of a raging epidemic, causing further success in the disease's transmission.

The decision to focus on the second wave of the pandemic was made preceding data entry so deaths were faithfully transcribed from 1 September 1918 to 31 December 1919.

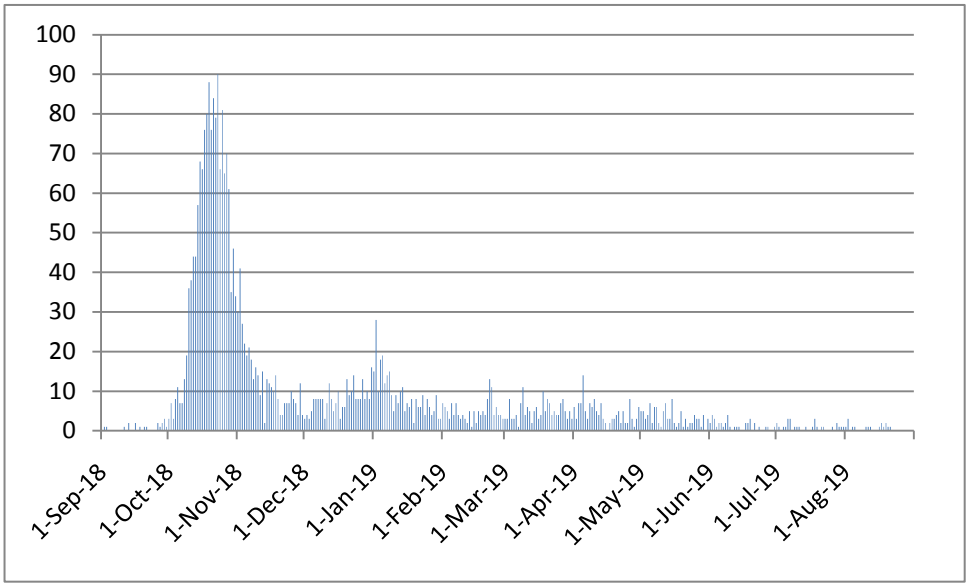


Figure 6.6 Influenza and Pneumonia Mortality, Toronto, 1 September 1918 to 31 December 1919

Figure 6.6 depicts the influenza and pneumonia mortality captured during this time.[107] From this graph it is clear that deaths continued well into 1919, suggesting the recognized 'end' of the pandemic was in many ways a cultural construct as in January alone, there were 281 deaths attributable to influenza and/or pneumonia (which

[107] The graph ends on 31 August as very few deaths occurred after this date.

represents more than twice as many deaths than were experienced during the entire 2009 H1N1 epidemic in Ontario).

By employing the analytical framework of Rosenberg (1989), one striking feature of pandemic mortality in the city of Toronto comes to light. All acts of this play were directed by the Great War. From ignorance, to recognition, to action, to end all stages of the epidemic were structured, in-part, by Toronto's investment in total war. The next chapter takes a closer look at how the Second World War shaped the health outcomes of Torontonians.

Chapter 7 War and Plagues: Spanish Flu and the First World War

Much is written within the anthropological literature about the intimate connection between war and disease (see, for example, Alland 1967; MacLeod 1992; Samuels 1982; Martin 1990). This discussion is often limited to how conditions of war exacerbate factors that lead to the severity of infectious diseases experiences. According to Sherman (2006:117)

> war involves overcrowding and intermixing of populations. Resources are diverted, and often famine and malnutrition increase. These so-called enabling factors in turn lead to decreases in personal hygiene and medical care and frequently in the breakdown of social structure. During wartime individuals are subjected to increased stress, they become more susceptible to new diseases, and endemic diseases may become more severe.

Furthermore Smallman-Raynor and Cliff (2004:5) suggest

> For all involved, resistance to infection may be compromised by mental and physical stress, trauma, nutritional deprivation, and the deleterious consequences of rapid exposure to multiple disease agents. Insanitary conditions, enforced population concentration and overcrowding, the destruction of health infrastructure, the interruption or cessation of disease-control programs, and the collapse of the conventional rules of social behaviour further compound the epidemiological unhealthiness of war.

These analyses focus on how resource diversion, intermixing of populations and breakdowns of social structure increase the severity of a disease experience, and they make little mention of how war may, in fact, inhibit the success of a pathogen. To address this question Steele and Collins (2009) put forth a novel way to access the relationship between war and plagues. Using simple correlations they examine the 1918-19 influenza pandemic experience in Austria-Hungary, Australia, France,

Germany, Canada, New Zealand, Portugal, Italy, Russia, Japan and the UK to test an interesting set of alternate hypotheses. At one end of the spectrum is the idea that increased levels of conflict participation led to a decrease in the ability of the state to confront pestilence. The alternative hypothesis states that increased conflict participation increases the ability of the state to confront pandemics. Their tentative findings suggest that conflict participation increases the ability of the state to confront epidemics. Both analyses rely on the availability of resources, population mobilization, and political authority.

Conflict Participation Decreases the Ability of the State to Confront Epidemics

The following section of this dissertation strives to add to this debate by examining how involvement in the Great War both hindered and helped the population of Toronto combat influenza when it appeared in the fall of 1918. Rather than relying on the simple quantitative measures used by Steele and Collins it draws from qualitative narratives to answer Nancy Scheper-Hughes' call to go beyond the numbers to address "what is being hidden from view in the official statistics" (1997:220). This discussion also strives to advance theories in evolutionary medicine by examining the relationship between pathogen virulence and the cultural vector hypothesis, political economy through exploring the severity of the structural violence created as a byproduct of Canada's involvement in the war, and syndemics theory via looking at the interconnectedness of multiple causation factors and subsequent morbidity and mortality.[108]

[108] For a complete discussion of these theories see chapter two.

It will begin by addressing the problems introduced by Smallman-Raynor and Cliff (2004) and Sherman (2006), namely the impact of resource diversion, mental and physical stress, impact on health infrastructure and issues pertaining to overcrowding and intermixing of populations within the city of Toronto. From there a discussion of the conditions at, Toronto's military hospital, will be provided to contribute to Ewald's theoretical analysis of disease transmission and pathogen virulence.[109]

War Weary!? - Psychological Strain and Physiological Discomfort

Miller (2002) presents an invaluable account of the conditions and reactions to the First World War within the city of Toronto. As a historian, he vividly captures the social conditions, patriotic spirit and determination of the inhabitants of the city at the time of the Great War. Although Miller's aim is not to explore the health of the citizens of Toronto, many inferences can be made from a close reading of his text. Miller depicts a city proud to be at war with its citizens depicted as martyrs to the cause.

When War broke out on 4 August 1914, it was met with excitement and volunteerism. The city of Toronto, whose population was largely made up of former residents of Britain, [110] was quick to take up arms to join the cause. Although most people believed that the military conflict would be short-lived, their moral support did not diminish in the four and a half years they were engaged in battle. The same however could not be said of manpower, supplies and individuals' thresholds for loss. The impact of the war on Toronto's residents was both physiological as well as

[109] For a more complete discussion of Ewald's ideas pertaining to pathogen virulence see chapter 2
[110] According to the Fifth Census of Canada (Volume 2 1913: 354) 92 per cent of Toronto's residents claimed British origin.

psychological. To capture both these elements, resource depletion (most notably expressed as shortages of food, labor and coal) and increased psychological strain will be explored with the end goal of understanding how they may have exacerbated Toronto's experience with the epidemic.

Food Shortages. Many studies have demonstrated the interconnectedness of malnutrition and susceptibility to infectious disease (e.g., Rice et al. 2000; Cohen 2000; Keusch 2003; Bhaskaram 2002). This relationship is facilitated by an increased vulnerability due to restricted immune function and a reduction in the functionality of nonspecific defense mechanisms (for a complete discussion see Scrimshaw and Sangiovanni 1997). Due to labor shortages and the necessity of sending food abroad, the nutritional status of the majority of Torontonians was in some way compromised. Although issues of supply and demand were present as early as 1915, the true shortages began in 1917. In January of that year a provincial Thrift Campaign was launched promoting conservation of foodstuffs at home. Women were encouraged to grow gardens and avoid using meat to prepare meals. They were further encouraged to sign a food service pledge and display a card that read "To Win The War, This Household is Pledged to Carry Out Conscientiously the Advice and Directions of the Food Controller" (*News* Sept 17: 1918:8). These cards were displayed prominently in the window of almost every household in Toronto (Miller 2002).

Figure 7.1 Advertisement from the Toronto Star (7 August 1917:6), designed to encourage Torontonians assist with the Summer/Fall Harvest

Although crop yields were abundant in the summer of 1917, the necessary manpower to harvest them was lacking. This problem was so profound that Premier William Hearst wrote an open letter to the residents of Ontario and their employers imploring them to abandon urban responsibilities and assist farmers whenever possible. The advertisement in Figure 7.1 appeared in the *Star* (August 7, 1917: 6). It depicts two farmers struggling to bring in their crops while fending off the grim reaper. From the left of the image a businessman comes to their aid. The caption, "Help Build the Bulwark Against Famine," appeals to the patriotic nature of Torontonians as it challenges them to build a barrier between themselves and the threats of death. These advertisements were effective ways to draw men out of the

city, but low wages served as a barrier to continued employment and as a result there were many problems associated with reaping the fall harvest.

The spring of 1918 saw further problems with food production. On 12 April 1918 Prime Minster Robert Borden cancelled all exemptions to the Military Service Act for men aged 20 to 22 (Miller 2002). One of the hardest hit groups was Canadian farmers, who were adamant that the best way for them to support the war effort was to continue to supply the necessary staples. Borden was unyielding in his resolve, however, and the men were conscripted. Again the harvest was hindered by the continued war effort.

There is no indication that the citizens of Toronto were without food, although it can be stated with a fair degree of certainty that the long-term effects of food rationing led to some macro- and micronutrient deficiencies. During World War II a group of researchers from Oxford University examined the impact of food rationing on overall nutritional status. Their surveys consisted of two groups of pregnant women.[111] Their results demonstrated protein and vitamin A and C deficiencies (Huxley et al. 2000). The reduction or omission of meat in the average Torontonian's diet may have led to a protein deficiency, which would have negatively impacted immune function as it is highly dependent on protein to aid in cell replication (to make more leuko/lymphocytes) as well as the creation of functional proteins (e.g. immunoglobulins and interleukins) (Scrimshaw and Sangiovanni 1997:467S). Reductions in the availability of fresh fruit and vegetables could have led to deficiencies in essential vitamins and nutrients. Bhaskaram (2002) has demonstrated

[111] The first was comprised of 120 working-class women who were surveyed in 1942 and the second was a group of 253 women surveyed in 1944.

the importance of vitamin A, vitamin C, iron, zinc, folic acid, vitamin B_{12} and other B-complex vitamins in the immune system's ability to resist infectious disease colonization. It is difficult to assess how significantly influenza susceptibility and mortality were affected by sustained food shortages. The multidimensional syndemics approach used here demands that one consider that nutritional deficiencies at any level may exacerbate other factors and therefore increase pandemic severity.

Labor Shortages. The absence of able-bodied men was not only felt in the agricultural sector, it was a systemic problem throughout the city. By the time voluntary enlistment had ended on 13 October 1917 "more than forty-five thousand local men had donned khaki uniforms" (Miller 2002: 146). Medical tests were performed as a condition of enlistment, and as a result of poor health some 40 to 60 per cent of those that volunteered were rejected for service. Contributing to the labor problems was the fact that the majority of the male population in the city of Toronto was comprised of either those not fit for service or the wounded. Married men who had left with the First Contingent were not granted furlough until 18 March 1918 (Miller 2002). Trained medical personnel were also required to travel overseas. Although it is difficult to gauge how depleted the physician and nursing staffs were when the epidemic hit, it stands to reason that the military action had caused their numbers to dwindle.

According to Rice (2003), one fourth of the nursing staff in New Zealand was overseas and Lezzoni (1999) estimates that 50,000 of the 140,000 physicians in the United States were serving in Europe. On the day the war with Germany commenced more than 200 women in Toronto volunteered to travel overseas to act as nurses

(Miller 2002). A correspondence dated 27 August 1916 between the commanding officer of Base Hospital and The A.D.M.S., Military District No. 2 also speaks of prolonged staffing issues. The Lieutenant Colonel states that his "shortage of 45 men on the total personnel of the Base Hospital should be made up for the present as follows: - Extra Officers 3, Extra Nurses 4 and Extra Privates 38" (LAC RG 24 Vol 4385 file 34-7-136 – Personnel Base Hospital), indicating shortages with medical personnel and support staff.

Labor shortages are not simply restricted to how many individuals were available, but who comprised the work force as well. Steele and Collins (2009) caution researchers to gauge the recorded availability of trained staff with a degree of trepidation, as their on-the-job experience may be negligible. In the battle against "invisible foes" Hastings stresses the importance of the trained, seasoned medical personnel. He states:

> …while all credit is due to the fine spirit of those who offered their services freely, the complex judgment involved in case-work were beyond them. It takes training to see with comprehension the various needs and remedies. While they may be able to meet simple problems, and are of inestimable value as auxiliaries working under direction of qualified workers, they cannot possibly solve complex, medico-social problems. (October 1918 Monthly Report Department of Public Health: 7)

Under-trained personnel did not just come from the ranks of the lay public, but were also represented by individuals who had not yet completed their schooling. On 17 October 1918 the Medical Faculty at the University of Toronto was closed and students were urged to volunteer their services (*The Globe* 17 October 1918: 6). On 18 October 1918 the Ontario College of Pharmacy discontinued classes until Wednesday, October 30 so that its students could aid in the epidemic effort (*The Star*

18 October 1918: 2) and on 9 October 1918, Hastings made an appeal to private nurses who had resigned from nursing to pursue domestic life to join in the fight against influenza (*The Globe* 9 October 1918: 8). There can be little doubt that the absence of trained personnel (who were absent due to involvement in the war or through illness and death) contributed to the overall mortality experienced within the city of Toronto.

Coal Shortages. Coal was a constant concern in Toronto from the commencement of the war. In 1914, Ontario was reliant on American companies for their supply of coal. Anticipating a short conflict, these organizations failed to expand their distribution networks. The result: coal was in short supply when the war extended into the fall and winter. This led to increased costs and significant backlogging of orders. The winter of 1916-17 was one of the coldest Toronto had experienced in years, causing already dwindling coal supplies to be stretched to the limit. This problem was further exacerbated by the embargo American coal companies placed on Canadian railroads, effectively closing all points of entry of coal into Ontario (Miller 2002). Cries of American profiteering and extreme hardship led the Toronto Board of Control to take action and acquire the necessary approvals and funds to launch Toronto into the fuel business. The difficulties experienced in the winter of 1916-17 were intensified in the beginning months of 1918, when Toronto was faced with an even colder winter than the preceding year (Pateman 1988). Throughout January and February it was not uncommon for temperatures to plunge to more than 20 degrees below zero Fahrenheit (-29°C), accompanied by vicious blizzards of snow and ice (*Telegram* 5 February 1918:6).

Numerous strategies were implemented to help conserve coal supplies without disrupting the war effort. These ranged from the Government's declaration of "heatless" Mondays until 18 March 1918 to the merging of congregations for Sunday service (Miller 2002). When these measures failed to adequately conserve resources the city was effectively shut down from noon on Friday to Tuesday morning with only essential services available. Even within these locations heat was kept to a minimum. In Ontario courtrooms were filled with overcoat clad judges and lawyers shivering as they worked (Miller 1999: 372).

When influenza hit Toronto in September of 1918 the city was already experiencing unseasonably cold weather (*The Globe* 30 September 1918: 7), which was of grave concern as citizens had just learned that orders for coal placed in the spring would not be supplied in full. Even those who had prepaid their orders at the end of April would be lucky to see a fraction of what they needed. The situation was much more severe for those who had not paid in advance as coal dealers were no longer taking orders (Miller 2002: 185).

The problems associated with the coal shortage were three-fold: 1) the shortage would have contributed to the economic hardships already exacerbated by the war effort, 2) September and October were unseasonably cold (according to newspaper reports) and so because of this systemic shortage, it is likely that Toronto residents were less buffered from their natural environment, and 3) this systemic shortages caused additional pressure, both physiological and psychological, which would have contributed to long-term stresses.

Economic hardships. By 1918 astronomical inflation and stagnation in wages began to expose fault lines across the province. Although the war managed to solve the problem of unemployment (Hart 1918), the overwhelming cost of the conflict compounded by the exportation of Canadian goods and an earnest desire to support troops overseas placed tremendous strain on every household in the city. Coal was just another commodity that heightened this problem. Overall inflation caused prices to rise 14 per cent in 1916, 36.2 per cent in 1917, 7.6 per cent in 1918, and 6.5 per cent in 1919 (Piva 1979: 40). During the month of October 1916 the price of bread rose 12.5 percent to 18 cents a loaf (Miller 1999).

By 1918, Toronto residents could no longer bend to the economic pressures created by the war. The workers' desire to not disrupt the war effort was palpable and resulted in the absence of strikes between 1914 and the first part of 1918,[112] even with an 80 per cent drop in wages since the war's commencement (Miller 2002). In the summer of 1918, unable to support families, Toronto city workers (about 1200 individuals strong) failed to report to work until a 20 per cent increase in wages was granted. Threatened by the promise of solidarity strikes by The Builders' Trade League, The Gloveworkers' Union, and over 1500 other machinists and specialists throughout the city, Toronto city officials voted to grant the wage increase to the city's striking civil servants (Miller 2002).

Further evidence of individuals' inability to cope with systemic shortages in the early part of 1918 can be viewed by the failure of the Patriotic Fund[113] to reach its

[112] The power of the union is evident from the Civic Employees' Union's walkout on 5 July 1918. Newspaper accounts retained a high level of patriotism throughout the strike and the clear desire to end the conflict quickly so that work toward the war effort could resume.
[113] For a more detailed discussion of this organization see pages 157-158

fund-raising goal of $3 million in three days. This was the first time in the organization's history in which it fell short of a targeted goal. According to Miller (2002), this failure was the byproduct of the cost of total war, through inflation and wage stagnation, rather than a lack of patriotism. Each year brought with it increased hardship as resources dwindled along with manpower and savings. It is evident that by the time influenza struck Toronto, residents were under a tremendous amount of strain, struggling to provide even the most basic of necessities to their families.

Cold-related Mortality. According to newspaper reports,[114] medical officials attributed the severity of the epidemic experience in the city to the unseasonably cold weather. This section examines the validity of this assertion and explores how resource depletion as a result of the war may have exacerbated the pandemic experience. Direct research that links deficiencies in environmental buffering and influenza morbidity and mortality have not been carried out, but researchers have demonstrated a general suppression of immune function by stress hormones during cold exposure (Ophir and Elad 1987; Dhabhar 2002). In addition, multiple regression analyses have established high instances of cold-related mortality with low living-room temperatures, limited bedroom heating and inactivity and shivering when outdoors (Hassi 2005). The Eurowinter Group (Keating and Donaldson 1997) suggested that excess mortality in winter was greater among individuals in lower socioeconomic classes because of 'fuel poverty.' As a result of these initial findings, a substantial body of research has been directed toward examining the relationship between socioeconomic status and excess winter mortality. The findings of these

[114] See for example *The Globe* 30 September 1918: 7, 8 October 1918: 6 and The Star 27 September 1918: 13, 4 October 1918:9, 7 October 1918: 2.

reports overwhelmingly suggest that there is no relationship between the two conditions (Gemmell et al. 2000; Gouveia et al. 2003; Hajat et al. 2006; Shah and Peacock 1999; vanRossum et al. 2001), but the researchers usually have noted that the proxies they used for poverty may not correlate well to fuel consumption or access.

Donaldson et al. (1998) looked directly at fuel availability and cold-stress related mortality within a fuel poverty framework utilizing participant observation and interview techniques. Their results demonstrated that mortality only increased when daily mean temperatures dropped below 0° C. Their research postulated that above this temperature individuals were able to safeguard themselves from the effects of cold-stress through increasing the number and items of clothing worn, increased amounts of physical activity and maintaining warmth in select rooms within houses. Information gathered from the Canadian Yearbook (1919) and Environment Canada support the newspaper's claim that temperatures were unseasonably low when the epidemic struck the city (8° lower in September, 2- 5°C colder in October, and 4-10°C higher in December[115]) (Canadian Yearbook 1920: 164-165). Even in light of these colder conditions data from Environment Canada suggests that temperatures did not remain consistently below the 0°C threshold until December.[116] If Donaldson et al.'s (1998) research is predictive for Toronto it would appear that fuel poverty did not contribute to the overall mortality experienced within the city suggesting public health

[115] The crest of the epidemic had already been reached by December so the fact that the average mean temperatures were unseasonably high would not have impacted public health officials interpretation of the relationship between cold and morbidity.

[116] The mean temperature for September was 13.6 (with a mean minimum temperature of 8.5˚ C and the extreme minimum temperature falling to 3.9˚ C). October boasted a mean temperature of 10.6˚ C (with a mean minimum temperature of 6.0˚ C and an extreme minimum temperature recorded as -1.1˚ C). November's mean temperature was 5.6˚ C with a mean minimum temperature of 2.0˚ C and an extreme minimum temperature of -7.8˚ C. December was the first month to witness a mean temperature below the threshold suggested by Donaldson et al. (1998) with an average temperature of -0.4˚ C (with a mean minimum temperature of -3.6˚ C and an extreme minimum temperature of -10˚ C).

officials, as represented by the popular media, were hasty to attribute increased pandemic severity to inclement weather.[117]

Psychological well-being. Although the relationship between cold-related mortality and influenza susceptibility may be hazy, there is no denying the relationship between stress and health. Studies have demonstrated that stress increases individual susceptibility to infectious disease as well as acts as an agent in the activation or reactivation of latent infections (Cohen and Williamson 1991). Within the context of Toronto, The Great War would have stimulated stress responses in the following ways:

1) War is inherently worrisome. Concerns about family members abroad and provisioning of basic necessities at home would have placed many Torontonians in a state of chronic stress.
2) 4 August 1918 marked the beginning of tremendous social disruption. In a matter of days numerous social support networks were broken down or extensively altered. Status and social rolls were distorted, leaving many in new, foreign and potentially anxiety-producing situations.
3) Patterns of sociability were irrefutably altered as many returning soldiers turned against non-British residents, at times terrorizing and torturing vulnerable groups of immigrants (Miller 2002).

The following analysis explores the physiological impact of stress, through an examination of how reactivating dormant pathogens may have resulted in an increase in influenza mortality. This will be done using tuberculosis as a potential co-morbid condition. It will also explore why changing social roles may have increased

[117] It is important to note that although the unseasonably cold weather may not have increased biological susceptibility to the virus, it may have had a social impact altering behavioral choices and thereby impacting the dissemination of the virus.

individual susceptibility to both infection and death. The end goal here is to demonstrate how the Great War give rise to conditions that imposed demands that exceeded individuals' ability to cope.

Stress triggers a cascade of physiological responses that have the potential to impede immune function. Mediated by the activation of the sympathetic adrenal-medullary (SAM) system, the hypothalamic-anterior pituitary-adreno cortex (HPA) system and other endocrine systems, which release hormones into the bloodstream. SAM activation is associated with the discharge of epinephrine, norepinephrine, and other catecholamines, whereas the HPA axis triggers the release of adrenocorticotrophic hormone and corticosteroids (Herbert et al. 1993; Flinn 2008; O'Leary 1990).

Cortisol and glucocorticoids have been documented to suppress immune function (Flinn 2008: O'Leary 1990). Yang and Glaser (2000) further suggest that individuals experiencing chronic emotional distress witness a shift in their T-helper (Th) cells from Th1 which operates in cell-mediated immune activities, to Th2 cells, which are associated with antibody production. This shift from Th1 can result in a marked decrease in cell-mediated immunity, potentially increasing susceptibility to viruses, fungi and mycobacteria (Yang and Glaser 2000). Stress may also impede immune function through direct innervation of the central nervous system and immune system (Cohen and Williamson 1991). In addition stress influences behavior, such as more frequent smoking or fewer hours spent sleeping, both of which may impede immune function (O'Leary 1990).

Increased and prolonged stress also has been demonstrated to reactivate latent microorganisms, which may occur through direct stimulation of pathogen reproduction or suppression of immune defenses which are effectively holding the disease in check[118] (Cohen and Williamson 1991). Numerous studies have linked an increase in the prevalence of herpesvirus infections (notably *Herpes simplex* type 1 [most commonly associated with cold sores], *Herpes simplex* type 2 [most commonly associated with genital lesions] and Epstein-Barr virus) to negative life events (Cohen and Williamson 1991; Glaser et al. 1999; Herbert and Cohen 1993). The same may be true of latent tuberculosis.[119] Although stress has not been identified as a risk factor for reactivation of tuberculosis, malnutrition[120], tobacco smoke, alcoholism,[121] and immune suppressive treatment, such as glucocorticoids[122] have (Lin and Flynn 2010). Considering the physiological and behavioral implication of stress, considering an increase in tuberculosis prevalence is a hypothesis worth considering.

Tuberculosis (TB), an extraordinarily efficient pathogen caused by the bacillus *Mycobacterium tuberculosis*, has coexisted with humans for thousands of years (McMichael 2001). Perhaps one if its most effective strategies is to cause latent infection in the majority of the individuals it inhabits. It has been suggested that primary infection results in active disease within the first two years in approximately 10% of cases, leaving some 90% of cases in a state of clinical latency (Tufariello et al. 2003). These individuals carry a 2-23% lifetime risk of developing reactivated

[118] Current research (Cohen and Williamson 1991; Glaser et al. 1999; Herbert and Cohen 1993) carried out linking emotional distress and reactivation of latent pathogens is suggestive, not conclusive.
[119] Due to the nature of tuberculosis infection and the lack of a comparable animal models, latency and reactivation of tuberculosis are poorly understood. For a more complete discussion see Flynn and Chan (2001), Tufariello et al. (2003), and Lin and Flynn (2010).
[120] The residents of Toronto were more likely undernourished as opposed to malnourished.
[121] Due to prohibition, over consumption of alcohol would not have been a consideration in 1918.
[122] Mentioned above as a stress related response.

infection (Parrish et al. 1998). The pathogen is able to lie-in-wait encapsulated within a granuloma, much the same way that a virus may hide in host cells. The granuloma is comprised of T lymphocytes, macrophages, B cells, dendritic and endothelial cells, fibroblasts and *M. tuberculosis* bacilli which have avoided elimination (Russell et al. 2010). The infection is effectively held in check by T cells and macrophages within individuals with a robust immune system. Analyses by Herbert and Cohen (1993) have demonstrated a decrease in T cells following long-term naturalistic stressors,[123] which is suggestive that chronic stress may impede the immune system's ability to keep these bacteria in check.

The potential increase in the number of individuals with active tuberculosis could have significant implications for influenza morbidity and mortality. Noymer and Garenne (2000) have demonstrated that TB infection is an important risk factor for contracting influenza, and, in addition, active tuberculosis and tubercular lesions decisively increase an individual's likelihood of developing secondary infections. More recent work by Noymer (2010) that concentrated on examining the long term impacts of epidemics has demonstrated that a portion of Spanish influenza mortality may represent borrowed future tuberculosis mortality. His work, which was carried out utilizing archival material from Massachusetts, demonstrates that tuberculosis death rates fell markedly in the years following the influenza pandemic (Noymer 2010).[124]

[123] This refers to social or environmental pressures in opposition to chemical stimuli.
[124] Sawchuk (2009) challenges Noymer's hypothesis. His work, exploring the pandemic experience in Gibraltar, demonstrates no clear change in the rate of tuberculosis mortality proceeding the 1918-19 epidemic. His findings suggest that the impact of influenza on pulmonary TB was only experienced by women and even that increase was very short in duration. Noymer (2010) has responded to Sawchuck's (2009) findings suggesting that due to the small population size his results have been misinterpreted due

This discussion illustrates how adopting a syndemic perspective may help elucidate the multifaceted nature of infection severity and transmissibility. In this instance the fatal synergy of social factors (in this case increased stress) and biological realities (tuberculosis and influenza pathogen interaction) may have increased the severity of the disease experience. These social and biological processes were orchestrated by deeper political and economic decisions (in the guise of structural violence created through involvement in the First World War and through reducing individual autonomy).

The structural violence created by Canada's decision to support the war effort was systemic throughout the city of Toronto. As has been demonstrated, war is inherently worrisome. Regardless of the extent to which Toronto residents supported the war effort, the unyielding hardship faced during total war undoubtedly took its toll on both the soldier and the civilian alike. Recent works carried out to understand physiological responses to stress have shown increases in cortisol levels stimulated by significant perceived uncertainty (Flinn 2008). From the preceding discussion it becomes clear that day-to-day life was plagued by uncertainty and adversity. Toronto residents struggled to make ends meet, ration provisions and support the troops. These hardships were further compounded by waves of bad news from the front. From the Second Battle of Ypres, fought in April 1915 where 6, 000 Canadians lost their lives, to the weeks leading up to the epidemic[125] in which 2,127 Toronto men became casualties of war with just over 51 men killed, wounded or missing each day (Miller 2002), Torontonians were inundated with reports of causalities from the front lines.

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to the high likelihood of a Type II error (fail to reject a null hypothesis when in fact the null hypothesis should have been rejected).

[125] 8 August to 17 September 1918

Most households mourned privately the loss of a brother, father, husband or son. Evidence of the brutality of the war can be further gleaned from the number of 'Old Originals' who returned home after being granted leave in March 1918. By the end of August 1916,[126] Canada had sent 87,300 men overseas. When their furlough was granted, 113 married men returned home from war (Miller 1999:). Even those who were returned home were not given reprieve for long as the German push between 21 March and 5 April 1918 necessitated the return of these men to the front (Miller 2002).

Participation also resulted in a breakdown of social support networks, radical shifts in occupation, and a disruption in social norms and expectations. Numerous studies have been published that examine the relationship between work-related stress and overall well-being and immune function (Marmot and Wilkinson 1999; McDade 2002; Sorensen et al. 2009). Research in this area shifts away from the identification of stress through direct physical or chemical measurements to theoretical and ethnographic explorations (Marmot et al. 1999). To this end, the theoretical framework of lifestyle incongruity has been adopted to examine the association between social disruption and stress created by the Great War.

Based largely on Weber's (1946) multivariate analysis of social class contingent on production, consumption and power, the lifestyle incongruity model strives to capture tension, conflict, and ambiguities that come about when individuals experience a shift in their sociocultural landscape (McDade 2002). Stress is facilitated when a "person occupying different ranks on different status indicators may have conflicting expectations about others' behavior and uncertainty about his or her own

[126] Miller (1999) reports that very few numbers are available for how many troops were sent overseas (as well this was an endeavor beyond the scope of this dissertation) so the number provided two years into the conflict was utilized as a proxy for how many men may have been shipped to the front in 1914.

behavior" (Dressler 1988: 79). Within an anthropological framework the overwhelming majority of studies that examine status inconsistency do so by focusing on cultural shifts that occur when Indigenous groups assimilate to Western ideologies or immigrate to Western nations (see, for example, Brown 1982; Bindon et al 1997; Dressler et al. 1987). Within these scenarios individuals become trapped between indigenous practices and Western ideals and therefore experience a period of liminality. The contention here is that a similar state was induced within Toronto when war broke out in Europe. The expectations of men shifted from heads of household to patriotic soldiers and women experienced an even more jarring transformation from rulers of the private sphere to active participants in the public realm. [127]

For men social status became embroiled within military effort. For those of non-European descent or who were reluctant to volunteer, social identity could be transformed from leaders within the community to social deviant in a matter of days. Although there are many instances in which evidence of this social transformation may be witnessed, for the sake of brevity only two will be explored here. On 9 August 1915 there was a large patriotic rally held at Riverdale Park which drew in 200,000 people. Adopting a shame-based campaign which had originated in Britain, women 'tagged' men not in uniform with a white chicken feather, marking the first time women were invited to publicly ridicule men (Miller 2002). A *Star* reporter wonderfully captured the practice:

[127] The reason that it is postulated that the shift was more severe for women than men is a by-product of the clarity each sex had with regard to their new expectations. Men unequivocally were encouraged to shift from citizen to soldier whereas women grappled with the pressure of their new dual role as both wife and husband.

> You would be standing with strained neck watching the bands in the valley. Someone would brush past and quietly lay something white on your lapel. It did not dawn at first what the white thing was. Then when you saw, in the dim light, your single violent impulse was to crawl, on hands and knees, out of the crowd and climb a tall tree. (The Star 10 August 1915: 5)

The second example demonstrates the tensions between non-British subjects and returned soldiers. On 2 August 1918 an angry mob of returned soldiers and sympathizers took to the streets from around six in the evening until the police were able to restore order at around two in the morning. During their irate rampage fifteen local restaurants, all operated by non-British residents, were broken into and looted. The total destruction resulted in $40,000 worth of damage, the humiliation of numerous non-British restaurateurs who were dragged into the street and made to salute the Union Jack, and injuries to scores of the city's inhabitants (Miller 2002).

The situation for women was quite different. Although there were opportunities to travel abroad to face the war effort head on as nurses and domestic aids, by-in-large the female fight was centered around the homestead. The challenge faced by women was how to retain their domestic duties as well as enter the public sphere when it was necessitated by the absence of men. This lifestyle incongruity was further intensified throughout the war, as the expectations of women were never static and any new status they may have been achieved was perceived as temporary.

Since the inception of the war, women were anxious to help out.[128] By examining the projects they undertook it becomes possible to witness the shift of women's activities from the private to public sphere. After the Second Battle of Ypres in April 1915 women's groups spearheaded a project to provide comfort to men fighting in France. Their three part plan was 1) to create home-like surroundings in

[128] For a more thorough discussion of women's volunteer efforts see pages 159-169.

areas where men were convalescing, 2) to establish refreshment canteens to provide men with an area for recreation and peaceful reading rooms, and 3) to distribute refreshments and other comforts to wounded soldiers passing through neighborhood rail stations (Miller 2002: 110).

Between August and November 1915 the scale and objectives of their efforts shifted. During this period 2500 women volunteered to sell tags to men in an effort to aid recruiters in the identification of potential soldiers and to raise funds to purchase machine guns and motor ambulances. In one day women raised $34,000 and demonstrated their ability to organize and effectively carry out a large scale fundraising campaign with the goal of going beyond providing comfort to soldiers to assisting them in the prosecution of their enemies. By January of 1916, women were working in munitions plants, running hotels and helping out as farm hands. In the words of Mrs. Huestis, they were "not looking for women to replace men permanently; we are not looking for a position that a returned soldier could fill; we are not looking for a position that an unfit man holds; but we are looking for positions that are held by men who ought to be in khaki" (*Globe* 13 June 1916: 6).

In order to fully understand the consequences of status incongruity within Toronto it is beneficial to utilize Selye's (1976) "General Adaptation Syndrome" further expanded in McDade (2002) to demonstrate how stress was achieved and how it altered individual and community level realities. The analysis breaks up the components and outcomes of stress into four components; stressor, response, consequence and moderators. A stressor is described by McDade (2002: 128) as "an environmental event or situation that disrupts normal functioning and poses an

adaptive challenge to the individual." Within this narrative the stressor is the Great War.

A response represents the attempt of an individual to restore homeostasis, or achieve stability around a new baseline, which is also referred to as allostasis (McDade 2002). Toronto's residents, claiming British origin, were quick to respond to the call to arms and as such, participation in the war effort became the new baseline around which social worth was measured. Those of non-British descent or unwilling to sacrifice their will to the war became socially marginalized. In effect all members of society struggled to define themselves within this novel experience. Gender, social, physical and psychological expectations were in a constant state of flux. The war brought with it tremendous social change and uncertainty as labor, resources, and social norms were all altered to cater to the war machine.

McDade (2002) suggests that consequences increase in severity the longer an individual or community experiences social liminality or unyielding stressors. The war marked a prolonged period of uncertainty, and shifting social norms. One of the clear consequences of prolonged exposure to stress is poor health. Given this reality it is not surprising that researchers have linked status incongruity to an increased susceptibility to infectious disease (McDade 2002; McEwen and Seeman (1998) Sorensen et al. 2009). Using the presence of Epstein-Barr virus (EBV) antibodies as an indirect biomarker of cell-mediated immune function Sorensen et al. (2009)[129] and

[129] Sorensen et al. (2009) study examined three village and three town populations within the Republic of Sakha (Yakutia), located in northeast Siberia. The researchers were interested in investigating the relationship between economic status, material wealth, residence location, cultural change and immune function by capturing the effects created by the post-Soviet climate in Siberia by comparing towns whose subsistence was to varying degrees impacted by the political restructuring.

McDade (2002)[130] have independently demonstrated the positive correlation between an increase in lifestyle incongruity and subsequent elevation of EBV antibodies indicative of reduced cell-mediated immune function.

Finally, moderators include developmental, genetic, or environmental variables that participate in the immune response process causing variability in individuals' vulnerability to stressors, responses, and consequences (McDade 2002), account for why different individuals respond to stress in a myriad of ways. The moderating effects of Toronto's changing social climate and the transport of individuals to the front would have posed a challenge to existing social networks, altering an individual's perception of available support opportunities or situational intermediaries.[131] According to Stansfeld (1999) social support can impact health through direct support or through buffering (thereby moderating the extent of acute and chronic stressors). The relationship between diminished social support and stress has also been well documented (Cohen et al. 2000; Uchino et al. 1996).

Military Matters

Much of the discussion thus far has been limited to the factors that may have intensified the epidemic experience among the city's civilian population, but Toronto's influenza narrative also illuminates some pertinent factors about the relationship between the conditions of war and their effect on disease transmission and severity.

[130] McDade (2002) was interested in Samoan youth. His study attempts to capture the social costs of Westernization.

[131] It is imperative to state in this section that the Great War also fostered a tremendous degree of altruism and reciprocity (see the section on women's work, this chapter) among the citizens of Toronto. It is not the contention here that social support networks were broken down as a result of the war but rather altered and perhaps perceived as less stable. In the majority of instances individuals would be more reliant on their community than family at this time.

Both Smallman-Raynor and Cliff (2004) and Sherman (2006) emphasize the deleterious impact of population aggregation and intermixing, as both these elements foster the efficient spread of pathogens. Although the conditions of war shaped everyday life within Toronto, its residents were not forced to contend with the physical reality of armed conflict. For the population of Toronto war did not result in overcrowding, squalid living conditions and an overwhelming loss of autonomy. For the men who trained, traveled abroad and returned home these factors were much more of a reality. This section will look at how the intermixing of populations and extreme population aggregation, both abroad and at home, may have provided an ideal breeding ground for the abnormally virulent virus responsible for the 1918 pandemic.

At a biological level the strain responsible for Spanish flu's virulence was largely facilitated by the conditions created by the First World War.[132] Soldiers were faced with intense psychological and physiological strain and, much like civilian populations, their immune function would have been substantially taxed. Secretions of testosterone and cortisol, hormones which have been shown to down-regulate immune function (Brunner and Marmot 1999; DaSilva 1999; Gluckman et al. 2009), would have been triggered by chronic stress, psychological strain and the pressures of active combat. Injuries and the prevalence of other pathogens (especially those associated with cardio-pulmonary conditions like tuberculosis or resource depletion like nutrient leaching macroparasites) would also facilitate the spread of a more virulent strain of influenza. Finally, the coming together of immunocompromised individuals huddled together in trenches and transport vehicles would have accentuated the pathogen's transmission opportunities.

[132] For a complete discussion see Chapter 2 pp. 6-10

If Humphries' theory regarding the origin of the virus from China and its subsequent global redistribution via infected Chinese laborers (see Chapter 3 pp. 33-35) is considered, a multidimensional understanding of the social conditions, political and economic realties created by the Great War which helped to nurture this opportunistic microorganism to flourish also come into view. The following discussion picks up where Humphries' work leaves off in an attempt to explore how political decisions may have had the unintended consequence of fostering and disseminating flu. The main focal points of this section will be a comparative assessment of military and civilian transport and care to gauge the experience of these two populations.

Care and its Consequences

The movement of military personnel not only presents a probable route of transmission for the virus,[133] it helps demonstrate how cultural practices may have aided in the spread of this virulent pathogen. On 13 October 1918 the chief coroner for the city of Toronto, concerned with Base Hospital's elevated mortality rate, ordered an inquest into the conditions of the facility. The inquest was centered on the death of one individual in particular, Cadet F.N. Davidson, R.A.F. Although the jury did not find either the staff or local military officials responsible for Base Hospital culpable, it condemned the Canadian government for failing to provide adequate supplies and lodging to its sick and wounded soldiers. The verdict read:

> We find that Cadet F.N. Davidson, R.A.F. came to his death at the Base Hospital on October 13 from influenza which developed into bronchial pneumonia. We also find and strongly censure the Military Medical

[133] see chapter 5 for a complete discussion

> Council of Ottawa for not providing better hospital accommodation for soldiers after four years' duration of the war. No.2, the largest military district in Canada, has sent the most men and it had been provided with the least accommodation by the Military Medical Council of Ottawa, and we think it not fair to the people of No.2 District. We also strongly condemn the Military Medical Council for the overcrowded condition at the Base Hospital, and we condemn the Base Hospital building as unsuitable for hospital purposes. (*The Globe* 8 November 1918:8)

The military desired to contain the treatment of military personnel in military hospitals and the conditions witnessed within these institutions was less than desirable. The best account of the epidemic experience at Base Hospital is provided in a report that was commissioned by A.D.M.S. M.D. No. 2. (RG 24 Vol. 4270 File 15-2-73 vol. 2 Influenza Epidemic October 1918 3 May 1918). The highlights of the document follow: From 23 September to 28 September daily admissions of flu sufferers were manageable. On 29 September 92 influenza victims were admitted, followed by 105 the next day, and 126 on 1 October. The peak number of admissions within the hospital occurred on 4 October, bringing the total number of flu patients to 714. Although the influx of patients slowed down after this point the number of cases of pneumonia increased. The heaviest work week was identified as the week beginning 20 October when 133 of the 452 cases in the hospital were pneumonic.

Every part of the building was commandeered for care from recreation rooms to staff sleeping quarters. Within the main building of the hospital every bed was utilized and 43 additional beds were placed within the corridors. Six winter tents were erected on the hospital grounds to house an additional 60 patients. No mention is made regarding the accommodations of the hospital's existing patient population. It is difficult to ascertain how many men were receiving care at the time. Reports from 1916, when the Hospital was established, suggest that the average patient population

fluctuated between 500 and 700 (LAC RG 24 vol. 4385 file 34-7-136 vol. 1 27 August 1916; 15 July 1916) with an additional 150-170 housed in the venereal section (LAC RG 24 vol. 4385 file 34-7-136 vol. 1 22 Sept 1916). The only reference to the existing patient population in 1918 is that in the summer the occupancy of Base Hospital had been reduced so much that certain sections had been closed off (RG 24 Vol. 4270 File 15-2-73 vol. 2 Influenza Epidemic October 1918 3 May 1918).

It is difficult to gauge how morbidity and mortality within Base Hospital compared to other institutions. A quote from *The Globe* (8 November 1918: 8) is suggests that mortality was more pronounced at Base Hospital than at other local area hospitals. The article reads "Coroner McCollum said little account could be taken of the comparative death rate of the Base Hospital with civilian institutions, as only the most serious cases were admitted to the latter whereas all cases were treated at the Gerrard street institution 'There is always a higher mortality where soldiers are congregated together in large numbers,' said the Coroner." However according to the report of the commanding officer of St. Andrews Military Hospital (RG 24 Vol. 4270 File 15-2-73 vol. 2 Influenza Epidemic October 1918 3 May 1918) "[t]he Statistics also conclusively prove that the results obtained, at the Base Hospital, compare most favorably with any other institution, Civil or Military, and that no patient who died would have had a better chance for life in the most modern hospital."

Clearly both individuals have a vested interest in representing the mortality profile at Base Hospital in a positive or negative light. Coroner McCollum would have favored the latter as it was his office that ordered the inquest into the death of Cadet Davidson, whereas the Lieutenant Colonel would have been more partial to the

former as he was serving as the commanding officer of the Base Hospital at the time of the epidemic. It is impossible to calculate the crude death rate for each institution as reliable morbidity data do not exist. In his report, the Lieutenant Colonel states that as of 24 October 1918, 1811 influenza cases, resulting in 71 deaths, had been cared for at Base Hospital. A report from the central registry dated 22 January 1919, which indicates all influenza mortality from military district number 2 hospitals, shows 1761 cases at Base Hospital with an associated 91 deaths (RG 24 Vol. 4270 File 15-2-73 vol. 2. 22 January 1919).

Table 7.1 Location of Death reported in the Death Registries for the City of Toronto for Spanish Influenza Mortality, 1 September to 31 December 1918

Place of Death	Number of Deaths
Home/Address Provided	1231
Toronto General Hospital	211
Western Hospital	157
St Michael's Hospital	130
Base Hospital	91
Hospital for Sick Children	91
Grace Hospital	44
Arlington Hospital	31
Wellesley Hospital	27
Isolation Hospital	12
Women's College Hospital	11
St. John's Hospital	9
Davisville Hospital	7
Exhibition Camp	4
Total	2056

Information compiled using place of death gathered from death registries for the city of Toronto confirm 91 deaths at Base Hospital although no further information is present in regard to number of cases treated. Table 7.1 further examines the mortality within the city of Toronto from information collected from the death registry from 1 September 1918 to 31 December 1918. From a cursory glance it would appear that Toronto

General Hospital, Western Hospital and St. Michael's Hospital experienced more deaths within their facilities. This is not, however, indicative of a higher crude death rate as these established institutions were larger and in all probability served a greater number of patients. Morbidity data may never be acquired as hospital records for the 1918 -19 epidemic have not been retained. [134] The only records that can be found for the number of cases treated within Toronto hospitals exist within newspaper articles.

Table 7.2 The situation in local Toronto hospitals 10 October 1918.

Hospital	Patients	Nurses	Deaths
St. Michael's….	34	8	2
Grace…….	23	21	0
Western……..	35	16	2
Wellesley…….	5	3	1
Sick Children's.	17	8	0
General……..	93	41	4
Totals…….	206	95	9

Source: The Star 11 October 1918:2

Table 7.3 The situation in local Toronto hospitals 24 October 1918.

Hospital	Cases	New	Nurses	Deaths
General…….	321	15	72	8
Western…….	130	26	17	5
St. Michael's…	65	3	6	3
Wellesley…….	46	3	18	1
Grace…….	46	1	10	2
Children's…….	87	15	14	3
Arlington…….	60	3	2	3
Totals…….	755	76	139	25

Source: The Star 25 October 1918:19

Table 7.4 The situation in local Toronto hospitals 27 October 1918.

Hospital	Patients	Nurses Ill	Deaths
General…….	340	60	7
Arlington…….	52	3	3
Grace…….	56	7	2
St. Michaels….	94	46	6
Western…….	131	14	10
Sick Children's.	98	14	8
Wellesley…….	48	14	5
Totals…….	814	157	36

Source: The Star 28 October 1918:2

[134] With the exception of, perhaps, The Hospital For Sick Children

Tables 7.2 to 7.4 were created by *The Star*, which used figures reported to City Hall (11 October: 2; 25 October: 19; 28 October: 2). All three tables have been provided to demonstrate the inconsistency with which the information is provided. No clear description exists as to whether the information provided is aggregate or representative of the date on which the information was provided (although the recorded number of deaths is indicative of the latter).

Although no clear consensus may be reached as to the crude death rate experienced within civilian and military medical facilities some qualitative information can be presented to help capture a snapshot of the conditions experienced within these institutions. Civilian hospitals did not exceed capacity. There are numerous newspaper reports of hospitals refusing to take new patients (for example, see the *Globe* 8 October 1918:6), whereas Base Hospital responded to the increased demand by creatively expanding its facility. According to a Globe article dated 24 October Hastings stated that the facilities at base hospital should not be used to treat more than 250 patients and as of that date is was believed that more than 800 soldiers were receiving care. The physical characteristics of the civilian and military facilities were also markedly different, primarily because of how long they were intended to be used. Base Hospital was established as a temporary facility and occupied a building deemed by the Board of Directors at the Toronto General Hospital as inadequate to provide care. Civilian institutions were permanent fixtures within Toronto's landscape invested in growth, resources and patient care standards of the time.

As a result of the permanence of these facilities, disease transmission capabilities would have been decidedly different. The problem of cross-infection was

apparent at the Base Hospital since its inception as a military facility. Between 3 November and 22 November 1916, eight individuals within the quarantine section of Base Hospital developed scarlet fever. Captain J.S. McCallum attributed this to the "unsanitary conditions existing in the Isolation Building of the Base Hospital" (RG 24 Vol. 4385 file 34-7-136 vol. 2 McCallum 23 November 1916). He cites insufficient accommodation, poor bathroom facilities, inadequate equipment, and lack of proper fumigation as the root causes of this ease of transmission throughout the ward. In his illustration of the meager accommodations McCallum states "at present Scarlet Fever cases are confined in rooms adjacent to Measles crossed with Scarlet Fever and both are attended to by the same orderly. In addition an Erysipelas case is at present in the Scarlet Fever Ward because there is no other place to isolate him. Chicken-pox and Mumps are isolated from each other by a doorway without a door" (RG 24 Vol. 4385 file 34-7-136 vol. 2 McCallum 23 November 1916). In order to combat the issue of cross-infection, the isolation section of the hospital was moved to the annex of the building. This area could comfortably accommodate 100 men. It was suggested that due to the physical composition of the rest of the building, halting the transmission capabilities of these viruses would have been a futile endeavor (RG 24 Vol. 4385 file 34-7-136 vol. 2 Isolation Section of Base Hospital 28 November 1916).

The qualitative evidence is suggestive that population aggregation, increased disease transmission capabilities and fewer resources were the defining characteristics of Base Hospital. These findings add credence to Paul Ewald's cultural vector hypothesis which explores how human behavior can facilitate pathogen transmission

and virulence[135] as well as demonstrate how political decisions impact individual autonomy and subsequent health. [136] Although the inquest into the death of Cadet Davidson did not find the staff of Base Hospital negligent in their care, it did condemn the Military Medical Council for failing to provide adequate medical facilities to men engaged in the war effort. The further decision to confine military cases to military facilities also contributed to the burdening of an already overtaxed system.

Conflict Participation Increases the Ability of the State to Confront Epidemics

Inasmuch as war confounds and exacerbates the conditions that lead to the proliferation of infectious disease agents, it also operates as a force which may unify communities, for as Hamilton and Seymour (1917) state, it serves to make sanitation and community health a common cause. It also operates as a force which evokes a spirit of social activism. This next section will explore how conflict participation increased the ability of the state to confront epidemics by exposing where improvements in public health were required as well as mobilizing altruistic groups throughout the city of Toronto.

The First World War helped to bring into focus the need for federal and provincial departments of health. In the years leading up to the Great War, Canada experienced a massive population boom which was facilitated by and helped to feed rampant industrialization and urbanization. The city's infrastructures could not keep

[135] For a more complete discussion see evolutionary medicine section in the framework chapter
[136] On an interesting side note a memo dated 17 October 1918 reads "There would be no practical value in disinfecting Ambulances engaged in carrying influenza cases. These Ambulances are too busy, and besides, their disinfection would be labour (sic) lost" (RG 24 Vol. 4270 file 15-2-73 vol 1. Disinfecting of Ambulances, Transfer of Influenza Cases). Due to the relationship between secondary infection and influenza mortality this practice may have increased the severity of cases and further supports Ewald's (1994) idea of the importance of transport vehicles in the severity of the epidemic.

up with this accelerated population growth and because of this slums, destitution, disease and crime helped to defined the new urban landscape (Bator 1979). Although public health reformers were hard at work prior to 1914, they lacked the investment of government agencies and the support of public participation. The First World War not only highlighted the overall ill-health of the population, but intensified the burdens already crippling the city's working class and poor.

One of the chief ways in which the city's poor health was demonstrated was through the rejection of men for active service. Even in light of relaxed medical standards, typically for every ten men who volunteered, three or four[137] failed their medical exam (Miller 2002:79).[138] Another way in which declining health was demonstrated was through the infant mortality rate. In March of 1915 the death rate for infants had climbed by one-third when compared to the same month one year earlier (Bator 1979). These tribulations associated with health and welfare were perceived as hindrances to the war effort and as such were tackled with as much vigor as the enemy. Charles Hastings was able to seize the opportunity to establish government refrigeration depots, more efficient and better funded social welfare programs, free distribution of biological products, health insurance and improved housing (Bator 1979; Canadian Public Health Association 1940; MacDougall 1990; Mercier 2006).

Not only did government spending on health increase during this time, but the willingness of the public to adopt a community conscience was also readily apparent. One of the best demonstrations of Torontonians willingness to support one another can

[137] It was not uncommon from 1916 on for this number to be as high as six or seven.
[138] It is important to note many men were denied for service for reasons other than poor health, for example poor eyesight or hearing impediments would have also rendered a potential soldier unfit.

be witnessed through the success of the Canadian Patriotic Fund. On 10 August 1914, Sir William Mulock, put forth the idea of creating a fund to support the dependents of soldiers at war. Hastily, he rallied politicians and influential public figures and got to work creating an organization that could provide aid to families in need. In September the Canadian Patriotic Fund was formed, Mulock's group augmented their by-laws and merged with the country-wide initiative (Morris c.1920: 8). From 1914 – 1918 the organization ran four successful campaigns raising $9,792,511 (see Table 7.5) (Morris c.1920: 226). In all, their efforts assisted 20,000 families, supplied 6,378 tons of coal to homes in need and provided $1,283,647 to help support the Toronto Branch of the Canadian Red Cross (Morris c.1920: 226). The work was supported by thousands of volunteers (mostly women), 300 physicians who provided their time free of charge, and seventy druggists who filled prescriptions also without charge (Morris c.1920: 227). As a result of those efforts, 97.3 cents of every dollar earned (see Table 7.6) was put back into the community (Morris c.1920: 223).

Table 7.5 Annual Campaign Contributions for the Canadian Patriotic Fund 1914-1917

Campaign Year	Amount Raised
1914	$1,014,482
1915	$2,364,356
1916	$3,205,292
1917	$3,208,381
Total	$9,792,511

Source: Morris c.1920.

Table 7.6 Allocation of Funds Raised Through the Canadian Patriotic Fund 1914-1918

Allocation of Funds	Cents
Paid to Soldiers Dependents	72.9
Paid to Toronto Branch Red Cross	14.7
On Deposit	9.8
Administration, equipment, etc	2.6
	100.0

Source: Morris c.1920

The health and welfare of one's neighbors came to be viewed as a social obligation. In this sense, as individuals experienced a loss of existing kin-based social support networks, a tremendous uprising of community-based social support systems sprang up to help meet the need of struggling families. Much in the same way that a mother was assisted when the sole wage earner went off to war, flu sufferers were cared for by community members who viewed one of the chief lessons of the Great War as drawing attention to the value of cooperation, coordination, and centralization, for it was believed that without these victory could not be achieved (Morris c.1920: 223).

Women's Work

The majority of this dissertation has focused on the efficacy of public health measures and political and economic realities to understand how influenza mortality was shaped within the city of Toronto. Actions such as those undertaken by members of the Canadian Patriotic Fund, suggest that community support may have been just as influential a factor in shaping the influenza experience within the city. Authors such as Jones (2005, 2007), Fahrni (2004), and Quiney (2002, 2003) have all demonstrated the unique power of female volunteer efforts. This final section will explore how participation in the First World War helped to shape these organizations and how they in turn altered the pandemic experience for countless flu sufferers.

At the conference of the American Public Health Association, held in Chicago in the last weeks of September, medical officials, who could come to consensus on very little, were unified in the belief that "The best method of treatment is to keep the patient in bed for several weeks – indeed for a number of weeks after apparent

recovery. This was considered essential for the prevention of the most frequent complication of influenza – pneumonia" (Price 1918: 368). Furthermore according to Charles Hastings, President of the Association as well as Toronto's Medical Officer of Health, and other physicians in attendance,

> the ordinary medical methods of treatment and therapeutic agents employed were admitted to be without much value, and, as President Hastings expressed himself, he felt like saying with Shakespeare, "throw physic to the dogs," as the most approved art of healing, at least in the case of influenza. The best thing that the physician can do for the patient is to leave the patient alone (Price 1918: 368).

During 1918 volunteer groups were not only essential in providing human power to care for the sick, but they were also paramount in organizing and implementing quintessential services for the general public. According to Dr. Hastings, 3514 influenza houses were visited by physicians and civic-minded volunteers (October 1918:6). [139] Although the Medical Officer of Health had ordered public health workers to take a survey of influenza sufferers, the majority of houses were identified through the efforts of industrious volunteer groups and the ability to help was facilitated by the foresight of other like-minded citizenry.

There were a tremendous number of volunteer organizations that coordinated pandemic relief, ranging from the Boy Scouts to the Women's Liberal Association, but three groups stand out above the rest: the Neighborhood Workers Association, the Sisters of Service and the Rotary Club's Rotarians (comprised of the wives of Rotary club members). All three worked in conjunction with the Public Health department to insure that proper care was obtained by all of Toronto's citizenry. However, although

[139] Hastings was also quick to point out that these were just the homes that were registered and "in the press of work, a considerable number had to go unrecorded" (October Monthly Report Department of Public Health, 1918: 6)

they were allied with the government agency they worked as autonomous agents which sought to creatively solve individual suffering associated with influenza infection. None of their energies were spent identifying a causative agent, and subsequent vaccine or other bureaucratic measures aimed at halting disease transmission; rather, their efforts were directed to patient identification and care. Working efficiently and effectively with one another they delivered relief to thousands of victims felled by Spanish influenza.

The Neighborhood Workers' Association

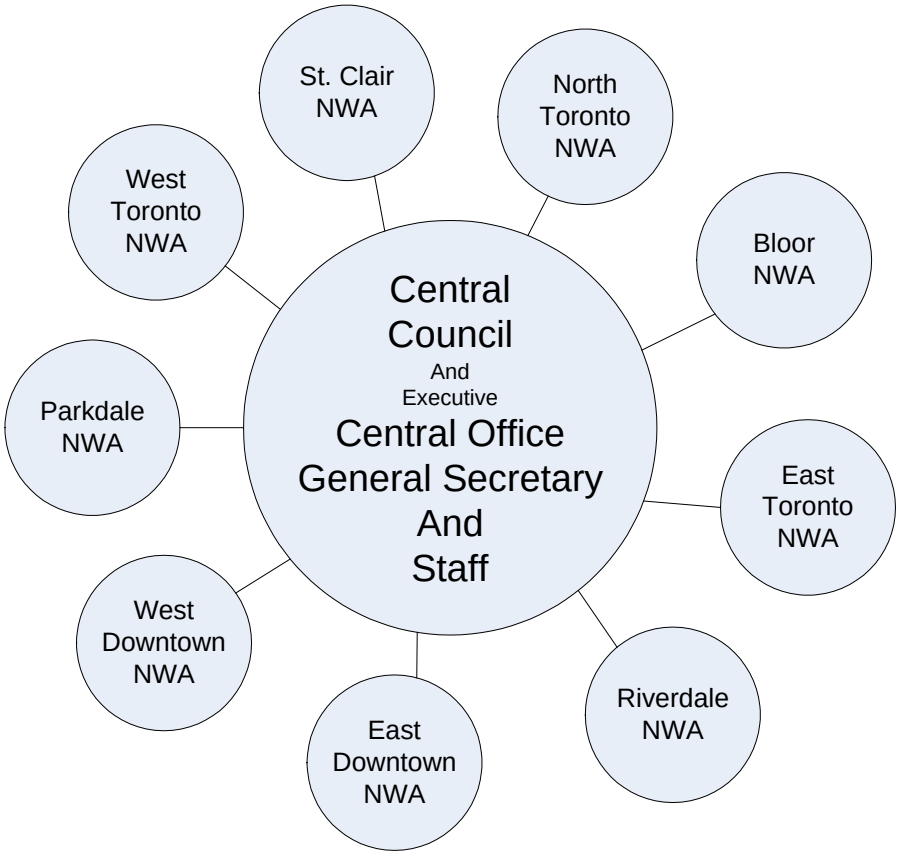

Figure 7.2 Geographic Distribution of the Neighborhood Workers' Association's Nine Central Offices in Toronto. Source: Stapleford (1919:384)

The Neighborhood Workers' Association (NWA) was founded in 1914 by a group of volunteers who were interested in addressing the growing levels of poverty facing many families within the city. At its inception the NWA served as an umbrella organization bringing churches and other charitable organizations together to coordinate relief efforts to families in need (www.fsatoronto.com/about/history.html).[140] Figures 7.2 and 7.3 were reproduced from Stapleford (1919: 384). They identify the location of the NWA's nine central offices (Figure 7.2) and the organizations with which they coordinated (Figure 7.3).[141]

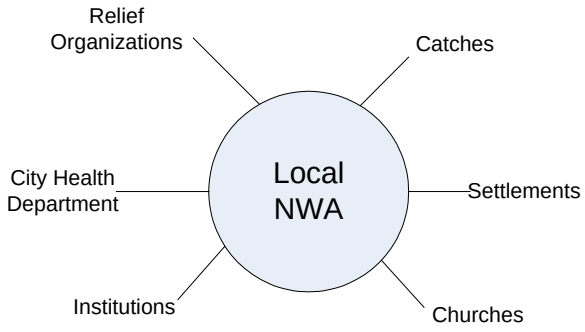

Figure 7.3 Organizations Affiliated with the Neighborhood Workers' Association in 1918. Source Stapleford (1919:384)

In 1919 the Neighborhood Worker's Association had grown to encompass 200 organizations staffed by over 600 workers and volunteers (Stapleford 1919) and although churches played a predominant roll in its workforce, the association prided itself on being "strictly non-denominational" (Stapleford 1919: 383). The aim of the organization was to help eliminate poverty, which the organization felt was "partly a

[140] Original documentation could not be consulted at this time as Family Service Toronto (formally The Neighbourhood Workers' Association) is in the process of transferring their archived material to the Toronto Archives. These records are currently inaccessible and will not be available for public viewing until late in 2011.

[141] These figures are important because they capture important information about how the city was divided as well as demonstrates their relationships with civic, religious and political institutions.

health problem, partly an economic problem and partly an education problem" (Stapleford 1916: 159). The projects undertaken by the NWA reflect this multidimensional view of the conditions leading to poverty. Their programs were designed to stimulate individual achievement to improve overall health by supporting preventive measures and providing basic care. Although the agency was headed by men, which was typical for the time, much of the work and administrative duties were carried out by women (Maurutto 2004).

On 18 October 1918 Reverend F.N. Stapleford, the General Secretary for the Neighborhood Workers Association, attended the Board of Education's general meeting to request the use of the kitchens connected to the Household Science Department so that members of his organization would be able to prepare proper nourishment for flu victims (soups, beef tea and custards) (*The Star* 18 October 1918: 21). Although his request was met with some resistance, as the debate over fuel consumption and cost was highly volatile, the Board agreed to the use of two kitchens. Additional rooms were also utilized for the preparation, collection and redistribution of pneumonia jackets.[142][143] In addition to the two kitchens established at The Central Technical School[144] and Annette Street School, the NWA oversaw the operation of two other local volunteer kitchens. On 21 October 1918 a facility at Earlscourt Central Methodist Church was opened which was supervised by Miss Sherridan, a graduate dietitian, and twelve other volunteers (25 October 1918: 12). Services at Orde Street

[142] A pneumonia jacket was a wrap, usually made of muslin, gauze or cotton that was used to keep the patient warm (Keeling 2010).
[143] In addition to the NWA, the Department of Public Health, Daughters of the Empire and the Women's Liberal Association also aided with the preparation of food and manufacture of pneumonia jackets
[144] The food prepared at the Central Technical School was not only provisioned to the local community but also served as the sole catering service for the Arlington, the temporary hospital established to assist flu sufferers (Globe 31 October 1918: 10).

were utilized to supply kosher food to 70 Jewish families in need (Globe 2 November 1918: 10).

The NWA also established 24 depots for the reporting, collecting and redistributing of goods and services. The depots were located around the nine existing local NWA headquarters (Figure 7.2) and were visited daily by F.N. Stapleford and Miss Emily Mohar to ensure that workers were getting the support and supplies they required (Star 25 October 1918:19). The organization relied on external reporting, word of mouth and newspapers to disseminate information about their services (Figure 7.4) which would be provided free of charge to people in need or at cost to those who could afford it. As of 29 October 1918 the NWA had raised $3,327.68 to help support families and cover the cost of provisions, $2,280.89 of which had been redistributed. On average, 500 homes a day were visited by charities associated with the NWA, 2,461 articles of bedding and clothing were distributed and 759 families had received food from the Central Technical School alone[145] (Globe 29 October 1918:10).

[145] This was comprised of 675 quarts of custard, 899 quarts of broth, 568 quarts of albumen, 689 quarts of gruel and 147 quarts of lemonade.

SUPPLY FOOD TO HOMES OF SICK

Stations for Rich and Poor Alike Opened by Neighborhood Workers

The Neighborhood Workers' Association has issued the following information:

Soups, jellies, custards, etc., can be secured from the depots listed below. These are supplied to all, rich and poor alike. Those who can afford to do so will pay for the cost of the materials. These stations are for the purpose of helping out those who are temporarily unable to have such food prepared in their own homes, owing to illness.

In cases of destitution, where sick-room supplies (linen, bedding, nightgowns, etc.) are necessary, these will be supplied through the central offices of the Neighborhood Workers' Association or through any of the depots.

Technical High School—Borden street entrance. College 7,300. Neighborhood Workers' Association, co-operating with the Women's Patriotic League and the I.O.D.E.

Orde Street Housekeeping Centre—Orde street, near McCaul street. College 4,553. All cases involving Jewish patients should be referred here. N. W. A., co-operating with the Council of Jewish Women.

Ryerson School — Dundas street west. College 581. Neighborhood Workers' Association.

Fred Victor Mission—Queen and Jarvis streets. Main 2,401. Neighborhood Workers' Association.

Broadview Y.M.C.A.—275 Broadview avenue. Gerrard 1,601. Women's Auxiliary, co-operating with the Women's Patriotic League.

Memorial Institute — 892 Richmond street west. Adelaide 2,648. Under own management, co-operating with the N. W. A.

St. James' Square Presbyterian Church—Gerrard street east. Main 6,627. Under own management, co-operating with the N.W.A.

Margaret Eaton School—34 North street. North 4,544. Co-operating with the N. W. A.

Sherbourne Street Methodist Church — Corner Sherbourne and Carlton streets. Main 7,056. Co-operating with the N. W. A.

Y.W.C.A.—21 McGill street. Main 7,220. Co-operating with the N.W.A.

Earlscourt Central Methodist Church—Boon avenue. Junction 2,742. Co-operating with the N.W.A.

Girl Guides, Boy Scouts and motors have been placed at the disposal of the central offices of the N.W.A. to look after the distribution of all requisitions for food and supplies, where people are unable to call for same in person.

For further information, offers of voluntary assistance or gifts of money and materials or the reporting of special cases, communicate with the Neighborhood Workers' Association, Central Offices 409-410 Ryrie Building, Yonge and Shuter streets. Telephone, Main 3,374.

Figure 7.4 Toronto Star article detailing the work of the Neighborhood Workers Association (October 18:21)

Sisters of Service

The Sisters of Service was a group established to provide moderately trained nursing care to the numerous sufferers throughout the city. The first publically recognized appeal to trained and untrained nurses was put out by Miss N.A. Gibson, secretary of the Home Bureau of Nursing, on October 7 in response to a shortage of nursing staff. It was her hope to elicit the help of married women who had subsequently abandoned the profession; Miss Gibson was also willing to receive the support of untrained women as well.

> "When trained help can not be secured, untrained help will be taken." she said this morning. "For in many homes in Toronto now there are whole families who have been taken down with the influenza who have no one to help them, and a woman who has had no experience can give medicine and prepare meals, etc. We will visit homes where these helpers are, so that we can follow up their work and give them instruction." Miss Gibson said that on Saturday, when cases were reported to her and help asked for, there were some five or ten families which she could send no one to. "Many nurses themselves have been taken down suddenly with the Spanish influenza, so that lessens the number to be called on. Of course we prefer the trained women, as the untrained require more work on our part, as they need supervision" (*The Star* October 7, 1918: 2).

Figure 7.5 Public health nurse visiting home. Photographer Arthur S. Gross 26 March 1912 Source: City of Toronto Archives series 372 subseries 32 item 95

The notion that there was an adequate supply of 'trained' women within Toronto was soon abandoned as the number of cases throughout the city began to swell. To meet the growing demand for nursing care within homes and the temporary hospitals established at the Mossop[146] and Arlington, the province turned to the Ontario Emergency Volunteer Health Auxiliary for assistance (McCullough 1918). On 16 October, Dr. Margaret Patterson initiated a three-lecture series aimed at training

[146] Although the Mossop never received patients, Sisters of Service were used to disinfect and arrange the building for occupancy.

women on the basics of patient care. She called graduates of the lecture series Sisters of Service (The Globe 16 October 1918: 4). Eighty-one members of The Imperial Order Daughters of the Empire, the Women's Canadian Club, the Graduate Nurses' Association, and the Women's Christian Association immediately volunteered, and by the time the lectures commenced in the afternoon volunteers packed the large auditorium, requiring many to listen from adjacent corridors (Miller 2002). By 17 October, four lectures had been delivered to over 1000 female volunteers and within a day over 100 were actually in attendance on cases of influenza in Toronto and about 20 others left for outside points to care for patients (The Globe 18 October 1918: 2). On 18 October local city school teachers, under the direction of Admiral Cowan, joined in the nursing of influenza patients (The Star 18 October 1918: 21). In Toronto alone, the Sisters of Service provided care to approximately 1000 families in need (McCullough 1918).

Rotary Means Service

The Rotary Club was formed by Paul P. Harris in Chicago in the winter of 1905 as a social network tool to combat the solitude of large cities. It was his belief that "[m]an is friendly in nature. The necessity for earning a livelihood under modern economic conditions should not force a man to sacrifice his natural instinct to have friends and to be friendly" (Rotary Voice 20 May 1919: 1). In 1912 the first Canadian Rotary Club was opened in Winnipeg (Rotary Voice 17 June 1919:1), and on 28 November of the same year a club opened in Toronto (The Rotary Club of Toronto 1999). One of the central tenets of Rotary was to serve one's country and community.

In keeping with its mandate the organization was active in soliciting funds and providing service for the war effort (Rotary Voice 8 July 1919: 1).

This organization occupied the unique position of strong social ties and solid government connections which enabled them to launch a very ambitious aid campaign. Concerned about the number of families that were homebound due to infection the Rotary Committee on Federation for Community Service was committed to identifying comprehensive information on the number of individuals throughout the city who were in need of care. The organization held a meeting at the King Edward Hotel at noon on 22 October where it was decided that they would launch a complete survey of the city with the end goal of identifying all those in need of assistance. It was their contention that the most efficient means by which to carry out this task was to employ the aid of the city's postmen and postal authorities. They would furnish postal workers with cards designed to ascertain the number of children and adults within a household, how many were ill, and if assistance was required (The Globe 24 October 1918:6). Within 11 hours Ottawa granted the Rotary Committee permission to utilize the services of postmen. Rotarians worked throughout the night and the following morning 10,000 cards had been printed and distributed to civic minded postal workers who went door-to-door personally filling out cards for the homes' inhabitants or making additional rounds to collect cards that had been left earlier in the day. By 24 October 30,000 cards had been hand delivered to Toronto homes (The Globe 24 October 1918:6). The first case reported under this system was from a home on Kingston Road. Twelve people resided within this dwelling, four adults and eight children, all sick with influenza and with no one to help. Within one hour of receiving

the information collected by the card system the Neighbourhood Workers Association had delivered soup and other food to this home, made arrangements to have this done at regular intervals and had furnished all other necessary aid (The Globe 24 October 1918:6).

War: The Great Help and Hindrance

Steele and Collins' (2009:192) tentative findings suggest that conflict participation increases the state's ability to confront an epidemic, which in many ways runs contrary to much of the research linking war and disease. This may be in large part a by-product of the fact that many scholars focus their studies on how the war alters and assists disease transmission patterns or how war's associated stress impacts individuals. As has been demonstrated in the first part of this chapter, these are important areas of inquiry, but they omit a large part of the story. Within the context of Toronto, the Great War inadvertently helped populations combat the epidemic, in that it cultivated volunteerism. Regardless of the increased strain placed on the health of individuals, it is clear that volunteer groups shaped in response to the Great War infused with a renewed feeling of community provided as much, if not more, support than government organizations. The influenza virus was depicted as yet another enemy and as such communities were encouraged to band together to fight it.

All of the evidence examined together indicates that we are not dealing with a simple dichotomy. Without war one may argue that the confluence of disease pools required to incubate such a virulent strain of influenza may not have existed. Nor would the newly evolved pathogen have been so successful in its worldwide transmission. However, even though the war brought individuals together and acted as

a prime means by which to spread the deadly virus, it also operated as a force which unified community membership.[147] This sustained community network provided unfettered surveillance and access to quality nursing care, and as stated by the medical community, it was this availability of this nursing care that defined the boundary between morbidity and mortality. No matter what the conclusion, there is no question within Toronto that the 1918 influenza pandemic can only be fully understood within the context of the First World War.

[147] It is important to stress that although aid was available to all of Toronto's residents, it may not have been distributed equally. MacDougall (2007) notes that fewer Sisters of Service were willing to provide care within the slums of Toronto. Problems surrounding the non-English speaking community members and groups associated with the enemy may not have benefited from the support of these organizations as comprehensively as British-born individuals. Very little information regarding the treatment of minorities is available. The only group discussed in any detail, the Jewish community, appeared to have access to all of the volunteer services within the city.

Chapter 8 Discussions, Conclusions and Future Directions

This dissertation provides a multi-approach descriptive anthropological analysis of the disease experience in Toronto to help contribute to the growing literature aimed at documenting and understanding this once forgotten pandemic. To this end three anthropological paradigms were adopted to help elucidate the disease experience. Rather than treating them as discrete units this dissertation strove to integrate these guiding frameworks into one overarching narrative. I felt this to be a necessity as the world is often not easily divided into disconnected components. In my conclusions however, I will present the findings of each of these theoretical lenses in order to make specific contributions to the work carried out within these areas. The final component of this discussion will be devoted to looking at Spanish flu as a predictive event. I will take the lessons learned within this dissertation and apply them to the Ontario response to the 2009 H1N1 threat.

The Toronto Pandemic Experience in a Nutshell

In all, Toronto displays the usual characteristics of the pandemic experience in major Western cities. The severe part of the epidemic lasted approximately five weeks (9 October 1918 - 2 November 1918) with an overall crude mortality rate of 4.1 per 1000, resulting in 2177 deaths from 1 September to 31 December 1918. Mortality appears to have been slightly higher among males than females and is characterized by the typical 'W' shaped mortality profile, with the highest number of deaths occurring

in individuals between the ages of 20 and 40. Initial evidence suggests that the disease did not demonstrate social neutrality, resulting in death disproportionately among individuals engaged in lower paying occupations. The disease appears to have been introduced into the city via infected military personnel either being shipped out to, or returning from the front. Although public health measures had a hand in reducing influenza mortality, the findings of this analysis are in line with Jones' (2007) conclusion that the majority of the population's health needs were met through "charity, volunteerism, and women's caring labour."

Findings Derived From a Political Economy Framework

Of the three paradigms adopted for this analysis, political economy is perhaps the most salient. This theory directed this dissertation toward an understanding of the relationship between the war effort and the trajectory of the influenza virus. Although it was my original intention to establish this work around a traditional application of this paradigm, how political and economic institutions limit individual autonomy, a novel use of a political economy perspective was also presented. Instead of simply focusing the negative effects that such hegemonic institutions have on the regions they influence, this dissertation also highlighted how political and economic institutions can improve the efficacy of community-based organizations.

Within a classical application of a political economically oriented analysis it has been demonstrated here that the continued war effort placed Toronto residents in a relationship with the Canadian government that fostered a state heightened vulnerability. Much like Hastings' representation of altruistic nurses and doctors, who put the needs of the suffering over that of their own, highly patriotic Torontonians

sacrificed provisions, autonomy, and welfare to support the Great War. The necessity of men to become soldiers and women to shift to heads of household prompted entrance into a stage of liminality. This status incongruity could have led to a significant degree of chronic stress that may have impeded immune function, thereby leaving residents at greater risk for contracting the virus. The conditions created by the war also impacted residents' vulnerability to infection and adverse reaction by enhancing nutritional and labor shortages as well as exposing residents to the physical realities of active combat, disrupting social networks and contributing to varying degrees of diminished autonomy.[148]

One of the other major contributions of this analysis is that it adds to the growing body of literature that conclusively demonstrates the relationship between troop movement and virus transport (Crosby 1989; Ewald 1994; Herring and Padiak 2008; Humphries 2005; Oxford 2001). Material collected from Library Archives Canada is highly suggestive that the movement of infected men from Niagara to Toronto was the route by which the virus entered the city.

From an economic standpoint there is suggestive evidence that demonstrates that socioeconomic status altered an individual's ability to deal with both the virus and measures put into place to inhibit pathogen virulence. Although never explicitly stated, the decision not to impose quarantine or enact mandatory reporting may have been driven by economic necessity. Prior to the epidemic the public health department was struggling. Although Hastings, the Minister of Health, had managed to increase the organization's base staff, they still were not in a position to adequately handle the

[148] These ranged from sanctions on grieving to forced conscription.

demands of the rapidly expanding city. As such, personnel were not available, both as a result of sickness and insufficient staffing, to uphold these measures.

Current research has also demonstrated that the decision to close schools creates different social and economic burdens for individuals from differing socioeconomic statuses (SES). Dr. McCullough, Ontario's Chief Health Officer, warned that without restrictions on movement, children might be at greater risk for contracting influenza if removed from the controlled atmosphere of educational institutions. Although not possible to examine in historical context, interviews conducted after more recent school closures in response to influenza infection, demonstrate that individuals from 'underprivileged backgrounds' are disproportionately disadvantaged both economically and ethically by this form of intervention (see for examples Cauchemez et al. 2009; Rutkowski 2010; Gift et al. 2010; Blendon et al. 2008; Johnson et al. 2008; Lempel et al. 2009). Hastings' mandate of municipal compensation to working mothers who, through necessity, were required to stay home and nurse sick children provides suggestive evidence that the economic burden on low SES households may have been palpable.

Although this work is nowhere near as definitive as studies conducted by Mamelund (2006) and Herring and Korol (2010), there is suggestive evidence that employment may have been a contributing factor for mortality. By looking solely at occupation by income it appears that the two employment arenas in which individuals were likely to bring home the lowest weekly income, working within the agricultural sector and being engaged in jobs relating to construction, suffered the highest proportionate mortality. Furthermore, individuals whose occupations brought them in

continued contact with the sick (e.g., housewives (likely acting as volunteer aids) and nurses) or whose workplace fostered continued population aggregation (e.g., school children and soldiers) may have also placed individuals at an increased risk for dying.[149]

The novel application of political economy theory in this dissertation concentrates on how political and economic institutions increase individual autonomy. Much of this discussion is born out of a reaction to Jones' (2007) work devoted to understanding the middle class experience of the pandemic in Winnipeg, Manitoba. Throughout her work she laments the class and cultural divide within the city. She notes that

> [t]he failure by municipal health authorities to invite the full participation and cooperation of immigrant and worker organizations in the fight against influenza … [is] a further indication of the attitudes dominating city hall. The state took an expert-driven, top-down approach to disease control, rather than one based upon community action and grassroots participation (Jones 2007: 169).

Hastings was very much a man of action more concerned with results than politics; as such the majority of his endeavors were aimed at improving the public's health over placating political and private stakeholders. Hastings' plan called for heightened communication and reform. As was outlined in the historical context of this dissertation, he was extremely successful in reducing infectious disease mortality within Toronto. He was also equally triumphant in expanding communication networks and fostering community involvement within the city. He was acutely aware that he lacked the staff to support the growing demands of the rapidly expanding city. To address these challenges he was an active participant in the formation of the

[149] Death registries only afford researchers the ability to comment on mortality. This increased mortality was most likely the by-product of increased morbidity within these populations.

Neighborhood Workers Association, and he funded community initiative and education programs and supported ethnic diversity within the city.[150] Although Hastings firmly believed in the virtues of a germ theory perspective he also respected the contributions of the lay volunteer. During the epidemic he quickly adapted his vision of care to embrace the assistance of these altruistic women's groups, charging local nurses with the task of supervising the partially trained volunteer care providers. There is no question, when attempting to understand the pandemic experience within Toronto, that the fostering of a relationship between civic and private agencies aided Toronto immensely in its 'battle' against influenza.

Adopting a Syndemic Analysis

This dissertation also speaks to the importance and challenges of a syndemic analysis. Clearly the big picture matters, but how does one incorporate all aspects of human suffering into one analysis? The honest answer is that this is not possible. However, adopting this framework challenges researchers to go beyond the obvious in order to fully understand the multiple components that contribute to a disease experience. This framework shaped how the sufferers' experiences were reconstructed within Toronto. This is no more evident than in the chapter relating to the relationship between war and plagues, which is a careful consideration of the numerous factors that may have influenced population health up to and including the pandemic experience. Incorporating features ranging from climate to stress, it paves the way for future research to help identify factors that increase individual risk, as well as provides a more comprehensive roadmap for understanding pandemic influenza mortality.

[150] MacDougall (1990) notes that Hastings attempted to disseminate information in multiple languages to be able to communicate ideas to the largest possible number of recipients.

For researchers interested in the reconstruction of pandemic mortality one of the largest stumbling blocks is the decision of which deaths to include. Some choose to simply incorporate influenza while others call for the inclusion of pneumonia as well. Inclusion criteria can drastically alter pandemic perceptions from mortality rates to timing to the clinical course of the disease. Consider, for example, how inclusion criteria for this project impacted the number of perceived deaths. If we were to examine disease causing death looking solely at deaths listed as influenza, 1142 cases would have been captured. If we relied upon immediate cause of death this number would plummet to 137, leaving us with an overall mortality rate of 2.2 per 1000 or 0.2 per thousand respectively, which is significantly different than the rate of 4.1 per 1000 reported in this dissertation. By adopting a syndemics framework is becomes apparent that the influenza virus alters host susceptibility to pneumonia, so much so that pneumonia may be considered a natural progression of the clinical course of pandemic influenza. These results further reinforce the inclusion of pneumonia mortality in the reconstruction of the Spanish flu experience.

Syndemics also challenged this work to situate this pathogen interaction within the social, political and environmental climate in which it was developed and maintained and encouraged a more holistic understanding of how biological realities shape, reinforce and are transformed by multiple social and cultural phenomenon, an idea which was explored by employing Rosenberg's ideas on epidemic narratives in chapter 6.

Incorporating Evolutionary Medicine

Day (2001) calls into question the validity of Ewald's hypotheses about pathogen virulence being linked to mode of transmission (see Ewald 1992, 1994), suggesting his work is largely based on verbal argumentation rather than observed fact. Although it was an aim of this dissertation to help support Ewald's cultural vector hypothesis and aberrant pathogen virulence by examining the conditions and mortality outcomes of public versus military hospitals, a lack of biological material and morbidity data put this goal out of reach. Historical materials provide evidence of conditions that fostered person-to-person transmission of a virulent pathogen within military installments. This evidence includes documentation of the failure to sanitize ambulances, insufficient accommodations in hospitals, and other factors. Although not amenable to statistical testing due to the absence of a clear population at risk, the inquest into the death of Cadet Davidson (see Chapter 7) as well as other military documentation add to the verbal documentation that underlie Ewald's thought provoking concepts.

Future Directions

The future research potential of this project is exciting. For example, the techniques outlined by Herring and Korol (2010) can be adopted to examine the spatial distribution of influenza mortality within Toronto and the role of socioeconomic and ethnic patterning in that mortality. Additional work is also required to fully contextualize the pandemic experience at Base Camp and within Toronto's permanent

hospital infrastructure.[151] It was not possible to examine the actual report created in response to the Davidson trial in this study,[152] but it may hold valuable insights into fully understanding the conditions of Base Hospital. All hospitals open in 1918 were contacted to ascertain if records of the pandemic had been retained. The Hospital for Sick Children indicated that, with required permission, access to their database may be possible. This could open the way to understanding and contextualizing childhood mortality and morbidity.

In an attempt to address early questions of the impact of informal networks of care, the Jewish population of Toronto was selected as a case study. Although this proved to be beyond the scope of this analysis, as it was first essential to capture the disease experience within the city, early results suggest that future research on this topic is warranted.

Figure 8.1 Toronto's the Ward c. 1910. Source: City of Toronto Archives Fonds 1244 Item 8031

[151] Certain institutions, like the Wellesley Hospital, responded to the epidemic but would not be fully functional for years.

[152] Archivists at Library Archives Canada believe the Davidson report may have been misfiled. A folder of inquest for an unknown individual dated 1929 did exist, but time and logistical constraints rendered it irretrievable.

The Jewish population's mortality rate was 2.1 per 1000[153] despite residing in one of Toronto's most deplorable slums (Figure 8.1). Mercier (2006) found a similar health advantage among the Jewish populations residing in Toronto's the Ward in his analysis of infant mortality at the turn of the century. This population is socially and geographically bounded making them ideal candidates for future study.

Finally, this project can also serve as a predictive and informative tool for future influenza pandemics, as it provides us with a framework by which to plan for and understand future epidemics. A discussion of the most recently recognized influenza pandemic and Ontario's response to that pandemic will be used to illustrate how knowledge of the past can help in dealing with epidemics of the present.

Public Health Response to H1N1; Summary of What Was and What Should Be

On 12 April 2009, the WHO recommended that the Mexican government respond to an outbreak of acute respiratory illness in LaGloria, Veracruz (Cordova et al. 2009). By 23 April the situation had been declared a public health emergency (Pourbohloul et al. 2009) and within hours schools within Mexico were closed and the world was on high alert (Cordova et al. 2009). What had been sequenced was a novel influenza virus. It appeared to be comprised of six genes from triple-reassortant North American swine virus and two genes from Eurasian swine virus lineages (Writing Committee of the WHO 2010). By 28 April the disease had been verified in Ontario (King 2010) and the next day the WHO moved the global pandemic alert for H1N1 to

[153] Between 1 September and 31 December 1918, 71 Jewish deaths can be identified within the city's death registry. According to the 1921 census 34377 Torontonians self-reported their religion as Jewish.

a Phase 5[154] (Fraser et al. 2009). By 4 May Mexico had experienced 29 confirmed H1N1 deaths out of 822 confirmed cases (Fraser et al. 2009).[155] By 11 May it appeared that the situation was under control within Mexico, schools were reopened and people resumed their daily routine, but the rest of the world braced itself for the 'coming plague'.

On 25 May, Ontario experienced its first confirmed H1N1 fatality (King 2010). The rapid global spread of the disease prompted the WHO to raise its pandemic alert from a Phase 5 to a Phase 6 on 11 June 2009.[156] By 17 June, 85 countries had officially reported 39,620 cases of swine-origin influenza and they had collectively experienced 167 deaths (Cauchemez et al. 2009). By June 2010 the majority of pandemic H1N1 influenza activity had waned.[157] Reports from March 2010 demonstrated that almost all countries had reported pandemic activity resulting in 17,700 laboratory-confirmed deaths worldwide (Writing Committee of the WHO 2010). The majority of illnesses consisted of an acute, self-limited disease phase and the highest attack rates were recorded in children and young adults. Overall the case fatality rate was 0.5%,[158] with 90% of deaths occurring in individuals under the age of 65 (Writing Committee of the WHO 2010). Identified risk factors included

[154] The WHO has six pandemic phases with associated descriptions and main actions. Phase 5 indicates that "The same identified virus has caused sustained community level outbreaks in two or more countries in one WHO region" (WHO). At this stage the WHO implements societal, pharmaceutical and individual aid (through leadership and resource allocation). The organization also becomes paramount in aiding the creation of contingency planning and monitoring and assessment of the pandemic threat.
[155] According to Fraser et al. 2009, there were 88 suspected deaths out of a possible 11,356 cases.
[156] "In addition to the criteria defined in Phase 5, the same virus has caused sustained community level outbreaks in at least one other country in another WHO region" (WHO). The main actions undertaken in Phases 5 and 6 are the same.
[157] Active areas of transmission at the time of this report included West and Central Africa, South East Asia and Central America (King 2010).
[158] Represented by a range of 0.0004 to 1.47 per cent.

pregnancy, obesity, and belonging to an economically or socially disadvantaged group[159] (Writing Committee of the WHO 2010).

The H1N1 influenza pandemic experience in Ontario was characterized by two waves. The first took place between 14 – 27 June and the second peaked between 25 October and 7 November (King 2010). The province's pandemic response was focused primarily on prevention, with the implementation of a provincial immunization program the driving force behind the public health response. On 24 September the province announced the schedule for its rollout of seasonal and H1N1 vaccination (King 2010). In just under a month Health Canada approved the vaccine for distribution and by 26 October mass immunizations for top priority groups had begun (King 2010).[160] By 19 November the vaccine was available for all Ontarians (King 2010). In conjunction with the vaccine rollout the province heightened surveillance,[161] reinforced communication networks with local, federal and provincial health networks, stockpiled antivirals,[162] and provided contingency plans to facilitate increased hospital admissions (King 2010).

The response in Ontario elicits recollections from Jones' work in Winnipeg in which an expert driven, top-down approach dominates patient care and pathogen prevention. Although this has proven to be extremely effective with other vaccine preventable diseases (e.g., polio, measles, mumps, tetanus), influenza's high rate of

[159] Within Canada Aboriginal groups were disproportionately adversely affected (Zarychanski et al. 2010).
[160] Individuals given first priority were pregnant women, children between the ages of six months and five years, people living in remote and isolated communities, people under 65 with chronic conditions, health care workers and household contact and care providers for infants under six months of age and individuals who were immunocompromised (Public Health Agency of Canada 2009).
[161] This included tracking calls to TeleHealth Ontario, monitoring hospitalizations and deaths, tracking school absenteeism and diligently observing the international situation (King 2010).
[162] Ontario purchased enough antivirals to treat 25 per cent of all Ontarians (Government of Ontario, Ministry of Health and Long Term Care).

mutation and efficient person-to-person spread suggests that not only do preventive plans need to be adopted but a consideration of what to do if prevention fails also needs to be considered. Even within a city as large as Toronto with unrestricted resources it still took seven months to manufacture and distribute the mass quantities of vaccine required to induce herd immunity and limit influenza morbidity. Furthermore the vaccine was not available to the general public until well after peak of the second wave had been experienced.

If the 1918-19 influenza experience in Toronto tells us anything, it is this—in a situation where the onset and spread of illness is rapid, access to basic care becomes paramount. In order to rally the support, necessary relationships with the community must already be in place (as demonstrated by a comparison of the disease experience in Winnipeg and Toronto). Public health agencies should make it a priority to uphold the recommendations of Toronto's first Medical Officer of Health, Charles Hastings, and reconnect with local charities and volunteer organizations so in that in the event of an emergency, communication networks and access to additional support are easily available.

This dissertation is a significant contribution to the academic community in that it provides information on a previously understudied city during the epidemic. It serves as a comprehensive guide for future research projects, it clearly identifies rich areas of potential inquiry and provides readers with a better understanding of the factors that increased mortality in the city of Toronto by offering insights into the intimate relationship between pathogens, environments and social circumstance. Finally, it is my hope that this work dispels some of the viral panic that surrounds

influenza and highlights how social support and altruism can go a long way in staving off illness and death.

APPENDIX 1

Table A1.1 All Pneumonia or Pneumonia Derivatives Listed in Toronto Death Registries Examined From 1 September to 31 December 1918

Cause of Death (ICD and DCD)	Number of Cases Recorded
pneumonia	616
bronchopneumonia	294
lobar pneumonia	81
pleuro pneumonia	15
double pneumonia	11
acute lobar pneumonia	7
pneumonia following influenza	7
double lobar pneumonia	6
tuberculosis bronchopneumonia	6
septic pneumonia	4
acute bronchopneumonia	3
pleural pneumonia	3
whooping cough and bronchopneumonia	3
broncho and lobar pneumonia	2
acute pneumonia	2
diphtheria + bronchopneumonia	2
pneumonia and diphtheria	2
pneumonia and influenza	2
probably pneumonia	2
s. fever and bronchopneumonia	2
septicaemia and bronchopneumonia	2
acute pluropneumonia	1
acute pneumonic tuberculosis	1
bilateral bronchopneumonia	1
bilateral lobar pneumonia	1
broncho pleuro pneumonia	1
debility – bronchopneumonia	1
endocarditis and pneumonia	1
enterios colitis and pneumonia	1
following pneumonia	1
gastro enteritis and broncho pneumonia	1
general eczema and bronchopneumonia	1
left lobar pneumonia	1
old age bronchopneumonia	1
peritonitis and bronchopneumonia	1
pneumonia and arteries sclerosis	1

pneumonia and croup	1
pneumonia and heart failure	1
pneumonia and nephritis	1
pneumonia and old age	1
pneumonia following labour	1
pneumonia following operation	1
pneumonia following Spanish influenza	1
pulmonary tuberculosis acute pneumonia	1
scarlet fever and broncho pneumonia	1
senile pneumonia	1
septicaemia with bronchopneumonia	1

Table A1.2 All Influenza or Influenza Derivatives Listed in Toronto Death Registries Examined From 1 September to 31 December 1918

Cause of Death ICD and DCD	Number of Cases Recorded
Influenza	1192
influenza and pneumonia	142
Spanish influenza	92
influenza and broncho pneumonia	40
epidemic influenza	15
influenzal pneumonia	14
influenza followed by pneumonia	9
influenzal broncho pneumonia	8
influenza with bronchopneumonia	6
Flu	5
probably influenza	5
acute influenza	4
influenza followed by bronchopneumonia	4
influenza with pneumonia	4
S. influenza	4
whooping cough and influenza	4
grippe	3
influenza and lobar pneumonia	3
acute influenza type Spanish	2
flu and pneumonia	2
influenza ‘Spanish”	2
influenza and miscarriage	2
influenza followed by double lobar pneumonia	2
acute influenza pneumonia	1
alcoholism and influenza	1
cancer and influenza	1

complicating influenza epidemic bronchopneumonia	1
convulsions following influenza	1
diphtheria, influenza	1
effects of influenza	1
epidemic influenza and bronchopneumonia	1
flu followed by pneumonia	1
flu: blood poisoning	1
Flue	1
flue and double pneumonia	1
flue and pneumonia	1
grippe and pneumonia	1
heart failure after influenza	1
inanition with influenza called when dying	1
infection from mother with flue in childbirth	1
infl pneumonia	1
influenza – stress birth baby week	1
influenza (pregnancy)	1
influenza (probably)	1
influenza (Spanish epidemic)	1
influenza (Spanish) + pneumonia	1
influenza and bronchitis	1
influenza and congestion of brain	1
influenza and convulsions	1
influenza and dilated hart	1
influenza and diphtheria	1
influenza and double pneumonia	1
influenza and empyema pneumonia	1
influenza and endocarditis	1
influenza and folliculitis	1
influenza and heart disease	1
influenza and influenzal pneumonia	1
influenza and lobar pneumonia	1
influenza and lung hemorrhage	1
influenza and mascoma	1
influenza and pleuro pneumonia	1
influenza and pneumonia and diaphragmatic pleurisy	1
influenza and whooping cough	1
influenza complicated with bronchopneumonia	1
influenza complicating aotic stenosis	1
influenza complicating labor	1
influenza effects	1

influenza epidemic	1
influenza following rheumatism	1
influenza hemorrhage	1
influenza pneumonia mial serais	1
influenza sporadic	1
influenza with pleurisy	1
influenza, bronchopneumonia and empyema	1
influenza, bronchopneumonia and pensilluar abscess	1
influenza, pneumonia and pleurisy	1
infuenzal bronchopneumonia and icterus	1
la grippe and toxic poisoning with ganue inflammation	1
la grippe causing rhaumaitis nosopharyngitis and compumtivitis	1
marasmus and la grippe	1
meningitis caused by influenza bacillus	1
meningitis flu	1
old age and influenza	1
prenatal “flu”	1
probably Spanish influenza	1
pruena galley influenza	1
result of influenza in mother	1
scarlet fever and influenza	1
Spanish flu meningitis	1
Spanish influenza and pneumonia	1
Spanish influenza followed by dbl pneumonia	1
toxemia following influenza	1
when called history of la grippe	1

APPENDIX 2

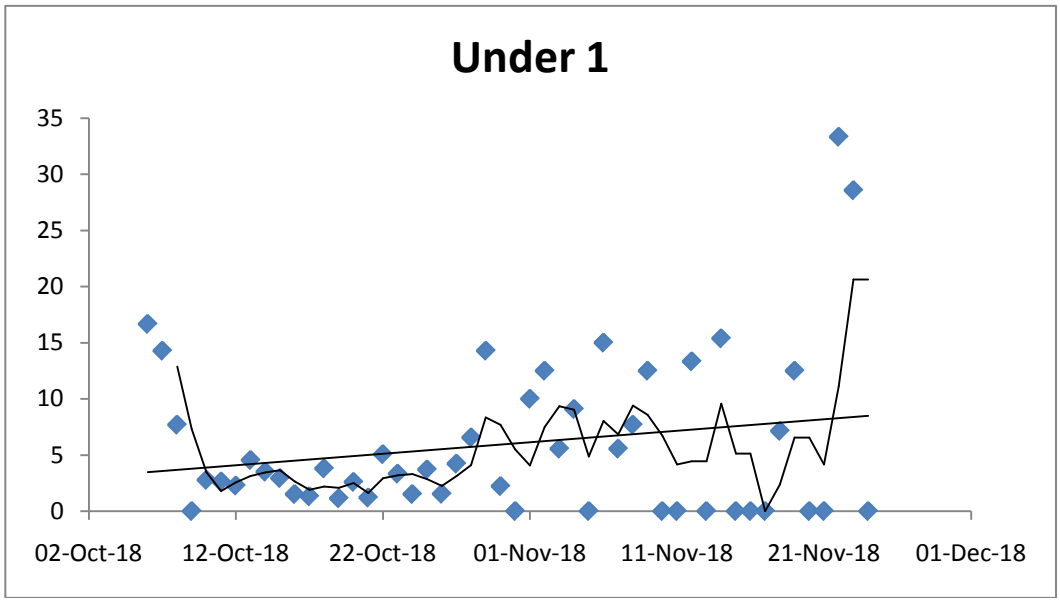

Figure A2.1 Proportionate Mortality of Individuals Aged 0-1, 8 October to 18 November 1918

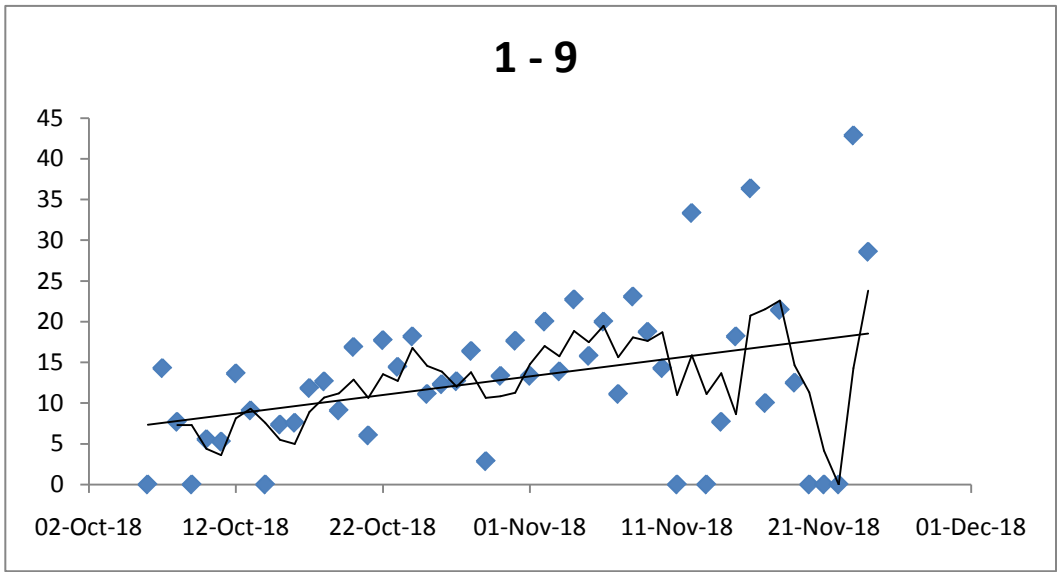

Figure A2.2 Proportionate Mortality of Individuals Aged 1-9 1, 8 October to 18 November 1918

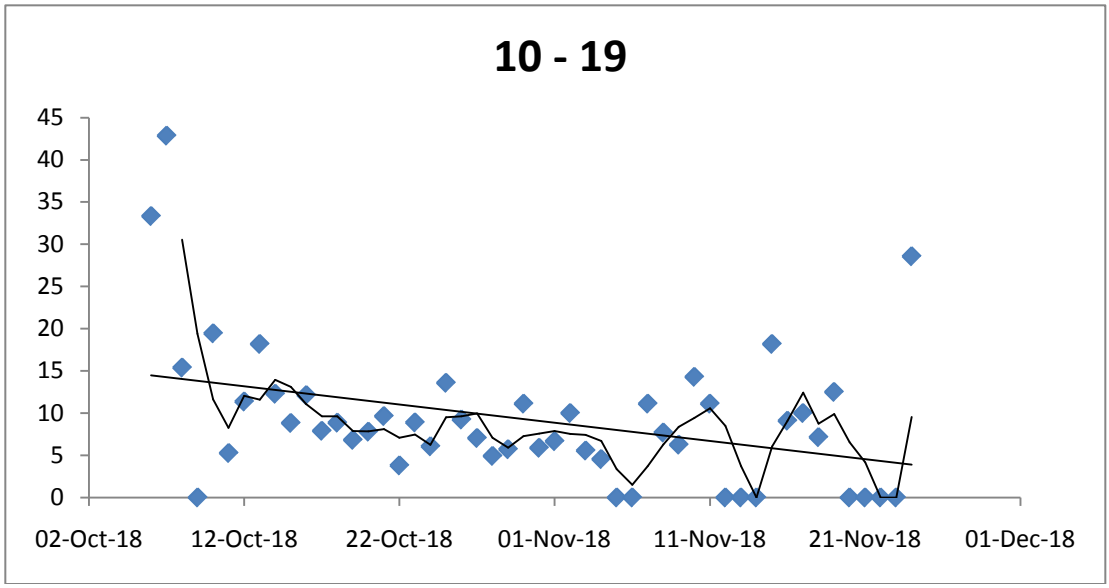

Figure A2.3 Proportionate Mortality of Individuals Aged 10-19, 8 October to 18 November 1918

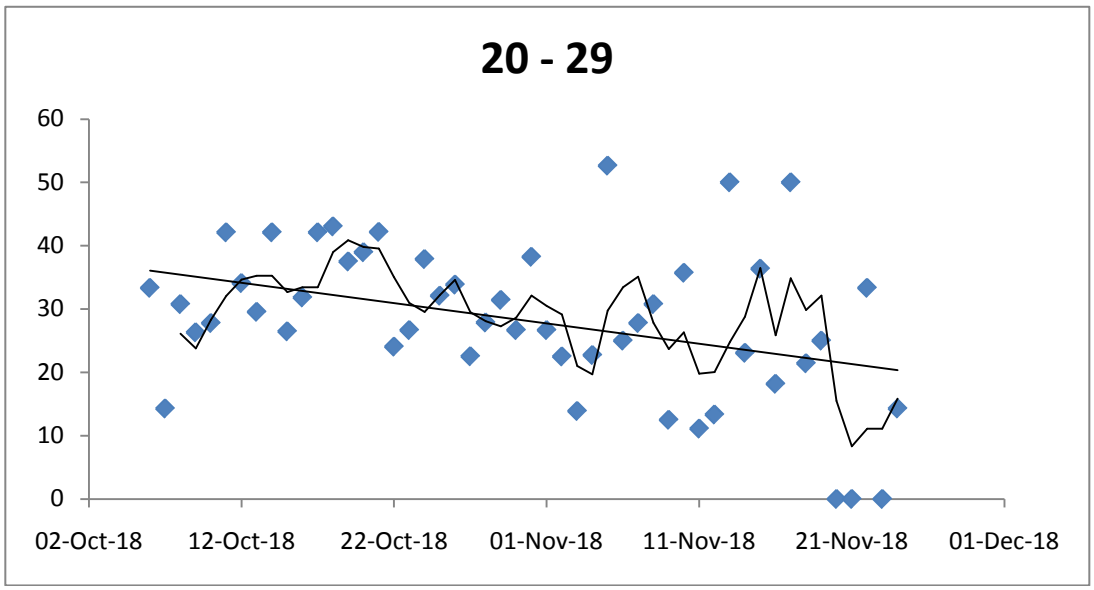

Figure A2.4 Proportionate Mortality of Individuals Aged 20-29, 8 October to 18 November 1918

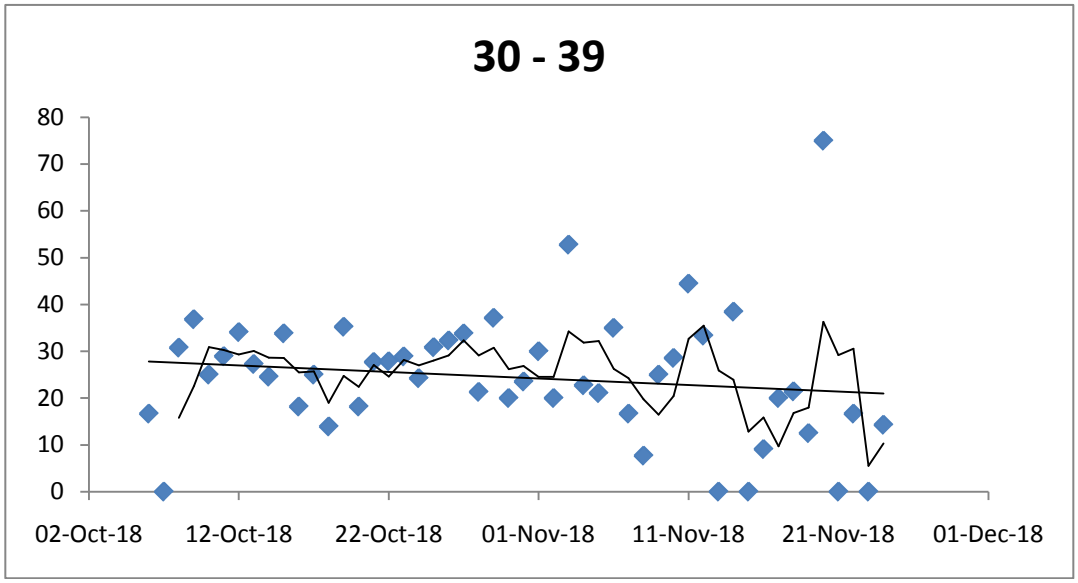

Figure A2.5 Proportionate Mortality of Individuals Aged 30-34, 8 October to 18 November 1918

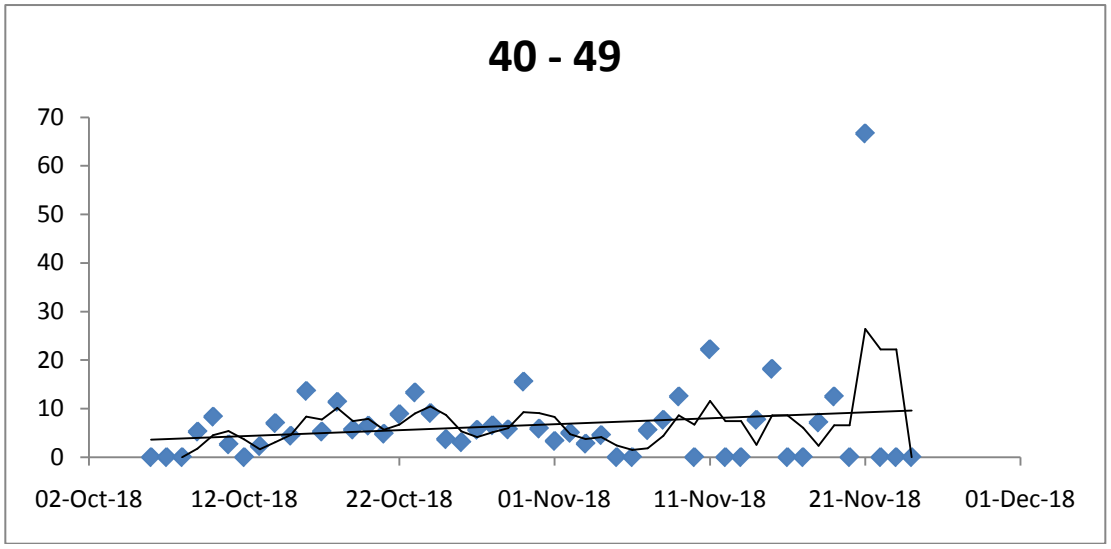

Figure A2.6 Proportionate Mortality of Individuals Aged 40-49, 8 October to 18 November 1918

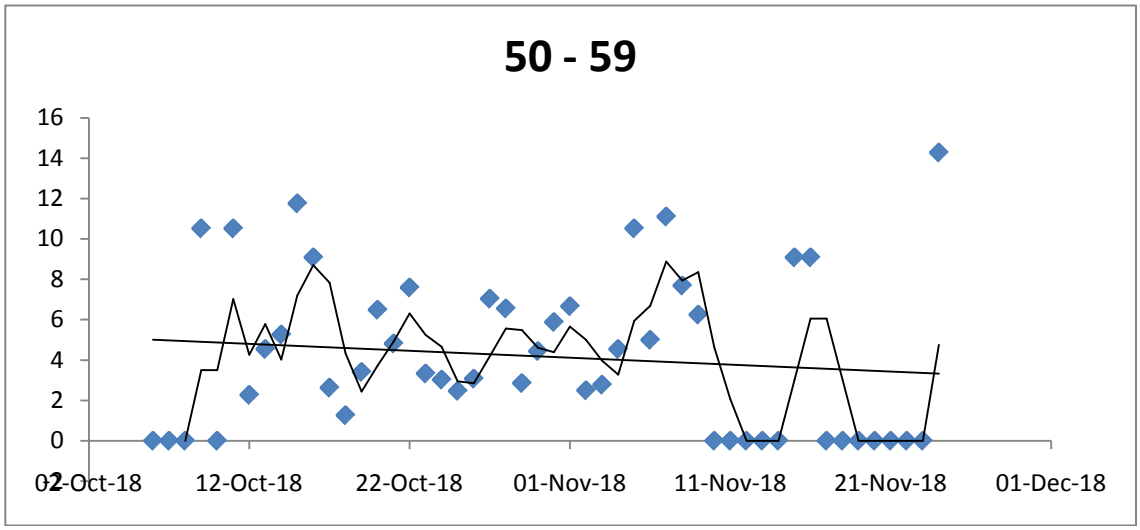

Figure A2.7 Proportionate Mortality of Individuals Aged 50-59, 8 October to 18 November 1918

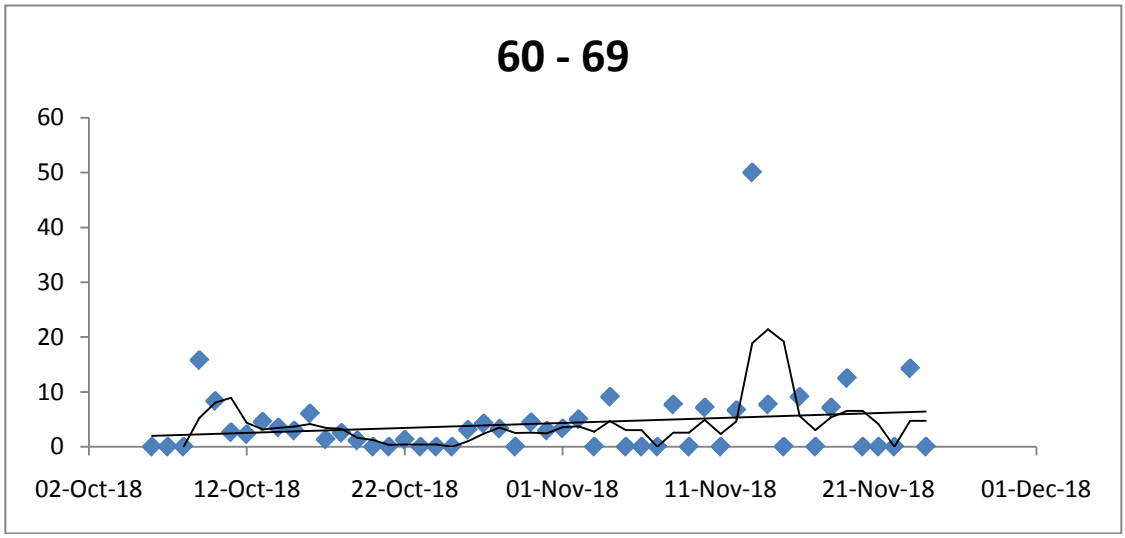

Figure A2.8 Proportionate Mortality of Individuals Aged 60-69, 8 October to 18 November 1918

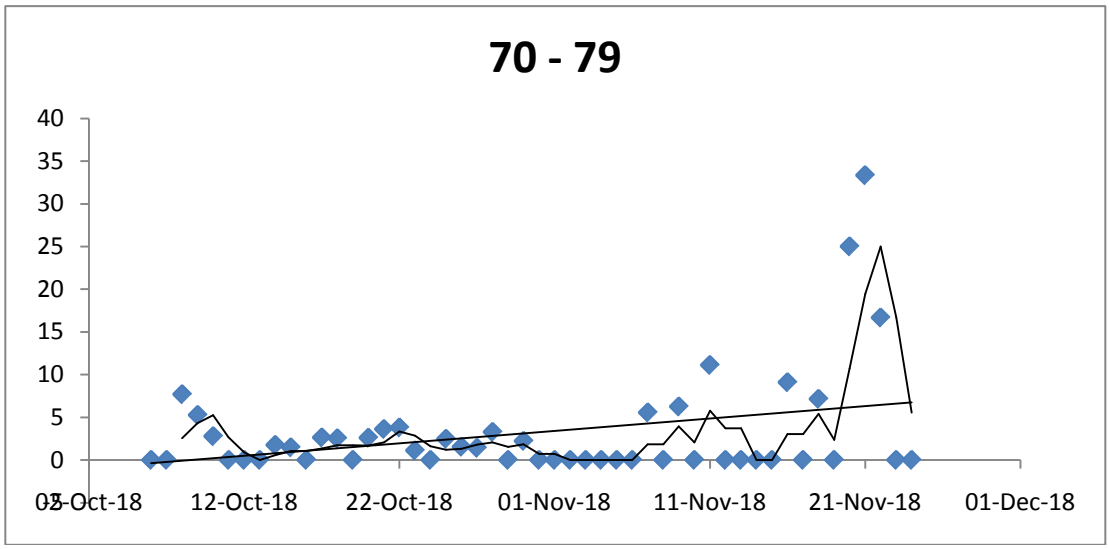

Figure A2.9 Proportionate Mortality of Individuals Aged 70-79, 8 October to 18 November 1918

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VITA

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