HISTORICAL POPULATION HEALTH: SPATIOTEMPORAL MORTALITY PATTERNS OF HAMILTON, ONTARIO 1880–1882 AND 1910–1912

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ABSTRACT

This dissertation empirically investigates multifaceted causes of health inequity by exploring historical connections between shifting economic activity, urban development, population change, and mortality. The purpose is to reveal the impact of changing socioeconomics on population structure and urban development, and the association of this impact on spatiotemporal mortality patterns. This research advances knowledge on the complex ecological interplay of population, behaviour, habitat, and subsequent health inequities, revealing a link between health disparities and economic transitions.

Examining Hamilton, Ontario, at two cross-sections (1880–1882 and 1910–1912), using purposive working-age and infant samples provides a snapshot of life and death before and during heavy industrial activity. Mixed-methods use data from census and death records, health reports, photographs, and maps to construct a profile of demography, epidemiology, and the urban environment (physical, built, and social). Two major findings are identified: 1) industrialization played a major role in emerging human health ecology risks correlating to the mortality patterns; and 2) industrialization tended to increase health inequities amongst the population and across the city.

The onset of predominately industrial economic activity caused further class divide, uneven urban development, and inequitable health outcomes. Statistical inquiry (multiple logistic regression) of the working-age mortality sample revealed an increased association between age at death and dying of tuberculosis, and an increased association between accidental death with biological sex, age at death, and birthplace. The infant mortality sample revealed a temporal shift with an increased association between diarrhoeal mortality and infant age, and between infectious respiratory mortality and infant age. Results from Historical GIS inquiry indicate residential working-class sections were more unhealthy environments than other areas of

the city. Historical documentation and photographs supported these results, presenting the likelihood of a social disparity to health outcomes.

The interplay between population, environment, and behaviour manifests into a spatiotemporal pattern of stressors related to socioeconomic status, urban development, and health disparity. Industrialization brought new stressors to Hamilton creating unequal opportunities for the rapidly growing working classes. Thus, without careful planning in urban development, concentrations of health risks lead to inequitable population health outcomes, especially those undergoing an economic transition, such as industrialization.

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CHAPTER 1
INTRODUCTION

1.1 Context

Historical health research can expose the multifaceted causes of health inequity both in the past and present, advancing the argument for more historical research in the social and health sciences. *Health inequities* are defined as inequalities that are unfair and avoidable between groups of people and are often related to social and economic conditions, and the effects of these conditions on health risks (Commission on the Social Determinants of Health, 2008). Such health inequities can leave a marked spatial pattern in an urban area. Thus, a comprehensive examination on the health of a city requires an understanding of the spatial distribution of the population. Spatial distribution of a population is determined by various aspects of the urban environment, such as the connection between an economic system, an urban structure, and the infrastructure (development). Since the urban environment is closely associated with health (Bunting, Filion, & Walker, 2010; Luginaah, 2009), urban planning—the regulation of urban development—can affect population health. Researchers in both population health and urban planning have acknowledged the importance of history in their respective fields, but note a lack of engagement in historical research (Campbell & Campbell, 2007; Commission on the Social Determinants of Health, 2008; Etches, Frank, Di Ruggiero, & Manuel, 2006; Filion & Bunting, 2010). Yet, envisioning the city as an unhealthy environment—as an attribute to the health of the population—has been extensively researched in historical epidemiology (Burke & Sawchuk, 2003; Emery & McQuillan, 1988; Hautaniemi, Swedlund, & Anderton, 1999; Herring & Korol, 2012; Luckin & Mooney, 1997; Sawchuk & Burke, 2000; Sawchuk, Burke, & Padiak, 2002;

Sawchuk, Herring, & Waks, 1985). Human health ecology is the theoretical foundation found in much work in historical epidemiology (Burke & Sawchuk, 2003; Hautaniemi et al., 1999; Luckin & Mooney, 1997; Sawchuk et al., 1985). This theoretical foundation infers that health interacts at various points: population, behaviour, and habitat. Thus, historical epidemiology offers tools to, retrospectively, examine complex causes of health inequity in an urban environment.

Historical epidemiological studies use mortality, coupled with additional sources of information, to examine health risks to the population (Burke & Sawchuk, 2003; Hautaniemi et al., 1999; Herring, Abonyi, & Hoppa, 2003; Herring & Korol, 2012; Janjua, 2009; Pelletier, Légaré, & Bourbeau, 1997; Sawchuk, 1993; Sawchuk & Burke, 2000; Sawchuk & Burke, 2003; Sawchuk et al., 2002; Sawchuk et al., 1985; Swedlund & Donta, 2003). Mortality is an obvious indicator of population health and is a health outcome measurement in epidemiology (Dicker & Gathany, 2007; Dicker, 2008), but it does not provide an indication of the quality of life. Thus, it is important to take a comprehensive approach when using mortality as an indicator of population health, as Swedlund and Donta (2003) suggest. Swedlund and Donta (2003), examined a scarlet fever epidemic using a mortality analysis, provide four domains of interest when undertaking historical epidemiology:

1. Historicity: situating an epidemic in time and place,
2. Medical history: understanding the historical perspective of the epidemic,
3. Epidemiology: using contemporary models to re-evaluate the available data, and
4. Molecular biology: understanding the pathogenesis and genetics of the disease.

In this dissertation, a mixed-method study design is used to analyze health ecological interactions (population, behaviour, and habitat) through empirical historical epidemiological research that comprehensively explores the connections between shifting economic activity, urban

development, population change, mortality, and the social gradient of health. The purpose of this research is to investigate the way that the changing socioeconomic constructs of a society influence population structure and urban development in an historical Canadian context, and how these factors contribute to spatiotemporal variations in mortality patterns.

Cities are resistant to change due to the resilience of urban structure (Filion & Bunting, 2010). Such resilience may preserve spatial disparities in health. These variations in a population's health status are influenced by population and environmental characteristics (e.g. socioeconomic status, identity, gender, and geographic location) that determine health (Public Health Agency of Canada, 2011). The social determinants of health are defined as a wide set of conditions that shape life, as people are born, grow, work, live, and age (World Health Organization, 2010). These conditions affect the health of individuals and tend to fall within three overarching sets of determinants, reflecting: 1) the social and economic environment; 2) the physical environment; and 3) the characteristics and behaviours of an individual (World Health Organization, 2010). There are a variety of frameworks used to address the social determinants of health, but all include a multi-level causal pathways approach: from the individual, community, organization, government, to the environment (Canadian Council on Social Determinants of Health, 2015). The Public Health Agency of Canada list determinants of health, which include: biology and genetic endowment; culture; education and literacy; employment and working conditions; gender; income and social status; health practices and coping skills; health services; healthy child development; physical environments; and social environments (Public Health Agency of Canada, 2011). The determinants correspond to the interactions found in the human health ecology model (Figure 1.1). Although this dissertation does not cover all of the

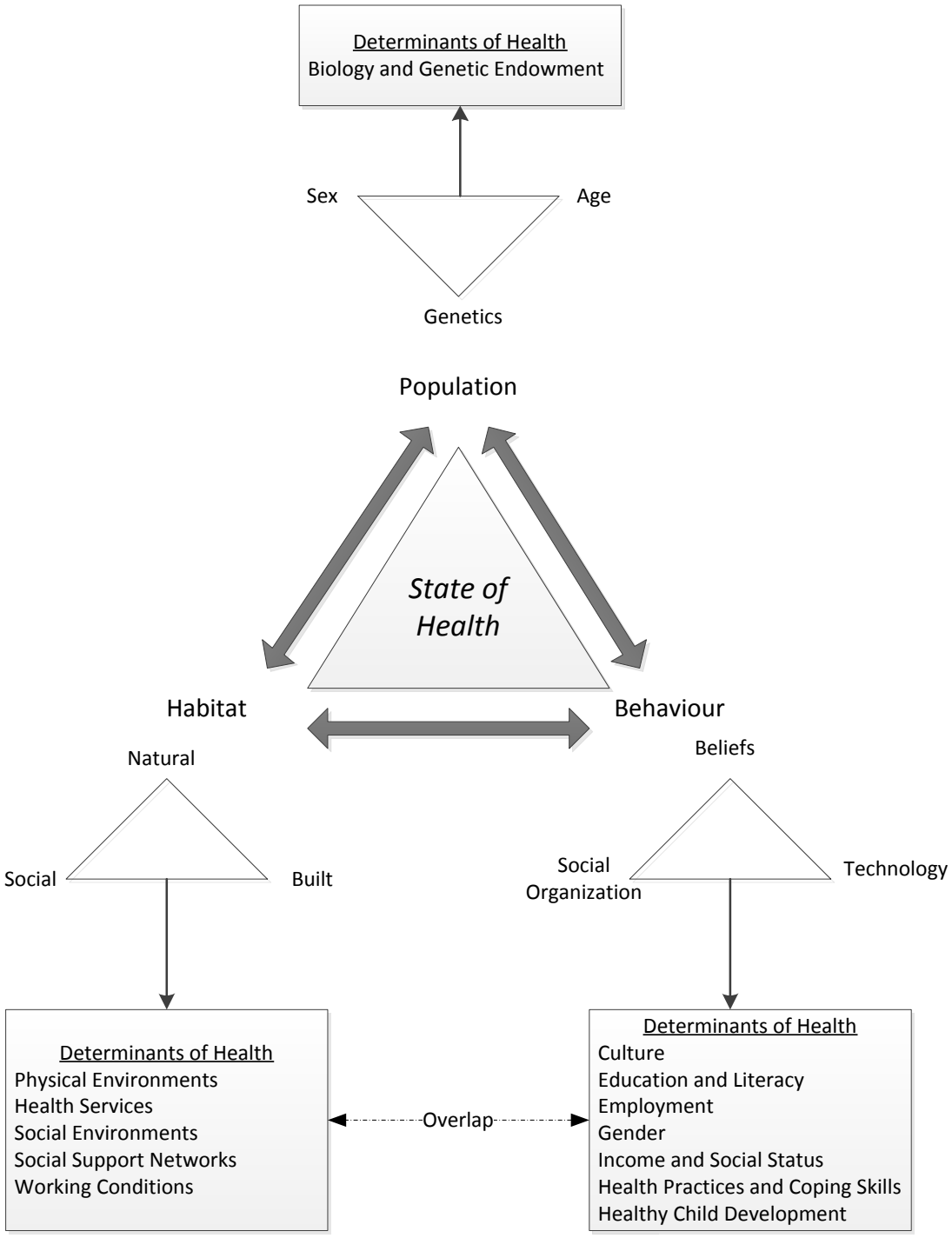


Figure 1-1. The Triangle of Human Ecology and Corresponding Determinants of Health (as adapted from Meade & Emch, 2010; Public Health Agency of Canada, 2011)

determinants of health, it is important to note the interconnectedness of these determinants. Such interconnectedness is the basis of human health ecology, because populations, their behaviours, and their environments govern the state of health for a given community.

Although a city's physical structure might be resistant to change, a transition in a city's social and economic structure will likely affect its demographic and epidemiologic characteristics. For example, early industrialization introduced new stressors to a city, which heightened intra-urban variation in both population structure and development, often manifested by variable overcrowding, class segregation, and an uneven distribution in the presence or quality of urban infrastructure (Burgess, 1967; Hautaniemi et al., 1999; Heron, 1988; Ludlow & Burke, 2012; Pelletier et al., 1997; Zunz, 1982). Both the first and second industrial revolutions brought about a widespread application of modern science. The second revolution specialized in production and heavy manufacturing on a national and international scale, and the emergence a new social and occupational class of capital, which advanced economic life (Deane, 1979; Landes, 2003). Although industrialization has made significant advancements on economy, technology, and society, understanding the health implications of a major developmental transition is necessary to ensure the use of best equitable urban development practices during economic transitions that lead to rapid population growth and urban development.

Current population health sciences developed from historical public health and urban planning approaches that took an ecological approach towards health and emphasized building healthy cities (Corburn, 2007; Frank & Mustard, 1994; Vlahov, Gibble, Freudenberg, & Galea, 2004). Cities in the past were seen as pathogenic and noxious. This "unhealthy city" image was reinforced as the spatial location of industry in the urban landscape later encouraged the development of crowded neighbourhoods with a predominately working-class population

(Burgess, 1967; United States Federal Housing Administration & Hoyt, 1939). At the turn of the 20th century, urban planners and public health officials began to work towards a common conceptual goal, the "healthy city" (Condran & Crimmins-Gardner, 1978; Corburn, 2009; Feldberg, 1995; Pelletier et al., 1997; Szreter, 2003; Woods & Hinde, 1987). Incorporating an historical population health and urban planning perspective can lead to further insights into the connection between historical urban development and the manifestation of past spatial health inequities, inequities that may even persist in current urban environments.

Causes of health disparities are multifaceted and thus require an ecological lens that analyzes the interactions between population, behaviour, and habitat. Historical mortality studies can help identify such connections. For example, Sawchuk et al. (1985) found that differences in infant mortality rates in Gibraltar from 1870-1959 between Jewish and Non-Jewish populations was related to weaning practices. Later weaning for Jewish infants protected them from bacteria in the water supply and unpasteurized milk that was harming non-Jewish infants (Sawchuk et al., 1985). Burke and Sawchuk (2003) also found that culturally-determined gender roles led to increased tuberculosis susceptibility among women after childbirth. According to Hautamiemi et al. (1999), industrialization in North America during the mid-19th century caused a spike in infectious disease mortality due to rapid population increase and the lack of public health infrastructure. Ludlow and Burke (2012) found that, historically, single-industry urban locales in Nova Scotia created a capitalist/labour dynamic in company towns that caused poor working conditions and poor living conditions, which, in turn, significantly affected the rates of tuberculosis and accidental mortality for the working-class. Although some class integration was found in Detroit neighbourhoods between 1880 and 1920, Zunz (1982) found urban development had a marked spatial inequality, connected to immigration status, ethnicity, class, and the

distribution of public services. While municipal water supply was accessible throughout Detroit, providing service improvements in working-class areas was a low priority for the municipal government, leading to spatial differences in the quality of the supply. Thus, the east side constantly lagged in public improvements compared to the west side (Zunz, 1982). These examples show that historical epidemiology can identify the plausible interplay of the ecological interactions that may contribute to health inequity.

Elements of space, place, and time are an important backdrop to understanding historical population health patterns. Space and place are key concepts in geographical studies because they both define locational phenomena across various sub-disciplines in geography. Human geographers are particularly interested in the interaction between humans and their environment and how this interaction affects human ecological experience (Cutchin, 2007; Dunn, Frohlich, Ross, Curtis, & Sanmartin, 2006; Dunn & Hayes, 2000; Goodchild & Janelle, 2004; Luginaah, 2009; Pampalon, Hamel, & Gamache, 2010; Vaughan, Clark, Sahbaz, & Haklay, 2005). Space emphasizes the importance of location on a given place relative to other on the distributions of patterns of one phenomenon in relation to another phenomenon (Baker, 2003; Goodchild & Janelle, 2004). Place, on the other hand, emphasizes the societal and cultural importance of a phenomenon within a given area, accounting for the complex association of people and their culture (Agnew, 1997; Anonymous, 2006; Goodchild & Janelle, 2004). Together, space and place present a dyadic representation for understanding how and why a phenomenon occurs in one location and not another. Using an urban space and place approach to health alongside an understanding of economy and society can show the effects of new urban landscapes on the health of the population. Disparities in health can assume a spatial pattern, due to a manifestation of inequalities in the environment (physical, social, and built). While space is

likely to reveal insights into health disparities between locations, place, however, can reveal societal or behavioural differences of such disparities between populations and, when combined with spatial analysis, provide a better understanding of the complex interplay that manifests into healthy or unhealthy locales.

1.2 The Healthy City: Binding the Disciplines

The history of population health is limited. In Canada, the concept of population health first appeared from Lalonde's (1974) report, *A New Perspective on the Health of Canadians*. Although the concept is not new, coming to a consensus on whether population health was a new field of study or an approach to public health remained a topic of discussion into the early 2000s (Kindig & Stoddart, 2003). The Public Health Agency of Canada (2011) defines *population health* as an approach that aims to improve the health of the entire population and reduce health inequities among population groups. Population health is considered to be a 'unifying force' that focuses on the interrelated conditions and factors that influence health, building on the public health and health promotion tradition (Public Health Agency of Canada, 2011). Public health tends to focus on prevention of disease through organized societal efforts (e.g. programs, services, and policies) (The Association of Faculties of Medicine of Canada, 2007). A key difference between public health and population health is that population health addresses a health issue from a broader lens, linking multiple disciplines from the biological to sociological sciences (The Association of Faculties of Medicine of Canada, 2007) whereas public health focuses on specific disease-prevention strategies. Thus, population health essentially takes a human ecology focus to understanding health outcomes by attempting to understand the interrelationships between humans and their physical, social, and built environments.

While population health is relatively new, compared to public health, public health concerns found in the historical record bridge an understanding of the inequities in the population and their connection to the physical, social, and built environment. Historically, public health, like population health, held a human ecology position in that the health of the population was intertwined with the health of the city (Hebbert, 1999; Szreter, 2003). Today, *Healthy Cities* is an approach defined by political and social agendas and is a strong movement for public health to create equitably healthy social and physical environments that address the determinants of health (World Health Organization, n.d.-b).[1] Although the term *healthy city* was not used until the 1980s, the concept of building healthy cities was in place. Building healthy cities and the role of the public health official rose as a consequence of the onset of industrialization, as cities notorious for being unhealthy and unsanitary spaces worsened under the industrial movement (Szreter, 2003). Publications, such as Chadwick's 1842 report, describe unsanitary conditions associated with the labouring population as hosts for infectious disease transmission (Ashton, 2009; Chadwick, 1842; Hautaniemi et al., 1999). As public health concerns spread throughout cities in the Western hemisphere, urban planning developed to build healthier cities (Corburn, 2004, 2007, 2009). Thus, public health and urban planning developed out of the need to improve the urban environment and, in turn, improve health for the population.

The following literature review shows how patterns of health were influenced by spatial patterns in urban development, socioeconomic conditions, and population structure. Historical

[1] The Healthy Cities approach was a programme developed in the 1980s, from which the Healthy Cities concept derived (World Health Organization, n.d.-b). The term *healthy cities* used in this dissertation refers to the concept, not the programme.

epidemiology, using a comprehensive health ecology approach, provides information on the historicity, medical history, epidemiology, and molecular biology that help decipher the complex factors that influence the overall health of a city and patterns of health inequity. In an historical epidemiological study, historical urban planning and population health can contextualize a city's environment, risks associated with the state of urban health, and the population at risk of those associated risks.

1.2.1 Urban Planning and the Healthy City

Urban planning is influenced by a system of beliefs that vary over time and across space which will influence development decisions (Fischler, 2000). Thus, development decisions are determined by the social, political, and economic influences that make up the system of beliefs. Understanding these underlying concepts can help to explain the variations and reasoning behind past and present urban planning decisions. One of the major factors in historical urban planning decisions was the desire to reduce uneven health outcomes caused by infectious diseases.

Historically, a clear connection between urban planning and public health emerged in the late 1800s, giving rise to the healthy cities movement in urban planning and public health. The late 19th and early 20th centuries was a period of industrial expansion in cities throughout North America and Europe, following on the rise of the Industrial Revolution beginning in Britain in the mid-18th century and diffusing outward. Infectious diseases increased during this period, spreading rampantly throughout predominately working-class section of urban areas, often because of overcrowding, abysmal water quality, a lack of adequate sewage and refuse disposal, poor food handling, and poor housing infrastructure (Vlahov et al., 2004). The spread

of typhoid fever, cholera, and tuberculosis in industrializing cities, for example, has been linked to unhygienic and unsanitary urban living conditions (Barkin & Gentles, 1990; Gagan, 1989; Leavitt, 1992; Sawchuk & Burke, 2000; Sawchuk & Burke, 2003). Increased knowledge of disease pathology with the onset of germ theory in the 1880s led to urban planning and public health movements at the turn of the 20th century. These movements focused on improving infrastructure by connecting water and sewer lines to houses, increasing housing standards (especially in regard to size and ventilation), and decreasing overcrowding of persons living in houses (Corburn, 2007; Cutler & Miller, 2005; Vlahov et al., 2004). In some cases, these concerns spurred concrete actions. Some diseases, such as typhoid fever and cholera infantum (or infantile diarrhoea), were directly mitigated by public health movements and showed large declines with improved public health infrastructure (particularly water and sanitation supply infrastructure) (Condran & Crimmins-Gardner, 1978).

Spatial differences in public health infrastructure and patterns of health were common throughout industrializing cities, revealing an uneven distribution of the health threats to the population. In the 19th century, a mortality analysis revealed that the cholera epidemic in Montréal and Quebec City mainly affected working-class neighbourhoods because these areas lacked the standard of sanitation and drinking water found in more affluent sections of the cities (Pelletier et al., 1997). In 1900, rates of tuberculosis and typhoid fever in Hamilton, Ontario, were highest in the densely populated areas surrounding production centres, with higher prevalence amongst the poor working-class population, who lacked adequate water supply and sewage systems (Gagan, 1989). In Cutler and Miller's (2005) study of mortality in United States in the early 20th century, they also found a health divide between cities that implemented clean water technologies and those that did not. The authors noted that the ad

hoc timing for adoption of clean water technology throughout American cities was influenced by the differences in beliefs about the cause of disease (miasmas or germ theory) and the difficulties sanitarians had in persuading city council to take action against poor water quality (Cutler & Miller, 2005). The public health and urban planning initiatives in the late 1800s and early 1900s were directly related to creating healthier cities and often identified practices and standards among the poorer urban populace and their urban environments as the cause of the unhealthy state of the city. Thus, a general improvement in the overall sanitary cleanliness of cities through clean water technologies and supply, proper sewage and garbage disposal, and the movement of livestock outside city limits, and in the cleanliness of city residents through improved hygiene practices, food preparation practices, and milk pasteurization was deemed important for creating healthy urban environments (Condran & Crimmins-Gardner, 1978; Leavitt, 1992).

1.2.2 Urban Environment and Historical Population Health

A key factor causing uneven patterns of health in urban areas appears to be the spatial inequality of the urban environment (access to clean water supply, proper sewage disposal, and well-ventilated low-density living arrangements) and the education of healthy living practices in line with disease aetiology (personal hygiene, milk pasteurization, and food preparation). In addition to this inequality and the occupational hazards found in the cities, working-class families' low wages and close proximity to industrial areas decreased their quality of health and standards of living compared to those living in higher-income sections of the city (Freudenberg & Galea, 2008).

As cities grew, economies grew simultaneously; however these economic developments also generated inequality (Freudenberg & Galea, 2008). Historically, residents living in cities faced increased risks to health compared to those living in towns or rural locales (Luckin & Mooney, 1997; Woods & Hinde, 1987), largely because of the higher rates of infectious diseases in urban locales. This urban/rural health dichotomy likely reflected the increased density and class variation found in cities, coupled with spatial variations in the urban development (such as housing quality, water access, and sewerage) (Anderson, Sorlie, Backlund, Johnson, & Kaplan, 1997; Araya et al., 2006; Hautaniemi et al., 1999; Walks, 2010). These variations in population and development in an urban area may influence disproportionate spatial risks to health.

The heterogeneous nature of an urban environment can create areas where health risks are heightened compared to other parts of the same city. This spatial differentiation was prevalent throughout North American cities in the late 1800s and early 1900s, largely due to spatial differences in living conditions, social class, and economic opportunities (Emery & McQuillan, 1988). The relationship between excess disease burden and internal urban variations was perhaps most evident in the distribution of tuberculosis, a disease that spread in part due to social inequalities. According to a mortality study conducted by Sawchuk and Burke (2000), the spread of tuberculosis in North American cities was partially caused by living conditions characterized by overcrowding, under-nutrition, improper ventilation in homes, and poor personal and domestic hygiene. These conditions would have caused weakened immune systems and provided an efficient mode of disease transmission, increasing susceptibility to not only tuberculosis, but also other infectious diseases. Locational disadvantages within cities can dispose individuals towards poorer health outcomes (Feldman, Warr, Tacticos, & Kelaher, 2009).

Indeed, the existence of locational disadvantages shaped life in cities, and several researchers have identified such poor environments in historical settings. Hillier (2002) found that areas in Philadelphia in the 1930s were colour-coded on insurance maps based on financial risk to insurance companies (a technique known as redlining), with the most hazardous areas, coded in red, which had low income status, poor living conditions, and high ethnic minority concentrations, being denied insurance and financial services. Historical accounts of Winnipeg show spatial ethnic segregation of non-British immigrants in areas with poor quality housing and poor economic opportunities (Artibise, 1975), both key determinants for the development of poor health. New York, by the end of the 19th century, began incorporating 'housing problems' into the planning agenda, because, in poverty stricken areas close to the city centre, tenement housing was notably problematic for disease spread, fire susceptibility, and extreme overcrowding (Beveridge, 2002; Fischler, 2000). Tenements in lower Manhattan were often occupied by immigrant groups and considered to be overcrowded slums (Beveridge, 2002). The cholera epidemics in Quebec City and Montreal during the 19th century occurred predominately in working-class neighbourhoods where water supply was poor and sanitary infrastructure was obsolete (Pelletier et al., 1997).

Understanding spatial patterns of the past is pertinent to determining historical health disparities and, in turn, can help identify recurring health trends related to urban development and at-risk populations. Consequently, an understanding of population health in the past can help explain how history affects current population health outcomes (see, Etches et al., 2006; Frank & Mustard, 1994; Freudenberg & Galea, 2008; Mustard & Frohlich, 1995; Vlahov et al., 2004). Nevertheless, population health research has been limited in its incorporation of an explicit historical approach to population based health research (Commission on the Social

Determinants of Health, 2008; Etches et al., 2006; Labonte, Polanyi, Muhajarine, McIntosh, & Williams, 2005).

1.2.3 Connecting Disciplines through an Historical Epidemiological Lens

Social hierarchies of advantaged/disadvantaged groups are still present worldwide and contribute to divisions in socioeconomic status (income, occupation, education), ethnic and religious groups, gender, age, geography, and so on (Braveman & Gruskin, 2003). Varying sociocultural practices among human populations can also affect the urban health patterns. In Sydney and Glace Bay, Nova Scotia, in the early 20th century, recruiting officers for the steel plant and coal mines, respectively, recruited immigrant labourers, often from rural Europe, because of the ability to offer low wages for unskilled work (Heron, 1980b). These men showed a disproportionately high mortality due to accidental deaths (Ludlow & Burke, 2012). In industrial spaces across 19th century United States, immigrants generally faced the worst hazards of urbanization and industrialization because they were concentrated in unskilled low-wage occupations (Hautaniemi et al., 1999). Social hierarchy contributes to unequal population health outcomes. Historical epidemiology and demography frequently reveal the connection between social hierarchy and disproportionate health outcomes (Curtis, 2008; Emery & McQuillan, 1988; Luckin & Mooney, 1997; Sawchuk & Burke, 2003; Sawchuk et al., 1985; Zunz, 1982). In this case, however, the selection bias that favours healthy robust younger immigrants from rural locations can have contradictory mortality effects for infectious disease transmission (Hautaniemi et al., 1999; Ludlow & Burke, 2012).

As the 20th century progressed, the influence of urban health threats, specifically communicable disease, waned and was accompanied by a decline in the initial healthy cities movement of urban planning (Cohen, 2000). Although tuberculosis continued to be a health

threat into the middle of the 20th century (Szreter, 1991), tuberculosis mortality for major American cities showed a decline in tuberculosis deaths as a percentage of all deaths between 1900 (11.1% of total mortality) and 1936 (5.3% of total mortality) (Cutler & Miller, 2005). Prior to the discovery of antibiotics, like penicillin in 1928, and the use of chemotherapy in the mid-20th century, social and public health improvements in living standards, personal hygiene practices, adequate sanitation (clean drinking water and adequate sewage disposal), and infectious disease control initiatives (e.g. sanatorium and quarantine) led to healthier urban environments and reduced rates of infectious disease (Farmer & Nardell, 1998; Grzybowski & Allen, 1999; McCuaig, 1999; Wherrett, 1977). Cutler and Miller (2005) found that, between 1900 and 1936, 32% of the reductions in infectious diseases (including tuberculosis) could be attributed to the implementation of clean water technologies. The implementation of clean water, and other social and public health interventions rose from the general acceptance of germ theory. Germ theory originated from John Snow's spatial epidemiological work in the 1850s identifying the Broad Street water pump in London as the source of the cholera outbreak in that location, and the discovery by Robert Koch of microorganisms like *Mycobacterium tuberculosis* and *Vibrio cholera* in 1882 and 1883, respectively (Koch, 1884, 1890; Snow, 1855). The decreasing connection between urban planning and public health may have been a by-product of the reduction of infectious diseases that resulted from an increase in healthy social and physical environments.

Within recent years, urban planning has shown a renewed interest in the healthy cities approach. This interest can be attributed to connections being made between chronic, non-infectious diseases with modifiable risk factors, and key determinants of health, and to a better

understanding of effects of such connections on the health status of a population (Araya et al., 2006; Corburn, 2007; Dunn & Hayes, 2000; Franzini et al., 2010; Gilliland, 2010; Walks, 2010; Wright & Muhajarine, 2008). According to Labonte et al. (2005, p. 5), "[t]he ideas underlying the term [population health] are not new and owe much to the legacies of nineteenth-century public health radicalism, Latin American social medicine and, more recently, social epidemiology." Public health and social and sanitary medicine were a response to the historical urban environments found throughout the 'Western' world, and thus the healthy cities concept continues to aid in the collaborative efforts towards creating healthy populations.

1.3 Filling the Gaps in Research

This study seeks to examine how changing social and economic factors brought on by industrialization affected population structure and urban development in the city of Hamilton, Ontario, and how, in turn, these factors contributed to variations in spatiotemporal mortality patterns at two time points, 1880–1882 and 1910–1912. Hamilton was chosen because its historical geography has been researched extensively, owing to its important contribution to Canada's economic growth (Cruikshank & Bouchier, 2004; Harris & Sendbuehler, 1992; Heron, 1980a; Nader, 1976). Moreover, some historical epidemiological research has already been conducted on Hamilton, due to its rapid urbanization occurring in the early 20th century (Gagan, 1989; Herring & Korol, 2012; Herring & Carraher, 2011; Toth, 2001). For example, Gagan (1989, p. 172) examined changing mortality rates between 1900-1914 to reveal the unequal distribution of mortality and disease, but noted that the 15 year timeframe may not have been sufficient to view the "changing health patterns of a large urban population" (p. 172). Toth (2001) used documented tuberculosis mortality rates to examine conceptual changes in public health and how that was applied to surveillance, treatment, and prevention tactics in the

Hamilton. As well, some work has been conducted on the two waves of influenza in the city, Russian influenza (1889-1890) and the Spanish influenza (1918), which showed spatial and social inequalities specific to these infectious diseases (Herring & Korol, 2012; Herring & Carraher, 2011). The research that has been conducted on Hamilton reflects the city's important place in Canadian history. Although there are similarities in the focus, no work has yet been published that uses a spatiotemporal approach to understand the potential impact of an economic transition related to industrialization on urban development and health.

The dates selected for this research (1880–1882 and 1910–1912) span a transition in Hamilton's economic system whereby the city moved from predominately commercial and light industry towards heavy industry and manufacturing (primarily steel manufacturing), becoming the most industrialized city in Canada (Harris & Sendbuehler, 1992; Nader, 1976). Although known for its steel industry, Hamilton attracted a wide variety of manufactories (Harris & Sendbuehler, 1992), which generated substantial growth, consequently introducing new stressors to the city. During the study period's 33 years, the city's urban landscape was radically transformed to reflect a highly industrialized economy, allowing for an examination of life and death before and during heavy industrial activity. Nevertheless, physical growth of the city was uneven, as the underlying physical geography and industrial needs (accessible water and transportation ease) constrained the patterns of urban growth eastwards. Over time, Hamilton's physical layout and design began to reflect its growing industrial economy, but continued to illustrate spatial differences in the urban social landscape, making Hamilton an excellent case to examine spatiotemporal mortality patterns and the connections of such patterns to the environment.

The healthy cities concept is a recurring theme found within urban planning and population health, once defined by infectious diseases, now influenced by connections of the urban environment and chronic degenerative/non-communicable diseases. In other parts of the world, however, infectious diseases like tuberculosis persist. Tuberculosis remains the leading cause of infectious disease mortality, with over 95% of deaths occurring in mid- and low-income countries (World Health Organization, 2014b). Historical epidemiological research can further understandings of current spatiotemporal trends, as some of the problems of the past continue or are repeated. Ill qualities of life sustain the notion of building healthy cities in response to inequities in the social determinants of health, whether past, present, or future. As such, growing fields of multidisciplinary urban research combining planning and health can be further linked through an historical epidemiological lens, allowing for a natural experiment of a phenomenon that has already occurred (Diamond & Robinson, 2010). A spatiotemporal structure of health research is necessary to fully comprehend past, present, and future population health trends. Rapid urban growth caused by an economic transition without proper planning will likely lead to spatial disease patterns associated with rapid population influx and lack of adequately developed urban space.

1.4 Description of the Dissertation

This dissertation uses a multidisciplinary approach to understand how changes in population structure and urban development influence the health patterns of a city's population over time. It focuses on synthesizing the data and methods from population health, urban planning, geography, and history as a means of assessing the relative impact of changing urban and population structures on the health of a Canadian city – Hamilton, Ontario. The

health disparities examined in this research will focus on selected determinants of health that relate to spatial population patterns in Hamilton, including biological sex, socioeconomic status, and social and physical environments. Specific causes of death are analyzed to understand the characteristics of the mortality sample, in other words, who was dying of a particular cause of death, and whether there are any temporal differences and spatial patterns. This requires a multidisciplinary approach in terms of both data collection and analysis. Central to this analysis is the use of specialized computer-based technology, such as Geographic Information Systems (GIS). GIS is designed to manage, analyze, and display geographic information (ESRI, n.d.-e). Historical GIS (HGIS) is used to analyze and visualize historical geographic data using GIS software. For this research, multi-method quantitative methods are used in conjunction with a custom-built HGIS, and qualitative textual and image inquiry. Combined, these methods assess changes to urban infrastructure, demography, and mortality, and their relationship to an economic transition of industrialization across space and over time. Using a mixture of methods allows this research to work with the limitations of the data available, while attaining a rich description of life and death in Hamilton.

Mortality is used in this study as an indicator for population health due to the availability of information in the historical record, allowing for a spatial examination of deaths. Data for mortality is readily available while morbidity data is lacking. It is entirely possible that morbidity would show a somewhat different spatial composition for the study period, as people who had a condition may not have died from that disease. Survival of an infection can, in some cases, be related to differences in socioeconomic status. For example, in Canada, prior to Medicare, persons of wealth would have been able to afford tuberculosis treatment and longer stays at the sanatorium, whereas lower income groups may not be able to afford such health care

services (McCuaig, 1999; Wherrett, 1942). Wealthier members of society may be infected but, for a variety of reasons (e.g. access to healthcare, healthier home and work environments, stronger immune system), may not die at the same rate as poorer members of society. Thus, both morbidity and mortality can reveal class-based variations. Spatial patterns of morbidity would give a more accurate picture of incidence of disease for a particular period. Morbidity, however, is not as available as mortality in the historical record and, thus, attempting to include it would provide an inaccurate picture of the period.

1.4.1 Purpose, Objectives, and Research Questions

As a case study, the purpose of this dissertation is to explore the ramifications of industrialization in Hamilton, Ontario, over the period 1880 to 1912, during which the city's urban structure changed rapidly, in order to understand how this rapid change influenced the spatial pattern of health in the city. Thus, the main objective is to determine if industrialization contributed considerably to altered mortality patterns found in Hamilton and if the altered patterns increased health disparity and inequity for Hamilton's populace. The following questions are used to accomplish this objective. First, what were the socio-demographic profiles in 1881 and 1911 Hamilton, and how did the profile change over time? Second, did these socio-demographic patterns reflect the temporal urban and economic development of Hamilton? Third, what were the mortality profiles for the 1880–1882 and 1910–1912 periods in Hamilton, were there any observed changes in the profile over time, and, if so, what are they? Finally, were there spatial differences in mortality for the 1910–1912 period across the city of Hamilton, and do those mortality patterns suggest health disparity in relation to socioeconomic organization and urban development?

A secondary objective is to reinforce the importance of careful urban planning during economic and urban development to address plausible spatiotemporal patterns of health inequities and bridge this health gap. By using a comprehensive health ecology approach as found in historical epidemiological studies, key insights can be gained using mortality alongside additional sources of information to examine changing population health patterns of Hamilton that resulted from changing economic structure. Due to the temporal change in its socioeconomic structure, Hamilton offers an opportunity to capture change in population characteristics, urban environment, and mortality during a period of rapid industrialization. Using two periods 1880–1882 and 1910–1912, and a purposive mortality sample (working age and infant sample), a snapshot of life and death before and at the height of heavy manufacturing (namely steel manufacturing) is provided. To do this, a mixed-method study design is required in order to move beyond an examination of mortality patterns towards a comprehensive understanding of the ecological interactions that impact the health of a population.

1.5 Organization of the Dissertation

This research study is presented in five chapters. The first two chapters provide an introduction to the research, the context of the study, and the methodological design; chapter three present the results and analysis; and chapters four and five discuss the results and conclude with the key contribution and implications of this research in the broader disciplinary context. The present chapter provides a background to the study that describes the purpose of the research, a literature review that frames the rationale for research by defining the gap in the literature and the importance for this research, and a description of the research objective. In

Chapter 2, the description of the study is explored and methods provided. The chapter begins with a high-level overview of urban models that help to explain the growth and development of Hamilton during the study period. From there an historical geography of the city up to and including the era of rapid industrial growth, and a description of the public and population health challenges that affected the city during the study period are provided. In the methods section, the research design describes the rationale for using a mixed method approach to examine health outcomes and mortality patterns. This is followed by a discussion of the data collection process, including the nature of the sources and the rationale for the sample selection strategy. From there, a summary of the data analysis, focusing on quantitative and qualitative methods, and the use of HGIS is given. Finally, the inherent limitations of conducting this type of historical research and the limitations present in this research are explored.

Chapter 3 is dedicated to the presentation and analysis of the data, presenting the quantitative, qualitative, and HGIS results of this research. Beginning with a temporal examination of the demographic data and then into the descriptive statistics of the mortality samples. Each mortality sample is analyzed sequentially, moving from general descriptive statistics to specific chi-square tests to identify independent variables that will build a profile of the mortality sample. Using these chi-square results, an in-depth examination using logistic regression is used to identify characteristics of those who died of a specific disease. These results are then integrated in the HGIS to analyze spatial mortality patterns. Qualitative results provide a descriptive text analysis of contemporary information pertaining to the state of health in the city of Hamilton at each period, as well as an analysis of photos relating to the growth of the city and state of health.

In Chapter 4, the research findings are discussed in response to each of the research questions. The methods are further synthesized to profile life and death in Hamilton. Finally, in Chapter 5, the study is summarized, key findings are identified, and implications of this research for practice are considered. Two major findings found in this research are as follows:

1. Industrialization played a major role in the emerging human health ecology risks correlating to the mortality patterns found among the two samples, and
2. Industrialization tended to increase health inequities amongst the population and across the city.

CHAPTER 2
METHODOLOGY

2.1 Study Site

Urban form can determine patterns of behaviour, the distribution of activities, and access to resources (Filion & Bunting, 2010). Further, changing urban structure will likely reflect changing population structure and economy, as well as other underlying forces. Study Site section, then, will focus on the physical, social, and built environments as key determinants of health. Rather than focusing on all aspects of the built environment, this dissertation is concerned only with those characteristics, such as housing and sanitation, which are likely to impact health during the study period. In doing so, a high-level overview of historical urban planning models is provided in order to produce an historical account of Hamilton's urban development driven by industrialization and link the changing urban development of Hamilton to possible population health risks and disease outcomes.

2.1.1 Historical Urban Planning Models

By the 1880s, Hamilton had undergone a slow transition from an economy based around shipping and commercial activity towards one that revolved around manufacturing, industrial, and transportation activities. The 1890s, however, marked a significant shift in the city towards heavy industrial steel manufacturing. This transition shaped the layout and structure of the emerging city. Steel production led to the formation of an industrial area close to the port with working-class neighbourhoods in close proximity (Cruikshank & Bouchier, 2004;

Nader, 1976). Historical urban planning models provide a theoretical pattern of urban development. Examining these models in the context of shaping of an industrial city can aid in better understanding the physical and urban forces that may have affected patterns of health disparity.

In the early 20th century, two urban models, zonal and sectoral, were developed in the United States to describe patterns in urban development found in North America (Figures 2-1 and 2-2, respectively). Based on a single city, Chicago, these two models emphasized class-based segregation in relation to industrial zones or sectors. For example, the zonal model suggests that poorer classes are often found just outside the central core of a city (Burgess, 1967). In the sectoral model, various residential areas, business districts, parks, and factories all occupy distinct urban locations; however, with the development of industry nearby residential areas became unfavourable, which caused those areas to become more working-class or lower-class areas (United States Federal Housing Administration & Hoyt, 1939).

Although the concentric zonal model is descriptively applicable to North American cities in depicting land use as a set of concentric rings around the central business district (CBD), the model does not adjust for physical features of the landscape, such as a harbour, river system, or lake. Cities are often located near water source to provide both a water supply for the population and a form of transportation. The variety of urban patterns found in American cities led to the development of the sectoral model which takes into account low-income sectors near major transportation routes and industry (United States Federal Housing Administration & Hoyt, 1939). In 1965, both models were adapted to show that residential and industrial zones

were divided by class partly based on prevailing winds (Mann, 1965). The Mann model also illustrates that

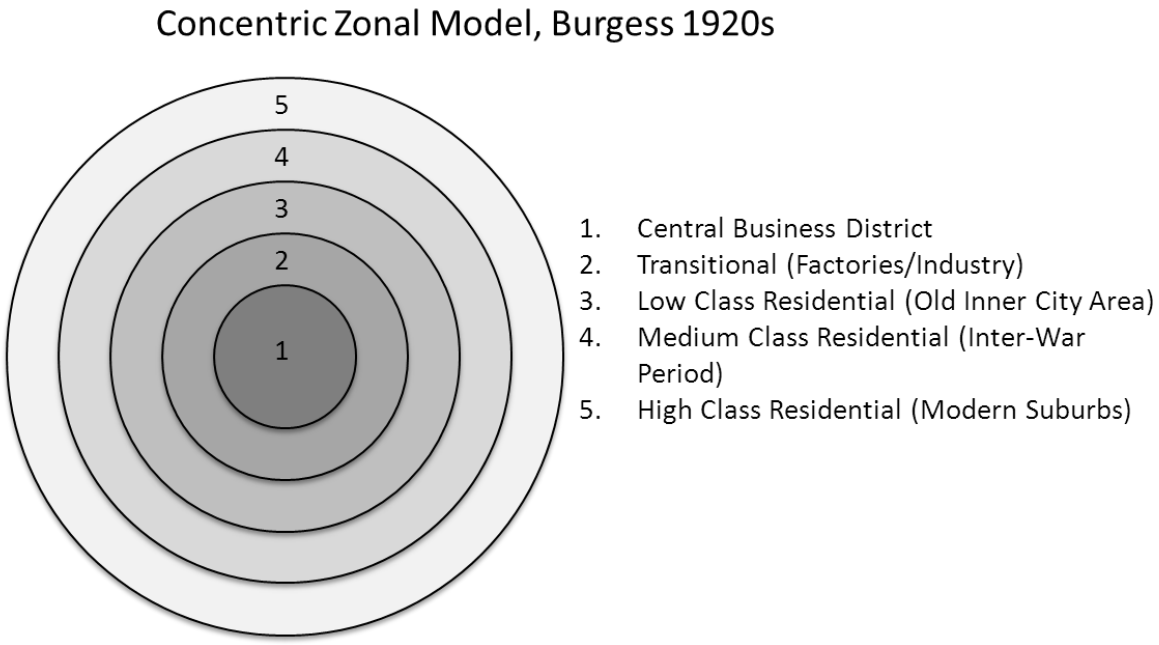


Figure 2-1. Illustration of Burgess's Zonal Model from the 1920s, Redrawn from Burgess (1967)

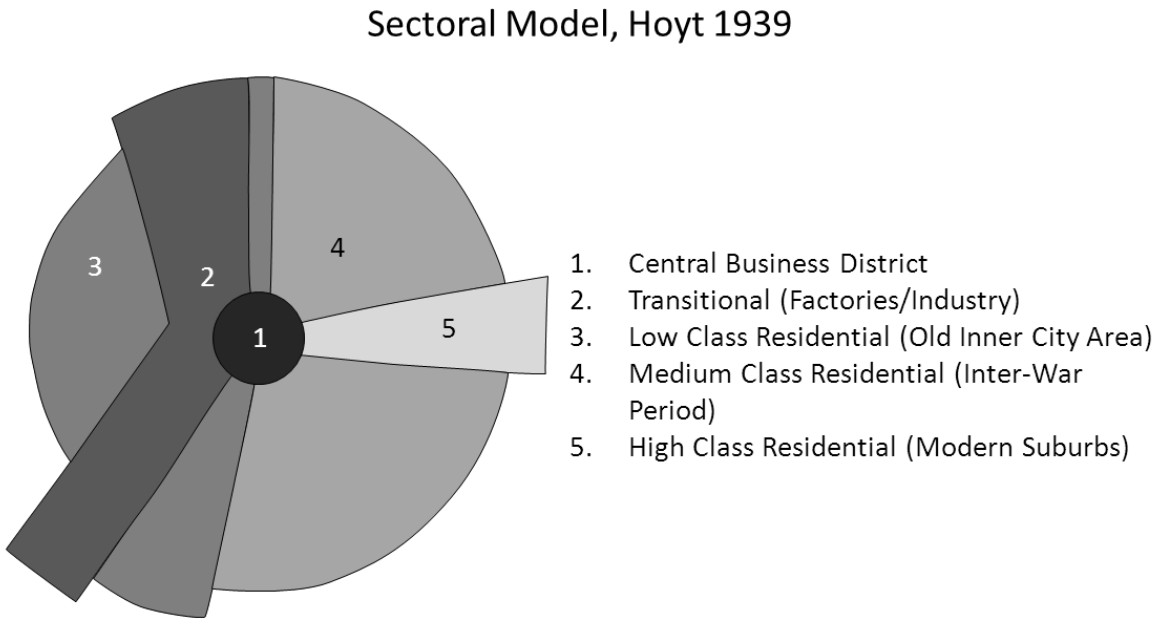


Figure 2-2. Illustration of Hoyt's Sectoral Model from 1939, Redrawn from the United States Federal Housing Administration and Hoyt (1939)

within the industrial sector, there are working-class residents (Figure 2-3). Mann's model of a British City best describes the city of Hamilton because its industrial development and class division resulted from this development.

Urban planning models are a generalized view of urban form and growth. Although cities may conform to certain aspects that are found in an urban model, the layout of planned urban sections or zones are likely to vary depending on the social, economic, and physical landscapes. All three models support the observation that working- and low-class residential areas are often found near or in the "transitional" industrial area. When compared to the zonal model, however, the sectoral model is more efficient at describing Hamilton's urban development. Hamilton's industrial area did not resemble a concentric zone around the CBD, but rather a sector of land located northeast of the CBD. In relation to Mann's model, prevailing westerly winds were also likely factored into Hamilton's industrial development plan to decrease the amount of exposure to pollution from the factories in the wealthier residential areas. Working- and low-class residential neighbourhoods could be found within and around this industrial zone/sector. Another important factor in the urban development of industrial space in Hamilton was the physical environment. The east end of Hamilton was 'undeveloped' from an urban perspective (predominately agricultural land) and was accessible to the harbour (for use in transportation of materials/products and industrial processing), making the northeast suitable for industrial development. A history of Hamilton's urban development accompanying industrialization will build upon the patterns of urban growth to give a better

understanding of how the city's physical, social, and built environments may have influenced the spatial and temporal patterns of health.

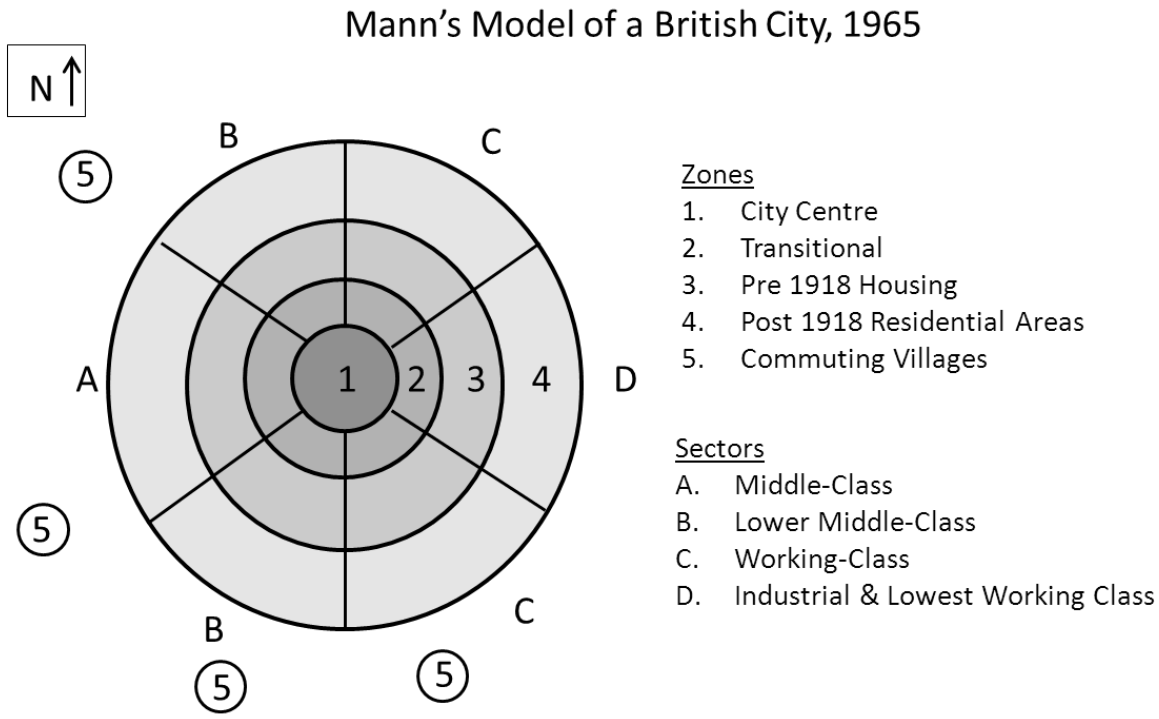


Figure 2-3. Illustration of Mann's Model of a British City from 1965, Redrawn from Mann (1965)

2.1.2 Towards Industrialization: Historical Geography of Hamilton

Hamilton's pattern of urban growth during the study period reflected both the physical and social environment. Such environments helped shape Hamilton's identity as 'Canada's Industrial City' because the social, cultural, economic, and political fabric of a city can and will influence urban development and demography. The pattern of urban growth was also confined by physical boundaries of the Niagara escarpment (known as the 'Mountain') and Lake Ontario, which restricted settlement patterns to the north and south. This section will discuss the regional and social geography of the city between 1881 and 1911 in order to contextualize the role of the urban development and the physical environment in shaping the identity of Hamilton.

2.1.2.1 Pre-industrial urbanization: Demographic and economic trends

The city of Hamilton is located in the Great Lakes-St. Lawrence region of Canada and occupies a strategic site at the head of Lake Ontario, west of the city of Toronto (Figure 2-4). Historically, the city was compact and was wedged between the Niagara Escarpment in the south and Hamilton Harbour (formerly known as Burlington Bay) on Lake Ontario. By the 1910s, however, the city had begun to extend development south onto the escarpment. Burlington Bay was a natural deep water harbour separated from Lake Ontario by Hamilton Beach (a sand bar that extends from the east side of the city) (Houghton, 2002). These physical features greatly affected early patterns of urban development and changed the socio-economic geography of Hamilton by constricting growth in an eastward direction that would eventually become the location of the industrial sector.

Hamilton presents a case study of how population growth and location is influenced by the structure of the city and the continual deep-rooted spatial concentration of society and class. Doucet and Weaver (Doucet & Weaver, 1984) give a comprehensive account of Hamilton's historical development as follows. In 1793, Hamilton was incorporated as a village, founded by land developer and merchant George Hamilton. Nine other land developers also helped the progress of urban development, and, along with Hamilton, each owned a section of the land for development and real estate. By the 1820s, the town of Hamilton took shape, with a central grid street patterned townsite, which became its CBD, located two miles from the bay.

By the early nineteenth century, this defined core set back from the bay would allow for a concentric peripheral development pattern. This early centralized placement of buildings, parks, and markets may suggest that the founders were planning for the physical expansion of

Hamilton from a town to a city. Another reason for placing the CBD two miles from the waterfront may have been the poorly drained land found at the base of the harbour, which would have been considered unsuitable for this function. Both the physical landscape and land owners'/developers' economic prospects likely determined the site for the CBD. Hamilton's physical location was vital to its economic prosperity. The geographical concepts of site (the physical nature of the area) and situation (its relation to other geographic features) can explain the city's physical location and rise to economic prosperity. In terms of site, Hamilton's location was strategic for trade connections along the Great Lakes with its natural harbour, but the Niagara escarpment to the north would cause the city to grow predominately east. As well, Hamilton's situation relative to other places enabled transportation connections for the import and export of goods. Both site and situation would have been very important for Hamilton's rise to industrialization as the city was not positioned near raw materials (e.g. coal or iron), but the natural harbour and transportation connections would have allowed the city to import raw materials to produce and export manufactured goods, such as steel.

Although Hamilton's early expansion may have been limited by the lake and escarpment, it was in a strategic location for urban commercial development and trade connections with the United States. Hamilton Harbour created a haven for ships during storms and, being naturally deep, allowed for commercial and transport activity (Nader, 1976). During the late 1840s, substantial improvements were made to increase commercial trade networks, transportation activity (Nader, 1976), and urbanization. Such improvements and strategies were implemented by the town fathers to boost Hamilton's economic growth and included: a) encouraging new modes of transportation by deepening and widening canals allowing for larger

ships into Hamilton Harbour and building railroads, b) enhancing civic services, for instance by constructing banks, and c) increasing the population by, for instance, dispatching immigration agents to other countries (Doucet & Weaver, 1984; Nader, 1976). The deepening of the already deep harbour increased commercial and transport activity, and likely encouraged new migrants to Hamilton, especially with the recruitment efforts described. As such, these efforts and strategies significantly influenced Hamilton's social geography and led to the city developing more industry.

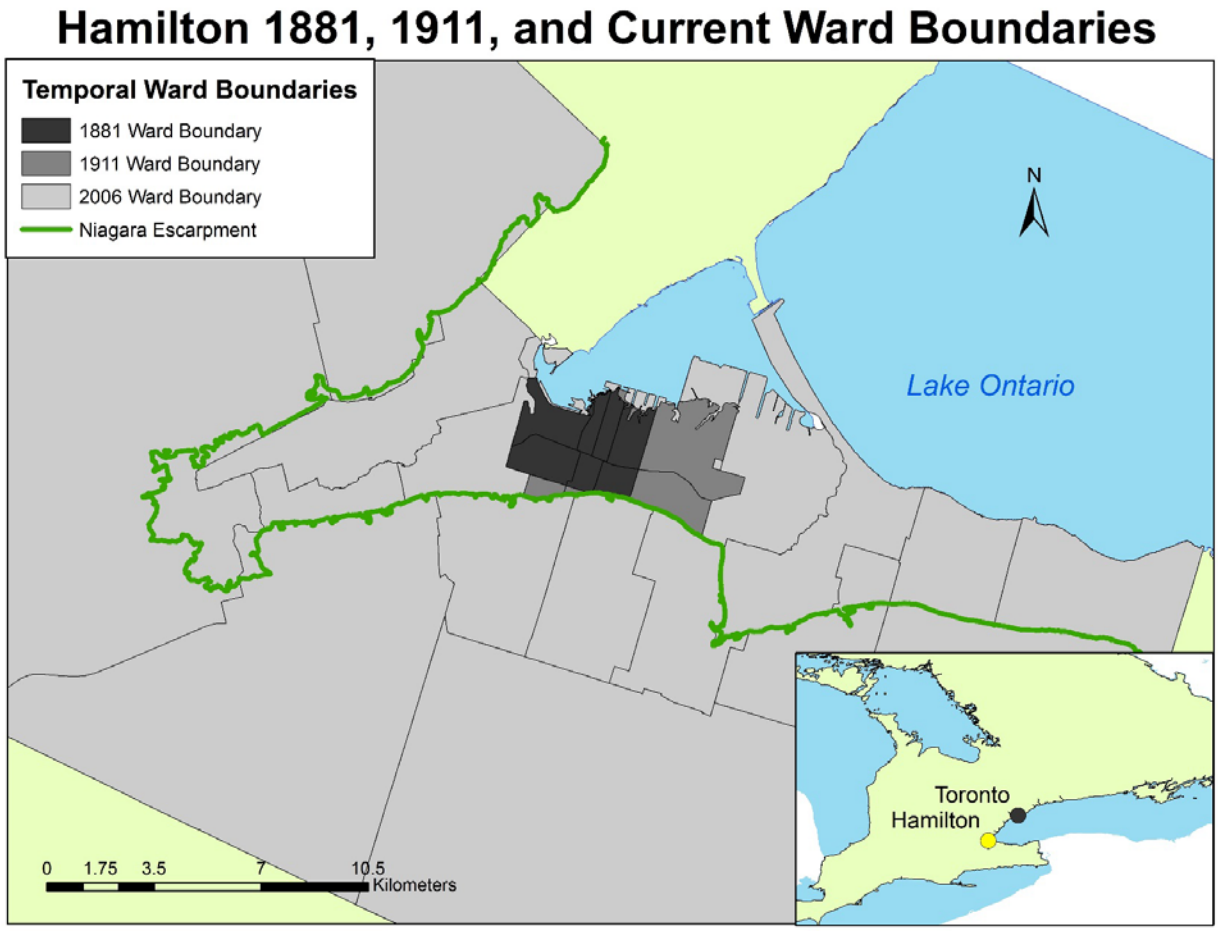


Figure 2-4. Urban Growth and Location of Hamilton, Ontario

During Hamilton's early years, the civic leaders were successful in their efforts to increase growth. After 1825, large numbers of immigrants were arriving in Hamilton and, by the 1830s, the efforts to encourage settlement in Hamilton turned the city into a boom town (Doucet & Weaver, 1984; Nader, 1976). By the 1840s, continued population growth fuelled by immigration from outside Canada led to a predominately immigrant population, mostly comprised of those arriving from the British Isles (England, Scotland, and Ireland) (Katz, Doucet, & Stern, 1978). Although the city prospered economically largely due to immigration, there was a continued growing class disparity between segments of the population, in relation to wealth distribution, property ownership, occupation, and ethnicity, which, in turn, was reflected in a more diverse settlement landscape.

Prior to industrialization, Hamilton presented a degree of spatial segregation among its residents: wealthier residents resided to the south near the escarpment on higher, better-drained land, while the city's working classes settled to the north on low, poorly-drained land near Hamilton Harbour (Cruikshank & Bouchier, 2004). As early as the 1830s, spatial segregation was prevalent in the landscape, with the southwest identified with 'exclusivity' (higher socioeconomic classes) and the east with 'relative inferiority' (lower socioeconomic classes) (Doucet & Weaver, 1984). Thus, even for the period prior to planning for industrialized space, Mann's urban model can be applied to early urban planning of Hamilton. Wealthier residents obtained land in the southwest away from the lake and near the escarpment, land that was therefore drier and more protected from the prevailing westerly winds that would later on carry smoke from the factories. Figure 2-5 presents a contour map of Hamilton

showing the elevation in meters. The city is partitioned into quadrants by James Street (north-south direction) and King Street (east-west direction).

Socioeconomic inequalities between religious groups and nationalities reflected historical Canadian ideas of immigration and favouritism based on ethnicity and nationality. For example, English Protestants were rarely, if ever, considered immigrants and often received citizenship status upon arrival to Canada (Avery, 1979). In the 1830s, Hamilton's sociocultural landscape

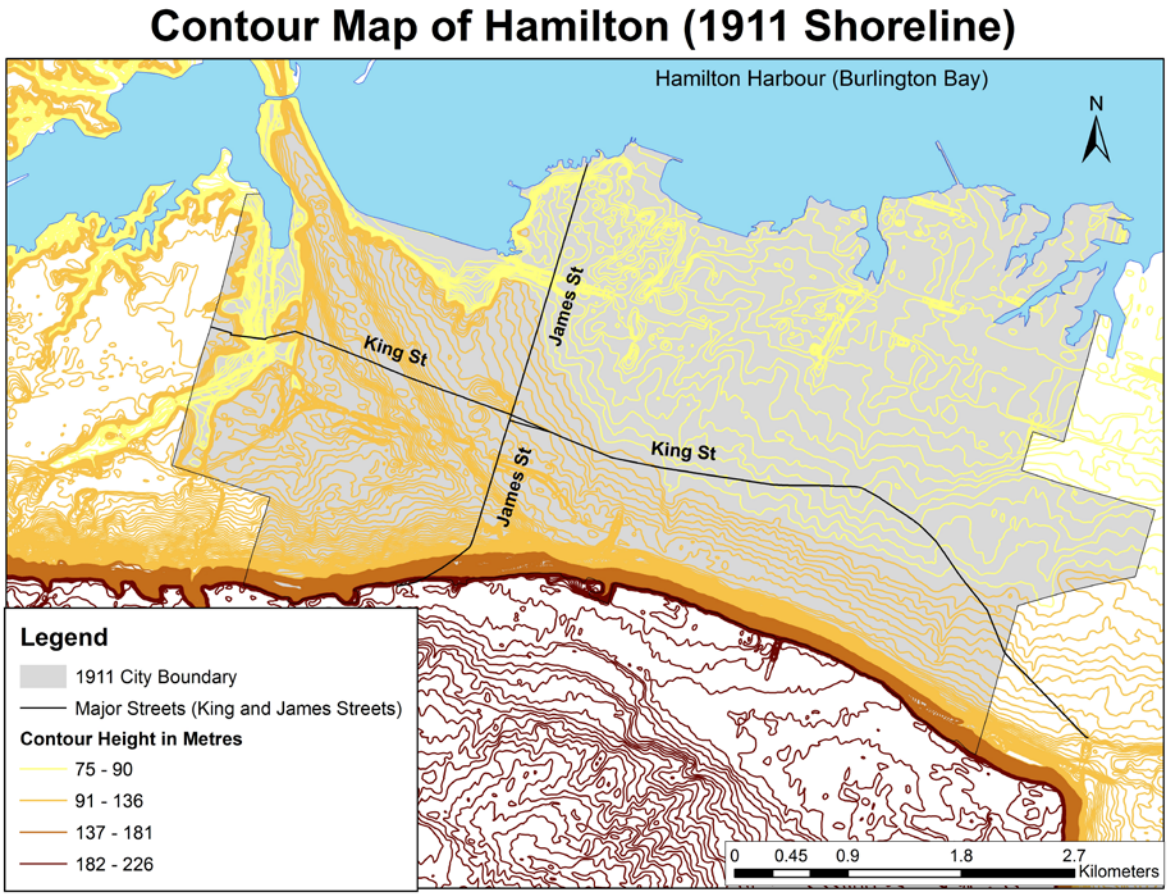


Figure 2-5. Contour Map of Hamilton

consisted predominantly of English Protestant and Irish Roman Catholic people, which, respectively, coincided with the elite and non-elite distribution of the urban population (Doucet & Weaver, 1984). By the 1830s and 1840s, Irish Roman Catholics were the second-largest population in Hamilton (English being the first), but held the lowest social status. Many of these resided in a designated part of the town known as Corktown, an area known for its poverty and poor living conditions (Doucet & Weaver, 1984, p. 84). As it is today, Corktown was located in the southeast section of Hamilton, bordered by the escarpment to the south, and James, Main, and Wellington streets to the west, north, and east, respectively (Bartlett,

2010). These divisions of upper- and lower-class neighbourhoods were firmly entrenched, according to Katz et al. (1978):

> The structure of inequality evident in 1851 existed with little change two decades later: the distribution of wealth; the relation between economic standing and occupation, property, and ethnicity; and patterns of social mobility all scarcely altered. Indeed, even the proportion of the population in the workforce hardly changed during early industrialization (p. 210).

These socioeconomic differences were also entrenched in the settlement patterns of the city.

By the 1850s, the immense population and economic growth of the first half of the 19th century had come to a halt. Based on aggregated census records (Canada Census and Statistics Office, 1911; Canada Department of Agriculture and Statistics, 1882), between 1848 and 1851, the population of Hamilton grew from 9,889 to 14,112, a growth of 42.7% with an annual rate of 14.23% over the three years. Prior to this date, population numbers found in online census data were available at the county level, but not at the city level. From 1851 to 1861, however, the growth rate declined. Over this ten-year span, the percent of growth was 35.31% with an annual rate of 3.53%. This decline in the rate of population growth between the 1850s and 1860s was due to a prolonged financial crisis that the city of Hamilton faced (Nader, 1976). The economic depression of the 1850s was widespread across Canada, but in Hamilton it was largely the result of the declining wholesale commercial industry (Nader, 1976). This proved temporary as Hamilton's economic conditions improved by the late 1860s, ushering in another era of rapid rise in population (Nader, 1976).

Acknowledging the decline of the city's commercial activity, by the 1870s Hamilton began to switch focus. As a predominately commercial economy, Hamilton was unable to successfully rise, economically, from the shadows of nearby Toronto. It appears that this highly

unequal neighbouring competition for commercial activity prompted the transition towards an industrial economy. Although some industrial activity was already underway, in the 1890s, Hamilton's municipality directed development policies that encouraged manufacturing expansion (Middleton & Walker, 1980). When the first blast furnace was fired in 1895, the city's economic focus became centred on industrial activity (Nader, 1976). As may be expected, the shift from commercialism to industrialization in Hamilton was not a swift transition. Although Hamilton's economic base shifted, the social structure of the city remained fairly stable, marked by entrenched uneven and unequal social structure that, in turn, was reflected in the patterns of urban development (Katz et al., 1978).

2.1.2.2 Industrial economy and urban growth

The slow transition from commercial to industrial activities, combined with the physical environment and the social, political, and economic structure of the city, likely contributed to Hamilton's predominately eastward urban growth pattern. According to the models of urban growth noted above, working-class neighbourhoods are often found close to the industrial sector (see, Burgess, 1967; Mann, 1965; Park & Burgess, 1921; United States Federal Housing Administration & Hoyt, 1939). While Hamilton had always shown class division, industrialization amplified socioeconomic segregation of classes in Hamilton, whereby working-class neighbourhoods were located in the eastern section of the city (Harris & Sendbuehler, 1992). Much of Hamilton's growth occurred in this location, referred to as East Hamilton, between the 1880s and 1910s.

The failure of Hamilton's wholesale/commercial economy increased the city's ambition to prosper. By the turn of the 20th century, Hamilton had established itself as Canada's most

industrialized city (Harris & Sendbuehler, 1992). Improvements that were made on the canals earlier and the construction of the Welland Canal in Welland, Ontario, opened transportation routes from Lake Erie to Lake Ontario and enabled the importation of low-cost coal from Pennsylvania (Nader, 1976). This canal likely caused Hamilton's rise to becoming Canada's 'Steel Town', as coal, once coked, is used in the smelting of iron ore and limestone to produce steel.

Although it is recognized for its steel industry, Hamilton drew in many other industrial enterprises, such as the manufacturing of rubber, chemicals, textiles, agricultural implements, and consumer goods, such as refrigerators, stoves, tobacco, and garments (Harris & Sendbuehler, 1992). Unlike Sydney, Nova Scotia, another major steel city in Canada, Hamilton did not have the characteristics of a single-industry town, but there are, nevertheless, similarities in the division of labour and power relations found at the turn of the 20th century in a highly industrial economy. The development of the city's industrial sector possibly cemented the continued divide amongst the classes.

The industrial development of Hamilton's eastside (predominantly in the northeast quadrant) was likely chosen for two main factors: the physical environment and municipal incentives. Industries were concentrated in the northeast and spread eastwards along the waterfront (Harris & Sendbuehler, 1992). Water is and was an important element for both industrial processing and the transportation of both raw and finished materials (Harris & Sendbuehler, 1992). Apart from the Niagara escarpment to the south, another cause for Hamilton's peculiar uni-directional urban spread can be attributed to prevailing westerly winds. Development of the industrial sector eastward would have helped offset the impact of polluted

air on the city, as prevailing winds would push the air eastward. Although Harris and Sendbuehler (1992) did not correlate prevailing winds directly with industrial land development, the authors do note that the west and southwest were more attractive residential sites, drawing in the city's elite. To attain the status of Canada's leader in the iron and steel industry (a position earlier held by Montréal due to its geographical placement along the St. Lawrence), policies created by Hamilton's municipality around the 1890s provided incentives for industrial developers in the form of lower taxes, cash bonuses, cheap land, and lower water rates (Middleton & Walker, 1980; Nader, 1976). The success of these incentives for industrial development created an extensive working-class suburb in Hamilton East comprised mostly of immigrants from the British Isles (Harris & Sendbuehler, 1992).

Hamilton's industrialized development represents a major transition not only in the city's geography of economic activity, but also for Canada as a nation. The patterns of settlement in Hamilton in part reflect its urban growth towards industrial activity and persistent class segregation over time. As will be seen, the rapid urban development due to the onset of industrial activity and the spatial divide amongst the classes may have manifested into unequal urban development in, for example, adequate public health infrastructure and possibly health disparities. Urban development and potential health risks for the city of Hamilton, therefore, must be examined together.

2.1.3 Identifying Health Risks: Historical Epidemiology, Urban Development, and Health Disparity

There are no extant annual reports from the Board of Health for Hamilton from the early years of this study period, however the city council did discuss health issues and concerns

pertaining to healthy city planning. Council minutes were divided by various committees or boards and included the Board of Works, the Hospital Committee, the Sewer Committee, and the Board of Health (City of Hamilton Council, 1878-1882). By 1884, a functioning municipal Board of Health was established and, by the later part of the study period, annual city health reports provide detailed descriptions of public health concerns in basic services and the public health practices of the population (namely the working-class population). Furthermore, these sources express concerns that persisted over time, and document the efforts of various city officials to build a healthy city, and to create awareness of the persistent issues plaguing the city in the face of rapid development due to industrialization.

The interrelationship between social standing, the built environment, and health has long been acknowledged in research. Poorer working-class populations have historically lived in areas with low-quality public health infrastructure, such as substandard housing, inadequate water supply, inadequate sewage disposal, and increased fire susceptibility (Acevedo-Garcia, 2000; Barkin & Gentles, 1990; Burgess, 1967; Condran & Crimmins-Gardner, 1978; Fischler, 2000; Gagan, 1989; Herring & Korol, 2012; Pelletier et al., 1997). While there were neighbourhoods that have a mix of income classes, this type of class segregation was common and affected the fabric of society. Spatial residential patterns, both past and present, are multidimensional reflecting the social, cultural, political, and economic fabric of a city and, more broadly, a society (see, Artibise, 1975; Beveridge, 2002; Cutchin, 2007; Herring & Korol, 2012; Vaughan et al., 2005; Zunz, 1982). The onset of industrialization, increased spatial class segregation and, at a time when the urban environment was considered 'unhealthy', the uneven development of basic public health provisions created considerable inequitable health

challenges. This was certainly the case in Hamilton, which consistently struggled to meet the demands of a rapidly growing city through the provision of basic infrastructure such as housing and proper sanitation. Additionally, public health issues pertaining to the quality of cow's milk (unpasteurized, adulterated), bottle-feeding practices, and general sanitary practices persisted and likely varied by class and, therefore, location. As the city transitioned towards an industrial economy and the industrial sector of the city grew, these inequities became even more pronounced.

2.1.3.1 Housing: Class segregation and residential health disparity

Historical working-class neighbourhoods in Canadian cities align with today's standard in that they are dense and close to the core, and, thus, support walkability and mass transit (Harris & Sendbuehler, 1992); however these neighbourhoods would have been considered unhealthy environments at the time. According to Harris and Sendbuehler (1992, p. 383), "subdivisions and homes were targeted at specific sectors of the labour force. Skilled machinists might expect to live in the better working class areas; unskilled labourers in the cheapest" (p. 383). Although it is likely that builders developed working-class suburban neighbourhoods such as those found in Hamilton's East End (e.g. Unions Park, Norwood/Beloit), owner-building was a common theme in the early 20th century (Harris & Sendbuehler, 1992). The lack of strict building regulations likely resulted in the increase in owner-built homes and working-class suburban neighbourhoods (Harris & Sendbuehler, 1992). Harris and Sendbuehler (1992) also note that "most started out with only a one- or two-room shack, sometimes just a kitchen, and then added rooms and storeys as time and finances permitted" (Harris & Sendbuehler, 1992, p. 385).

Variations in economic class structure in Hamilton reveal a similar picture of ethnic inequality persistent in early Canadian labour history. Irish Catholics lived in considerable poverty in the city of Hamilton compared to English Anglicans and Scottish Presbyterians (Katz et al., 1978). Such discrimination supported economic disparity, as obtaining high-wage work was extremely difficult for non-British immigrants (Artibise, 1977; Avery, 1979; Heron, 1980a; Heron, 1988). Irish Catholics were commonly employed as poorly-paid labourers in Hamilton (Katz et al., 1978). By the early 1900s, the increase in immigrants from locations outside northwestern Europe began to change these policies, as the Irish Catholics began to 'seem' more like the English and Scottish in terms of social standing when compared to the arrival of Polish, Slavic, and Jewish immigrants (Artibise, 1977; Avery, 1979; Heron, 1980a). The pattern of migration revealed that wealthy individuals moved much less frequently than poorer people (Katz et al., 1978), which may be tied to homeownership among the wealthy class and the removal of working-class owner-built houses as Hamilton's urban development began to catch up to the influx of its population in the industrial sector. As well, the compact form of the city meant that most of Hamilton's residents were able to commute to work by either streetcar or on foot (Harris & Sendbuehler, 1992).

2.1.3.2 Public health infrastructure and persistent public health concerns

Today, basic infrastructure services found in higher income countries are, for the most part, mandatory and must be provided prior to development regardless of socioeconomic standing of the residents (Harris & Sendbuehler, 1992). Historically, however, piped water and sewers were often limited to higher income neighbourhoods and locations within Hamilton's city limits (Harris & Sendbuehler, 1992). Such services were expensive and would have caused

increased taxes for the residents (Harris & Sendbuehler, 1992). Residents sometimes petitioned against these services (Harris & Sendbuehler, 1992), or would 'tap in' illegally without approval from the city. Lack of such services would likely have affected the health of those in certain parts of the city.

A variety of extant sources (e.g. Provincial Board of Health, City Council Meeting Minutes, and Hamilton's Board of Health) report on the major public health issues that occurred in Hamilton during both periods of this study, 1880–1882 and 1910–1912. Such reports add depth to a mortality analysis and provide context to living standards and behaviours, and ultimately a better indication of the population at risk. The Provincial Board of Health was formed in 1882; prior to this date, health issues were reported on an ad hoc basis throughout the province (Harris, n.d.). This limits the ability to understand health during the earlier periods. Hamilton's official Board of Health was formed in 1884; prior to this date a police officer was sanctioned as the 'Health Officer' representing a Board of Health to report to council when diseases were rampant in the city (Harris, n.d.). Over time, likely coinciding with continued reporting of health, public issues and concerns became better documented through annual city Board of Health reports. In the earlier periods, however, these issues can be found in the city council minutes and the provincial Board of Health annual reports. These documents, along with secondary sources, provide a sense of early urban public health planning, spatial inequalities in the growing city, and the early conceptualization of building a healthy city.

The building of Hamilton's waterworks in 1859 is an example of an attempt to create a clean healthy supply of water for the city's residents. Prior to the building of the water works,

the first public water supply consisted of five wells strategically placed throughout the centre of the city: the first at Gore Park near the Market, Hughson and King streets (southeast), the second at the corner of James and Cannon streets (north), the third at the corner of King and MacNab (southwest), and the fourth and fifth at an unknown location (James & James, 1978). This first public water system was believed to contain relatively clean water for cooking, cleaning, and drinking (James & James, 1978). As James and James (1978) note, however, the assumption that the water was clean ignored the reality:

> Before the construction of the waterworks, life in Hamilton lacked both comfort and security. There had been a long history of devastating fires which could, and often did, spread rapidly from building to building; of epidemics of cholera and other water-borne diseases, which recurred with such seasonal regularity they almost became an accepted fact of life in the city; and of problems arising from clouds of dust stirred up by increasing traffic on the dirt roads of the city. (James & James, 1978, p. vii)

Much of the problem in supplying basic sanitary services seems to have been due to the ad hoc nature of sewer and water main development. Apart from the public wells, the Great Lakes, which were simultaneously used for disposing untreated wastewater, were a common drinking water source in Ontario; not coincidently, the contaminated water from the Lakes caused outbreaks of typhoid fever (Cook, 2014). In turn, epidemic disease spurred advancements in public health interventions. The spread of typhoid fever throughout North America eventually led to advances in clean water technology, such as chlorination (Cook, 2014; Cutler & Miller, 2005). Similarly, after the 1854 virulent cholera pandemic, Hamilton's city council approved the construction of the waterworks; prior to the pandemic, they had considered this service unnecessary and postponed its construction due to its considerable cost (James & James, 1978). Nevertheless, not all benefitted. The inability of the city to supply adequate housing to

newcomers in the height of rapid industrialization caused a surge in construction of houses in areas where water and sewer lines were not yet available.

During the late 1870s and early 1880s, concerns surrounding the city's sanitation persisted. The growing east end lacked a sewerage and city council noted in 1879 that this section of the city may have had problems with sewer and water contamination (City of Hamilton Council, 1878-1882). Nightsoil (human excrement) collection was the common form of sewage removal, especially in the east end where there was not yet a main sewer line. At this point, the west end sewer was nearly completed. The Sewer Committee, in the 1879 city council minutes, stressed the importance of completing the city's sewer system and noted that residents tapped into the sewer without the council's permission (City of Hamilton Council, 1878-1882). Thus, city council was aware of the inadequacy of sewer infrastructure in Hamilton. In the 1880 city council minutes, the Board of Health requested a one year contract to hire individual(s) to collect nightsoil and suggested that persons also be paid to remove garbage (City of Hamilton Council, 1878-1882). In the 1880 minutes the Sewer Committee states that the cost of sewer construction was an inhibiting factor for completing construction; this committee also requested a by-law be introduced to forbid persons from tapping or connecting with any common sewer without permission because it could cause considerable damage, such as pipe breakage that would injure the sewer system (City of Hamilton Council, 1878-1882). In 1882 the Sewer Committee noted that sewer pipes in place were now considered too small to allow for the necessary amount of water to flush out the pipes (City of Hamilton Council, 1878-1882), suggesting that the city was beginning to outgrow the sewer system but that cost continued to hinder the construction of new larger sewer mains.

In 1879, the Board of Water Works reported that the city had fairly good water access because the construction of high-level pumping engines and a reservoir were adequate to fulfill the required duty. Some areas (location not provided in the minutes), however, were still receiving water by wagons and carts (City of Hamilton Council, 1878-1882). The city also had problems with surface water drainage as shown by the presence of stagnant water; in other cases stagnant water was caused by residents discarding waste water into the city streets (City of Hamilton Council, 1878-1882). In 1880, the Board of Health Committee informed the council of the need for work and material to drain the stagnant pool of water in ward five, located in the northern part of the city (City of Hamilton Council, 1878-1882). Stagnant water is a health risk because it provides a breeding ground for disease vectors (e.g. mosquitoes) and bacteria that can then transmit diseases to humans (Centers for Disease Control and Prevention, 2011b; World Health Organization, 2015b). The Board of Water Works recommended in the 1880 minutes that water mains and a hydrant be constructed on MacNab south between Herkimer and Markland Streets to supply the several first-class houses now being built there, while water mains might be extended to individual houses providing the homeowner had the necessary pipe (City of Hamilton Council, 1878-1882). Although the Board of Water Works reported that the water system was adequate, there were instances that suggest otherwise. Additionally, there appears to have been some class-divide in the order of houses being supplied water mains with wealthier areas receiving main extensions earlier on, though water main extensions for the most part depended on owners having the acceptable pipes to allow water main hook-ups.

In the early 1910s, the state of Hamilton's public health infrastructure continued to be of concern. The reports note the lack of sewerage and water supply, especially in the industrial districts and in working-class and immigrant neighbourhoods. The 1909-1910 Board of Health report noted the lack of sewerage in the rapidly growing manufacturing district and called for better enforcement of the rules governing the construction, cleaning, and disinfection of dry earth closets (composting toilets) (Roberts & City of Hamilton, 1910). Houses found in the industrial sections were reported to have been overcrowded and unhygienic; furthermore, the medical health officer suggested the need for thorough and regular inspections of these premises which he considered to be too numerous and constantly increasing (Roberts & City of Hamilton, 1910).

Medical officer Dr. Fred B. Bowman for the year 1911-1912 reported on the inspection of homes in sections of the city where there was an influx of artisans and labourers and observed that these areas lacked sufficient housing to support the demand (Bowman & City of Hamilton, 1912). Although the report does not clearly point to the specific section of the city under scrutiny, it is likely, based on the previous 1910 reports and literature that the medical officer for the 1912 report was also talking about the industrial northeast quadrant of Hamilton. Information provided in the report stated that all overcrowded premises had poor sanitary conditions, and that some families had no water service, had only an unsanitary privy vault, and/or lacked the provision for waste water except for the back yard, the alley, or an open drain (Bowman & City of Hamilton, 1912).

The city began daily examinations of the city's water supply to test for bacteria in the water in 1912. In the annual report, a monthly average of bacterial counts revealed that the

summer and autumn months had the highest bacterial count (Bowman & City of Hamilton, 1912). There is no mention in the report as to how these averages were obtained; however, the standard plate count has been used since the 1800s to test bacteria in water distribution systems using the number of colony-forming units per one millilitre of water (cfu/ml) (Robertson & Brooks, 2003). Colony count was recognized in the late 1880s as an excellent indicator of filtration performance in the water system (Robertson & Brooks, 2003). Low and consistent levels of heterotrophic bacteria counts would mean that the treatment system is working effectively and that other coliforms should not be present as they are more susceptible to disinfection (Robertson & Brooks, 2003). The current Canadian drinking water standard for coliform bacteria or *E. coli* (*Escherichia coli*) is that none are detected per ml (Health Canada, 2015). The average bacterial count for summer and autumn months was 30.43 with the highest count in September (39.9) and the lowest count in November (17.7). These inconsistent levels suggest that there were issues with water treatment in Hamilton. The report did not describe the types of bacteria found, but did report on *B. coli* (*Balantidiasis coli*) with no counts found (Bowman & City of Hamilton, 1912). *B. coli* is a parasite that can be transmitted through the fecal-oral route caused by contaminated food and water (Centers for Disease Control and Prevention, 2013).

The issues that arose from the lack of these basic services in Hamilton during the study period caused an increased concern for the public health of the city's population because several common diseases of the time spread due to poor infrastructure. Diseases such as typhoid fever and cholera, which are both caused by the presence of human excreta in water and food sources, are often associated with public health infrastructural issues and individual

practices, and therefore can be good indicators for related health risks. For instance, the 1832 cholera epidemic that hit Montréal and Quebec City mainly affected the working-class neighbourhoods because of a lack of proper sanitation and adequate supply of drinking water (Pelletier et al., 1997). A classic case study of how poor individual-level practices can spread infectious disease is Typhoid Mary (see Leavitt, 1992). Through historical research, Leavitt (1992), found that the cook, Mary Mallon, an asymptomatic carrier of typhoid fever, contaminated food through improper hygiene practices causing a cluster of typhoid cases in New York among those who ate her food (Leavitt, 1992).

Similarly, tuberculosis is associated with poor public health infrastructure, overcrowding, and poor housing. Tuberculosis in humans is an infectious bacterial disease caused by *Mycobacterium tuberculosis*, often affecting the lungs (pulmonary tuberculosis) but it can also affect many other areas of the body (extrapulmonary tuberculosis). Pulmonary tuberculosis is predominantly transmitted between humans through respiratory droplets in the air (e.g. coughing and sneezing). Dr. Peter Bryce, Chief Medical Inspector of Immigration for Canada (1882—1904), stated that tuberculosis was a disease of 'house life' in densely populated cities with slums, overcrowded work-rooms, and factories (Bryce, 1916). In 1882 Koch identified that tuberculosis could also spread from cows to humans and, with cows living within city limits, risks to human health would have come in the form of contaminated milk supply of infected cows and airborne interpersonal infection due to close proximity (Davies, 2005). Unpasteurized milk, additionally, would have grave implications for non-breastfed infants because they would be ingesting large quantities of bacteria that would increase the prevalence of infant diarrhoea. Even controlled for via certification and pasteurization, the

additional cost for higher quality milk may have led to practices of watering down milk and purchasing milk from cheaper, less sanitary sources (see, Czaplicki, 2007; Sawchuk et al., 2002; Speake, 2011). In Canada and the United States, attempts to address the issue of pasteurization were progressing in the 1900s, however it was not until 1938 that Ontario amended the public health act making pasteurization compulsory (Speake, 2011).

At the turn of the 20th century, public health movements were centred on improving conditions of the home by connecting water and sewer lines, and in the home by limiting crowding and increasing ventilation (Corburn, 2007; Cutler & Miller, 2005; Vlahov et al., 2004). Public health concerns about living conditions, sanitary practices, disease prevention, and food and milk quality were brought up during both periods of this study. City Council attempted to take a proactive approach to the health of the city and its people. In addition to concerns raised regarding the public health infrastructure, the Board of Health noted its support in the City Council minutes for 1879 for a sanitary by-law that was used to promote the health of the city (City of Hamilton Council, 1878-1882). Requests were also made to provide medicine for the poor and a special committee was formed to address a lack of employment for the poor with the goal of devising a scheme to provide employment opportunities for the unemployed labouring class (City of Hamilton Council, 1878-1882). In the 1881 council minutes, the Board of Health noted that people disposed of garbage and ashes into publically-accessible places (City of Hamilton Council, 1878-1882). Also in the 1881 council minutes, the Board of Health requested extra expenditure for vaccination and care for smallpox patients (City of Hamilton Council, 1878-1882). The Provincial Board of Health (1883) remarked that smallpox was practically under control throughout Canada, with less than a dozen deaths in a population of

almost two million, which the Board attributed to activities of the local authorities. By 1883, a special committee on increased fire protection was formed and City Council noted that the improvements made to the water works system produced ample water for domestic and manufacturing but the pressure was insufficient to ensure protection against fire (City of Hamilton Council, 1878-1882). Thus, public health concerns were being brought forward to the City Council to take action on proper disposal procedures of water, sewage, and refuse, provision of health care for the population, and effective fire protection.

In 1884, the Provincial Board of Health delivered a report before the Hamilton Literary Association detailing public health concerns in Hamilton. The problems they detailed include sewer gases in homes caused by improper drainage, and the occurrence of diarrhoea caused by poor food handling, contamination of water, unhealthy cow stables, unclean milk cans, and sewer seeping from privies into wells (The Legislative Assembly of Ontario, 1884). Improper handling of milk products was noted to be a cause of disease, as zoonotic tuberculosis is usually transmitted to humans through unpasteurized milk from unsanitary cows infected with *Mycobacterium bovis* (de la Rua-Domenech, 2006). The Provincial Board of Health's address to the Legislative Assembly of Ontario (1884) expressed that preventative measures needed to be adopted at multiple levels: individual (personal cleanliness and cleanliness of clothes), household (cleanliness and ventilation), private grounds (filth in yards), and public sanitation (removal of excreta and the adoption of all necessary sanitary precautions). Currently, multilevel approaches like this are ecological and considered key to disease prevention as they bridge the gap between individual and community. Focusing on multiple levels of disease causation connects these

environments and aids in the development of healthy cities (Diez Roux, 2011; Golden & Earp, 2012).

The concerns raised by the Ontario Board of Health predominately pertained to infectious disease control in Ontario cities through public health measures. The message of the address given to the Hamilton Literacy Association was likely then adopted by the city of Hamilton to begin promoting healthy city initiatives across different levels towards the prevention of infectious disease. During the latter period of this study, continued concerns relating to infectious disease control were prominent in the annual health reports for the city of Hamilton. The 1909-1910 annual report suggests the city was keeping with the public health progress, but, with the rapid population increase, infant survival was a constant struggle (Roberts & City of Hamilton, 1910). The causes behind the high infant health risk were said to be related to feeding practices, the poor quality of cow's milk, 'the maternity problem' (the lack of education for parental responsibilities), poverty, housing, the environment, and social life (Roberts & City of Hamilton, 1910).

Typhoid fever in 1910 was the most prevalent since 1906 but, for the most part, could not be traced to a contaminated milk or water supply; however, the "…general conditions of insanitation tend to superinduce disease" (Roberts & City of Hamilton, 1910, p. 21). While the origin of the disease could not be traced, cities along the Great Lakes notably used the lakes for drinking water, as well as to discharge their sewage, and, in cities like Chicago, typhoid fever mortality rates declined with interventions, such as the opening of the main water channel and the cleaning of 'filth' (Benidickson, 2007; Pearse & Tolman, 1923).

Furthermore, the medical health officer also suggested that the use of Women Health Visitors, as found in England, would benefit an industrial city like Hamilton, with its large increasing foreign population (Roberts & City of Hamilton, 1910). In England, Women Health Visitors were women who were trained to instruct women on sanitary practices of the home, and infant and child health (Cochrane, 1908). They would visit the homes of lower socioeconomic class citizens, instruct women on care and feeding of children, distribute literature on sanitary matters, and aid in cleanliness and health practices (Roberts & City of Hamilton, 1910).

As noted previously, concerns about the state of housing were prevalent during the latter period. To relieve the congestion in the industrial districts of the city and potentially prevent disease, Dr. Bowman provided a variety of suggestions to the city, which included:

- Providing municipal housing for the working classes,
- Supporting the work of charitable organizations by which wealthy classes invested money toward the benefit of the municipality,
- Developing rapid transit—subway or rail system—from the centre to outlying districts and the decentralization of industries,
- Enacting a proper building or housing by-law with provision for its enforcement, and
- Monitoring the maintenance of houses in terms of sanitary conditions by the supervision of the Health Authorities (Bowman & City of Hamilton, 1912).

The suggestion to decentralize industry was not implemented, as location of the industrial sector and working-class neighbourhoods in Hamilton are in the same location today they were in 1895 when the first blast furnace was fired. Hamilton, however, made progress to provide better quality milk to the population through increased inspections of dairies. The 1911-1912 annual report noted that the inspection method had created friendly rivalry between dairies towards improved quality of milk by using installed refrigerators, immediately cooling milk once

pasteurized, and bettering cow's living conditions (Bowman & City of Hamilton, 1912). The increased cost of these measures, however, may have led to a divide in access to or purity of pasteurized or certified milk. During the latter study period, these growing public and population health concerns, in light of industrialization, would have affected the city's ability to progress in improving its urban health effectively.

2.2 Methods

Changing economic structure in an urban area can affect the overall urban development and the physical structure of that space. By using a mixed-methods approach, this research considers whether the city's demographic and epidemiologic structures were affected by Hamilton's economic transition into an industrial powerhouse in Canada. Conducting research across time and in the historical record does have its limitations. Yet, the ability to draw insights across time can aid in furthering knowledge of how various spatial patterns, such as mortality, demographic, and urban development can influence health outcomes. The materials used, methods employed, and limitations faced in answering the research questions are presented. This research was exempt from the University of Saskatchewan's research ethics review process because the work falls under the category of the writing of modern history and it draws on publicly accessible data.

2.2.1 Research Design

Understanding the multi-level interplay of population, habitat, and behaviour that together help to determine health requires a multidisciplinary approach. The term *multi-level* in this research refers to the human health ecology approach, which examines the interplay of levels in the human ecological system—population, behaviour, and habitat—leading to a better understanding of the connection between the characteristics of the population (composition) and

characteristics of the location or environment (context) (Meade & Emch, 2010). Additionally, the incorporation of mixed methods and multi-level techniques into health-based research can contribute to a holistic interpretation of the interplay of these factors. In other words, more complex research designs, such as the design presented for this research, can help explore and answer, in more detail, questions on the who, what, where, when, and why of population health disparities.

2.2.1.1 Choosing the research design

Through the combined knowledge of a wide array of academic and applied disciplines, researchers can address health disparities, understand the multifaceted causes, and evaluate polices towards reductions in population health disparities (Oliver & Cookson, 2000). Although multidisciplinary research is becoming more prevalent, there are challenges. A key challenge is overcoming the epistemological perspectives that define disciplines and the disciplinary narratives, which reflect a constructed worldview found within a discipline (Mattingly, 2010). Yet, drawing on disciplinary expertise supports a comprehensive understanding of plausible causal factors for health outcomes to better understand the interconnections between urban development and population structure (e.g. Corburn, 2009; Frumkin, 2004; e.g. Galea, Freudenberg, & Vlahov, 2005; Gilliland, 2010; Haines, Godley, & Hawe, 2011; Harris & Lewis, 1998; Jackson, 2003; Johnson et al., 2008; Labonte et al., 2005; Luckin & Mooney, 1997; Park, 1967; Walks, 2010). Furthermore, a human health ecology focus, frequently found in social science disciplines, such as anthropology and human geography, presents a multi-layered approach that includes consideration of biocultural and sociocultural differences and the physical and built environments to understand health variations (e.g. Barbosa, 1998; Dunn & Hayes,

2000; Herring & Swedlund, 2003; Johnston & Low, 1984; Meade & Emch, 2010; Rainham, McDowell, Krewski, & Sawada, 2010).

Mixed method approaches to research combine objective and subjective methods into a research design. As a synthesis that includes qualitative and quantitative ideas, mixed method research continues to develop, being one of three methodological paradigms (the other two being quantitative and qualitative) (Johnson, Onwuegbuzie, & Turner, 2007). The rationale for using mixed methods is based on the assumption that collecting diverse types of data provides a more complete understanding of the research problem than either quantitative or qualitative data on their own (Creswell, 2014). Given the multidisciplinary nature and complexity of factors in understanding what determines health, population health research benefits from mixed method research designs. Previously, population health research was predominately quantitative, until critiques on the lack of context and subjectivity led to an increase in qualitative research (Labonte et al., 2005), and also towards the inclusion of more mixed methods approaches. Dunning et al. (2008) note that, while using mixed methods is not new in quality of life research projects, there is a large gap in the literature on the methodology involved in mixed method approaches. Additionally, according to McKendrick (1999), the term *multi-method* has been widely misunderstood whereby *method* is too often interpreted as a *technique*, and thus the research process is left out. An understanding of the models used in mixed method research can and should be used as a building block to the process of generating an appropriate research methodology.

While many mixed method research designs exist, three primary models can be found in the social sciences that can then be used to build more advanced mixed method strategies (Creswell, 2014). In the first model, the *convergent parallel model*, the researcher converges or

merges qualitative and quantitative data to provide a comprehensive analysis to the research problem. In the second model, the *explanatory sequential model*, the researcher first conducts the quantitative component of the research, and then further explains the quantitative findings using qualitative research methods. The second model allows for a strong quantitative orientation while the incorporation of a qualitative phase allows for a more in-depth exploration into the research problem. The third model, the *exploratory sequential model*, is technically the reverse of the explanatory sequential model, beginning first with the qualitative data collection and analysis, which is then used to build the second, quantitative, phase of the research. In this third model, the qualitative phase can be used to determine the best approach for the quantitative phase (Creswell, 2014). As the models show, like other research methods, there is a variety of ways to approach a research study. Ultimately, the research purpose, hypothesis, and questions are guiding factors in selecting any research design.

2.2.1.2 Study design

The design of this research draws from multiple disciplines and uses the explanatory sequential mixed-method model to analyze life and death patterns in Hamilton through a multi-level human ecology lens. Due to the economic transition towards a highly industrialized urban space, Hamilton provides an excellent case from which to assess the dynamics of urban development and population patterns and their impact on population health outcomes. By examining two periods of time, 1880–1882 and 1910–1912, insights can be gained into temporal and spatial demographic and epidemiologic profiles of Hamilton before (1880s) and during (1910s) a period of heightened industrialization.

Historical research presents a unique opportunity to examine health disparity through case control studies and natural experiments. Case control studies are observational because

there is no intervention; instead one is able to examine cases that have occurred against controls, making them retrospective by design (Lewallen & Courtright, 1998). This type of study can also examine various risks and their association with the cause or case (Lewallen & Courtright, 1998). For example, it is possible to compare risk factors of past individuals with an outcome with those without the outcome (e.g. compare those who died of a particular disease to those who did not die of that disease). Unlike randomized control trial (RTC), natural experiments or the comparative method concern interventions that are not completely manipulated by the researcher; rather they are 'natural' (Dawson & Sim, 2015). Epidemiology is essentially the study of natural experiments on human populations, because it uses observation to better understand disease patterns (Diamond & Robinson, 2010). Research using this type of experiment can be used to fill gaps in the evidence base because of the ability to observe the outcomes and impact, or lack thereof, of an intervention, policy (Dawson & Sim, 2015; Petticrew et al., 2005), or event in time.

2.2.1.3 Sources

Due to the retrospective nature of historical research, data quality is often a key disadvantage (Lewallen & Courtright, 1998) to be considered. The ability to use raw individual civil death and census records provides information on diseases that presented a risk to the population and on the characteristics of that population at a given period in time. These records can also support longitudinal analyses as, in many instances, they were the major source of continuous monitoring of vital statistics (Mahapatra et al., 2007), although changes in data gathering protocols may lessen the value of such studies. Annual health reports from a city, province, and/or country provide context to the health concerns that prevailed in a location in the past, thereby situating the mortality patterns in space and time, as well as aiding in an

understanding of medical understanding (i.e. historicity and medical history). Historical maps provide spatial details of an area at a given point in time, such as urban development, and, when plotting mortality, reveal clusters or patterns of a disease. Additionally, historical photos illustrate and even bind the various sources of information together by providing a visual context of the past. Thus, multiple sources of information, when used in concert with one another, can deliver a more holistic analysis of a particular problem and the underlying factors that may cause an outcome.

The explanatory sequential mixed-methods model uses an iterative sequence of phases, with each drawing upon the analysis of the preceding phase (Bazeley, 2009; Creswell, 2014). This research uses both descriptive and inferential statistics, working with the available sources of information and their limitations, to provide an analysis of Hamilton's historical socioeconomic transformation and its impact on population and mortality. Beginning with a demographical analysis, this study identifies the population at risk for both periods and uses this information to inform the analysis of sociodemographic and socioeconomic changes that occurred in Hamilton between the two periods. This is followed by an in-depth statistical analysis of temporal mortality patterns. Spatial analysis drawing on HGIS techniques is then used to provide a snapshot of mortality for the latter period. Limitations in available spatial data did not allow for the mapping of mortality patterns for the earlier period; therefore temporal comparisons of spatial analysis were not possible. Finally, descriptive narrative and photo-analysis are used to contextualize the quantitative and HGIS results of this research. To study the impact of socio-economic and urban mortality patterns, a purposive mortality sample, a sample that is specifically chosen based on characteristics of interest to the research, is used.

2.2.1.4 Sample selection and data collection

Data for this study of Hamilton were collected from a variety of historical sources (Table 2-1) generated during two periods in time, 1880–1882 and 1910–1912. To examine the impact of the economic transition and resultant urban development on population health, aggregate census data and individual disaggregated civil death records were the main sources of data collected for both periods. Aggregated 1881 and 1911 censuses provide information on the population structure and/or the population at risk (the denominator) and are used to examine the living population. To address the research objectives, a series of strategic choices were made about the sample selection of deaths (numerator) found in the civil death registry for the city of Hamilton at both periods.

A purposive sample of all deaths for two selected groups was chosen: infant deaths under one year of age (not including stillbirths) and working age deaths 15-64 years of age. Infant mortality is often used as an indicator to measure health, well-being, and level of development in a society because the varied causal factors involved can also affect the health of a population (Centers for Disease Control and Prevention, 2014; Vaid, Mammen, Primrose, & Kang, 2007). Working-age mortality is likely to be sensitive to changes in occupation that follow the process of industrialization and can also be used to further investigate the social gradient of disease (Marmot et al., 1991). Thus, examining the historical mortality patterns of both infants and working age individuals can inform the potential impact the changing economy had on urban development and spatiotemporal disparities of health in places like Hamilton.

Table 2-1. List of Archival Materials and Use for Research

Information Source	Method for Analysis	Citation
Census of Canada 1881 1911	Quantitative demographic analysis over time and space	(Canada Census and Statistics Office, 1911; Canada Department of Agriculture and Statistics, 1882; Department of Trade and Commerce, 1915)
Death Registry 1880-1882 1910-1912	Quantitative mortality analysis over time. 1910-1912: spatial mortality patterns using addresses	(Government of Canada, 1869-1938, 1869-1942)
Historical City Maps 1882 & 1911	HGIS: Base layer for developing Hamilton Maps	(City Engineer, 1910; The Canadian Almanac, 1882)
Fire Insurance Map 1911-1916	HGIS: Historical Hamilton addresses 1910-1912 for spatial mortality analysis	(Hamilton Ontario, 1911)
Photos of Hamilton	Photo Analysis: Use as a visual source to analyze social knowledge, social understanding, and insights into the environment.	(Local History and Archives, n.d.; Roberts & City of Hamilton, 1910)
Ontario Department of Agriculture Colonization and Immigration 1881	Discussion of the results. Demographic analysis. Hamilton as a port of entry, information on population immigrating	(Commissioner of Immigration, 1882)
Provincial Board of Health Annual Reports 1882, 1883, & 1912	Discussion of the results. Health By-laws, summaries and reports of infectious and contagious diseases	(Province of Ontario, 1883; The Legislative Assembly of Ontario, 1884, 1912)
City Board of Health Reports, Hamilton 1909 & 1911	Descriptive analysis of health issues raised in Hamilton	(Bowman & City of Hamilton, 1912; Roberts & City of Hamilton, 1910)
City Council Minutes 1880-1883	Descriptive analysis into concerns and requests of these committees	(City of Hamilton Council, 1878-1882)
City Directories 1911-1912	Data Checking. If address information is missing in the	(Vernon, 1911)

	death registry, city directory can be accessed to obtain a better spatial sample for individuals.

2.2.1.5 Characteristics of the sample

The disaggregated nominal census would be an ideal data source for a spatial demographic analysis, however a comprehensive analysis of the nominal census was not feasible for the present study as transcription and analysis of the vast number of data points would have been prohibitive. The published census, however, does provide information about the living population (denominator), a useful source to analyze change in population over time and, as such, was consulted for this research (Canada Census and Statistics Office, 1911, 1914, 1915; Canada Department of Agriculture and Statistics, 1882; Department of Trade and Commerce, 1915). Table 2-2 provides the total population count and percent of the total population for infants and working age people as found in the 1881 and 1911 census for each year (Canada Census and Statistics Office, 1911, 1914; Canada Department of Agriculture and Statistics, 1882). Information of interest for this research found in both censuses includes age and sex composition, nativity (origin of the population), and occupation.

Table 2-2. Population Number for Each Time Period, Selected Sample, and Total Population, Data Source: Census of Canada, 1882 & 1912

Year	Infant: < 1 year of age		Working Age: 15-64 years		Total Population
	n	%	n	%	n
1881	954	2.65	22306	62.04	35956
1911	1767	12.15	57807	70.50	81996

Changes in how information was aggregated occurred between the 1881 and 1911 censuses. For example, in the 1881 census categories (sex, religion, and origin of the

population) were broken down at the ward level but, in the 1911 census, these same categories were broken down by a west and east division based on James Street. Another change to aggregation across the periods was in the structure of occupation of the populations. In the 1881 census, occupation type was defined individually and, where applicable, by sex; in the 1911 census, however, a more detailed level of information was provided for occupation, including workers by sex, nativity, and age. Unfortunately, occupations of the people were not recorded geographically (e.g. by ward or east/west section) in either census record. Although such discrepancies persist in the data structure over time, valuable population information can still be obtained to offer supporting evidence that informs the research questions and purpose.

A civil death registry can provide substantial information about historical population health disparities (see, Herring & Korol, 2012; Ludlow & Burke, 2012; Madrigal, 2003; Sawchuk et al., 2002; Sawchuk et al., 1985; Swedlund & Donta, 2003; Swedlund & Herring, 2003). Death registries contain individual-level information on the causes of death found in any given year (Ontario Vital Statistics, 1880-1882, 1910-1912). When conducting historical research using death registry data, two factors must be considered. First, over time the type of information included in the registry may vary and affect the ability of the researcher to analyze categorical independent variables (i.e. the inclusion or exclusion of information). Second, concepts of disease aetiology change over time (e.g. miasmas to germ theory), which can alter how deaths were classified. This second factor will be discussed later in the data analysis section. Temporal inferences can still be acquired, but these two factors must be considered in the interpretation and analyses of the data. Although such factors can influence how the information is interpreted, empirical historical research is essential to advance knowledge of Canada's mortality history because "…only the analysis of individual-level data can show

important aspects of mortality, such as social class differences and patterns of mortality by cause of death" (Emery & McQuillan, 1988, p. 136).

A sample was taken from the death records for the purposes of the analysis (Ontario Vital Statistics, 1880-1882, 1910-1912); Table 2-3 provides the count of each sample for each period. In most cases, the variables were consistent across both periods (e.g. date of death, sex of the deceased, age of the deceased, occupation, birthplace, and cause of death). One key variable, however, was lacking in the earlier period: location of death. By the later period, spatial information was included in the record as place of death and, if different from residence (e.g. in hospital), the place of residence was also intended to be included. Unfortunately, residence was not consistently recorded. Other inconsistencies in data collection hindered the ability to compare data across periods. In the earlier period, religious affiliation was included, but left out in the later period. In the later period, race was included in the record, but excluded from the earlier period. In any event, the category *race* as it was used in the death registry cannot be used for analysis due to inconsistencies in the information provided. In some cases actual racial identifiable terms (e.g. white, coloured) were used, but in other cases nationality was given (e.g. Canadian, English, and Irish).

Table 2-3. Number of Deaths for Each Time Period and Selected Sample, Data Source: Ontario Death Registry, 1880-1882 & 1910-1912

Year	Infant Deaths	Working Age Deaths
1880–1882	513	623
1910–1912	783	1319

While inconsistencies occurred in the characteristics of the data source over time, the information still provides important insights into the population at risk of dying, those who have succumbed to death, and the urban processes that may impede urban health (e.g. the public

health infrastructure). Such characteristics in the available data sources determine the approach to the analyses.

2.2.2 Data Collection

Census information pertaining to population age, sex, origin, occupation, and religion were transcribed into tables from the published 1881 and 1911 censuses into Microsoft Word and Excel Files for demographic analyses (Microsoft Corporation, 2010 & 2013). Data from the civil death records were collected from the online source 'Ancestry.ca', as well as from microfilm obtained from the Archives of Ontario (Government of Canada, 1869-1938). Initially, all deaths from 1880–1882 and 1910–1912 were transcribed 'as is' into two MS Excel files, one for each period. Additional columns were subsequently included in order to maintain location reference: the page reference in 'Ancestry.ca' and the schedule page number in the registry. The death registry schedule page number was used to identify missing entries in the 'Ancestry.ca' record, which were then accessed from the national death registry microfilm (Government of Canada, 1869-1942), obtained via interlibrary loan from the provincial Archives of Ontario. The purposive selected sample of infant deaths (under one year of age not including stillbirths) and working age deaths (15-64 years of age) from 1880–1882 and 1910–1912 were then selected out and put into SPSS (22.0) (IBM Corporation, 2013) for the data cleaning, coding (e.g. age groups, cause of death, birthplace, sex, seasonality), and analyses.

Several historical maps of Hamilton were collected for the HGIS analyses, dated as closely as possible to 1881 or 1911 (Table 2-4). Library and Archives Canada in Ottawa have a rich collection of Canadian maps, some that have been made available online through digitization. Two City of Hamilton maps (1882 and 1910) and one Fire Insurance Plan of Hamilton (1878) were accessed through the Library and Archives Canada online database (City

Engineer, 1910; Hamilton Ontario, 1878; The Canadian Almanac, 1882). The condition of access for all three maps was 'open' (access code 90), meaning the records were unrestricted and may be consulted for research and reproduction purposes. Hard copies of the Fire Insurance Map Plans (1911 with revisions to 1916) were located for the later period through the Lloyd Reeds Map Collection at McMaster University (Hamilton Ontario, 1911) and have since been digitized as part of this research. Again, these maps were older than 90 years and were therefore within the public domain for research and academic use as per copyright restrictions. With permission granted, a digital high-resolution image of the Fire Insurance Plan was assembled from plates digitized using an SLR camera. The photos had to be taken while the plates remained in their protective plastic casing, which in some cases created a glare on the image. The information was for the most part still discernable for creating historical addresses for the HGIS layers for each period. Photography was repeated during the second data collection period for cases where the digitized copies were not legible.

Table 2-4. List of Maps Collected, Map Date, and Location of the Maps

Map Title	Map Date	Location of the Map
Map of the City of Hamilton	1882	Library and Archives Canada [Online]
Map of the City of Hamilton	1910	Library and Archives Canada [Online]
Fire Insurance Plans of Hamilton	1878	Library and Archives Canada [Online]
Fire Insurance Plans of Hamilton	1911-1916	Lloyd Reeds Map Collection, McMaster University

Local History and Archives was the main source for collecting copies of photographs and images for the photo/image analyses. Most visual media were collected during the second data collection trip. After receiving permission, photos taken around each period that were relevant to the study were copied digitally using an SLR Camera to get the best resolution of each image (Lamb, 2014-2015). As well, Local History and Archives have uploaded a number of photos

onto their website, which can be downloaded and used for research purposes (Local History and Archives, n.d.).

Other sources of information were collected to add qualitative context to the results of the study. In doing so, sources such as annual reports and city council minutes provide details of the urban environment and population health in Hamilton from various boards and/or committees, such as the health, labour, immigration, water, and sewer boards. As Table 2-1 above shows, these documents range in dates of availability. For example, the province of Ontario did not have a board of health until 1882, and Hamilton did not have one until 1884. Most city-level information for the earlier period came from the City Council Minutes microfilm reels obtained from the Local History and Archives, which contained a large amount of information. Annual City health reports were available for the later period. Reports came from one of three locations: Local History and Archives; Provincial Archives of Ontario; and the Medical History Library and Archives at McMaster University.

The variety of informative sources collected for this study facilitates the mixed methods approach. Mortality and census information were used to quantitatively examine life and death in Hamilton. Various maps were used to build the HGIS layers that create an historical representation of Hamilton and analyze spatial mortality patterns during the 1911 period with the best possible accuracy. Analyses of photos provide qualitative context to understand the social and built environment that can build connections between people and their human and built environments. Finally, textual documents such as city council minutes and annual reports can be used to highlight concerns raised by the municipality, but also to contextualize the results of the quantitative analyses. Together, these resources generated a synthesis of life and death in Hamilton, at the end of the nineteenth century and at the start of the twentieth.

2.2.3 Data Analyses

This research employs a mixed-method design for data collection and data analyses. Although this research tends to follow the explanatory sequential mixed-methods design where HGIS and qualitative methods are used to help explain the quantitative results, it was not necessary to follow a sequential procedure in the data collection phase due to the focus on a historical rather than a current population. The analysis of the data collected, however, does follow the explanatory sequential process. First, demographic patterns using the census were analyzed for each period to contextualize the population at risk (the denominator). Second, mortality patterns using the death registry were analyzed for each period using descriptive and inferential statistics. The results from inferential statistics are integrated with an HGIS analysis that maps mortality patterns of significant sample characteristics for the later period. Tying all of the results together was a photo analysis and textual documentation to inform the evidence and contextualize life and death in Hamilton. The three methods within this mixed-method model—quantitative, HGIS, and qualitative—will be explained here separately.

2.2.3.1 Quantitative methods

Quantitative methods for this research were designed to examine changing demographic patterns over time and, where possible, across space. The temporal and spatial mortality patterns assist in understanding the risks faced in Hamilton at two points in time. The analytic processes of the two key sources, the 1881 and 1911 Censuses and 1880–1882 and 1910–1912 death records are described in this section.

Censuses

Information from the published 1881 and 1911 censuses have already been categorized. Although the aggregated categories can hinder comparisons over time, the information can be

useful for understanding changing demographics in the city of Hamilton. Consequently, the method of analyses does not allow for a straightforward spatial comparison between these two periods, but can still contribute to an understanding of the changing demographic patterns that were likely the result of increasing industrialization in Hamilton.

To get a sense of who was living in Hamilton in 1881 and 1911, the censuses provide a breakdown of the population into age and sex (Canada Census and Statistics Office, 1911, 1914; Canada Department of Agriculture and Statistics, 1882). Although age and sex information were not given a spatial attribute, the information provides a general examination of the population structure for the city and any changes that occurred over time. Age and sex information was used to create population pyramids, to generate sex ratios, and as the denominator for crude and specific (age, sex, and cause) mortality rates.

The history of Canadian migratory patterns reveals that, as the Canadian economy grew rapidly in both the industrial and agricultural sectors, immigration from countries other than the British Isles correspondingly increased. This phenomenon was especially prevalent at the turn of the 20th century and has been termed the *Great Migration* by labour historians such as Avery (1979) and Heron (1979; 1988). Urbanization and industrial development caused rapid population growth due to the influx of large numbers of immigrants from non-British countries required to fill less skilled industrial positions, such as labourers (Avery, 1979; Heron, 1988; Zunz, 1982). Using demographic information on population origins can lend credence to the process of industrialization as an explanatory factor for changing urban demography in Canadian industrial cities, such as Hamilton.

Occupation can be used as a proxy for socioeconomic status when categorized appropriately because the type of work can reflect social status. For some occupations, it can

also be used as a basic health risk indicator in terms of safe and unsafe work environments, described more recently as key determinants of health by the Public Health Agency of Canada (Public Health Agency of Canada, 2011). Together, as a socioeconomic and a work-related indicator, occupational status can also be used as a proxy to better understand spatial urban health patterns that may result from unequal quality of living conditions and indicate groups most likely to be affected by such urban public health infrastructural disparities. Occupation was not aggregated by ward (1881) or cardinal east/west (1911) census. Nevertheless, the secondary literature suggested that in Hamilton class-based segregation in the 1880s was arranged north/south (lower working-class and higher-class, respectively), before it shifted spatially by the 1910s, by which time class segregation cleaved along an east/west divide (lower working-class and higher-classes, respectively). Supporting literature along with primary sources can help to identify probable occupational and health divisions in Hamilton. As well, the later 1910 census aggregates workers by sex, nativity, and age, which is useful for understanding variations in the types of work (i.e. skill) by sex, immigration, and age. The information examined from the census can help to understand the population at risk, which will lead into quantitative analyses of working-age mortality and infant mortality.

Civil death registry records

Attention to limitations of data variation, ideas of disease causation, classification of historical causes of death, and sample size are important considerations in the data analyses of mortality patterns. Four databases were created to contain deaths by sample and by year: 1880–1882 Infant Mortality, 1910–1912 Infant Mortality, 1880–1882 Working-age Mortality, and 1910–1912 Working-age Mortality. In creating four databases, analyses can be conducted within each period for each sample and then used to understand any changes that occurred over time.

Creating two databases by sample would have led to overgeneralized coding for some of the independent variables (such as occupation, birthplace, accident mortality) due to the lower frequency of such factors in the earlier period, which could have hindered insights into the socioeconomic changes that industrialization caused in Hamilton. As a result, this research, while modest and conditional on subjects dying, seeks to examine each period and sample independently as a method to understanding changing mortality patterns over time and across urban space.

Comparing causes of death with current western medical disease aetiology can be used to analyze past health risks however, it also has its difficulties. Concepts of disease aetiology and disease nomenclature have changed over time; in turn, the body of ideas within the western epistemology are best understood as products of the era when they were in use. Recorded causes of death were shaped by the local and historical contexts and, thus, analyzing historical mortality is confounded by changing disease environments, diagnostic criteria, and terminology (Anderton & Hautaniemi Leonard, 2004). There were cases where historical causes of death were symptomatic (relate to symptoms or complications rather than primary cause), included multiple primary causes of death and incorporated vague, misused, and inconveniently employed nomenclature (Alter & Carmichael, 1999; Beemer, 2009; Moriyama, 1993; Padiak, 2009). Such cases can therefore be lost to the analyses because they do not fall within the current standard for classifying diseases, the International Classification of Diseases (ICD) 10 (World Health Organization, n.d.-a).

Since the inception of international disease coding practices, revisions have been conducted on a decennial basis to ensure the best representation for causes of death and defining diseases in line with current trends and transitions in medicine (Moriyama, 1993;

Whitehead, 2000). The predecessor to the ICD, titled International List of Causes of Death (ILCD), was first published in 1900, with its 2nd edition published in 1909 (Department of Commerce and Labor, Bureau of the Census, & Durand, 1911). The ILCD2 specifically accounted for cause of death classification by numbering specific causes of death and identifying alternative nomenclature. For example, the cause of death title *Tuberculosis of the Lungs* (#28) includes designations that refer to deaths from tuberculosis of the lung, such as *consumption*, *phthisis*, and other perhaps less known names such as *caseous bronchitis* and *specific pneumonia* (Department of Commerce, Bureau of the Census, & Harris, 1914). Additionally, each cause of death categorized in the ILCD2 (Department of Commerce and Labor et al., 1911) is found within a broader grouping of diseases (Table 2-5). General Diseases (I.), however, appears to be a catch-all category, including infectious diseases, cancers, and diabetes. While the system in use at the time may be idiosyncratic and based on out-dated concepts, knowledge of current disease aetiology and corresponding ICD10 entries can aid in analyzing or translating past classification schemes, thereby developing appropriate substitutes to code cause of death.

The revisions of the ICD, according to Moriyama (1993), were too detailed for classifying historical causes of death, which the author attributes to the traditional principal of single cause mortality. Although single primary cause mortality is often practiced in recording deaths (with secondary cause separate), there were problematic cases when a primary cause is assigned two or more causes of death. Typically in a situation, for example, where three causes were listed, the first mentioned is the primary cause with the second and third as underlying causes, respectively (Broderick, 1955). Yet there were exceptions, such as when the wording refers to the secondary listed as primary causes (e.g. pneumonia followed by measles) or when

the second listed disease is considered the prominent cause of death (e.g. chronic bronchitis and measles) (Broderick, 1955; Department of Commerce et al., 1914). In 1914, the problem of joint cause mortality was addressed through the development of the Index of Joint Causes of Death (Department of Commerce et al., 1914).

Table 2-5. International List of Causes of Death, as found in Department of Commerce and Labor et al. (1911)

Disease Group	Causes of Deaths	Classification Numbers
I	General Diseases	1-59
II	Diseases of the Nervous System and of the Organs of Special Sense	60-76
III	Diseases of the Circulatory System	77-85
IV	Diseases of the Respiratory System	86-98
V	Diseases of the Digestive System	99-118
VI	Nonveneral Diseases of the Genitourinary System and Annexa	119-133
VII	The Puerperal State	134-141
VIII	Diseases of the Skin and of the Cellular Tissue	142-145
IX	Diseases of the Bones and of the Organs of Locomotion	146-149
X	Malformations	150
XI	Early Infancy	151-153
XII	Old Age	154
XIII	External Causes	155-186
XIV	Ill-Defined Diseases	187-189

Classifying historical causes of death should undergo multiple coding schema, starting with single nomenclature classification using a temporally relevant classification system and moving into more general disease classifications (in accordance with the research purpose) that take into account historical and current disease nosology. Using an historical and current approach to coding mortality data allows an historical epidemiologist to deal with unknown disease nomenclature, overgeneralized grouping of mortality, and multiple primary causes of death. Thus, best practice in classifying historical cause of death is to code mortality using

disease classification schema that best reflect the period of time, while still maintaining the integrity of current knowledge of disease aetiology and informing the purpose of the research. In this research, specific causes of death are either left separate due to their significance in representation in the sample or grouped into classes or categories of death, as described above. The term *causes of death* is used herein to refer to both the causes of death and classes of death that are found in the mortality record.

When sample sizes are either too small or too large, errors or biases in a quantitative analysis can occur and, thus, analysis can lose statistical power (Lenth, 2001; Nemes, Jonasson, Genell, & Steineck, 2009; Tanaka, 1987). In a logistic regression model, the odds ratio can be skewed in small sample sizes showing extreme value estimates, but this extreme value estimate decreases as the sample size increases (Nemes et al., 2009). Thus, overestimation of an exposure or even a result of small sample size (reporting larger effects than larger samples) is known as *small study effect* (Nemes et al., 2009). Although large sample sizes produce more precise analyses, there is a point when a larger sample size does not substantially improve the precision of an analysis (Sullivan, 2012). A sample size that is too large may also lose statistical power or become overpowered, where the chances of detecting potentially non-substantive differences increase, with the possibility of the results becoming meaningless as everything becomes significant (Lenth, 2001; Tanaka, 1987). Determining an adequate sample size is study specific (Nemes et al., 2009). Additionally, determining the variables is also study specific. According to Burke (2001), overfeeding regression models with independent variables that have no found statistical association can lead to the loss of explanatory power to interpret the model. Frequency analyses can be used to help code and select variables for analyses. A cross-tabular chi-square test can be used to confirm the absence of complete separation between dependent and

independent variables for a multinomial logistic regression analyses. This study takes into account the concerns raised above in the coding and analyses of the quantifiable death registry data.

In each of the four databases created for this study, variables (date of death, sex, age, occupation, birthplace, and cause of death) were coded using SPSS (22.0). Causes of death were first coded by individual disease classification using the ILCD2. Where multiple primary causes were listed, the Index of Joint Causes of Death (Department of Commerce et al., 1914) and the Grammars of Death database (Hautamiemi Leonard, Anderton, Swedlund, Beemer, & Robinson, n.d.) were consulted. Additionally, when necessary, the current ICD10 was consulted to gain additional knowledge of disease aetiology and classification for more general disease groups. This coding process and frequency analyses of causes led to the first generalized cause of death schema for working age and infant mortality (Tables 2-6 and 2-7, respectively), which also allowed for the creation of cause-specific mortality rates. The term *all other* represents a catch-all category for causes of death peripheral to those of immediate interest to this study. It is expected to see a large percentage of deaths attributed to the 'all other' category as it includes deaths from a variety of causes, such as those that occurred from childbirth (puerperal), premature births, haemorrhaging, additional chronic diseases (such as diabetes), diseases of the nervous system (such as paralysis), and external causes (such as sun stroke, exposure, suicides, murders). *Ill-defined* refers to deaths that were considered ill-defined (e.g. old age < 70 years or symptomatic cause: undefined fever) in the ILCD2 or where cause was *unknown* or not given. Throughout this dissertation, *tuberculosis* refers to respiratory tuberculosis; *infectious respiratory cause* refers to all infectious respiratory diseases except tuberculosis; and *infectious non-respiratory cause* refers to the remaining infectious causes of death.

Table 2-6. Frequency and Percent of All Causes of Death for the Working-age Sample (age 15-64 years), Data Source: Ontario Death Registry, 1880-1882 & 1910-1912

Cause of Death	Frequency		Percent	
	1880–1882	1910–1912	1880–1882	1910–1912
Tuberculosis (Respiratory)	154	178	24.7	13.5
Infectious Respiratory Cause	80	108	12.8	8.2
Infectious Non-Respiratory Cause	59	83	9.5	14.3
Heart Disease	47	96	7.5	7.3
Cancers & Malignant Tumours	29	116	4.7	8.8
Accidents (non-violent)	20	117	3.2	8.9
All Other/Ill-defined	234	622	37.6	47.2
Total	*623*	*1319*	*100.0*	*100.0*

Table 2-7. Frequency and Percent of All Causes of Death for the Infant Sample (age < 1 year), Data Source: Ontario Death Registry, 1880-1882 & 1910-1912

Cause of Death	Frequency		Percent	
	1880–1882	1910–1912	1880–1882	1910–1912
Diarrhoeal Cause	114	155	22.2	19.8
Infectious Respiratory Disease	78	101	15.2	12.9
Nutritional Cause	68	174	13.3	22.2
All Other/Ill-defined	253	353	49.3	45.1
Total	*513*	*783*	*100.0*	*100.0*

Other variables that were also coded from the mortality dataset to provide information on their potential as explanatory variables include date at death (seasonality), age at death, sex of the deceased, birthplace, and occupation (for the working-age sample). Date of death was coded into the following seasons: spring (March, April, and May), summer (June, July, and August), autumn (September, October, and November), and winter (December, January, and February) (Tables 2-8 and 2-9). Gastrointestinal-related mortality and infectious respiratory-related mortality (pulmonary tuberculosis not included) have a seasonal pattern: the former are prevalent during warmer months (summer and autumn), and the latter during winter and spring months (Madrigal, 2003; Sawchuk & Burke, 2003). This follows a pattern found in historical cases as,

for instance, infant diarrhoea (or weanling diarrhoea) in 1860s Gibraltar, was exacerbated by the long-standing water supply problem during the summer and autumn months (Sawchuk & Burke, 2003).

Infant mortality from diarrhoeal causes, such as cholera infantum, was the result of "…an interplay of factors, biological, environmental, and economic…" (Thompson & Keeling, 2012, p. 472). Thus, understanding seasonal spikes (especially in the infant mortality sample) assists in understanding conditions in the urban environment, especially in relation to accessible clean water supply. During the first year of life, infant diarrhoeal causes have a seasonal effect, usually heightened risk during the summer and autumn months where there is an increased facility for bacterial pathogens (Cheney, 1984; Sack, 1997). Urbanization likely compromised the quality of food items, such as milk and meat, prior to the period of refrigeration, whereby spoilage increased with distance between rural and urban centres (Craig, Goodwin, & Grennes, 2004). There is also some evidence supporting a seasonality of pulmonary tuberculosis in the late winter and early spring potentially related to vitamin D variations, increased indoor activity, immune function, and food availability (Fares, 2011). Thus, analyzing seasonality for working age deaths assists in determining the importance of this independent variable against dependent mortality variables (e.g. infectious respiratory cause of death).

Table 2-8. Frequency and Percent of Seasonality of Death for the Working-age Sample (age 15-64 years), Data Source: Ontario Death Registry, 1880-1882 & 1910-1912

Season	Frequency		Percent	
	1880–1882	1910–1912	1880–1882	1910–1912
Spring	153	356	24.6	27.0
Summer	146	325	23.4	24.6
Autumn	138	324	22.2	24.6
Winter	186	314	29.9	23.8
Total	*623*	*1319*	*100.0*	*100.0*

Table 2-9. Frequency and Percent of Seasonality of Death for the Infant Sample (age < 1 year), Data Source: Ontario Death Registry, 1880-1882 & 1910-1912

Season	Frequency		Percent	
	1880–1882	1910–1912	1880–1882	1910–1912
Spring	107	166	20.9	21.2
Summer	180	255	35.1	32.6
Autumn	116	224	22.6	28.6
Winter	110	138	21.4	17.6
Total	*513*	*783*	*100.0*	*100.0*

Age at death is another important variable to consider as part of the analyses. Tuberculosis among working age cohorts reveals susceptibility peaks among adolescents between the age of 15-19 years and young adults from 20-30 years of age (Donald, Marais, & Barry, 2010). During the early 1900s, single-industry company towns in Cape Breton showed age-related tuberculosis susceptibility for young working-age males (Ludlow & Burke, 2012). Diarrhoeal causes were often exacerbated among postneonatal infants, with peak age of incidence for diarrhoea occurring between the ages of 6 months and 18 months, coinciding with the time of weaning and the introduction of new foods (Sack, 1997). For example, in the mid-nineteenth century in Massachusetts, infants being weaned from their mothers were known to succumb quickly to cholera infantum (Swedlund, 2010).

Furthering disease risk would have been declining breastfeeding rates during the late 19th century United States, when breast milk was commonly replaced with cow's milk (Thompson & Keeling, 2012). According to Swedlund (2010), age at death could be important for infant cholera because weaning and supplemental feeding would likely begin between ages one and two, and since teething occurs at approximately six to seven months, deaths in the first six months were likely indicative of alternative feeding practices prior to teething. In this

dissertation, the working-age sample was divided into three age categories: young working age (15-29 years), middle working age (30-49 years), and older working age (50-64 years) (Table 2-10). The infant sample was divided into two age categories: neonatal (up to 28 days after birth) and postneonatal (after 28 days to < 1 year) (Table 2-11). These age categories follow the same age definitions as presented by mortality statistics from the Centers for Disease Control and Prevention (CDC) (Centers for Disease Control and Prevention, 2009).

Table 2-10. Frequency and Percent of Age at Death for the Working-age Sample (age 15-64 years), Data Source: Ontario Death Registry, 1880-1882 & 1910-1912

Age at Death	Frequency		Percent	
	1880–1882	1910–1912	1880–1882	1910–1912
Young Working Age (15-29 years)	196	347	31.5	26.3
Middle Working Age (30-49 years)	232	488	37.2	37.0
Older Working Age (50-64 years)	195	484	31.3	36.7
Total	*623*	*1319*	*100.0*	*100.0*

Table 2-11. Frequency and Percent of Age at Death for the Infant Sample (age < 1 year), Data Source: Ontario Death Registry, 1880-1882 & 1910-1912

Age at Death	Frequency		Percent	
	1880–1882	1910–1912	1880–1882	1910–1912
Neonatal	145	264	28.3	33.7
Postneonatal	368	519	71.7	66.3
Total	*513*	*783*	*100.0*	*100.0*

Another explanatory variable considered in this research was biological sex of the deceased (Tables 2-12 and 2-13). Biological sex can reveal health risks that have a predominately male or female pattern of susceptibility. Sex differences in mortality are well-established: for instance, women generally live longer than men (Crews, 2003; Rogers, Everett, Onge, & Krueger, 2010). Risk factors can include an array of genetic, environmental,

behavioural, and cultural factors (Crews, 2003) and are age-related. Death rates vary between the sexes following birth, during adolescence (accidents and trauma kill more boys/men), and during reproductive years (maternal-related causes) (Crews, 2003). Thus, with industrial occupations prominent in this society, accidental mortality would likely occur more commonly among males than among females (e.g. Caplan, 2005; Ludlow & Burke, 2012). An important question to be explored in the analysis is to what extent there were sex-based differences in the mortality profiles, and if these change over time.

Table 2-12. Frequency and Percent of Biological Sex for the Working-age Mortality Sample (age 15-64 years), Data Source: Ontario Death Registry, 1880-1882 & 1910-1912

Sex	Frequency		Percent	
	1880–1882	1910–1912	1880–1882	1910–1912
Male	304	680	48.8	51.6
Female	319	639	51.2	48.4
Total	*623*	*1319*	*100.0*	*100.0*

Table 2-13. Frequency and Percent of Biological Sex for the Infant Mortality Sample (age < 1 year), Data Source: Ontario Death Registry, 1880-1882 & 1910-1912

Sex	Frequency		Percent	
	1880–1882	1910–1912	1880–1882	1910–1912
Male	289	431	56.3	55.0
Female	224	349	43.7	44.6
Not Given	-	3	-	0.4
Total	*513*	*783*	*100.0*	*100.0*

The birthplace variable was also considered for the quantitative analyses (Table 2-14 and 2-15). Birthplace can provide inferences into socioeconomic status and health risks, especially when documented inequalities (such as employment attainment) persisted between immigrant and non-immigrant groups. Nationality and race can also be used to infer any such inequalities; however, place of birth, as found in the historical death record, is a more consistent and, perhaps,

less socially constructed variable. Canadian labour history supports that inequalities in employment opportunities occurred: Canadian-born and British-born people were able to attain better positions (Avery, 1979; Heron, 1988). Furthermore, historical immigration policies on screening and quarantine protocols in countries such as Canada (Beiser, 2005; Gushulak & Williams, 2004) may lessen the burden of infectious diseases among foreign-born immigrant populations. Thus, place of birth can provide insights into health inequalities and geographical and social buffers that can affect mortality risk.

Table 2-14. Frequency and Percent of Birthplace for the Working-age Mortality Sample (age 15-64 years), Data Source: Ontario Death Registry, 1880-1882 & 1910-1912

Birthplace	Frequency		Percent	
	1880–1882	1910–1912	1880–1882	1910–1912
Hamilton	70	177	11.2	13.4
Other Ontario	47	328	7.5	24.9
Canada	106	125	17.0	9.5
United States & North America	41	56	6.6	4.2
England & Wales	137	260	22.0	19.7
Ireland	130	69	20.9	5.2
Scotland	64	65	10.3	4.9
Other Europe	21	68	3.4	5.2
All Other	0	6	0.0	0.5
Ill-Defined	7	165	1.1	12.5
Total	*623*	*1319*	*100.0*	*100.0*

Table 2-15. Frequency and Percent of Birthplace for the Infant Mortality Sample (age < 1 year), Data Source: Ontario Death Registry, 1880-1882 & 1910-1912

Birthplace	Frequency		Percent	
	1880–1882	1910–1912	1880–1882	1910–1912
Hamilton	422	660	82.3	84.3
Other Ontario	37	30	7.2	3.8
Canada	42	16	8.2	2.0
United States & North America	5	5	1.0	0.6
England & Wales	3	8	0.6	1.0
Ireland	-	-	-	-
Scotland	3	2	0.6	0.3

Other Europe	-	1	-	0.1
All Other	-	-	-	-
Not Given/Ill-Defined	1	61	0.2	7.8
Total	*513*	*783*	*100.0*	*100.0*

The last variable, occupation, is used for the working-age sample only (Tables 2-16 and 2-17). Occupation as an indicator for socioeconomic status can be used to inform spatial health risks and possible urban divisions of class. Classifying occupation requires knowledge of historical occupation categories (Van de Putte & Miles, 2005). The Historical International Standard Classification of Occupations (HISCO) is a published classification scheme of occupational titles found in the 19th and 20th centuries and is tailored to aid in coding occupations in various parts of the world, including Canada (Van Leeuwan, n.d.). HISCO was consulted along with the Government of Canada's National Occupational Classification to develop a classification scheme that enabled the examination of industrial and non-industrial occupations (Government of Canada, 2011; Van Leeuwen, Maas, & Miles, 2002). An additional category, *home duties*, was included. Although not considered an occupation in the traditional sense of the term, *home duties* encompass a large proportion of the female working-age mortality sample (79.3% of all females with occupation listed), and could capture environmental exposure in a neighbourhood given the amount of time these women would spend in that location. Thus, the category provides a representation of females that would otherwise be considered *ill-defined*. Females working outside of the home, however, were included in their respective categories. For example, domestics would be classified under *service*.

Frequency analyses of the first round of coded variables from the mortality dataset provide insights on the potential explanatory variables (age at death, date of death, sex of the deceased, birthplace, and occupation), and the key causes of death per year and per sample.

Following from the coded data above, the purposive sampling of infants and working-age people explores mortality patterns in relation to urban health and economic change. Thus, the quantitative analyses examine crude and specific mortality rates, moving into the cross-tabular chi-square analyses of the key variables to determine any absence of complete separation, and concluding with the multinomial logistic regression analyses of a bivariate categorical dependent variable in each analysis. Cross-tabular chi-square analyses tested the independent variables for significant association and for determining the final coding selection to avoid overfeeding the regression model and losing statistical power.

Table 2-16. Frequency and Percent of Occupations using HISCO Classification Schemes for the Working-age Mortality Sample (age 15-64 years), Data Source: Ontario Death Registry, 1880-1882 & 1910-1912

Occupation	Frequency		Percent	
	1880–1882	1910–1912	1880–1882	1910–1912
Professional, Managerial, Technical (HISCO 0-2)	29	54	4.7	4.1
Clerical, Sales, Service (HISCO 3-4)	77	161	12.4	12.2
Agriculture, Forestry, Fishery, Animal Husbandry (HISCO 5)	8	30	1.3	2.3
Industrial, Trades (HISCO 7-9.8)	125	301	20.1	22.8
Labourers (HISCO 9.9)	58	135	9.3	10.2
House Wife, Home Duties	105	300	16.9	22.7
All Other/Ill-Defined	221	338	35.5	25.6
Total	*623*	*1319*	*100.0*	*100.0*

Table 2-17. Frequency and Percent of Industrial Occupations for the Working-age Mortality Sample (age 15-64 years), Data Source: Ontario Death Registry, 1880-1882 & 1910-1912

Occupation	Frequency		Percent	
	1880–1882	1910–1912	1880–1882	1910–1912
Non-Industrial	106	215	17.0	16.3
Skilled Tradesmen	125	301	20.1	22.8
Labourers	58	135	9.3	10.2
House Wife/Home Duties	105	300	16.9	22.7
All Other/Ill-Defined	229	368	36.8	27.9

Total	*623*	*1319*	*100.0*	*100.0*

Logistic regression, as a multivariate statistic, allows the inclusion of other confounding factors or independent variables (such as age, occupation, birthplace, seasonality, and sex). The logistic regression model developed for this research follows from similar studies that have developed mortality-related measures, such as the mortality odds ratio (MOR) (Burke & Sawchuk, 2003; Lee et al., 2003; Miettinen & Wang, 1981). The mortality odds ratio in this research evaluates a specific cause of death and its relative distribution across groups (i.e. independent variables) when compared to 'other' causes of death. According to Miettinen and Wang (1981), if the rate for the 'other' causes of death is the same for the exposed as for the nonexposed, then the MOR equals the rate ratio and, therefore, the observed/expected ratio. In this study, the logistic regression models employed were conditional on dying from some cause to assess whether inputted independent variables significantly predicted specific cause mortality. Thus, the multiple levels of analyses for the mortality profiles appears as follows: first, crude and specific mortality rates for each sample; second, cross-tabular chi-square analyses; and third, multiple logistic regression to obtain mortality odds ratios. The results obtained from the quantitative analyses are further qualified through the HGIS analyses and qualitative analyses.

2.2.3.2 Historical GIS

Using a map to visualize historical data can lead to better conclusions about history (Bailey & Schick, 2009). HGIS often integrates multiple sources of information—typically archival sources—and is highly interdisciplinary combining scholarship with GIS (Gregory & Ell, 2007; Gregory & Healey, 2007; Knowles, 2000). GIS technology is geographic in nature but is a tool used across many disciplines such as history, public health, urban planning,

archaeology, and, of course, geography (e.g. Cromley & McLafferty, 2002; Cunfer, 2008; Harris, 2002; Knowles, 2008; Vaughan et al., 2005). Creating an HGIS is often quite time consuming (Gregory & Healey, 2007), because of the use of multiple sources of information that often need to be digitized and georeferenced to essentially create a map that reflects the historical time and geography of the project. Georeferencing is the process of aligning data that have geographic attributes to a known coordinate system to be "…viewed, queried, and analyzed with other geographic data."(ESRI, n.d.-b). HGIS is similar to GIS in structure, except that HGIS typically uses historical and archival sources to give historical spatial information (Knowles, 2000). Features that have a specific location can be aligned using a current basemap and georeferencing techniques (Bailey & Schick, 2009). One way to align them is to link historical points to their equivalents on a modern accurate map, a process known as *rubber sheeting* (Rumsey & Williams, 2002), whereby a digitized historical map is corrected to fit the geometry of a current map to account for errors in surveying and imagery of the historical source. In practice, there are limits to the effectiveness of this procedure. As Rumsey and Williams (2002, p. 6) note: "it is almost impossible to perfectly align an old map to modern coordinate systems because mapping methods before the age of aerial photography often only very imprecisely represented scale, angle, distance, and direction" (p. 6). Yet, the value of the information more than compensates for the residual errors that occur in georeferencing (Rumsey & William, 2006).

The following flowchart (Figure 2-6) illustrates the process used to develop the Hamilton HGIS for this research, a current Hamilton GIS database was obtained from the City of Hamilton to create a basemap (City of Hamilton Information Services, 2010). This file contains a large source of information in vector format that includes street centrelines with addresses, buildings, cemeteries, ward boundaries, escarpment, municipal boundaries, shoreline, and lake features,

providing a significant amount of detail found in present-day Hamilton. Shapefile (GIS data storage format) data from this current city was transferred into a file geodatabase (database for GIS data) and features (map objects), then used as base layers for editing, georeferencing, and geocoding (assigning an address location) historical maps and information for both study periods, 1881 and 1911 (ESRI, n.d.-a).

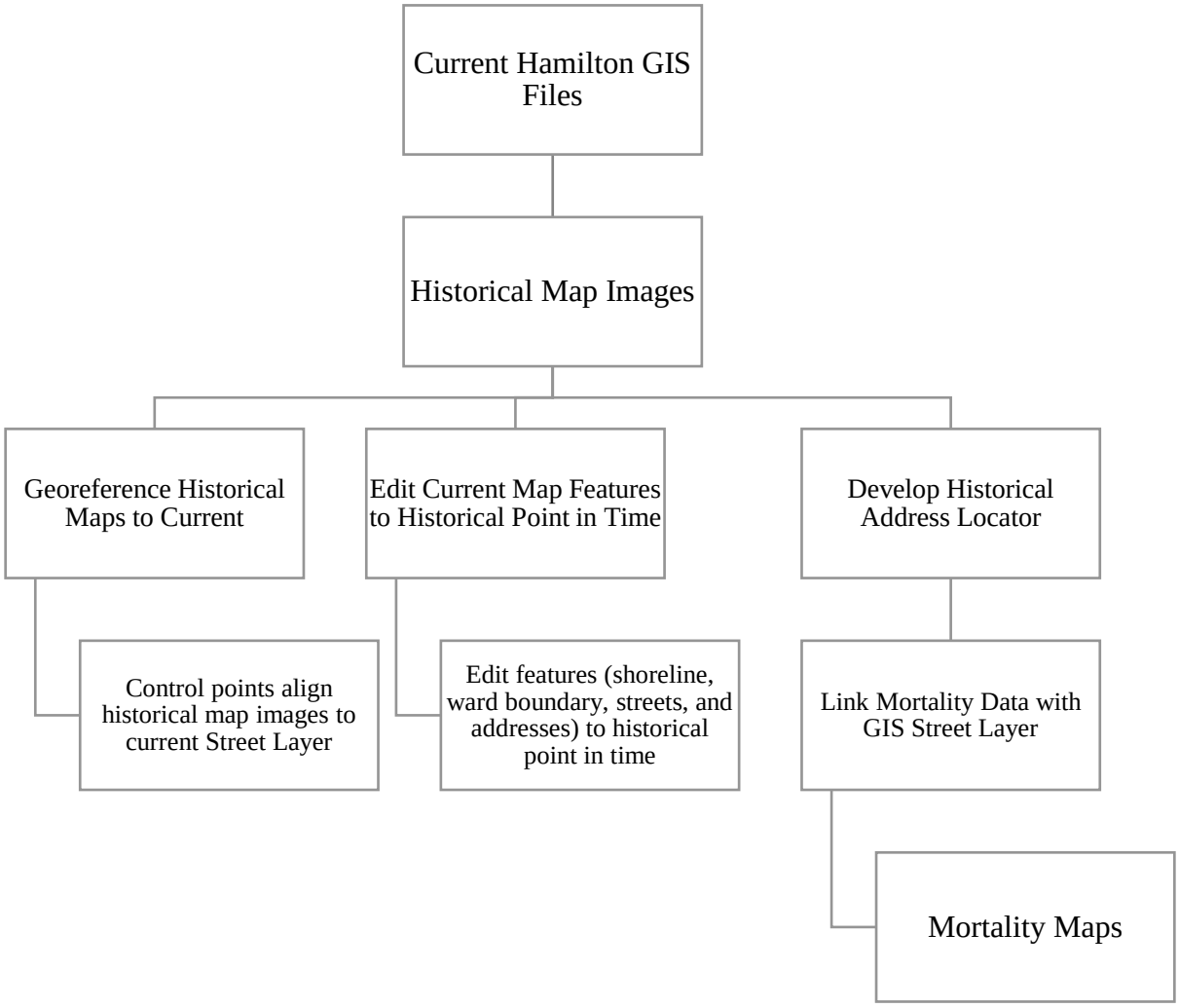


Figure 2-6. Flowchart of the HGIS methods used in this research

The City of Hamilton 1882 and 1910 maps were used initially to delete all current Hamilton map data that did not fall within the boundaries of each period. This was done, first,

by inputting the 1882 and 1910 digitized raster image of each map into a respective geodatabase (Hamilton Basemap 1881 gdb. and Hamilton Basemap 1911 gdb.). Second, each map (jpeg raster image) was georeferenced onto the basemap layer. Third, a polygon feature was created to delete features from the basemap layer (clip tool) that occurred outside the respective 1911 and 1882 map boundaries, creating a basic geographic outline for each period. GIS databases, in the past, have been quite poor at handling uncertainty, incompleteness, inaccuracy, and ambiguity (Gregory & Healey, 2007), which is why a modern basemap is a useful starting point for HGIS. Each map in this study was georeferenced using various control points at approximate centres of street intersections in the historical map raster and connecting the control point to its respective street intersection in the current GIS basemap. Although some inaccuracies occurred in the rubber-sheeting process, such as streets not aligning perfectly with the current basemap, overall the 1882 and 1910 map images were aligned accurately enough for HGIS editing of the street level data.

Both City of Hamilton maps, 1882 and 1910, displayed street level information (street names and location) that was used to edit current street centreline data (delete, create, and rename streets) and shoreline data to best reflect the geography for each period. As Hamilton grew, the shape of the shoreline changed, likely due to the filling in of inlets and building of wharves and docks to optimize the port for transportation and industrial purposes. Ward boundary maps were not located during data collection. Although the 1882 City of Hamilton map labelled wards, clear lines were not drawn to indicate actual boundaries. A textual document, however, was acquired that contained information on changing ward boundaries from 1813-1985, including hand drawn ward boundary maps (Kingdon, 1995), and was consulted to create ward boundary polygon features for each period.

Fire insurance maps are a useful resource for historical urban research as they provide detailed city plans of the built environment. Such maps often reveal information about the location and footprint of buildings, the material used for buildings, the names of buildings, and the number of stories and use of a building (Gilliland & Novak, 2006; Oswald, 1997). Additionally, these maps are an historical tour of cities' economic and civil structure:

> Fire insurance maps were the footprints of America's industrial revolutions ...[and were] enduring relics that bear witness to the mortality of businesses, industries and cities...With uniformity and clarity, these maps relay architectural details, of residential, commercial and industrial buildings. They provide at a glance, through the use of symbols, colors and labels, a snapshot of the built environment. (Oswald, 1997, p. 7).

Another important attribute found in Fire Insurance maps is street address information. Street address information is a vital source of data for research that examines historical spatial patterns of demography and epidemiology, as it can provide an accurate address location for a period in time, useful for geocoding historical information of where a person lived and died.

As the *characteristics of the sample* section explained, deaths from the earlier period did not include spatial information. Furthermore, the 1878 Fire Insurance Plans of Hamilton only included six plates covering a small geographic proportion of urban space. These limitations for the earlier period prevented the ability to plot mortality spatially. The plates for the Fire Insurance Plans of Hamilton, 1911-1916, were georeferenced to edit current street addresses to match the later period of this study. The current City of Hamilton street centreline data included addresses in the dual ranges style. This style provides address information on the right and left of the centreline. The US Address Locator – Dual Ranges style enables the input of house ranges on the right and left of the centreline (Tufts University, 2005 - 2012). This style was then used when creating an Address Locator for HGIS street addresses for the 1911 city of Hamilton.

Common errors found using dual ranges are positional accuracy of the streets and the accuracy of the street information (Tufts University, 2005 - 2012). The development of historical addresses to geocode historical information (e.g. demographic and epidemiologic) will likely provide a more accurate picture of spatial patterns than plotting the same historical information on a current street address map. Although recreating an HGIS street address map is quite time consuming, the outcome is a more accurate spatial examination of the information as streets undergo name, address numbering, and, in some cases, locational changes over time:

> Accurate spatial boundaries were key to all calculations derived from geographically located data. Without using historically correct and accurate unit ward boundaries, one cannot tell whether statistical changes reflect changes in population, changes in boundary lines, or both. (Knowles, 2000, p. 452).

Thus, the HGIS street centreline data for Hamilton reflects the 1911 period, allowing for the geocoding of the mortality data using the created Hamilton 1911 Street Address Locator. The mortality maps created show specific locations of cause and associated factors as per the results of the quantitative analyses. This spatialization of mortality provides an in-depth picture of potential spatial health inequities, leading to a better understanding of the possible health determinants at play, the locations that may have a higher health risk, and the populations at a higher risk. Although ward level mortality rates would have been a useful method to aggregate data first by a rate to control for bias of population density and disease clustering and then into specific mortality location, limitations of the level of aggregation provided in the published 1911 census led to mapping the data based on frequency.

Death registration between 1910 and 1912 included a section on place of death and residence. When a death occurred in an institution, for example, residence was also supposed to be included; however, residence was not always provided. Including deaths using the institution as the address would not provide an accurate picture of where people lived when they died, and,

thus, may provide an overrepresentation in areas where these institutions were located and underrepresent possible residential areas of higher health risk. Particularly when considering the environment, as this study does, using institutions can potentially skew the spatial results. For example, the 'infants home' and 'rescue home' were both located in the southwest quadrant of the city; thus, including deaths where an institution is the only spatial locator may suggest the southwest was an area of risk. Similarly, Herring and Korol (2012) omitted from their analysis those deaths where only institutions were listed as place of death in the registry. Cases missing an address in the death registry were then cross-referenced using the 1911 City Directory to obtain, when possible, a spatial location for the individuals (Vernon, 1911). Of the total causes of death included in the HGIS working-age sample (n = 392), 89 cases (22.70%) lacked a spatial property in the registry. The total causes of death included in the HGIS for the infant sample (n = 256), 32 cases (12.50 %) lacked a spatial property in the registry. While difficulties occurred due to the nature of the historical record and the lack of complete information, the use of HGIS techniques in creating historical maps can increase the accuracy of the information plotted. Thus, while it may not always be possible, researchers interested in using GIS in historical research can benefit from developing a GIS that is as close as possible to the period under study. A number of street features changed in Hamilton, from renaming of whole streets to changing street numbers. The use of multiple maps to identify and edit such changes can, and likely did, improve the matches made when geocoding historical features.

2.2.3.3 Qualitative methods

As Creswell (2014) notes, in the explanatory sequential mixed-methods design, the quantitative results not only inform the sampling procedure but can also drive the qualitative analysis. Thus, the results obtained from the quantitative analyses and HGIS were further

qualified using textual and visual archival documents that describe urban development and health concerns in Hamilton around each period of time (1880–1882 and 1910–1912). Furthermore, photos and images collected from the archives were analyzed using historical photo analysis techniques. The orientation of this explanatory sequential mixed-method design uses the qualitative information to support the results from the quantitative. By moving through the various sequences of statistical analysis and HGIS, the qualitative results are analyzed after the HGIS to enrich the story of life and death in Hamilton.

Additional archival information produces a narrative that adds substance to the numbers presented in the quantitative results. The use of historical sources in this manner has been found in other studies by researchers working in historical epidemiology (e.g. Herring et al., 2003; Meindle & Swedlund, 1977; Sawchuk & Burke, 2003). For example, Herring et al. (2003) used documents from the period to describe the destitute conditions found in northern Aboriginal communities. Sawchuk and Burke (2003) used historical health reports to describe the housing conditions and state of public health in Gibraltar. Using additional historical sources of information as narrative to supplement the quantitative results appears to be a fundamental element to an historical epidemiological approach. According to Swedlund and Donta (2003), medical history is one of four domains that encompass a comprehensive view of historic epidemics from a bio-anthropological perspective (the others are historicity, epidemiology, and molecular biology). Medical/health information can often be found in historical annual health reports. When annual reports are not available, other sources, such as community council minutes, may contain information regarding health concerns. This study uses supplemental documents such as Board of Health Annual Reports (both City and Provincial), City Council

Minutes, and Provincial Immigration Reports to understand not only Hamilton's medical history, but also aspects of urban development that may indicate potential health risks.

Photographs and other visual content can provide a wealth of knowledge concerning the social and built environments that shape a geographical space, in other words, as a visual source for social knowledge, social understanding, and urban development. Essentially, a photograph itself is a source of information (Peters & Mergen, 1977). Photo analysis as a research method emerged from the social sciences, predominately anthropology and sociology, whereby the researcher generates the visual data (Borchert, 1982). These photos can be created by the researcher or by study participants (e.g. PhotoVoice). With historical photos, the researcher is limited by what is available in the record. Often photos will lack information about the context or content of the picture (Borchert, 1982). As well, photographs, as with other documentation, are cultural artefacts and can be the product of bias, such as the agenda of the photographer or agency (Borchert, 1982; Margolis, 1988). Although a picture may distort reality by being taken out of context, misrepresented verbally, or manipulated, there is a presumption that something exists (Davidson, 1981). Thus, historical photo analysis seeks to understand the literacy of the visual, to bring the context and content back to the photograph, and/or to better understand the historical period that is under study, rather than, as Davidson (1981) suggests, just to illustrate the text. For instance, Phillips (2003) incorporated photographs to show and describe the types of labour that occurred at the Oneida asylum in the 19th century. Although, in some cases, in-text descriptions are not used, the images themselves tie into the text and add richness to the historical content (Swedlund, 2010). In this study, an historical photo analysis approach is used in combination with written documentation to highlight historical ideas of society and culture, and to illustrate spatiotemporal change in an urban locale.

2.2.4 Data Limitations

A variety of limitations can occur when conducting historical research. The reality is that researchers only have access to what remains in the record and has survived the tests of time, and, thus, there will ultimately be relevant information that is unavailable to the researcher. Smith (2003, p. 311) states that, although the only difference between historical and current research is the provenance of the data, the reliance on "…archival data imposes its own special constraints [and] grants its own freedoms…" (p. 311). Historical research requires careful consideration of how to interpret the data; in addition, the limitations of missing or illegible data can pose potential problems with a research design. Historical research limitations will be discussed here followed by a description of the research limitations in this study.

2.2.4.1 Historical research limitations

Limitations faced by historical researchers relate to the data itself and the process of data collection. Often information in census or death records will be filled out completely, but occasionally portions of such information are missing, illegible, or ill-defined. What was recorded and what was not recorded can be attributed to the information that was considered most vital by the registrar or the information that was provided to the registrar. For example, age at death, gender, and cause of death are usually deemed crucial information and recorded in the death registry. Other information however, was missing or inappropriately categorized, such as race, nationality, residence, or occupation. With respect to occupation, for example, occupations may be listed but are ill-defined or lack additional information, such as *retired* without any previous occupation listed. In other cases, occupation is simply stated as *not available* or left out altogether. At the same time, information that appears consistent in the death record (e.g. age at death), particularly in older records, might be inaccurate. This follows from similar concerns of

age accuracy as noted in the 1911 census. Bulletin XVIII of the 5th Canadian Census states (1914):

> It is impossible to claim absolute accuracy for census statistics of ages because (1) some people do not know their true age, (2) some people appear to deliberately mis-state their age and (3) in many instances the report of ages is not made by the person or persons concerning whom the enumerator is prosecuting the enquiry as to age. (p.1)

The bulletin continues this explanation by stating that, in the case of boarders and lodgers, inaccurate reports of age were especially common, and that there was a tendency towards age heaping by rounding up or down (e.g. 20 or 25 years of age rather than 21 or 24 years of age) (Canada Census and Statistics Office, 1914). This is not to say that the information is false or unusable; rather, the information can still provide a rich context concerning the population under study.

Historical material that is hand-written can be illegible, which can lead to transcription errors. Transcribing hundreds, even thousands, of individual-level death entries, for example, even under a skilled eye, can lead to errors in the database. Recorder error may also occur when archives and/or genealogical databases digitize historical documents. Frequently, archival resources are being digitized for user accessibility and preservation of the original. Missing data and illegibility can occur by accidentally missing pages during preservation or through poor image quality during the scanning process. Information can also be lost due to poor conservation over time. An excellent example of a poorly maintained archival source was seen during previous master's research by this author (Ludlow, 2009), where steel company accident reports (fatal and non-fatal) for Sydney, Nova Scotia, during the early 20th century had, at some point in time, been exposed to moisture, leaving only a few pages of the large textual document legible.

Conducting temporal analyses in the historical record can be quite difficult as the researcher is at the mercy of what information was collected and how information was collected.

Censuses and the death registries offer excellent examples of probable limitations of temporal-based research, as over time, the information included can change. Other considerations of temporal limitations in the historical record may relate to the availability of annual reports, maps, photographs, and other sources of historical information. At what point in time, for example, annual reports were implemented will factor into the ability to compare health concerns over time, especially if other documentation (e.g. council minutes) is not available in the record.

Essentially, all research has its limitations. Historical research, however, is conditioned on the availability of archival sources as data. With few exceptions, new historical data cannot be created under controlled circumstances. How the recorder interprets the information is also a potential limitation, as improper interpretation can lead to loss of data. The condition of the source, whether original or digital, will affect its legibility, and errors can occur during the transcription process, perhaps leading to missing or inaccurate data. Whether working with original or digitized documentation, the problem of missing data, recorder error, and/or illegibility are the realities of historical research.

2.2.4.2 Limitations of this research

Although a working age and infant sample was chosen for the purpose of this research, all deaths from 1880–1882 and 1910–1912 were originally collected for a total of 5,722 deaths (n = 1,901 and n = 3,821, respectively). The original Excel database contained the ancestry.ca page number and the death registry schedule page. During the coding of the selected samples for analyses, any concerns or discrepancies were then back-referenced to the file in the ancestry.ca database to be confirmed and/or fixed. This was useful to confirm that missing data was not due to a transcription error (e.g. occupation and place of death/residence). Another example where the data appeared to be a possible transcription error were cases when cause of death was

labelled ‘still-born’, but the infant was given an age at death. These cases were then back-referenced to be corrected or established ‘as is’ in the ancestry.ca database. Also, although names of the deceased were not used in this research, keeping the name of the deceased in the database helped to locate and delete any duplicate cases that occurred. The death registry also included a section on the names of the decedent’s parents, which was useful when searching for infant addresses by locating the father in the city directory (see, Vernon, 1911).

Inconsistent availability of data sources can lead to new approaches in examining information in the historical record. For example, live births were available in the 1881 census but not in the 1911 census, instead in the annual reports. Furthermore, in the annual reports, live births were not disaggregated by sex. A proxy was used for infant mortality to be able to construct specific mortality rates using sex and cause of death. This was done using the census population under one year of age. Infant mortality rates constructed in the historical record and in current countries with known poor reporting of infant death are often scrutinized for underreporting of live births—therefore deaths—which can underestimate the infant mortality rate upwards to 40% (Anthopolos & Becker, 2009; Atlas, 2013; Janjua, 2009; Kleinman, 1986; McCarthy, Terry, Rochat, Quave, & Tyler, 1980). Acknowledging the limitation of available information to obtain specific infant mortality rates and issues of underestimating, the proxy used in this study allowed for a more detailed analyses. Infant deaths labelled *stillbirth* but noting the infant was alive at birth was used with census living population under one year to estimate infant mortality, which would give a more accurate number of deaths than disregarding all infants labelled *stillbirth*. This proxy may lead to an overestimation and make it difficult to compare with other conventional infant mortality rates for the time period. However, this author suggests future studies in historical infant mortality may require innovative techniques to employ detailed

analyses of infant death in the past and, that infant mortality rates alone should be reviewed with caution in historical work.

When archival information is copied into another medium, the process of copying (microfilm, microfiche, and/ or digital) can produce poor quality images of the original source. There were instances in the death registry and city directory (on either microfilm or digital image) when the image was too dark to be legible. Illegibility of a data source was also attributed to the data collection phase of this research. Collecting large amounts of data in a short amount of time create instances when image quality is overlooked. For example, the city directory was copied from a microfilmed image where the images varied in brightness, contrast, and focus. This likely led to some of the copied images being darker, lighter, or slightly out of focus. When consulting the city directory, most images were legible, but some required viewing adjustments to get a better quality image. Lastly, when collecting photographs, some photos were attached to a photo album, which could have led to the image being distorted due to album binding. To account for this, and with permission from archivists, weights were used to lay each respective photo album page as flat as possible without causing any damage to the source.

As is the case with most historical research, many roadblocks occur, causing a researcher to use a variety of tools in concert to inform the research. This particular project, as the limitations express above, was no different. Using data from information collected for other purposes can create problems or pitfalls: “data for spatial analysis often arise from several different sources, and have seldom been gathered with the interests of the geographical epidemiologist in mind” (Staines & Järup, 2000, p. 15). Although such problems persist when using more modern sources that were created for a different purpose, these sources were the essential instruments in historical research. Historical research is presented with the unique

limitation of only being able to access what has survived in the record. As Swedlund and Herring (2003) note, what survives in the archives "… were 'accidental' data-sets. That is, the data were gathered for purposes quite different from the ways in which we employ and interrogate them today." (Swedlund & Herring, 2003, p. 2). Acknowledging that the data are not precise is crucial in historical research, as we cannot go back in time to ensure its accuracy. Thus, it is best to approach the results of an historical study as patterns or trends and not an absolute value (Sawchuk & Burke, 2000).

2.3 Conclusions

As the city of Hamilton continued its transition towards industrialization, spatial disparities in health were maintained. Hamilton conformed to the early urban development models and, while a concentric form was initiated with the CBD at the core of the city, the physical landscape presented the fundamentals of Mann's model of a British city—an incorporation of both concentric and sector models. Whether this is a by-product of civic leaders' pre-urban planning or a representation that perhaps some Canadian cities resemble the British urban model rather than the American urban model is beyond the scope of this dissertation. In scope, however, is that these urban models provide a contextual backdrop to understanding the potential 'locational' aspects of the population and urbanization. Understanding the spatialization of urban development shapes the context of Hamilton through an historical, physical, and social geographic lens. This chapter focused on providing a history of Hamilton's economic transition towards becoming Canada's industrial city and the potential spatial health inequities that arose as the city grew. Industrialization of Hamilton brought a rapid influx of the population, predominately working-class population. This led to continued socioeconomic segregation of class. The south and southwest were considered attractive

residential locations, which contrasts with an industrial sector in the northeast allowing the prevailing westerly winds to push polluted air eastward away from the city. As well, the examination of Hamilton's public health issues that arose with industrialization suggests the growing occurrence of a health divide that would create marked health inequities among the city's classes.

This chapter also restated the purpose of this research, provided detailed description of the research plan, and gave information of the limitations found in historical research. The mixed-method research design follows a similar approach to what Creswell (2014) defines as the explanatory sequential model, an iterative process using additional methods to explain the results of quantitative analyses. Two additional methods are used, HGIS and qualitative (descriptive text and image), to expand on plausible causal factors of where mortality was occurring and why it was occurring there.

CHAPTER 3
RESULTS

3.1 Introduction

The following chapter will present the results of the statistical data analyses, the HGIS results, and the qualitative findings. Analyses, using descriptive statistics, will inform on Hamilton's population structure around 1881 and 1911, and mortality patterns for the years 1880–1882 and 1910–1912. Following the descriptive analysis, is an integration of the inferential statistical results (logistic regression using mortality odds ratio) and spatial analysis to produce an in-depth mortality profiles. A qualitative inquiry into health issues that faced the population through historical documentation (both textual and visual) follows.

3.2 Demographic Analyses

As industry developed, Hamilton's population grew and changed in structure in response to the growing industrial economy. The urban landscape and geography also changed as a result of industrialization. Through statistical analysis of Hamilton's demographic profile using the 1881 and 1911 Census of Canada, the demographic phase of this research will reveal the results of changing demography and urban geography around 1881 and 1911 and will seek to respond to two research questions:

> What are the socio-demographic profiles in 1881 and 1911 Hamilton, and how did the profile change over time?
>
> Do these socio-demographic patterns reflect the temporal urban and economic development of Hamilton?

3.2.1 Quantitative Demographic Analyses

Demographic information collected from the Census of Canada returns for 1881 and 1911 was analyzed (Canada Census and Statistics Office, 1911, 1915; Canada Department of Agriculture and Statistics, 1882). In 1881, Hamilton's total population was 35,956, with 17,410 males and 18,546 females (48.42% and 51.58%, respectively). By 1911, Hamilton's total population was 81,996, with 42,178 males and 39,818 females (51.44% and 48.56%, respectively). The total population more than doubled over the 30-year span of the study period (Figure 3-1). Between 1881 and 1891, the city's population grew by 36.16%, at an annual rate of 3.62%. The lowest period of growth occurred between 1891 and 1901, with population growth of 7.50% over that time, or at a rate of 0.75% annually. The largest increase in population occurred in the last decade, 1901-1911, when the population of Hamilton grew at 55.79%, an annual rate of 5.5%. Thus, in total between 1881 and 1911, the population of Hamilton grew 128% at an average rate of 4.27% annually. Some of this growth may be attributed to physical growth of the city as it absorbed nearby hamlets and towns through amalgamation into the expanded city limits. Much of the land to the east, where most of the physical growth took place, however, was agricultural land; therefore, it would not have made as significant an impact as a growing migratory population.

The growth in total population over the period affected the age and sex structure found in Hamilton. Population pyramids show the age and sex structure at both points in time (Figures 3-2 and 3-3). Both 1881 and 1911 population pyramids present expansive population patterns, which is defined as the occurrence of a larger proportion (percentages) of the population in the younger age groups, with older age groups usually being smaller in size (United Nations Population Fund, n.d.). The 1881 pyramid shows a growing population with a high birth rate and

low life expectancy, but a slowing death rate as represented by the triangular shape and steepness of the sides—a population structure that is typically young and growing.

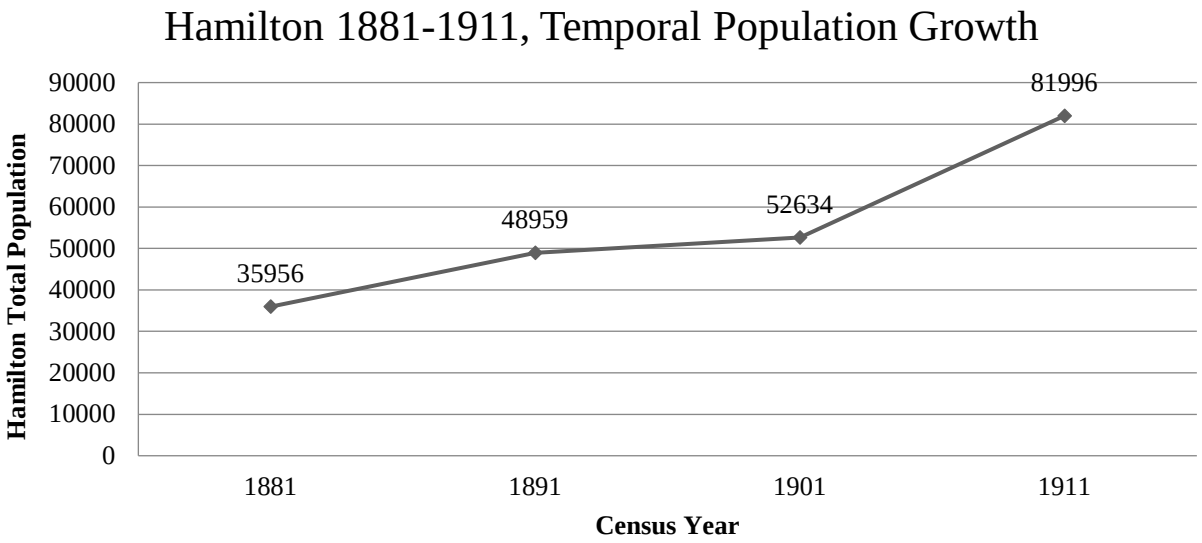


Figure 3-1. Population Growth of Hamilton Spanning 30 Years

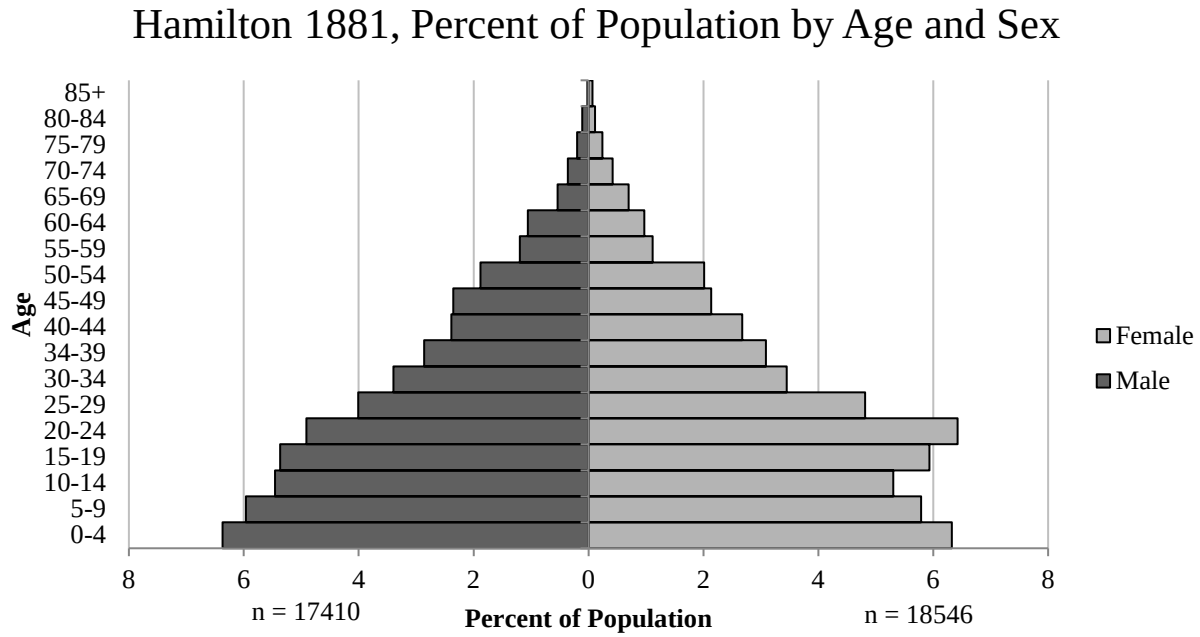


Figure 3-2. Population Pyramid for 1881 Hamilton

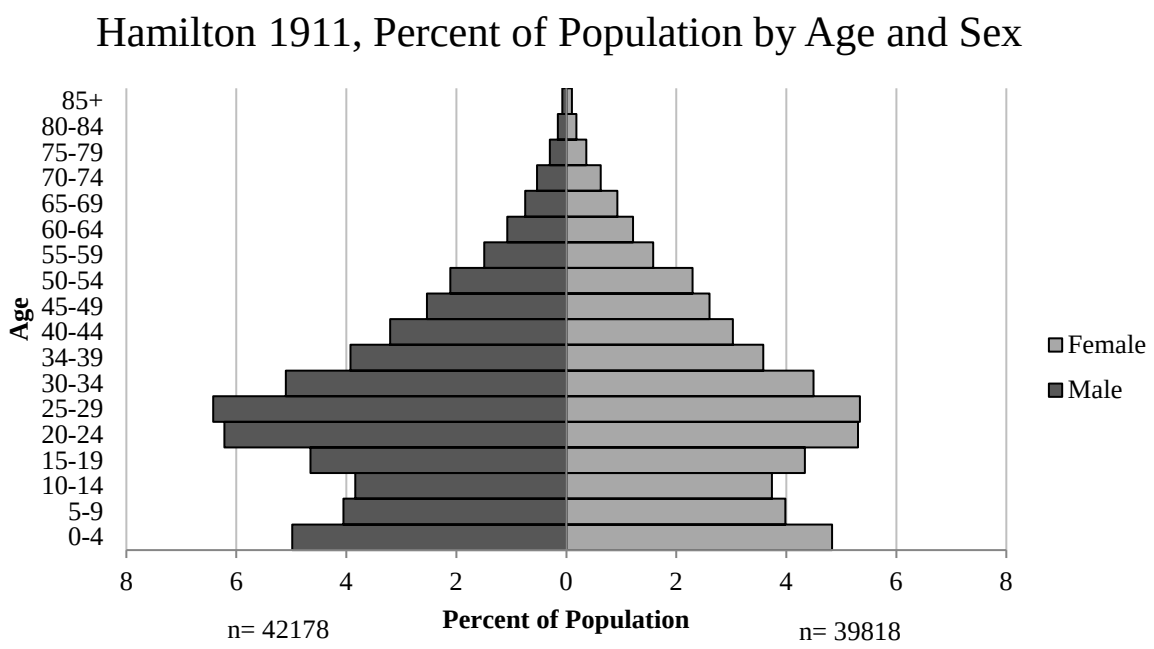


Figure 3-3. Population Pyramid for 1911 Hamilton

The 1911 pyramid also shows a growing population; however, the birth rate is lower and, as represented by the steepness and concavity of the sides, suggests that the life expectancy remained low with high death rates in the older ages. Apart from interpreting a population pyramid based on natural increase (birth rates and death rates), migration is another determining factor in the shape of a population pyramid (Population Reference Bureau, 2013). There were more young working-age women in 1881 when compared to young working-age men. In 1911, however, this finding was flipped as there were more working-age men when compared to working-age women. The bottom of the 1911 pyramid is restricted around ages of 5–19 years, but show expansion at ages 0-4 years. Restricted pyramids are often described as having declining births or high infant mortality. An alternative explanation, however, is the migration of individuals in their twenties. The larger proportion in this age group would cause an increase in younger age cohorts due to reproduction—similar phenomenon to the baby boom generation.

Further summary analyses are required to determine the reason for changing sex structures of males and females between 1881 and 1911 in Hamilton.

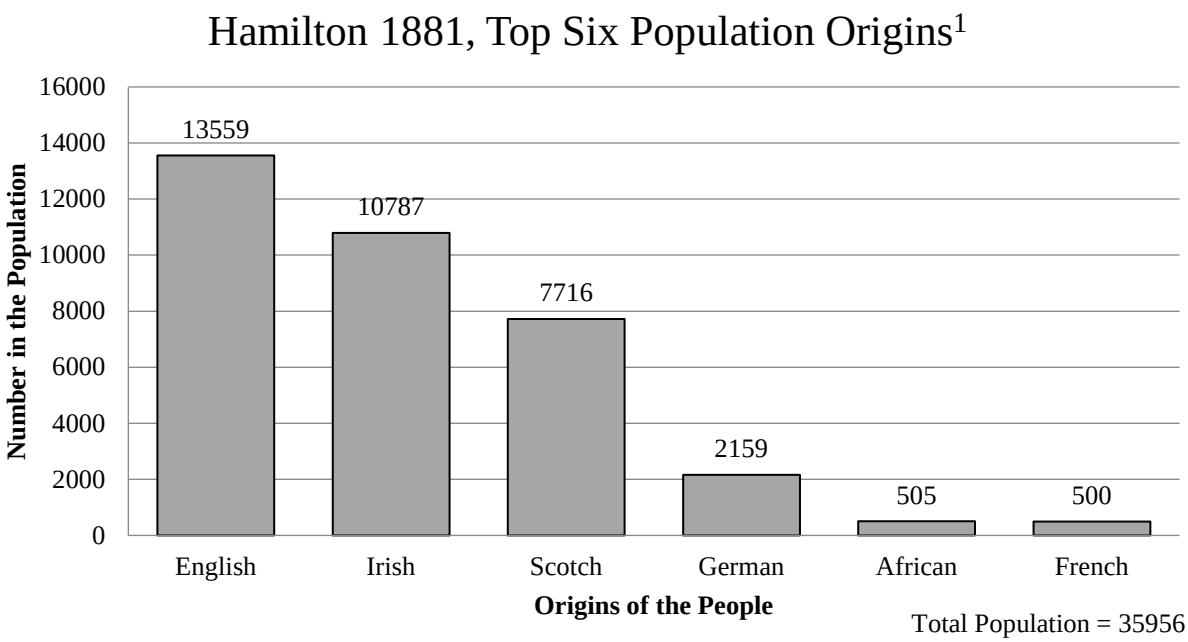
Sex ratio analyses conducted for Hamilton at both periods, 1881 and 1911, include the total sex ratio, the total working age sex ratio, and working age sex ratio broken down by age category (young, middle, and older working ages) to capture the distinct sex variations found within this cohort (Table 3-1). For the total 1881 population, there were fewer males than females (93.88 males per 100 females). By 1911, however, the sex ratio shifted and there were more males than females in Hamilton (105.93 males per 100 females). The sex ratio differences are more pronounced among the working age population for each period (1881 = 90.15 males per 100 females and 1911 =108.72 males per 100 females) and even more pronounced among the young working age population (1881 = 83.19 males per 100 females and 1911 = 115.39 males per 100 females). The results obtained from these sex ratios led to a further examination into temporal patterns of the origins of the people in Hamilton.

Table 3-1. Sex Ratios and Age-Specific Sex Ratios for 1881 and 1911, Hamilton

Sex Ratio	**1881**	**1911**
Total Sex Ratio	93.88	105.93
Total Working Age Sex Ratio	90.15	108.72
Young Working Age Sex Ratio 15-29 years	83.19	115.39
Middle Working Age Sex Ratio 30-49 years	96.86	107.69
Older Working Age Sex Ratio 50-64 years	100.68	91.85

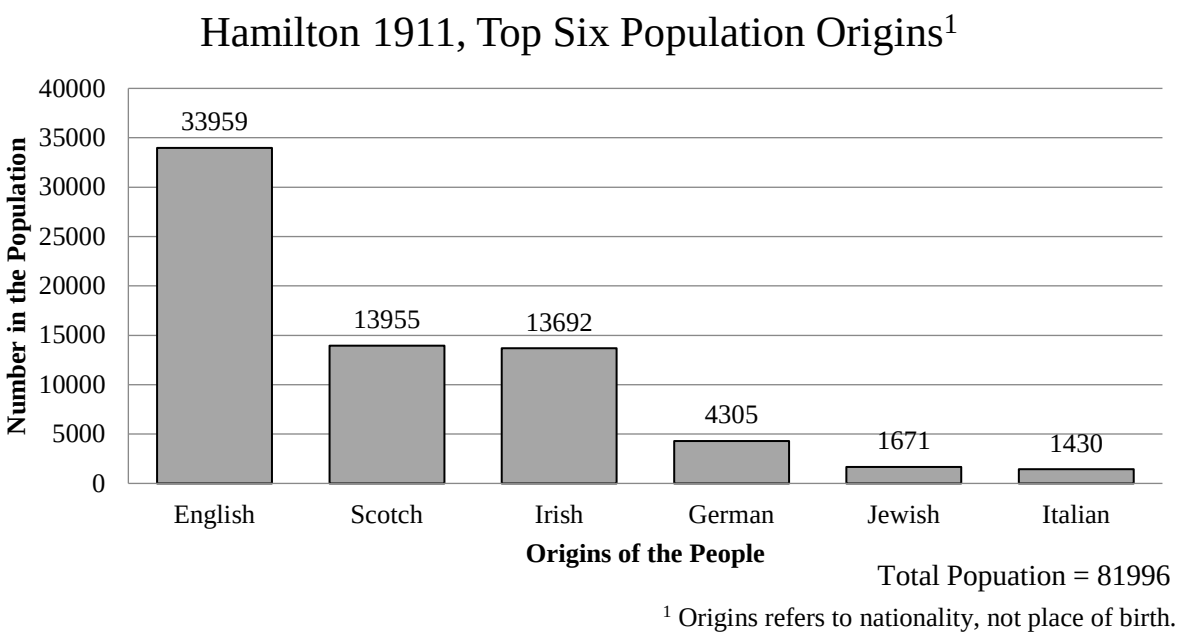
Origins of the population were examined for each study period and the top six origins are presented here (Figures 3-4 and 3-5). Origins of the population does not provide place of birth, but rather a sense of changing nationality and ethnicity in the population structure of a location. This was one example of a limitation to using aggregate census, as 'origins' was consistent in

both records, but birthplace was not consistently used. In 1881, population origins were mostly English (37.71%), Irish (30.00%), or Scottish (21.46%), being 89.17% of all origins of the people in the city of Hamilton. In 1911, a lesser proportion of the population, though still the majority, were English (41.42%), Scottish (17.02%), or Irish (16.70%), making up 75.14% of the total origins of the people. The slightly lower proportion of these origins in the population was likely due to an influx of people from other parts of Europe in the 1911 period or that, perhaps, immigration from the United Kingdom and Ireland was beginning to decline. At the beginning of the 20th century, immigrants to Canada mostly originated from the United States or the United Kingdom (Boyd & Vickers, 2000). During the 1910s and 1920s, however, the number of individuals originating from other European countries began to grow, although this growth was slow at first and rose to its highest levels between 1961 and 1971 (Boyd & Vickers, 2000).



[1] Origins refers to nationality, not place of birth.

Figure 3-4. Frequency of the Top Six Population Origins for Hamilton, Ontario in 1881



[1] Origins refers to nationality, not place of birth.

Figure 3-5. Frequency of the Top Six Origins of the Population for Hamilton, Ontario in 1911

A special report published in 1915 defined the *foreign born* population as persons who were born in a country other than the United Kingdom or dependencies of the United Kingdom (Department of Trade and Commerce, 1915). The information provided in the report only included information on individuals defined as *foreign-born*. In the 1881 census, the foreign-born population of Hamilton was 2,443 (Canada Department of Agriculture and Statistics, 1882), which made up 6.79% of the population of Hamilton. The 1915 report noted that, prior to 1890, only 1,188 of the population were classified as foreign-born. According to that report, the total foreign-born population in 1911 was 7,693, which made up 9.38% of Hamilton's total population (Department of Trade and Commerce, 1915). These results suggest a slight increase in the proportion of the population originating from outside the United Kingdom (and dependencies of the United Kingdom), which may have been caused by increasing industrial activity in the city. In order to assess this increase, an examination of temporal occupational differences was conducted.

Occupations of the people for both periods were categorized using HISCO (Van Leeuwan, n.d.; Van Leeuwen et al., 2002) and examined for any trends in occupational structure (Figure 3-6). At both points in time, production, equipment operators, and related comprised the largest percent of employment, 61.90% and 66.82% for 1881 and 1911, respectively. HISCO categorizes these together because workers are directly associated with the extraction of materials, manufacturing processes, operation of equipment (e.g. steam boilers, air gas compressors, and heating and ventilation), and transportation of materials (Van Leeuwan, n.d.). Manufacturing steel requires individuals to operate equipment such as the blast furnace, coking system, and other machinery, and transport raw coal, limestone or iron ore or partially processed materials (coke, smelted coal) on-site to be processed into the final product. The 1881 census did not supply a column for females, but rather included a female category only for specific occupations (domestics, nurses and midwives, teachers, nuns, laundresses, and dressmakers). Of the total working population in 1881, 87.57% were male and 12.43% were female. Of the total working population in 1911, 78.56% were male and 21.44% were female. There were 9% more women contributing to the workforce by the later census year. Temporal occupational patterns were further analyzed for males and females.

For both periods of time, 1881 and 1911, males made up a high percentage of workers in production, as equipment operators, and in related occupations, 70.47% and 74.01%, respectively (Figure 3-7). The top five male occupations within the production, equipment operators, and related group for 1881 and 1911, respectively, made up 63.34% and 68.85% of the total men in this group (Tables 3-2 and 3-3). During both periods, labourers and related workers were the largest subgroup (1881 = 20.39% and 1911 = 36.61%). In 1881, clothing production (textile industry) made up the second largest subgroup (17.35%). By 1911, metal processing

became the second largest subgroup at 11.73%, attributed to the increase in manufacturing industry, especially steel.

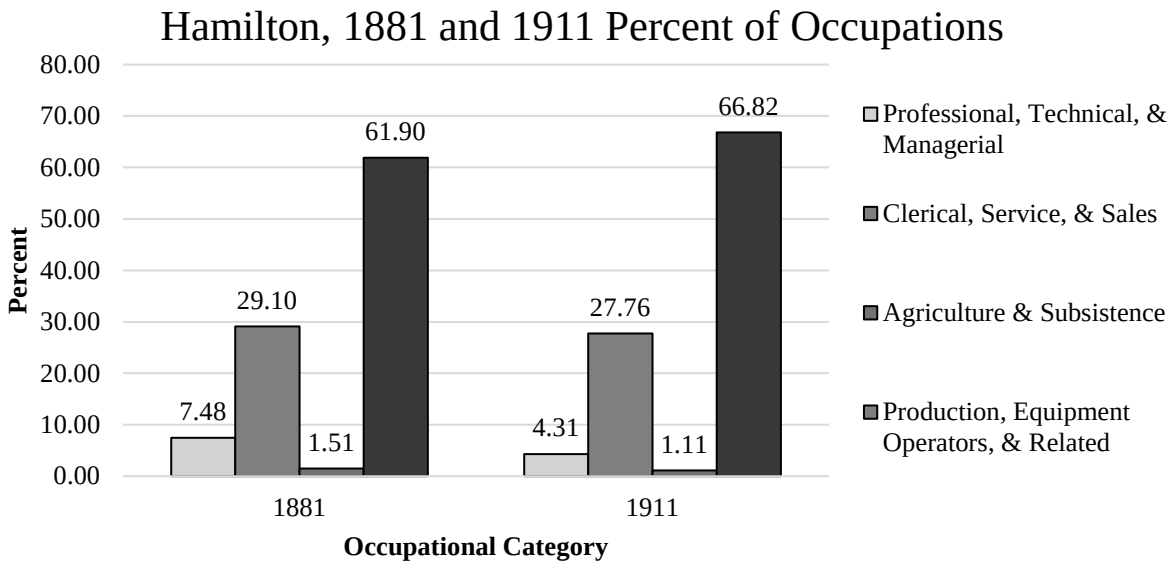


Figure 3-6. Percent of Occupations for Hamilton, Ontario 1881 and 1911, using HISCO Occupational Classification

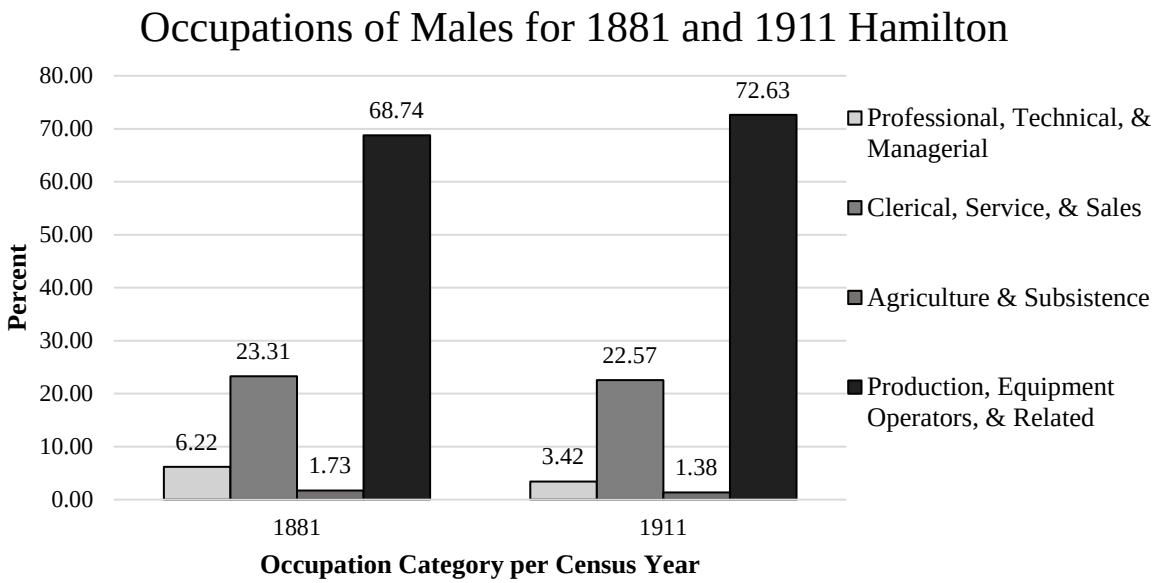


Figure 3-7. Percent of Male Occupations in Hamilton, Ontario for 1881 and 1911, using HISCO Occupational Classification

Table 3-2. List of the Top Five Male Occupations in the Production and Equipment Operators, Related group for 1881 Hamilton, Data Source: Census of Canada, 1882

Occupational Title: Production, Equipment Operators, and Related Group	**Number**	**Percent ***
Labourers, Related	1,604	20.39
Clothing Production, Related	1,365	17.35
Building, Construction, Related	826	10.50
Machine Production, Related	683	8.68
Transport &Equipment Operators, Related	504	6.41
Top Five Total	*4,982*	*63.34*

*Percent taken from total number of males found in the production and equipment operators, and related occupation group in 1881 Hamilton = 7866

Table 3-3. List of the Top Five Male Occupations in the Production, Equipment Operators, and Related group for 1911 Hamilton, Data Source: Census of Canada, 1912

Occupational Title: Production, Equipment Operators, and Related Group	**Number**	**Percent***
Labourers, Related	7,659	36.61
Metal Processing, Related	2,454	11.73
Building & Construction, Related	1,998	9.55
Transport & Equipment Operators ,Related	1,199	5.73
Metal Smiths & Toolmakers, Related	1,094	5.23
Top Five Total	*14,404*	*68.85*

* Percent taken from total number of males found in the production and equipment operators, and related occupation group in 1911 Hamilton = 15163

Working-age females revealed a more prominent change in occupational structure between the two periods (Figure 3-8). Clerical, sales, and service occupations remained the most common occupational category for females in 1881 (69.91%) and 1911 (46.79%). By 1911, however, women showed a higher percent of workers in the production, equipment operators, and related occupations when compared to the earlier period (1881 = 13.69% and 1911 = 45.50%). The top five occupations for females in 1881 and 1911 are shown in table 3-4 and 3-5. In 1881, the top five occupations made up 98.04% of all working females, with 66.62% of all working females found in the domestic and related occupational group (Table 3-4).

By 1911, the top five female occupations made up 64.85% of all working females (Table 3-5). Although domestic service and related occupations continued to represent a high proportion of all female workers, it was a much smaller percent (16.24%) of the total working females. A decrease of 50.38% in domestic service work between the two periods indicates a major shift in women's labour. The top five breakdown of occupations supports this shift, with more occupations found in production, equipment operators, and related group, namely in clothing production and related and weaving production and related being the first (20.73%) and third (12.86%) highest occupational group for women, respectively (Table 3-5). Thus, most working women in Hamilton by the later period were employed in the textile industry, increasing by 7.04% from the earlier period. In the early 20th century, Glenn (1991) found that most employment opportunities for young women in the United States were in factories, particularly the garment industry, which drew in disproportionately large numbers of young Jewish immigrant women. By 1911, the fifth most common nationality was Jewish. From this analysis, it appears that industrialization may have brought more opportunities to the female workforce. The 1911 census provides a further breakdown of the occupational data for nativity and sex, and working age and sex, allowing for an additional analysis of the 1911 census data. Labourer and related occupations were disaggregated from the production, equipment operators, and related group in response to the labour history literature noting inequalities in job attainment for what was defined, historically, as *foreign born* groups in Canada (Avery, 1979; Caplan, 2005; Heron, 1988).

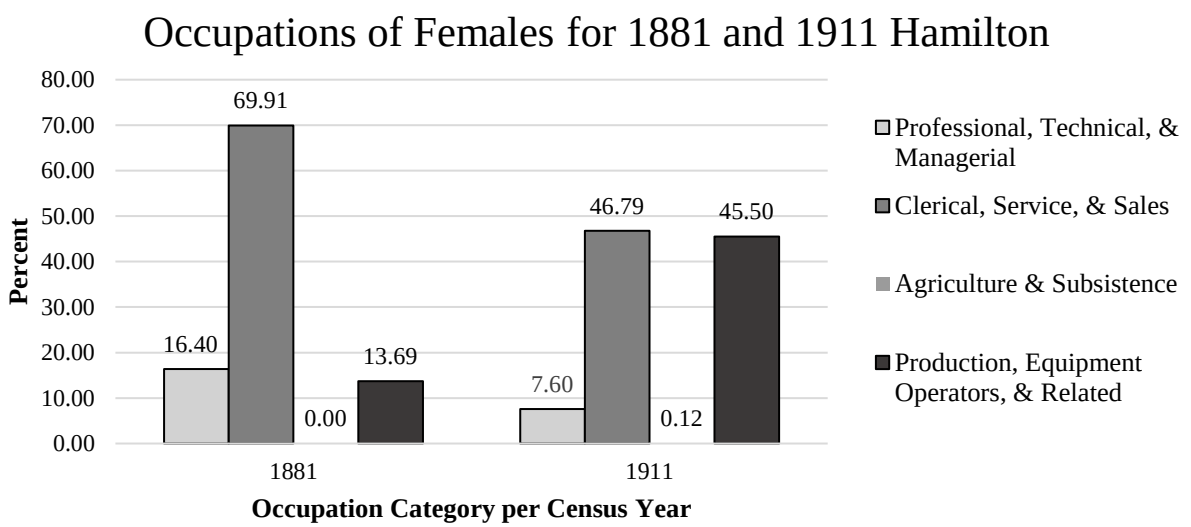


Figure 3-8. Percent of Female Occupations in Hamilton, Ontario for 1881 and 1911, using HISCO Occupational Classification

Table 3-4. List of the Top Five Female Occupations for 1881 Hamilton, Data Source: Census of Canada, 1882

Occupational Title	**Number**	**Percent***
Domestic Service and Related Workers	1,056	66.62
Clothing Production and Related Workers	217	13.69
Teachers	172	10.85
Workers In Religion	57	3.60
Laundresses and Related Workers	52	3.28
Top Five Total	*1,554*	*98.04*

*Percent of total number of working females in 1881 Hamilton = 1585

Table 3-5. List of the Top Five Female Occupations for 1911 Hamilton, Data Source: Census of Canada, 1912

Occupational Title	**Number**	**Percent***
Clothing Production and Related Workers	1,599	20.73
Domestic Service and Related Workers	1,253	16.24
Weaving and Related Workers	992	12.86
Saleswomen and Related Workers	678	8.79
Telephone Operators and Related	460	5.96
Top Five Total	*4,982*	*64.58*

*Percent of total number of working females in 1911 Hamilton = 7714

Nativity and occupation for working males in 1911 Hamilton is shown in Figure 3-9. Of the total working males, 51.41% were Canadian-born and 48.59% were immigrants. Canadian-born males occupied a fairly high percentage of the jobs within the professional, technical, and managerial group (69.81%), as well as the clerical, sales, and service occupational groups (64.50%). Workers in the production, equipment operators, and related group showed slightly more Canadian-born males than immigrant males (53.53% and 46.47%, respectively). Immigrant males, however, made up a much larger proportion of labourers and related workers compared to Canadian born males: 64.42% and 35.58%, respectively. Although the Canadian-born and immigrant male workers were close in terms of percentage of the total population, the breakdown within each occupational group presented substantial variations.

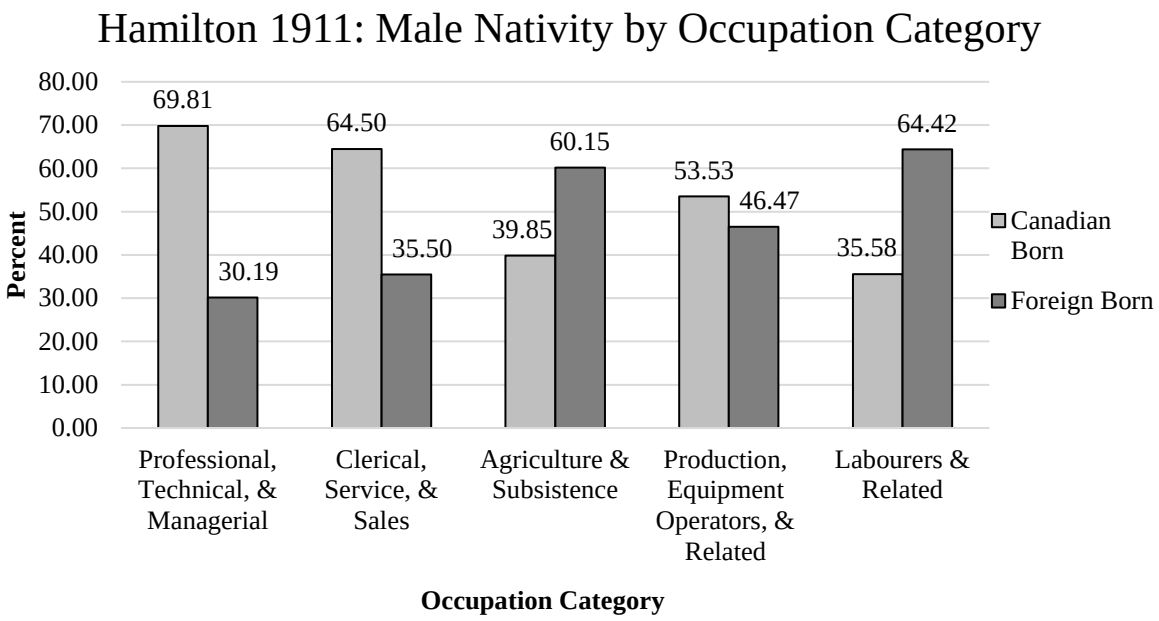


Figure 3-9. Percent of Males per Occupational Category by Nativity for 1911 Hamilton, using HISCO Occupational Classification

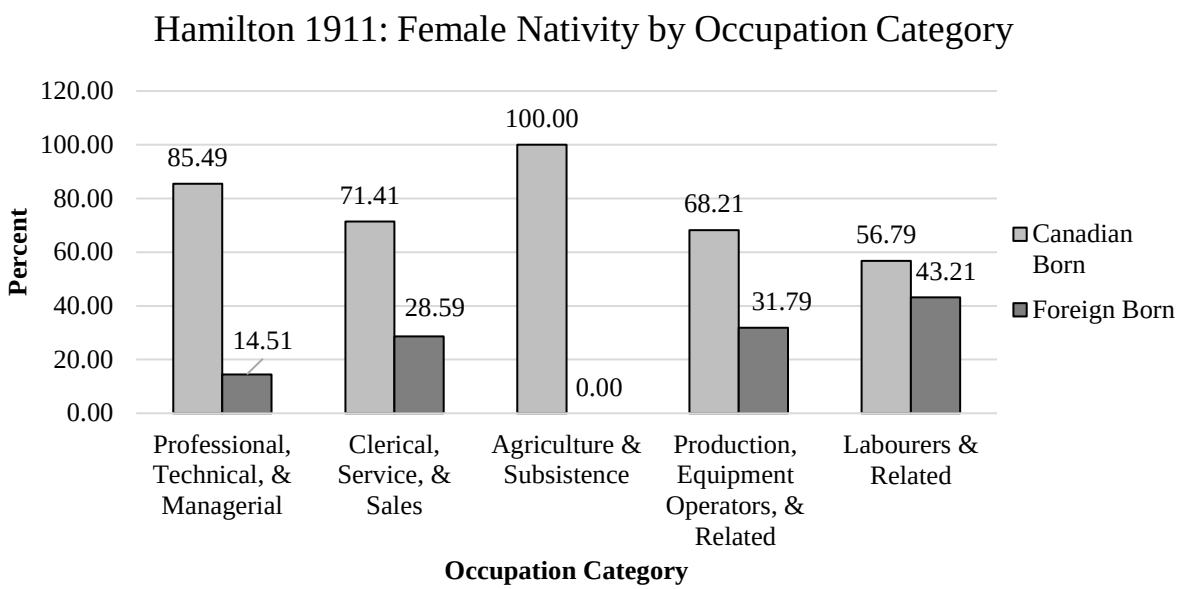


Figure 3-10. Percent of Females per Occupational Group by Origin for 1911 Hamilton, using HISCO Occupational Classification

Working age and occupation were analyzed for males in 1911 Hamilton (Table 3-6). The age groups in the 1911 census were not organized into evenly-distributed age groupings, but, instead, ages were listed as 10-14 years, 15-24 years, 25-64 years, and 65+ years, which may skew the interpretation of the data. Overall, the distribution of male workers in 1911 was 0.69% for 10-14 years of age, 27.12% for 15-24 years of age, 69.53% for males aged 25-64 years, and 2.66% for males aged 65 years and older. Male workers under the age of 25 years made up 27.86% of all workers. Child labour was common in industrial Canadian cities (Bullen, 1986; Hurl, 1988; Tucker, 1988). The introduction of wage-labour allowed women and children to become part of the labour workforce; children were often seen as part of the wage unit in a family (Hurl, 1988; Tucker, 1988). Many working-class families depended on second and third wage earners to stay above the poverty line (Bullen, 1986). Although some limitations and restrictions on child labour occurred in Ontario's factories in the 1900s, inspectors were aware of

and sensitive to family hardships, especially for widowed mothers whose welfare depended on their children's wages (Hurl, 1988).

Table 3-6 provides the breakdown of the percentage of occupational groups per age group. The occupation group with the highest percent of males aged 10-14 years was labourers and related occupations (50.26%). Although the overall number of males aged 10-14 years was much lower (n = 193) than the other ages, young boys, if working in industrial work, would often be found in labourer-type jobs. This type of job required little to no skill. The Sydney steel plant in Nova Scotia (circa 1910s), for example, employed young boys in the 'yards' to carry raw materials (e.g. coal and iron ore) to different sections of the plant for processing by more skilled men (Caplan, 2005). The second highest occupation group for male workers between 15-24 years of age was the labourers and related workers groups (32.28%). For male workers aged 25-64 years, a similar number of males worked in the clerical, service, and sales occupational group (22.19%), and the labourers and related workers occupational group (25.18%). As well, a similar percentage of male workers aged 65 years and older worked in the clerical, service, and sales occupational group, and in the labourers and related workers occupational group (28.05% and 24.43%, respectively). With the exception of men aged 10-14 years, the highest percentage of males worked in production, equipment operators, and related. Although the interpretation might be skewed given the breakdown of the age groups in the 1911 census, most males in Hamilton were working in the production, equipment operators, and related group.

Table 3-6. Male Occupations per Working Age for 1911 Hamilton, Data Source: Census of Canada, 1912

Occupational Group	**Age Groups**			
	10-14 Years % (n)	15-24 Years % (n)	25-64 Years % (n)	65+ Years % (n)
Professional, Technical, & Managerial	0.00 (0)	1.54 (117)	4.07 (793)	5.91 (44)
Clerical, Service, & Sales	14.51 (28)	21.12 (1,605)	22.19 (4,324)	28.05 (209)
Agriculture & Subsistence	1.55 (3)	1.29 (98)	1.34 (262)	3.49 (26)
Production, Equipment Operators, & Related	33.68 (65)	43.78 (3,327)	47.21 (9,198)	38.12 (248)
Labourers and Related	50.26 (97)	32.28 (2,453)	25.18 (4,905)	24.43 (182)
Total Number per Age Group	*193*	*7,600*	*1,9482*	*745*

Working age and occupation was also analyzed for females in 1911 Hamilton (Table 3-7). As with the male age groups, the uneven age groupings (ages 10-14 years, 15-24 years, 25-64 years, and 65+ years) may skew the interpretation of the data for female working ages. Overall, the distribution of female workers in 1911 was 1.66% for females aged 10-14 years, 52.03% for females aged 15-24 years, 45.36% for females aged 25-64 years, and 0.95% for females aged 65 years and older. Over half of the women employed were under 25 years (53.69%). Prior to the 1900s, it was common for women (except poor married women) to leave the workforce upon marriage; however, in the United States, the early 1900s saw a shift in that married women remained in the workforce (Kessler-Harris, 2003). Table 3-7 provides the breakdown of occupational groups per age group. Although the industrial process provided additional opportunities for female employment, those who relied on wages earned from mills and factories would often have to rely on two, and in some case three, wage earners to make ends meet (Kessler-Harris, 2003). Loss of wages due to accidents at work would therefore require women to stay in or re-enter the workforce. It is worth noting that forms of social security (e.g. worker's compensation, mother's pension) were emerging shortly after this study period;

however old age pension did not materialize until later in the 20th century, even though discussions were occurring as early as 1908 (Guest, 1997).

Results found that the clerical, service, and sales occupation group had the highest percent of female workers aged 15-24 years (48.11%), 25-64 years (47.41%), and 65 years and older (59.46%). Females aged 10-14 years, however, largely worked in the industrial and manual occupational group (66.92%). The production, equipment operators, and related group was the second most common occupation for female workers in the above-14 years age groups, with 43.98% of females aged 15-24 years, 37.76% of females aged 25-64 years, and 21.62% of females aged 65 years and older. Similar to the male data, the data interpretation might be skewed given the breakdown of the age groups, but, in 1911 Hamilton, most females in each of the working ages worked in the clerical, sales, and service occupational group or production, equipment operators, and related occupational group.

Table 3-7. Female Occupations per Working Age for 1911 Hamilton, Data Source: Census of Canada, 1912

Occupational Group	**Age Groups**			
	10-14 Years % (n)	15-24 Years % (n)	25-64 Years % (n)	65+ Years % (n)
Professional, Technical, & Managerial	0.77 (1)	3.17 (129)	12.52 (444)	16.22 (12)
Clerical, Service, & Sales	24.62 (32)	48.11 (1,957)	47.41 (1,681)	59.46 (44)
Agriculture & Subsistence	0.00 (0)	0.07 (3)	0.17 (6)	0.00 (0)
Production, Equipment Operators, & Related	66.92 (87)	43.9 (1786)	37.59 (1333)	21.62 (16)
Labourers and Related	7.69 (10)	4.74 (193)	2.31 (82)	2.70 (2)
Total Number per Age Group	*130*	*4,068*	*3,546*	*74*

3.3 Descriptive Mortality Analyses

The progression of Hamilton to a highly industrialized city after 1881 led to the changing population patterns described above. These changing patterns in the population at risk are

important to understanding population health outcomes, like mortality. Mortality patterns provide insights to urban health determinants through knowledge of disease pathogenesis and impact on the population. Using government death records from Ontario (Government of Canada, 1869-1938, 1869-1942), this research analyzed mortality of working-age and infant deaths that occurred between 1880–1882 and 1910–1912 to answer the third research question:

> What are the mortality profiles for 1880–1882 and 1910–1912 periods in Hamilton, are there any observed changes in the profile over time, and are these observations expected?

For each purposive sample (working-age and infants), a detailed quantitative mortality analysis was conducted using the data collected for 1880–1882 and 1910–1912 deaths for Hamilton from the Ontario Vital Statistics Death Records (Ontario Vital Statistics, 1880-1882, 1910-1912). This section will use broad mortality categories (major causes of death, and crude and specific mortality rates), and then move into the descriptive cross-tabular chi-square to test for independent variables. Using an iterative design, the results from each quantitative analyses will inform the next quantitative step, all building up to development of the logistic regression model that will be presented in subsequent sections.

3.3.1 Working-age Mortality Profile

A crude mortality rate (CMR) is calculated by dividing the total number of deaths in an area by the total population in an area. It is considered crude because it does not take into account the age or sex composition of the population, and so comparison between different populations may be problematic. However, in this case CMRs were calculated for the working age cohort for each of the two census years using the 1881 and 1911 census working-age population (n = 22,306 and n = 57,807, respectively) as the denominator.

An average mortality rate of the years combined was also conducted for each period. Table 3-8 presents the results for the CMRs. During the earlier period (1880–1882), the average mortality rate was 9.31 working-age deaths per 1,000 living working-age population. The highest mortality rate in the earlier period for the working-age sample was in 1882 with a rate of 10.31 working-age deaths per 1,000 living working-age people. Overall, the rate in 1882 was the highest mortality rate for either period. Although mortality rates among the working-age sample declined by the later period (1910–1912), the average mortality rate for 1910–1912 was 8.07 working-age deaths per 1,000 living working-age people. The lowest mortality rate for the later period occurred in 1910 with a rate of 6.59 working age deaths per 1,000 living working-age people. The rate in 1910 was the lowest mortality rate across both periods. Sex-specific mortality rates for each period were conducted using the average number of deaths of each sex (numerator) for each period over the working-age population of each sex (denominator) obtained from the respective census (1881 or 1911).

Table 3-8. Crude and Average Mortality Rates for Working-age Sample (15-64 years), Hamilton, 1880–1882 and 1910–1912

	Working Age Deaths per 1,000 Working Age Population
Year	**Crude Mortality Rate (CMR)**
1880	8.74
1881	8.88
1882	10.31
Average CMR 1880–1882	*9.31*
1910	6.59
1911	9.38
1912	8.23
Average CMR 1910–1912	*8.07*

Table 3-9 presents the results from this analysis. The average sex-specific mortality rate for each period revealed a general decrease for both males and females. During the 1880–1882

period, the average sex-specific mortality rate for males was 9.58 working-age male deaths per 1,000 living working-age males. For females, the average sex-specific mortality rate for same period was 9.06 working-age female deaths per 1,000 living working-age females. Males and females showed the highest mortality rate in this period during 1882, with the same rate of 10.31 per 1,000 living for each sex. The year 1880 also had a high male mortality rate of 9.46 working-age male deaths per 1,000 living working-age males. By 1910–1912, the average male working-age mortality rate was 7.53 working-age male deaths per 1000 living working-age males. For females, the average working-age morality rate was 7.69 working-age females per 1,000 living working-age females. The mortality rates per year for the later period (1910–1912) show that working-age males had the lowest mortality in 1910 (6.48 working-age male deaths per 1,000 living working-age males).

Table 3-9. Sex-Specific Mortality Rates Working Ages, Hamilton, 1880–1882 and 1910–1912

Working Age Deaths per 1,000 Working Age Population

Year	Male MR*	Female MR**
1880	9.46	8.10
1881	8.98	8.78
1882	10.31	10.31
Average MR 1880–1882	*9.58*	*9.06*
1910	6.48	7.08
1911	7.74	7.91
1912	8.37	8.09
Average MR 1910–1912	*7.53*	*7.69*

*Numerator = Working age male deaths, Denominator = working age male population.
** Numerator = Working age female deaths, Denominator = working age female population.

3.3.1.1 Major causes of death

A frequency assessment of all causes of death was conducted to determine the distribution of major causes (those deaths occurring most frequently in the mortality sample) of working-age death for Hamilton at each period, 1880-1881 and 1910–1912. Major causes of death are

grouped into classes of diseases, unless a single disease was shown to be substantive. Overall, the major causes of death account for 66.13% of the total causes for 1880-1881 and 58.15% of the total causes for 1910–1912. Table 3-10 presents the major causes of death for each year. Not surprising, given the two periods, tuberculosis was the primary cause of death for both 1880–1882 and 1910–1912 periods, with 24.72% and 13.5%, respectively, decreasing by 11.22% by 1912. Tuberculosis was declining by the turn of the 20th century due to improvements in living and social conditions (Davies, 2005; Feldberg, 1995; Grzybowski & Allen, 1999; Vynnycky & Fine, 1999; Wherrett, 1977). Individuals susceptible to tuberculosis tended to develop the disease around child-bearing and child-rearing ages, which would restrict the number of child born to sufferers: in other words, there was less fertility among individuals more susceptible to this disease (Davies, Tocque, Bellis, Rimmington, & Davies, 1999). Additionally, the introduction of sanatoriums removed infected persons from their homes and city (Fairchild & Oppenheimer, 1998), which decreased the potential for further infection.

Table 3-10. Major Causes of Death Working-age Sample, 1880–1882 and 1910–1912 Hamilton, Data Source: Ontario Death Registry, 1880-1882 & 1910-1912

Cause of Death	**1880–1882**		**1910–1912**	
	Number	Percent*	Number	Percent*
Tuberculosis	154	24.72	178	13.50
Infectious Respiratory Cause	80	12.84	108	8.19
Infectious Non-Respiratory Cause	59	9.47	82	6.22
Heart Disease	47	7.54	96	7.28
Cancer & Malignant Tumour	29	4.65	116	8.79
Bright's Disease & Nephritis	23	3.69	70	5.31
Accident (non-violent)	20	3.21	117	8.87
Total Major Causes	*412*	*66.13*	*767*	*58.15*

*Percent of all causes found in the sample

Although, in Canada, sanatoriums attempted to provide free accommodation for those unable to pay, the shortage of beds in the early 1900s and financial pressure meant that none were actually designated for the poor population and, unless one was willing to pay, the average

length of stay was about four months (Ferguson & Ferguson, 1935; Larmour, 1987; McCuaig, 1999; Wherrett, 1977). Free tuberculosis treatment for citizens was not implemented until late 1920s, beginning in Saskatchewan, which would set the stage for Canadian Medicare (Larmour, 1987; McCuaig, 1999). Prior to free treatment, however, the inability to accommodate poorer patients and the financial pressures on them meant patients would return home after a few months, likely without full remission from tuberculosis, as the average recommended stay in a sanatorium was at least one year (Larmour, 1987; McCuaig, 1999). Given that tuberculosis was a disease of poverty, those who would need treatment most would not be able to get it, which illustrates the inequities to healthcare access.

By the later period, 1910–1912, accidents and cancer mortality increased to be, respectively, the second (8.87%) and third (8.87%) of all-cause mortality, whereas, from 1880–1882, accidents only made up 3.21% of deaths and cancer 4.65% of deaths. Accidents occurring at the steel plant in Hamilton were not recorded with any consistency prior to 1910; however injuries and fatalities throughout the factories and mines were a common occurrence during this time in Canadian and American history due to poor working conditions (Caplan, 2005; Fitch, 1969; Heron, 1988; Ludlow & Burke, 2012; United States Department of Labor, n.d.). In the United States, the rise of the mill industry in Holyoke in the mid-1800s was coupled with notably poor working conditions with long hours, frequent accidents, fires, and crowded and poorly ventilated spaces (Hautaniemi et al., 1999). Industrialization brought about marked differences in occupational risks: in Saguenay-Lac-Saint-Jean, Quebec, from 1840-1971, for instance, workers with some skill had notoriously high accidental hazards in the workplace compared to unskilled workers (Gagnon, Tremblay, Vézina, & Seabrook, 2011). This is expected as skilled

workers operated dangerous machinery and would be in close proximity to dangerous materials such as molten steel (Caplan, 2005; Gagnon et al., 2011; Ludlow & Burke, 2012).

The increase in cancer and malignant tumours is an important finding because it identifies an increased burden of cancer among Hamilton's working age population, which could suggest increased environmental risk and/or a major change in occupational structure. Steel working is a known risk for cancers due to exposures to various chemicals; however, the period of this study is too early to see the repercussions of such exposures (Fitch, 1969; Ludlow, 2009). Other explanations describe the historicity and medical history of cancer and are plausible justifications for the increase in cancer found in this sample. Generally, the accepted explanation for the increase in cancer along with other chronic degenerative diseases is the decline from infectious diseases, which allowed people to live longer and, in turn, increased exposure to cancer-causing agents. There are, however, some biases with this explanation due to changes in how physicians named and described diseases (Hautamiemi Leonard et al., n.d.). Clow (2001) examined the history of cancer care in Ontario and found that, in the early part of the 20th century, there was little consensus on what caused cancer, which may affect how physicians classified this cause. As well, in the late 19th century, cancer was considered a women's disease associated predominately with female reproductive system and an infectious disease (Moscucci, 2005). Such aetiological concepts could present a bias in a mortality sample. Even later than this, a report in the United States analyzed incidence data for 30 years prior to 1973 and found that, during that period, there were changes in the definitions of the disease, diagnosing patterns, and classification of the disease, which affected the registration of cancer data (Lashof, Banta, Gough, Fensterheim, & Gelband, 1981).

While some transition in the epidemiological profile appears, with an increase in chronic degenerative diseases and accidental deaths, infectious diseases remain prevalent. In the earlier period (1880–1882), however, infectious respiratory causes (12.84%) and infectious non-respiratory causes (9.47%) were, respectively, the second and third highest categories of mortality when compared to all other cause mortality. These major causes of death likely substantially affected the at-risk population's health. Some of these major causes of death are more likely to reveal potential relationships between urban development and population health disparities in a growing urban industrial economy than other causes given the connection of diseases to the conditions of the environment.

3.3.1.2 Specific mortality rates for major causes of death

Specific mortality rates were used to better understand the sex- and cause-specific mortality patterns for six of the top causes of death to further determine appropriate causes of death for this study. Using the major causes of deaths, cause-specific working-age mortality rates were conducted for each period (Figure 3-11). Although tuberculosis was the primary cause of death for both periods for the working-age sample, the tuberculosis working-age mortality was twice as high in the earlier 1880–1882 period than in the 1910–1912 period (6.90 tuberculosis working age deaths per 1,000 living working age population and 3.08 tuberculosis working age deaths per 1,000 living working age population, respectively).

In the United States, tuberculosis declined by about one third between 1850 and 1890; however, the decline was not steady, as social and geographical variations persisted (Feldberg, 1995). Tuberculosis in Hamilton did decline at a rate that was similar to that found throughout Canada and Ontario (Toth, 2001). Toth's (2001) analysis of deaths found in annual reports showed a drop in tuberculosis rates between 1906 and 1908 followed by a spike in rates between

1909 and 1911. This aligns with the heightened period of population growth and industrial development that occurred between 1906 and 1911 in Hamilton (Wood, 1987).

From 1880–1882, the cause-specific mortality rates for respiratory infectious diseases and infectious non-respiratory diseases were 3.59 and 2.65 deaths per 1,000 living working-age population, respectively. These infectious diseases had also declined by 1910–1912 to, respectively, 1.87 and 1.42 deaths per 1,000 living working age population. Rates decreased by 1.72 for respiratory infections and 1.23 for other non-respiratory infections. Non-respiratory infections, such as typhoid fever, are often connected to the environment and hygiene practices.

Throughout the 19th century, infectious disease epidemics scourged American cities, prompting urban sanitation projects (Leavitt, 1992). Today, infection rates for enteric diseases (e.g. typhoid fever, dysentery, and cholera) are highest in low-income areas where general standards of living, water supply, and sanitary conditions are low or inadequate (Corner, Dewan, & Hashizume, 2013; Kelly-Hope et al., 2007). While infectious non-respiratory diseases declined by the later period, their continued persistent raises questions about the environment in Hamilton: were there spatial differences, and what were the attributable factors? Respiratory infections are less predictable, but have a seasonal effect (Beveridge, 1991). Throughout history, however, there have been influenza epidemics that became pandemics. One occurred between 1889 and 1890, known as the Russian Influenza, after this next was the great pandemic of 1918-1919 (Beveridge, 1991; Herring & Carraher, 2011), giving a plausible explanation for the decline in respiratory infections.

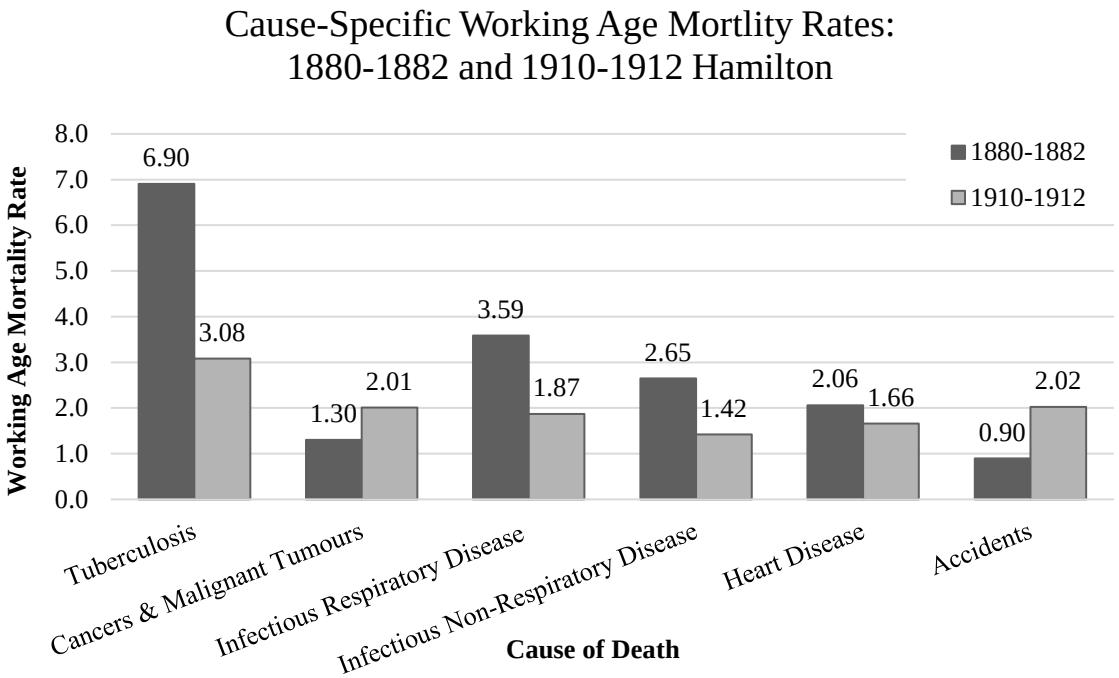


Figure 3-11. Cause-Specific Working Age Mortality Rates for 1880-1882 and 1910-1912 Hamilton

By the later period, 1910–1912, a higher mortality rate for accidents (2.02 deaths per 1,000 living working-age population) was found when compared to 1880–1882 (0.90 deaths per 1,000 living working-age population). Given the rise of industrial activity, the known working conditions, and the frequent accidents associated with these occupations during early industrialization, an increase is expected. Although more women were working by the later period, men continued to dominate employment in the notoriously dangerous factories. The increase in cancer and malignant tumours death rates from 1.66 deaths in the earlier period to 2.06 deaths in the later period per 1,000 living working age population is associated with the changing epidemiological structure: lower maternal deaths enabled the increase of cancer and malignant tumours.

The sex- and cause-specific working-age mortality rates revealed some sex differentials for cause of death for 1880-1881 and 1910–1912 (Figures 3-12 and 3-13, respectively). For the

1880–1882 period, males and females showed similarly high mortality rates for tuberculosis with 7.19 male deaths per 1,000 living working-age males and 6.65 female deaths per 1,000 living working-age females; a difference of 0.54 for males. For the 1910–1912 period, overall rates had declined (2.82 male deaths per 1,000 living working-age males and 3.36 female deaths per 1,000 living working-age females), and women faced a slightly higher tuberculosis death rate of 0.54 in excess of men. The rate difference for either period is small, however, when compared to that found in some other situations. In New York during the 1900s, the sex-based ratio for tuberculosis mortality was 2:1 for males (Frieden, Lerner, & Rutherford, 2000). In 2012, the global sex-based ratio for tuberculosis was 1.9:1 males per female (Nhamoyebonde & Leslie, 2014). The lower rate difference among females, in both historical and current contexts, may be an artefact of underreporting and underdiagnosing, but may also because of gender-based societal roles, risk behaviours, and activities (Nhamoyebonde & Leslie, 2014). The lower difference in the later period may reflect gender-biased occupational roles causing increased accidental mortality found among men—men dying from accidents in the factories before succumbing to tuberculosis death.

Accidents, for both periods, revealed sex-based differences. For the earlier period, this difference aligned with the lower rate of accidental deaths overall. The accidental death rate for males in 1880–1882 was 1.61 working-age deaths per 1,000 working-age males. For females, the rate was 0.26 deaths per 1,000 working-age females, with a rate difference of 1.35 for males. By 1910–1912, both the mortality rates and the difference increased substantially, with a rate difference of 2.56 for males (the mortality rate for males was 3.25 deaths/1000 working-age males and the mortality rate for females was 0.69 deaths/1000 working-age females). Accidental deaths showed the widest sex-specific mortality gap for the later period, which may suggest

occupational risk differences between males and females. Lifestyle risks, however, must also be considered in accidental cause mortality. In another early 20th century industrial city, dominated by steel manufacturing, accidents caused more deaths amongst the male cohort than tuberculosis (Ludlow & Burke, 2012). More recently, unintended deaths among young males are attributed to occupational fatalities, as well as use of poor judgement leading to drowning, motor vehicle crashes, and gunshot wounds (Stiglets, 2001). Thus, occupational and behavioural risks are likely to account for the sex-based differences for accidental deaths.

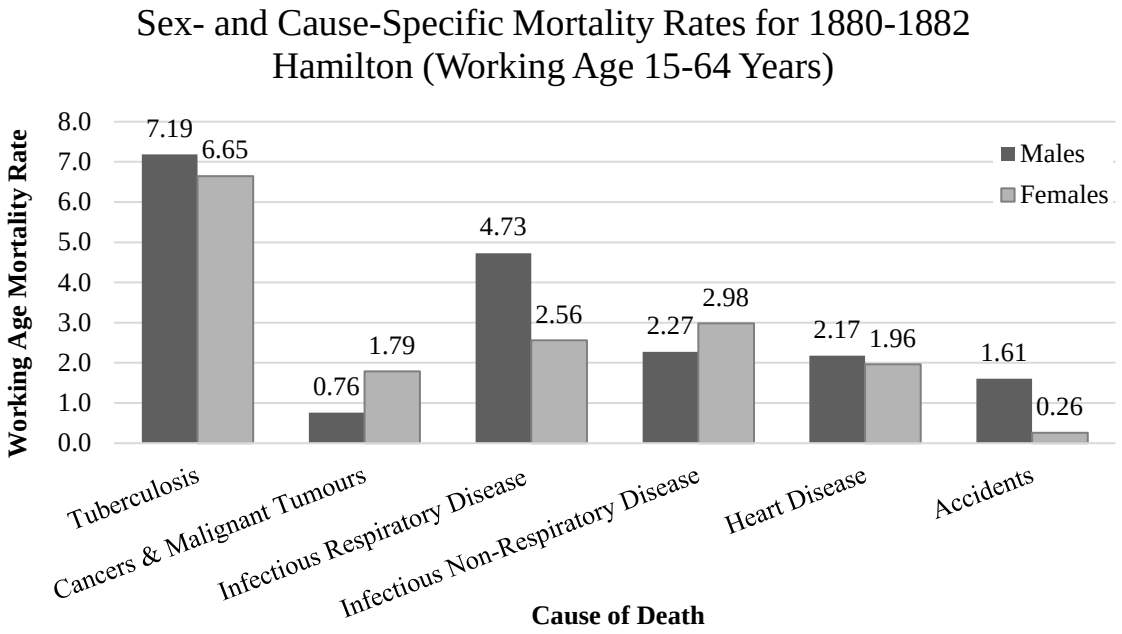


Figure 3-12. Sex- and Cause-Specific Working Age Mortality Rates for 1880-1882 Hamilton

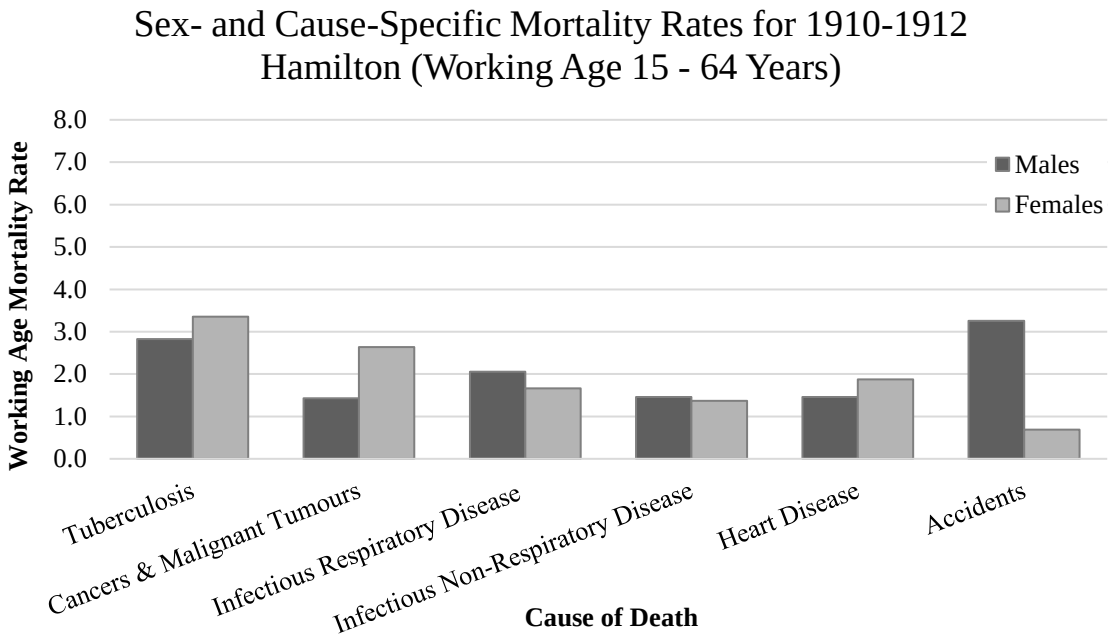


Figure 3-13. Sex- and Cause-Specific Working Age Mortality Rates for 1910-1912 Hamilton

Other sex-specific mortality rates did not show substantial variations or these differences appear to have lessened over time. The slight increase in cancer does support conceptions in medical history of cancer as a female disease. It is possible that the sex ratio is the result of men dying at earlier ages as they would be less likely to succumb to cancer. Over time, specific mortality rates for cancer show an increasing gap between males and females. For 1880–1882, the rate difference was 1.03 more females, but, by 1910–1912, the ratio increased to 1.21 for females. There is evidence of a negative correlation between maternal death and reproductive cancers: with a decline in maternal death, more women are available to die from reproductive cancers. Currently, in lower-income countries, there are 2.5 maternal deaths for every death from breast or cervical cancer in women between the ages 15 and 49 years (Institute for Health Metrics and Evaluation, 2011). Future trends for reproductive cancers in lower-income countries further suggest that, by 2025, maternal deaths are expected to fall, while breast and cervical cancers are expected to rise amongst women of reproductive age (Institute for Health Metrics

and Evaluation, 2011). Such trends currently occurring in lower-income and pre-industrial countries are important because of the similarities in terms of epidemiological risk of infectious diseases to that in historical North American cities. Yet, known gender-based misconceptions of cancer would cause underreporting in the mortality sample for men, skewing the results and confidence in reporting on cancer mortality at the turn of the 20th century.

Sex-based differences for other infectious diseases also declined. The biggest decline can be found for infectious respiratory disease: in 1880–1882, the rate difference was 2.17/1,000 higher for males than females, and by 1910–1912, this difference was 0.4/1,000 more for males. Lifestyle, behavioural, and socioeconomic differences have been suggested to explain higher male mortality for respiratory infections in recent studies (Falagas, Mourtzoukou, & Vardakas, 2007). Closing the gap on the sex-based differences, where a disease did not show a substantial decline over time (e.g. infectious non-respiratory disease), suggests that males and females were becoming equally at risk of dying from these diseases.

The mortality rates examined here give a high-level composition of the population at risk of dying in the working-age cohort. For the purpose of this research, tuberculosis, infectious respiratory disease (other than tuberculosis), infectious non-respiratory disease, and accidental causes were selected for an in-depth statistical analysis using chi-square tests for independence and multiple logistic regressions to further examine the characteristics of the sample. These causes of death indicate health in relation to the environment and understanding occupational hazards for the population at risk.

3.3.1.3 Cross-tabular chi-square analyses

Cross-tabular chi-square analyses were conducted to reveal the statistical relationship of the variables found in the working-age mortality sample for both periods. The coding of

variables (dependent and independent) and the analyses are tested for independence and to avoid overfeeding the multinomial logistic regression model. The first round of analyses focus on establishing a relationship, if any, between the independent variables and are also used to determine whether these variables require more generalized coding. These analyses, however, are not used to determine an association with causes of death and are, therefore, excluded from this results section, but are presented in Appendix A. Independent variables were then tested with the causes of death (tuberculosis, infectious respiratory, infectious non-respiratory, and accidental causes of death) to associate any statistical significance (Table 3-11).

Table 3-11. Significance of Independent Variables with Cause of Death* by Year

Year	P-Value					
	Biological Sex	Age	Occupation	Birthplace	Marital Status	Seasonality
1880–1882	< 0.001	< 0.001	Cell violation	Cell violation	n/a	Cell violation
1910–1912	< 0.001	< 0.001	Cell violation	0.007	< 0.001	< 0.001

*Causes of death included in the chi-square are tuberculosis, infectious respiratory, infectious non-respiratory and accidental causes

The 1880–1882 death registries did not report marital status in the record, so this variable could not be tested. In other cases, there was a cell violation, meaning that, within the chi-square, at least one cell had an expected count of less than five, which violated the assumption of the test. This violation could provide misleading probabilities on the distribution between cause of death and the respective variable. In the case of cause of death and occupation in the later period, however, the results showed some significance, and the violation might have been caused by too many classifications. In the 1910–1912 working-age mortality sample, two cells violated this assumption: 1) respiratory infectious disease for professional, managerial, and technical

occupations; and 2) infectious diseases for professional, managerial, and technical occupations. Further grouping of occupations may have led to non-violated chi-square assumptions in the later period (e.g. industrial and non-industrial occupational groups) and, given the significance, could be considered in the logistic regression model. The results presented below to test whether proportions were different in the independent variables use a chi-squared (χ^2) test of independence with alpha (α) equal to 0.05 as criterion for significance.

Findings for the chi-square test of cause of death and biological sex in the 1880–1882 are presented in Figure 3-14. There were more observed accidental deaths among males (n = 17; 85.0% of accidental deaths) than would be expected by chance (n = 9.8), but fewer observed accidental deaths amongst females (n = 3; 10.2% of accidental deaths) than would be expected by chance (n = 10.2). These findings produced statistically high-significance results (χ^2= 20.262; df = 4; $p < 0.001$), supporting a correlation between cause of death and biological sex.

Chi-square test for cause of death and biological sex results of the 1910–1912 working-age mortality sample are presented in Figure 3-15. The working-age mortality sample exhibited a high number of observed accidental deaths for males (n = 98; 83.3% of accidental deaths) than would be expected by chance (n = 58.7), but showed a lower number of observed accidental deaths for females (n = 19; 16.2% of accidental deaths) than would be expected by chance (n = 58.3). An excess of observed 'all other' deaths among females was found (n = 392; 56.4% of all other deaths) than would be expected by chance (n = 346.3). In contrast, a relative deficiency observed for 'all other' deaths among males (n = 303; 43.6% of all other deaths) than would be

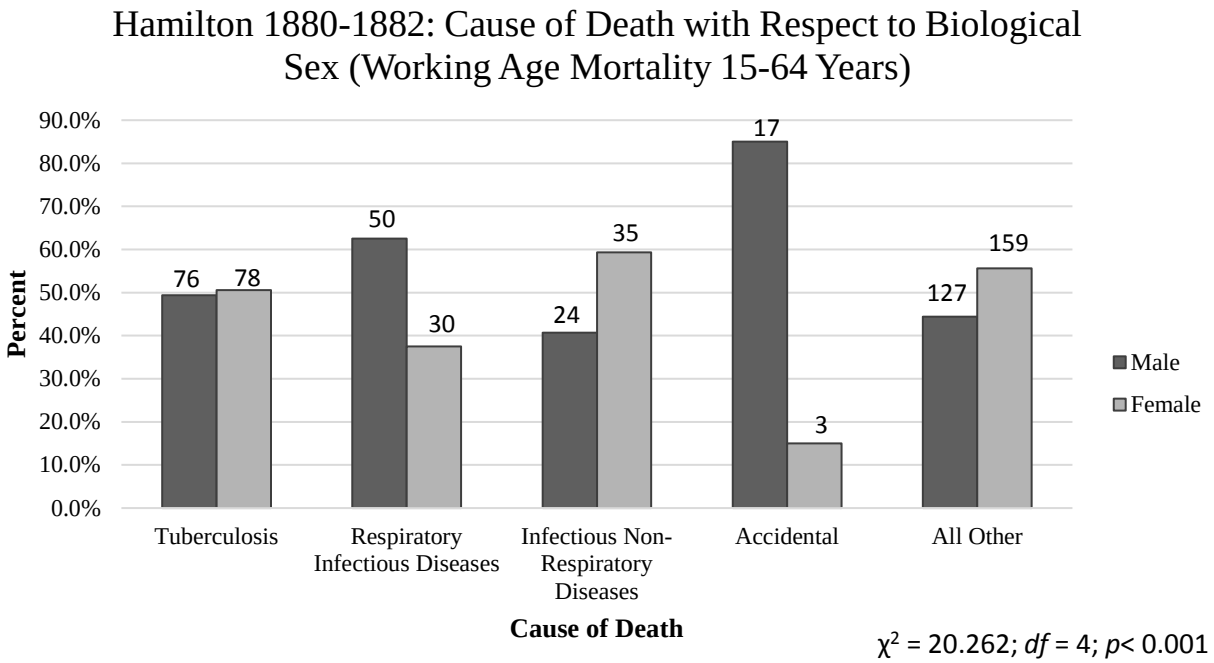


Figure 3-14. Analysis of Cause of Death with Respect to Biological Sex, Hamilton 1880-1882 Working Age Sample

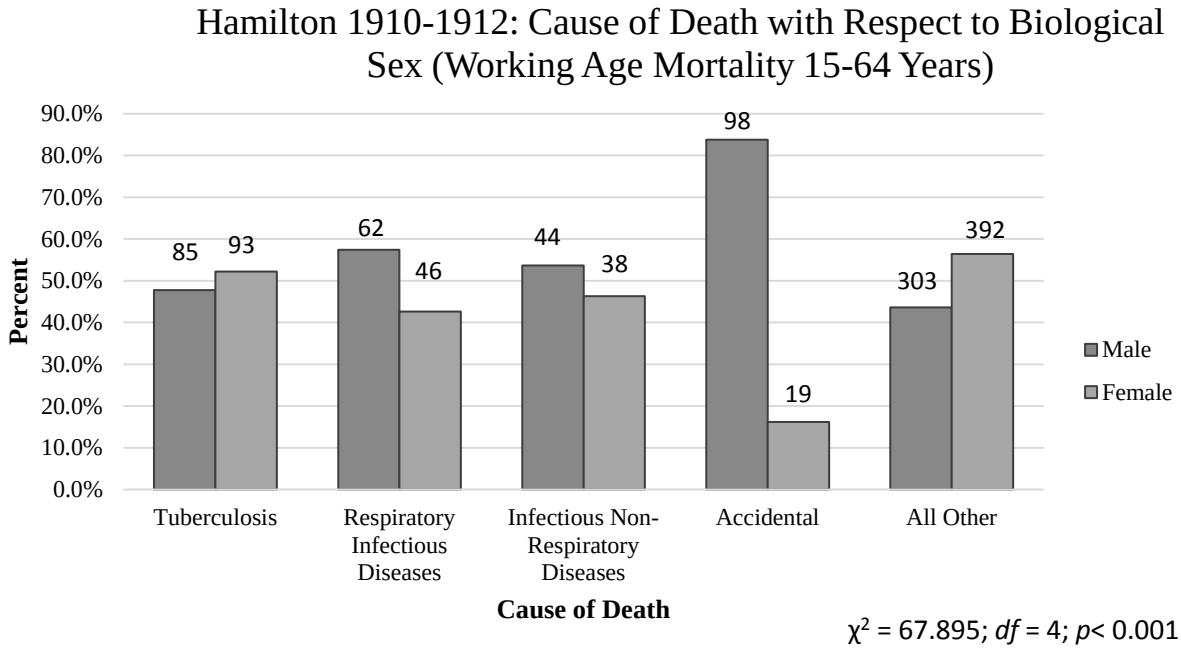


Figure 3-15. Analysis of Cause of Death with Respect to Biological Sex, Hamilton 1910-1912 Working Age Sample

expected by chance (n = 348.7). Recall, 'all other' is a catch-all for causes of death not under examination in this research. The chi-square analysis for cause of death and biological sex for the 1910-1912 working age mortality sample produced statistically significant results (χ^2 = 67.895; df = 4; $p < 0.001$). Thus, the inference that cause of death and biological sex are related (dependent) is supported.

Results of the 1880–1882 working-age mortality sample for cause of death and age at death are presented in Figure 3-16. Within the working-age mortality sample, there were more observed tuberculosis deaths for young working age (n = 70; 45.5% of tuberculosis deaths) than would be expected by chance (n = 49.4), but there were fewer tuberculosis deaths in relation to older working age (n = 25; 16.2% of tuberculosis deaths) than would be expected by chance (n = 47.3). Infectious non-respiratory diseases showed a higher relative excess for young working age (n = 39; 66.1% of infectious non-respiratory disease deaths), but showed a relative deficiency among the older working ages (n = 6; 10.2% of infectious non-respiratory disease deaths) than would be expected by chance (n = 18.9, n = 18.1, respectively). In contrast, all other deaths showed a relative excess for older working age (n =118; 41.3% of all other deaths) than would be expected by chance (n = 87.9), but all other deaths showed a relative deficiency for younger working age (n =57; 19.9% of all other deaths) than would be expected by chance (n =91.7). This chi-square analysis supports a correlation or cause of death and age at death for the 1880–1882 sample as revealed from the statistically high-significance results (χ^2 = 81.180; df = 8; $p< 0.001$).

Chi-square test for cause of death and age at death produced significant results for the 1910–1912 working-age mortality sample (Figure 3-17). A higher relative excess of tuberculosis was found for young working age (n = 83; 46.6% of tuberculosis deaths) than would be expected

by chance (n = 48.1), while a lower relative deficiency was found for respiratory tuberculosis among older working age (n = 18; 10.1% of tuberculosis deaths) than would be expected by chance (n = 64.0). Infectious non-respiratory diseases exhibited more observed cases for young working age (n = 46; 56.1% of infectious disease deaths) than would be expected by chance (n = 22.2), but also showed a relative deficiency for older working age (n = 16; 19.5% of infectious non-respiratory disease deaths) than would be expected by chance (n = 29.5). There was also a relative excess of accidental causes found among the young working ages (n = 46; 39.3% of accidental deaths) than would be expected by chance (n = 31.6), and in contrast, fewer observed cases found among older working age (n = 23; 19.7% of accidental deaths) than would be expected by chance (n = 42.0). Alternatively, all other deaths showed a relative excess for older working age (n = 320; 46.0% of all other deaths) than would be expected by chance (n = 249.0), but all other deaths showed a lower relative deficiency for younger working age (n = 117; 16.8% of all other deaths) than would be expected by chance (n = 187.9). These results support the inference that cause of death and age at death are related (dependent) as the chi-square produced statistically high-significance results (χ^2 = 160.453; df = 8; p< 0.001).

Results of the 1910–1912 working-age mortality sample for cause of death and birthplace are presented in Figure 3-18. These results showed a higher number of observed accidental causes of death for foreign-born people (n = 17; 17.3% of accidental deaths) than would be expected by chance (n = 10.0), but also showed a lower number of observed accidental causes for Canadian-born people (n = 37; 37.8% of accidental deaths) than would be expected by chance (n = 54.4). This analysis for cause of death and birthplace for the 1910–1912 working-age mortality sample produced statistically high-significance results (χ^2 = 20.897; df = 8; p = 0.007), supporting the inference that cause of death and birthplace are related (dependent).

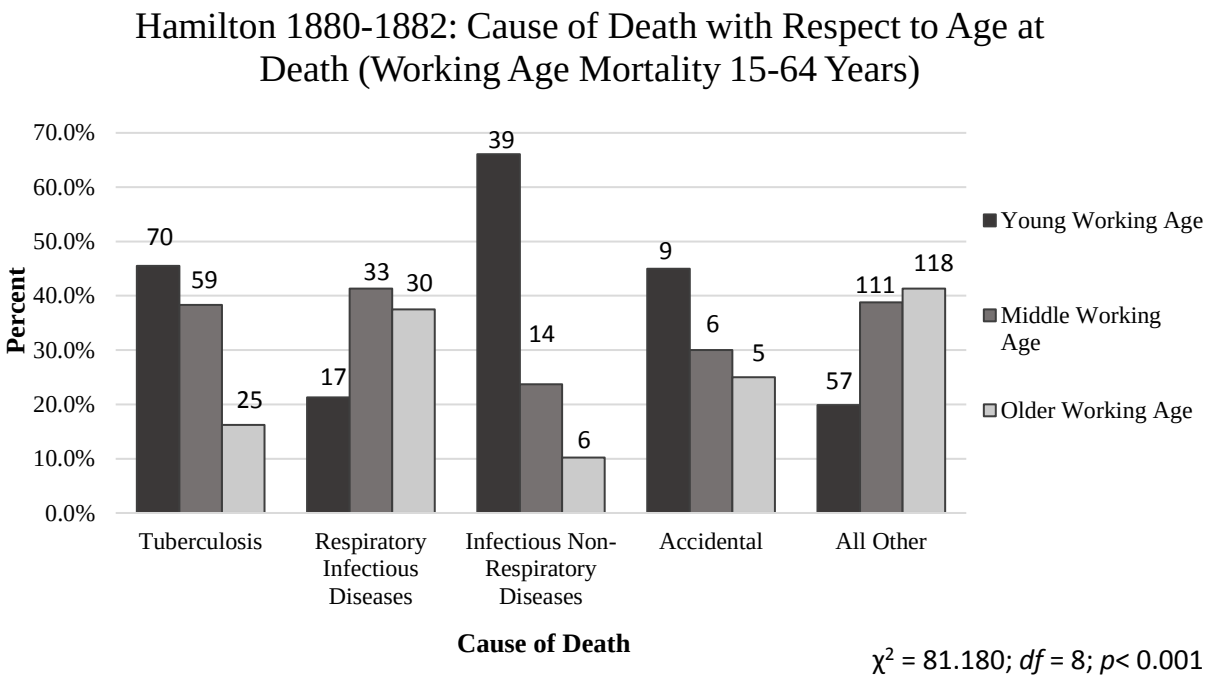


Figure 3-16. Analysis of Cause of Death with Respect to Age at Death, Hamilton 1880-1882 Working Age Sample

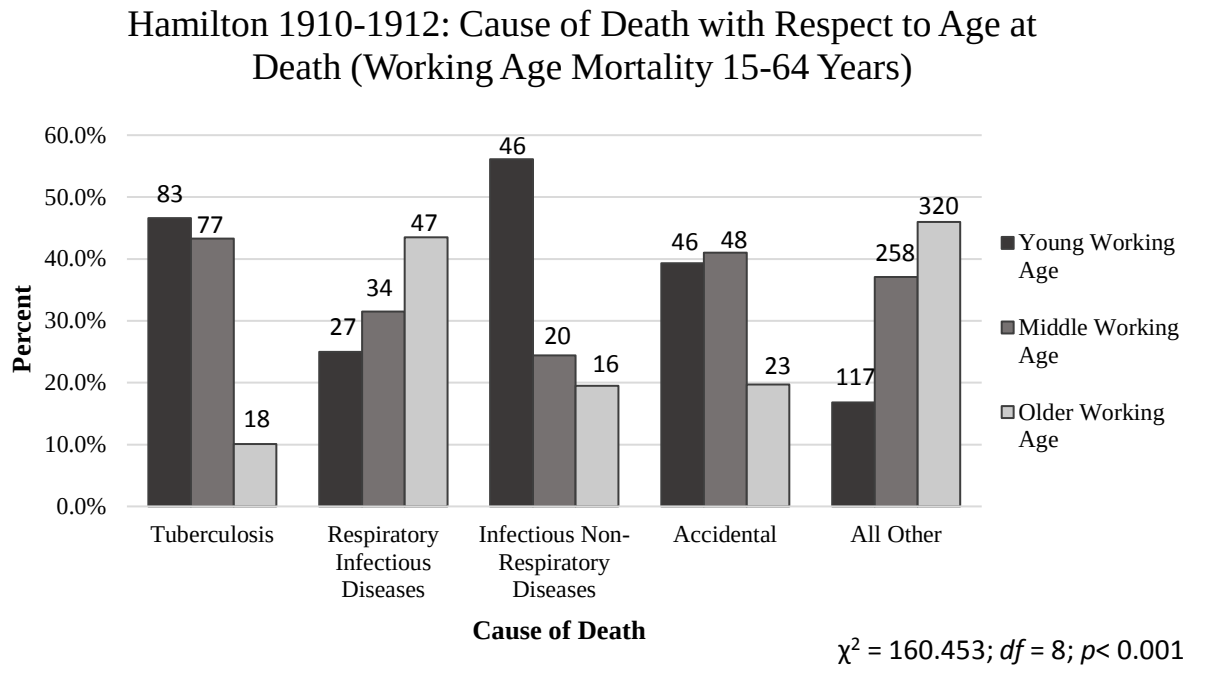


Figure 3-17. Analysis of Cause of Death with Respect to Age at Death, Hamilton 1910-1912 Working Age Sample

An analysis for marital status and cause of death for the 1910–1912 working-age mortality sample was conducted and the findings presented in Figure 3-19. Within the working-age mortality sample, a relative excess was found for tuberculosis and single marital status (n = 84; 48.6% of tuberculosis deaths) than would be expected by chance (n = 52.3). As well, a relative deficiency was found for both married (n = 81; 46.8% of tuberculosis deaths) and widow (n = 8; 4.6% of tuberculosis deaths) marital statuses than would be expected by chance (n = 103.6 and n = 17.1, respectively). Infectious non-respiratory disease and accidental causes also showed a relative excess for single marital status (n = 32; 45.7% of infectious non-respiratory disease deaths; n = 50; 47.5% of accidental deaths) than would be expected by chance (n = 21.2; n = 32.0). Thus, these three causes of death categories showed an excess among single marital statuses than would be expected by chance alone. Alternatively, all other causes showed a relative excess for widow marital status (n = 85; 13.0% of all other deaths) than would be expected by chance (n = 64.6), but showed a relative deficiency for single marital status (n = 140; 21.4% of all other deaths) than would be expected by chance (n = 198.0). The chi-square analysis for cause of death and marital status for the 1910–1912 working-age mortality sample produced high statistically significant results ($\chi^2 = 81.767$; $df = 8$; $p < 0.001$). This analysis then supports the inference that cause of death and marital status are related (dependent).

The 1910–1912 working-age mortality sample for cause of death and seasonality results, however, did not violate any assumptions and are presented here (Figure 3-20). Among the working-age mortality sample, a relative excess of observed cases were found for respiratory infectious diseases during both spring (n = 44; 40.7% of respiratory infectious disease deaths) and winter (n = 36; 33.3% of respiratory infectious disease deaths) than would be expected by

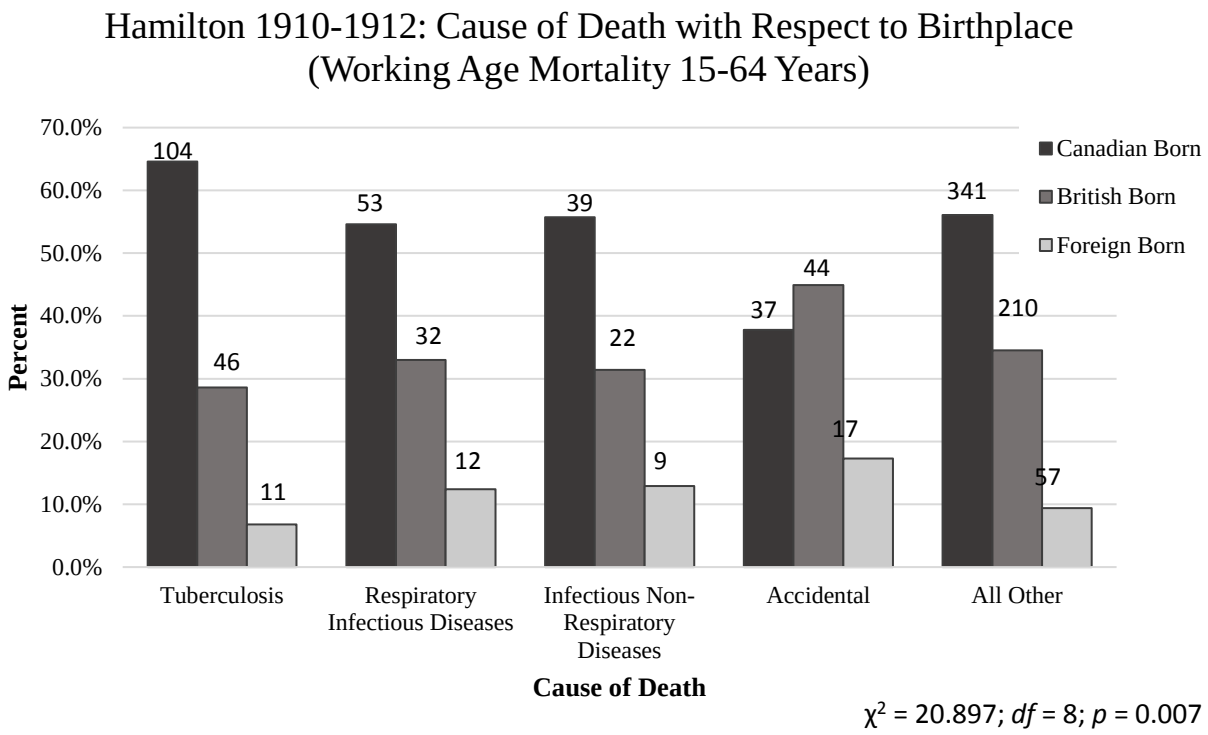


Figure 3-18. Analysis of Cause of Death with Respect to Birthplace, Hamilton 1910-1912 Working Age Sample

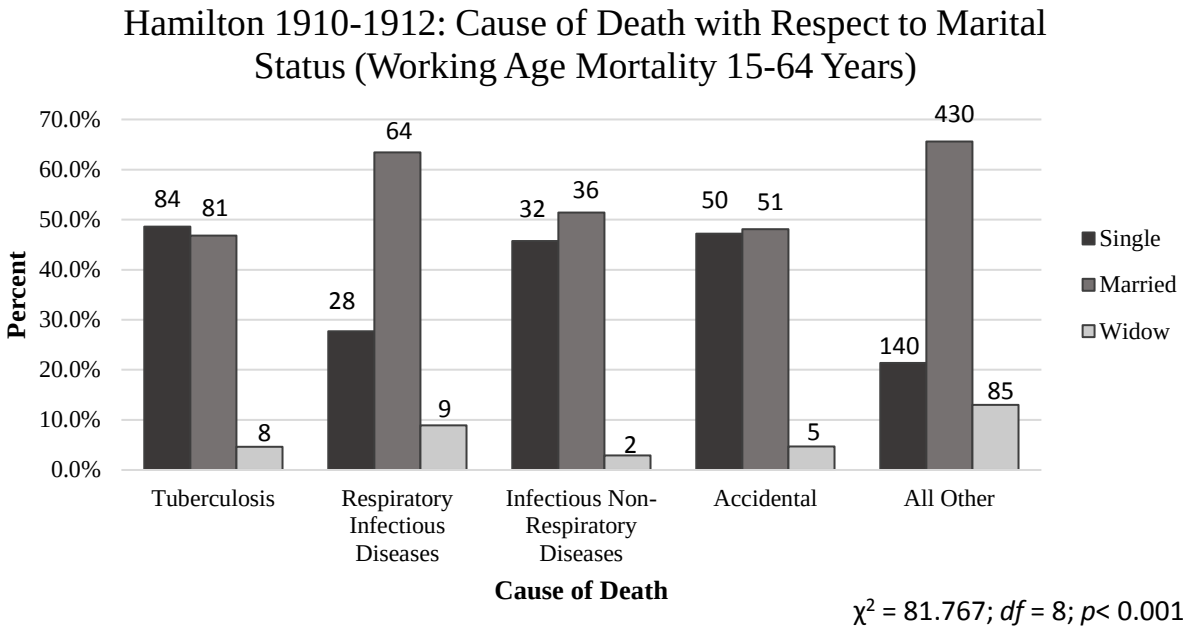


Figure 3-19. Analysis of Cause of Death with Respect to Marital Status, Hamilton 1910-1912 Working Age Sample

chance (n = 29.3 and n = 25.4, respectively). A relative deficiency of cases was observed for respiratory infectious diseases during the autumn season (n = 11; 10.2% of respiratory infectious disease deaths) than would be expected by chance (n = 27.0). This chi-square test for cause of death and seasonality produced high statistically significant results (χ^2 = 36.454; df = 12; p< 0.001), supporting the inference of cause of death and seasonality are related (dependent).

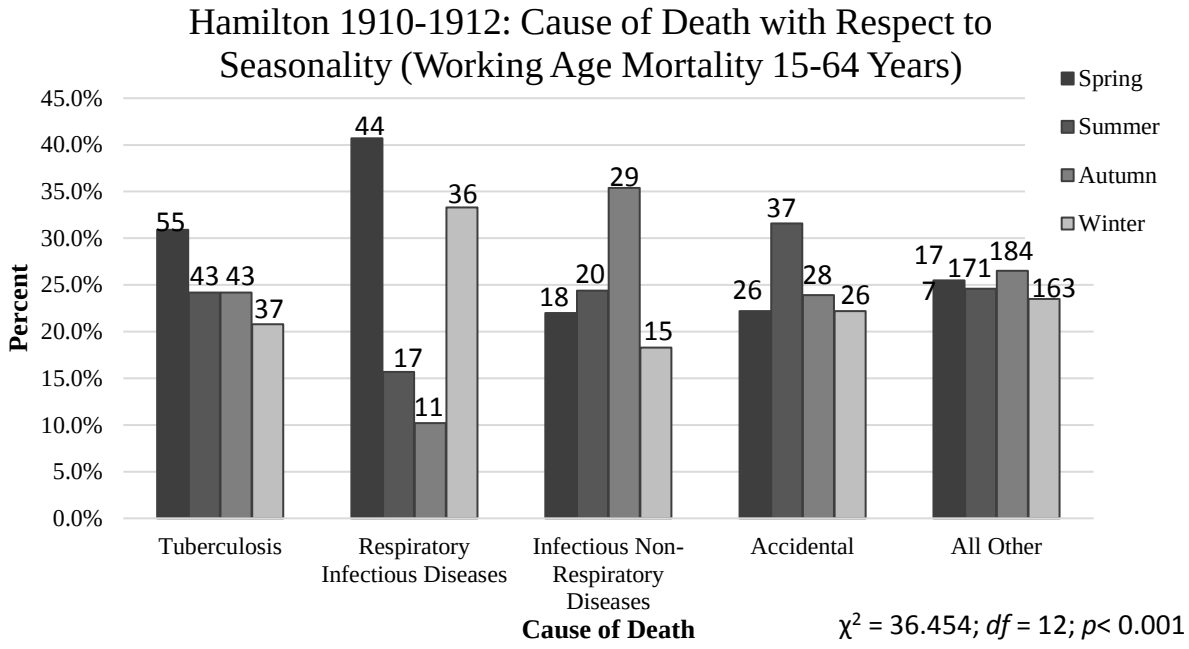


Figure 3-20. Analysis of Cause of Death with Respect to Seasonality, Hamilton 1910-1912 Working Age Sample

The test for independence helps to show which variables have a relationship (are dependent), thereby removing any null (independent) relationships from inclusion in inferential statistical analyses. Understanding these relationships will help identify the independent variables to avoid complete separation in building the multiple logistic regression model for causes of death. Based on the chi-square test, infectious respiratory disease causes of death did not show any observed anomalies, with the exception of seasonality in the 1910–1912 working-age sample, so it does

not need further inferential testing against multiple factors. Tuberculosis, infectious non-respiratory disease, and accidental causes of death did show observed anomalies with different variables at both periods and will be used to build the model. Variables that revealed significant relationships for the 1880–1882 working-age mortality sample were biological sex and working age. Variables that revealed significant relationships for the 1910–1912 working-age mortality sample were biological sex, age at death, birthplace, marital status, and, once re-categorized as industrial/non-industrial, occupation.

3.3.2 Infant Mortality Profile

Proxy infant mortality rates (P-IMR) were examined for the infant (< 1 year) sample for each year using the 1881 and 1911 census infant-under-one-year population (n = 954 and n = 1767, respectively) as the denominator. When collecting infant deaths, this study reviewed the age of the infant labelled *stillbirth* to account for some underreporting of infant deaths. An average P-IMR was also calculated for each period. Table 3-12 presents the results of the P-IMR. The average P-IMR in the earlier period (1880–1882) was 179.25 infant deaths per 1,000 live births. The highest P-IMR in either study period was in 1882 with a rate of 187.63 infant deaths per 1,000 living infant population. While P-IMRs had declined by the later period (1910–1912), the average mortality rate for 1910–1912 was still high, with 147.90 infant deaths per 1,000 living infant population. The highest mortality rate for the later period occurred in 1912 with a rate of 168.65 infant deaths per 1000 living infant population. The lowest P-IMR occurred in both 1910 and 1911 with the same rate of 137.52 infant deaths per 1,000 living infant population. This proxy showed an overestimation in the rate of 18.13 for 1881 and 20.75 for 1911. The IMR for 1881 was 163.21 infant deaths per live births and for 1911 the IMR was 116.77 infant deaths per live births. Given the likelihood of an underestimation for the actual

IMR, as supported in the literature (e.g. Atlas, 2013; Kleinman, 1986), the actual rate of infant mortality in Hamilton likely fell somewhere between the IMR and the proxy-IMR. In either case, to contextualize just how large these mortality rates were, the IMR in Canada for 2011 was 4.8 infant deaths per 1,000 live births (Statistics Canada, 2015). Outside of the two study periods, in 1901, the IMR for Canada was 139.00 infant deaths per 1,000 live births (Haines & Steckel, 2000). A five year average for the years 1921-1925 showed that IMRs in Canada and Ontario were, respectively, 98.70 and 82.7 infant deaths per 1,000 live births (Bélanger, 2000). By 1925, the Hamilton IMR was estimated as 89.62 infant deaths per 1,000 live births (Janjua, 2009). These figures suggest that IMRs continued to decrease through the early 20th century. Specific mortality rates follow this analysis to gain more insight into the infant population at risk of dying.

Table 3-12. Proxy Infant Mortality Rate, Hamilton, 1880–1882 and 1910–1912

Infant Deaths per 1,000 Infant Living Population

Year	Proxy Infant Mortality Rate (P-IMR)
1880	168.76
1881	181.34
1882	187.63
Average Mortality Rates 1880–1882	*179.25*
1910	137.52
1911	137.52
1912	168.65
Average Mortality Rates 1910–1912	*147.90*

3.3.2.1 Major causes of death

To begin identifying major causes of mortality among the infant sample, a frequency assessment of all causes of death was conducted to determine the major causes of infant deaths for Hamilton at both periods, 1880-1881 and 1910–1912. These causes of death are grouped into

classes using both historical and current knowledge of disease aetiology (Anderton & Hautaniemi Leonard, 2004; Department of Commerce et al., 1914; Department of Commerce and Labor et al., 1911; Hautamiemi Leonard et al., n.d.; Sawchuk et al., 2002; Sawchuk et al., 1985; Thompson & Keeling, 2012; World Health Organization, n.d.-a). The term *causes of death* refers to both specific causes and grouped classes of death. Table 3-13 presents the major causes of death for each year. Overall, the eight major causes of infant death accounted for 82.26% of the total infant deaths for 1880-1881 and 82.91% of the total infant deaths for 1910–1912. For the earlier period, 1880–1882, diarrhoeal causes contained the highest percentage of deaths (22.22%) of all mortality. Infectious respiratory disease and nutritional causes followed with 15.20% and 12.88% of all causes of death, respectively. By the later period, however, nutritional causes held the highest percentage (20.66%) of mortality causes. Closely following nutritional causes were diarrhoeal causes (19.77%) and then premature births (14.82%) of all deaths. In the later period, however, infectious respiratory disease persisted as a major cause of infant death, comprising 12.88% of all major causes of death.

Table 3-13: Major Causes of Death Infant Sample, 1880–1882 and 1910–1912 Hamilton, Data Source: Ontario Death Registry

Cause of Death	**1880–1882**		**1910–1912**	
	Number	Percent*	Number	Percent*
Diarrhoeal Causes	114	22.22	155	19.77
Infectious Respiratory Causes	78	15.20	101	12.88
Nutritional Causes	68	13.26	162	20.66
Infectious Non-Respiratory Causes	46	8.97	30	3.83
Convulsions	46	8.97	17	2.17
Weakness and Debility	34	6.63	17	2.17
Premature Births	32	6.24	117	14.92
Congenital Malformations	4	0.78	51	6.51
Total Major Causes	*412*	*66.13*	*767*	*58.15*

*Percent of all causes found in the sample

3.3.2.2 Specific proxy infant mortality rate for major causes of death

Using the major causes of deaths, diarrhoeal causes, nutritional causes, and infectious respiratory diseases, cause-specific P-IMRs were conducted for each period (Figure 3-21). High IMRs can indicate poor standards of living, physical condition, and maternal health and welfare (Reid, 2001). For the earlier period (1880–1882), diarrhoeal causes presented the highest P-IMR (119.50 infant diarrhoeal cause deaths per 1,000 living infant population). By the later period, however, nutritional causes presented the highest P-IMR (98.47 infant nutritional causes per 1,000 living infant population). Sex- and cause-specific mortality rates for each period were further analyzed to determine any differentiation (or risk) between male and female infant cause mortality.

The sex- and cause-specific P-IMRs revealed some sex-differential mortality patterns for infants for the 1880-1881 and 1910–1912 periods (Figures 3-22 and 3-23, respectively). Infant mortality is generally higher for boys than girls. In early life, males tend to show more vulnerability to environmental conditions (malnutrition, infection susceptibility, weaning stress) (Wells, 2000). Sex-based differences in diarrhoeal, infectious respiratory, and nutritional causes present strong support for environmental causal factors. Diarrhoeal causes in the earlier period were highest for both males and females (male P-IMR = 131.21 and female P-IMR = 106.43). Rates were down by 1910–1912: for males the P-IMR was 93.92 and for females the P-IMR was 80.72. The difference between male and female P-IMR also decreased over time, from a difference of 24.78 to 13.2. The factors associated with infant mortality from diarrhoea include the lack of piped water, the absence of flush toilet, poorly built homes, and household overcrowding (Victora et al., 1988).

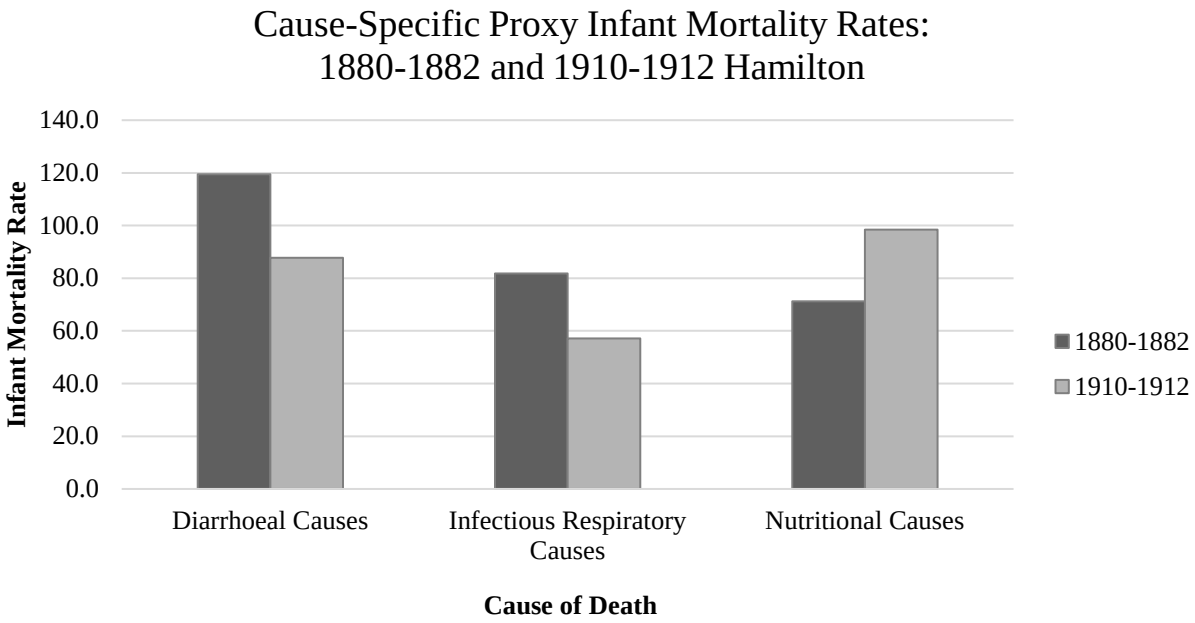


Figure 3-21. Cause-Specific Proxy Infant Mortality Rates for 1880-1882 and 1910-1912 Hamilton

The largest P-IMR gap between infant males and females in 1880–1882 occurred for infectious respiratory mortality (male P-IMR = 95.43 and female P-IMR = 66.52), a rate difference of 28.91 for males. By 1910–1912, however, infectious respiratory causes showed a higher female rate (female P-IMR = 61.45 and male P-IMR = 52.29), a difference of 9.16. Males are at higher risk for prematurity and respiratory conditions in the perinatal stage of infancy; an increase in the survival of premature infants can affect the sex balance of mortality (Drevenstedt, Crimmins, Vasunilashorn, & Finch, 2008). Breastfeeding has been found to have a protective effect against respiratory disease among infant girls (Libster et al., 2009). Thus, changes in mothers' practices may account for the increase in infant female mortality for respiratory infections. In the United States, artificial feeding practices and earlier weaning practices increased throughout the late 19th and early 20th century (Cheney, 1984). This change led to breast milk being replaced with poorer quality or insufficient foods, which created

stressors such as an increased contact with contaminated foods (Cheney, 1984) due to a contaminated water supply or lack of refrigeration (Keusch et al., 2006)

The sex-based mortality gap was smallest in 1880–1882 for nutritional cause mortality (male P-IMR = 79.52 and female P-IMR = 62.08), a difference of 13.56. Nutritional causes heightened by 1910–1912 for both male and female infants (female P-IMR = 89.16 ad male P-IMR = 106.72), as did the mortality gap, which showed a higher male rate difference of 17.56. Males have an increased susceptibility to nutritional insults (Wells, 2000). The overall increase in nutritional causes suggest a shift in nursing behaviour and weaning time, which, where sex differences occur, can be a reflection of altered feeding practices for female or male infants (Humphrey, Bello, & Rousham, 2012). Such shifts increase risk to infectious disease, such as diarrhoeal causes, by exposing infants to contaminated foodstuffs, water, and feeding

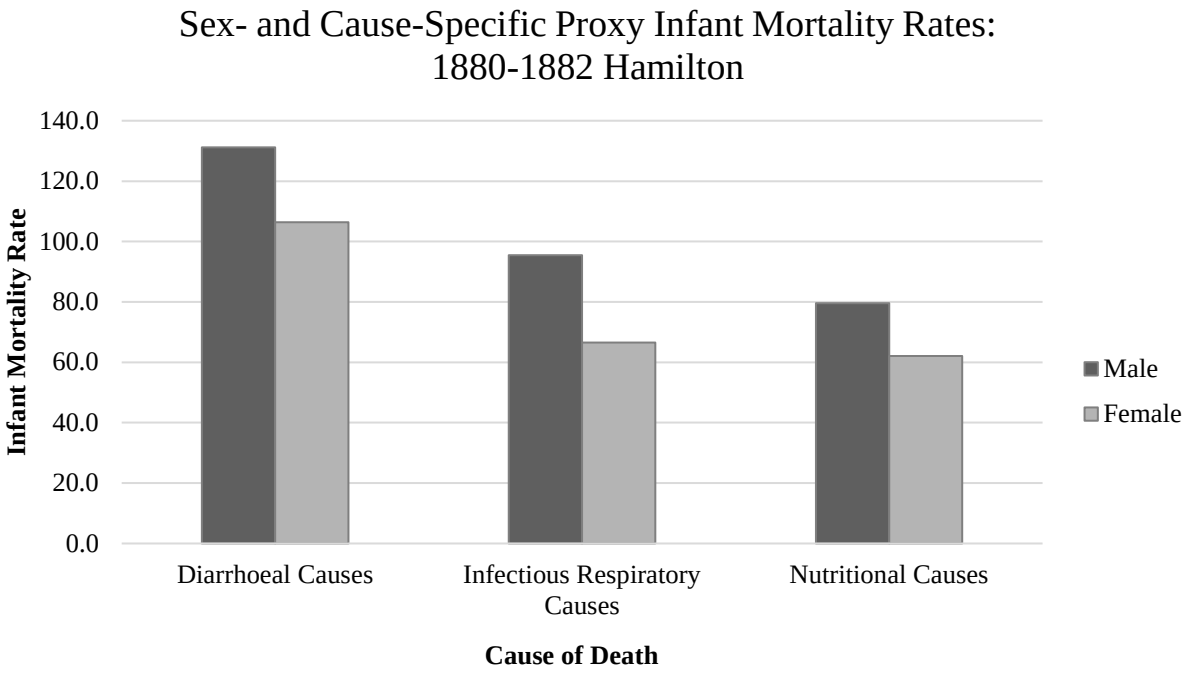


Figure 3-22. Sex- and Cause-Specific Proxy Infant Mortality Rates for 1880-1882 Hamilton

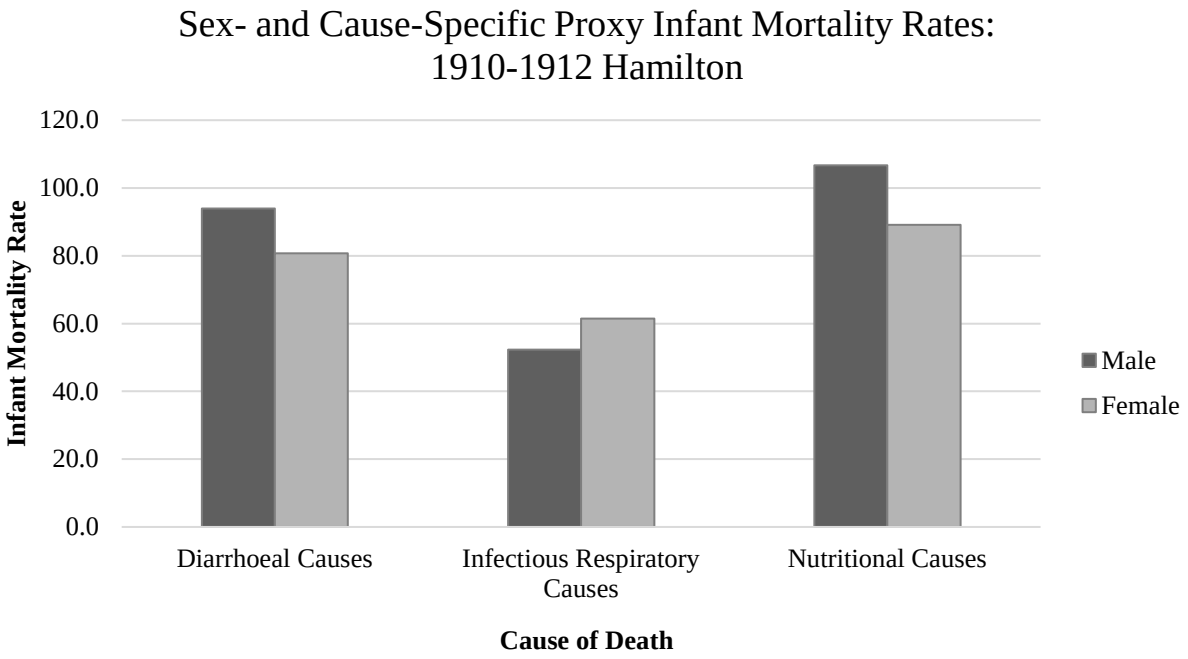


Figure 3-23. Sex- and Cause-Specific Proxy Infant Mortality Rates for 1910-1912 Hamilton

implements (Sawchuk et al., 1985). Excessive mortality after three months can imply the cessation of breast milk and uptake of artificial feeding, but also reflects an increased exposure to inappropriate or contaminated foods (Humphrey et al., 2012). The trends in infant mortality point to two causal factors: maternal practices and inadequate environment. Further examination into the characteristic of the infant mortality sample begin to identify who was dying in 1880–1882 and 1910–1912 Hamilton.

3.3.2.3 Cross-tabular chi-square analyses

The cross-tabular chi-square analyses presented here are used to reveal the statistical relationship of the variables found in the infant mortality sample for both the 1880-1881 and 1910–1912 periods. The first round of analyses focuses on establishing a relationship, if any, between the independent variables. These are presented in Appendix B for further information, as they do not pertain directly to the logistic regression model, but rather inform the development

of categories for the model and capture any relationships that occur between the independent variables. Table 3-14 presents the significance of independent variables with cause of death for each period.

Table 3-14: Significance of Independent Variables with Cause of Death* by Year

Year	P-Value			
	Sex	Age	Birthplace	Seasonality
1880–1882	0.561	< 0.001	Cell violation	< 0.001
1910–1912	0.557	< 0.001	Cell violation	< 0.001

*Causes of death included in the chi-square are diarrhoeal, infectious respiratory, and nutritional causes.

No significant relationship was found between causes of death and biological sex for either cross section (1880–1882 and 1910–1912). Birthplace for both periods showed a cell violation, which violated the assumption of the test. The remaining results for the chi-square test for independence revealed a significant relationship with the causes of death. To test whether proportions were different in the independent variables, a chi-squared (χ^2) test of independence with alpha (α) equal to 0.05 as the criterion for significance.

Within the infant mortality sample, diarrhoeal cause mortality revealed higher than expected results (Figure 3-24). More observed cases among postneonatal age infants (n = 106; 93.0% of diarrhoeal cause deaths) were found than would be expected by chance (n = 82.2), but fewer observed cases among neonatal age infants (n = 8; 7.0% of diarrhoeal cause deaths) were found than would be expected by chance (n = 31.8). A relative deficiency was also found for infectious respiratory cause mortality for neonatal age (n = 8; 10.3% of infectious respiratory deaths) than would be expected by chance (n = 21.8). Nutritional cause mortality, however, did show a relative excess among neonatal age infants (n = 28; 41.20% of nutritional cause deaths) than would be expected by chance (n = 19). Results of the 1880–1882 infant mortality sample

for cause of death and age at death produced highly statistically significant results ($\chi^2 = 59.422$; $df = 3$; $p < 0.001$), supporting the inference that cause of death and age at death are related (dependent).

Figure 3-25 presents the chi-square results for cause mortality and infant age. Within the infant mortality sample, a relative excess was found for diarrhoeal cause mortality among postneonatal age infants ($n = 149$; 96.1% of diarrhoeal cause deaths) than would be expected by chance ($n = 103.8$). A lower relative deficiency was found for diarrhoeal causes amongst neonatal age infants ($n = 6$; 3.9% of diarrhoeal cause deaths) than would be expected by chance ($n = 51.2$). Infectious respiratory cause mortality also presented a higher number of observed cases for postneonatal age infants ($n = 93$; 92.1% of infectious respiratory cause deaths) than would be expected by chance ($n = 67.6$), but a lower number of observed cases for neonatal age infants ($n = 8$; 7.9% of infectious respiratory cause deaths) than would be expected by chance ($n = 33.4$). Similarly, nutritional cause mortality had a higher observed excess for postneonatal age ($n = 142$; 82.6% of nutritional cause deaths) than would be expected by chance ($n = 116.5$) and an observed deficiency for neonatal age ($n = 32$; 18.4% of nutritional cause deaths) than would be expected by chance ($n = 57.5$). All three disease categories showed a higher than expected count among postneonatal deaths. Results of the 1910–1912 infant mortality sample for cause of death and age at death produced high statistically significant results ($\chi^2 = 258.839$; $df = 3$; $p < 0.001$). The inference that cause of death and age at death are related (dependent) is supported.

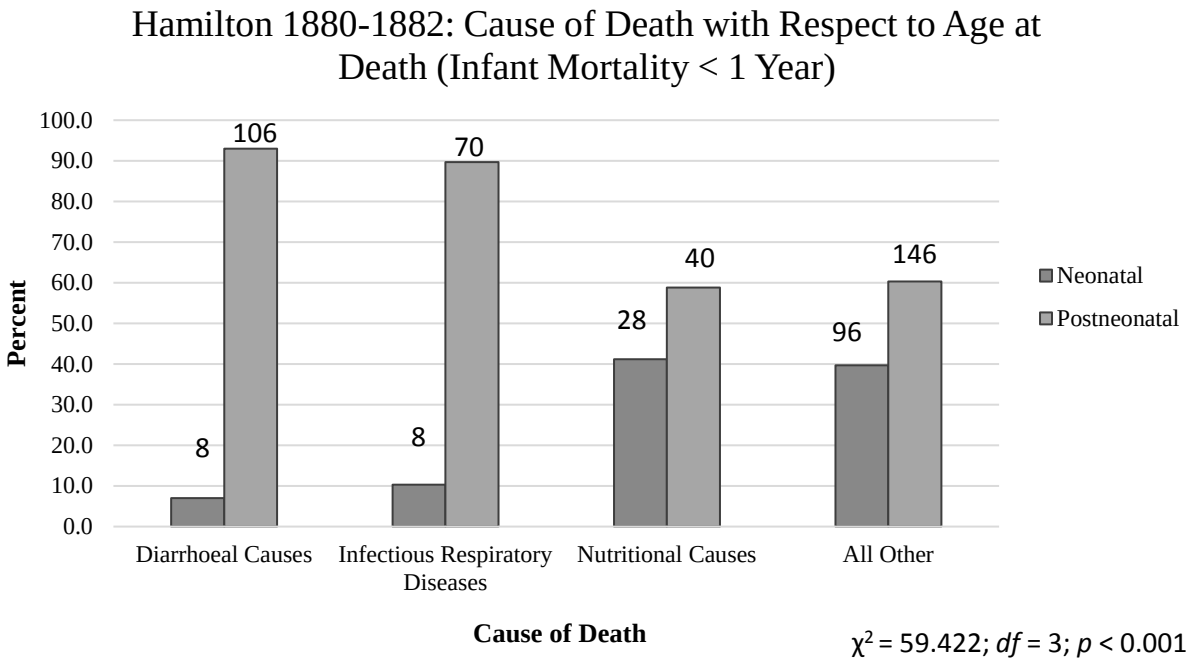


Figure 3-24. Analysis of Cause of Death with Respect to Age at Death, Hamilton 1880-1882 Infant Mortality Sample

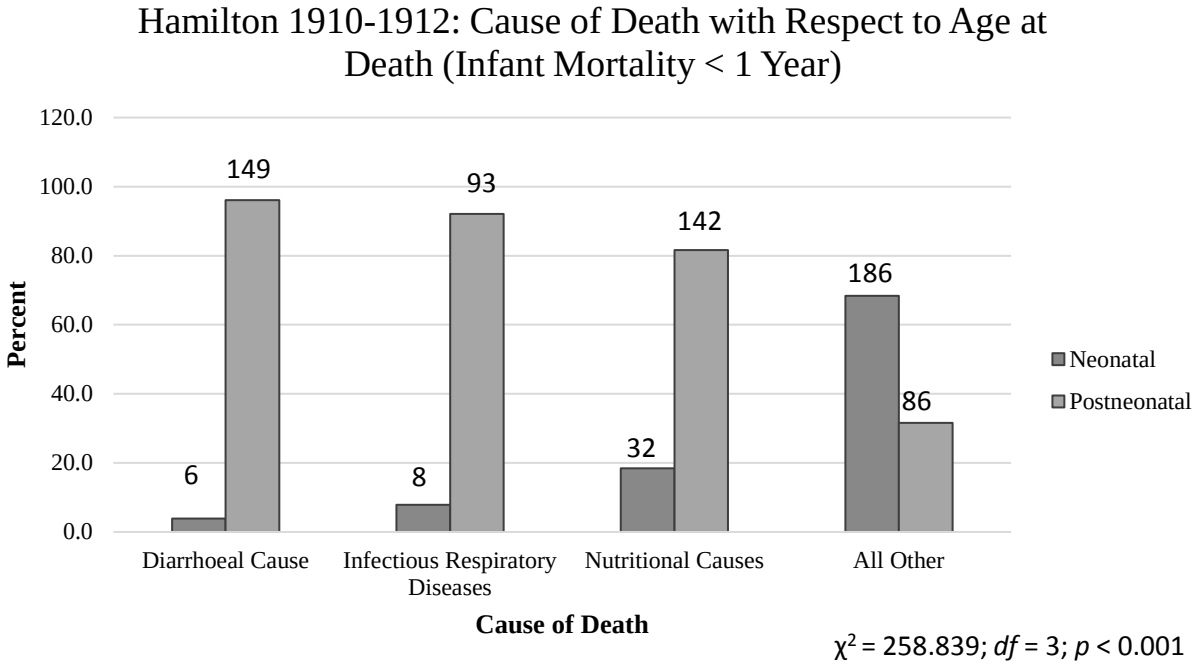


Figure 3-25. Analysis of Cause of Death with Respect to Age at Death, Hamilton 1910-1912 Infant Mortality Sample

The last set of chi-square analyses tested for cause of death and seasonality. Figure 3-26 presents the results for the 1880–1882 infant mortality sample with respect to cause of death and seasonality. There was a higher number of observed cases of diarrhoeal cause mortality during the summer months (n = 76; 66.70% of diarrhoeal cause deaths) than would be expected by chance (n = 40.0). A lower number of observed cases of diarrhoeal deaths during the spring and winter months (n = 4; 3.5% and n = 5; 4.4% of diarrhoeal cause deaths, respectively) than would be expected by chance (n = 23.8 and n = 24.3, respectively). When observing infectious respiratory cause mortality, a high relative excess for spring seasonality was found (n = 31; 39.7% of infectious respiratory deaths) than would be expected by chance (n = 16.3), while a lower relative deficiency for summer seasonality was found (n = 11; 14.1%) than would be expected by chance (n = 27.3). The results of the 1880–1882 infant mortality sample support a correlation between for cause of death and seasonality produced high statistically significant results (χ^2 = 102.465; df = 9; p< 0.001).

The chi-square results of the 1910–1912 infant mortality sample for cause of death and seasonality are presented in Figure 3-27. These findings revealed a higher number of observed cases of diarrhoeal cause mortality occurred during the summer and autumn months (n = 79; 51.0% of diarrhoeal cause mortality, n = 61; 39.4% of diarrhoeal cause mortality, respectively) than would be expected by chance (n = 50.6 and n = 45.4, respectively). There was, however, a lower number of cases observed for diarrhoeal cause mortality during the winter months (n = 11; 7.1% of diarrhoeal cause deaths) than would be expected by chance (n = 27.2). Infectious respiratory cause mortality did have a higher relative excess for winter seasonality (n = 33; 32.7% of infectious respiratory cause deaths) and a relative excess for spring seasonality (n = 31; 30.7% of infectious respiratory cause deaths) than would be expected by chance (n = 17.7 and n

= 20.7, respectively). These results for cause of death and seasonality revealed a high statistical significance ($\chi^2 = 96.9888$; $df = 9$; $p < 0.001$), supporting the inference that cause of death and seasonality are related (dependent).

While a number of the results presented here did not produce statistical results or violated the needed assumption to analyze the chi-square, variables that showed relationships can be furthered examined to determine the level of relationship between cause of death and independent variables using a multiple logistic regression model for each cause of death. Based on the chi-square test for independence, nutritional causes of death did not show any observed anomalies, with the exception of age at death in both the 1880–1882 and 1910–1912 infant sample, and thus does not support the need for further inferential statistical analysis. Diarrhoeal and infectious respiratory causes of death did show observed anomalies with two variables (infant age and seasonality) at both periods and will be used to build the models.

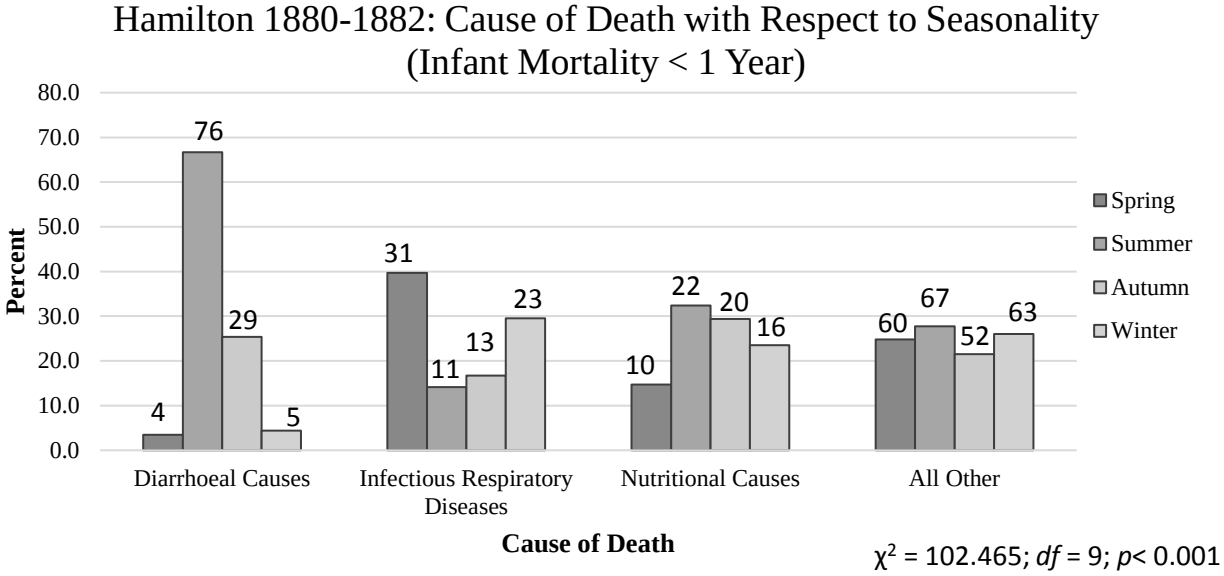


Figure 3-26. Analysis of Cause of Death with Respect to Seasonality, Hamilton 1880-1882 Infant Mortality Sample

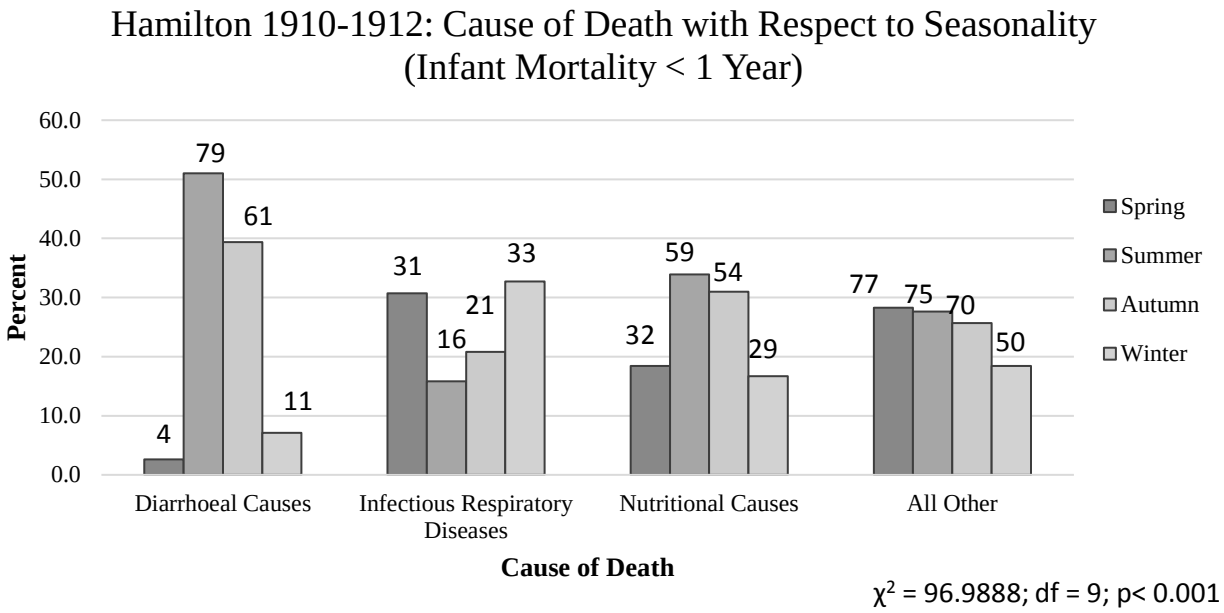


Figure 3-27. Analysis of Cause of Death with Respect to Seasonality, Hamilton 1910-1912 Infant Mortality Sample

3.4 Analysis of Mortality Profiles and Patterns

This section is broken down by sample and cause, allowing for the synthesis of the methods and a comprehensive view of each disease in response to the third and fourth research questions:

> What are the mortality profiles for the 1880–1882 and 1910–1912 periods in Hamilton, are there any observed changes in the profile over time, and what are they?
>
> Are there spatial differences in mortality for the 1910–1912 period in Hamilton, and do the mortality patterns suggest health disparity in relation to socioeconomic organization and urban development?

The logistic regression models presented here consider various characteristics associated with the likelihood of dying using the mortality odds ratio. Independent variables were chosen from descriptive chi-square results. HGIS techniques were used to examine the spatial patterns of the significant independent variables identified in the logistic regression. This HGIS divides the city into four quadrants (northwest, southwest, northeast, and southeast). King and James

streets have been used in other research to show spatial divides either as north/south or east/west (Harris & Sendbuehler, 1992; Herring & Korol, 2012). The temporal pattern of urban development supports that this segregation would likely provide better insights into health disparities than if the city was separated only by east/west or north/south. Together, the results from the logistic regression and HGIS illustrate the characteristics of the mortality sample and where these individuals lived prior to dying. The qualitative inquiry provides context, helping reveal plausible causal factors relating to the urban environment, population, and their behaviours using contemporary observations and imagery.

With any empirical spatial analysis, the use of defined boundaries such as city limits, ward boundaries, or provincial or state boundaries can create potential biases, such as boundary effects and the modifiable areal unit problem (MAUP). Boundary effects occur when arbitrary boundaries are imposed on unbounded spatial data; similarly, edges effects occur when these boundary problems are ignored or distorted (ESRI, n.d.-c). MAUP occurs when the same data is spatially aggregated into different areal units, thereby presenting different results of that data (ESRI, n.d.-d). The limitations of the data in this research render it impossible to calculate and map ward-based rates, which limits the ability to compare spatial patterns at a finer scale across the city. Nevertheless, analyzing frequencies can still be useful as it identifies where deaths were occurring and provides added detail which, when combined with the qualitative analysis, begins to show the patterns of mortality in Hamilton as it underwent rapid industrialization. Thus, the maps generated here provide a visual representation of the spatial patterns of mortality that were reflected in the death registry and further build the story of life in industrial Hamilton.

3.4.1 Working-age Mortality Sample

Logistic regression models presented here use the chi-square test for independence (or the absence of complete separation) results. Significant independent variables (the predictors of mortality cause) chosen for the 1880–1882 working-age sample included biological sex and working age. Likewise, independent variables chosen for the 1910–1912 working-age sample included biological sex, working age, birthplace, occupation, and marital status. Not all death records contained spatial information, leading to a smaller sample from which to conduct the HGIS analyses (Table 3-15). Out of the total number of cases (n = 378) in the registry, 95 cases (25.1%) were unmatched to a spatial location. This means that, of the regression model mortality causes, 74.9% of the cases (n = 283) in the working-age sample could be mapped. Accident cases had the highest number of unmatched cases (41.38%) in the geocoding process because resident addresses were not reported in the death record. One possible explanation for this is that accidents occurred outside the residence and therefore residence was not recorded.

Table 3-15 Cause of Death Cases Unmatched during Geocoding, Working-age Sample (age 15-64 years), Hamilton 1910–1912

Cause of Death	**Number of Cases**	**Cases Missing Spatial Property (Unmatched)**	**Percent of Unmatched Cases**
Tuberculosis	178	29	16.30%
Infectious Non-Respiratory Causes	84	24	28.57%
Accidental	116	42	41.38%
Total	*378*	*95*	*25.13%*

3.4.1.1 Working Age Tuberculosis Mortality

Tuberculosis in humans is an infectious bacterial disease caused by *Mycobacterium tuberculosis*, often affecting the lungs (pulmonary tuberculosis) but occasionally affecting other

areas of the body (extrapulmonary tuberculosis). This disease is predominately transmitted between humans through respiratory droplets in the air (e.g. coughing and sneezing), but can also be transmitted interspecies. The bovine form of tuberculosis (*Mycobacterium bovis*) has a wide host range, found in wild and domestic mammals, and can infect humans through contact with an infected animal (zoonosis) (de la Rua-Domenech, 2006). The zoonotic form of tuberculosis in humans is spread through ingestion, inhalation, and sometimes by contact with broken mucous membranes and broken skin of an infected mammal (de la Rua-Domenech, 2006). Primarily the risk of contracting this form of tuberculosis is through the consumption of infected unpasteurized milk and tuberculous mammal carcases (de la Rua-Domenech, 2006; Pritchard, 1988). This form is clinically and pathologically indistinguishable from *Mycobacterium tuberculosis* and likewise can infect humans both as extrapulmonary and, to a lesser extent, pulmonary tuberculosis (de la Rua-Domenech, 2006; O'Reilly & Daborn, 1995). The pulmonary zoonotic form is more often associated with those working closely with infected cows (O'Reilly & Daborn, 1995). Tuberculosis remains a significant cause of death today; especially in countries where HIV rates are high, where public health infrastructure is inadequate, where milk remains unpasteurized, and where regions are transitioning rapidly towards urbanized industrialized locales (Byarugaba, Etter, Godreuil, & Grimaud, 2009; Farmer, 1997; Lawn & Churchyard, 2009; Lienhardt et al., 2012; Lonnroth, Jaramillo, Williams, Dye, & Raviglione, 2009; Murray et al., 2009; O'Reilly & Daborn, 1995). While, geographically, tuberculosis is now mostly spread in lower income countries, tuberculosis was common at the turn of the 20th century in the rapidly expanding urban and industrial landscapes of the western world.

Logistic regression model: predicting for tuberculosis mortality

Mortality odds ratio tests various characteristics associated with the likelihood of dying due to tuberculosis versus all other causes of death, based on the results from the chi-square test independence. The logistic regression model for 1880–1882 tuberculosis mortality (Table 3-16) suggests that, conditional on death, working age significantly predicted tuberculosis-related mortality. The young working-age (15-29 years) group was over three times more likely to die from tuberculosis (OR = 3.891, $p < 0.001$) when compared to older working-age groups. The middle working-age (30-49 years) group had over two times the odds of dying from tuberculosis (OR = 2.346, $p = 0.001$) than older working-age groups (50-64 years). Other independent variables in the model were not significant predictors for tuberculosis death.

The logistic regression model for 1910–1912 tuberculosis mortality (Table 3-17) also suggests that, conditional on death, working age significantly predicted tuberculosis-related mortality. Both young working age and middle working ages exhibited a significant relationship with tuberculosis cause of death. The youngest working-age group was over seven times more likely to die from tuberculosis (OR = 7.746, $p < 0.001$) than older working-age group, while the middle working-age group was over six times more likely to die from tuberculosis (OR = 6.318, $p < 0.001$) when compared to older working-age group. Other independent variables in the model were not significant predictors for tuberculosis. Similar to the 1880–1882 logistic regression model results, the 1910–1912 model suggests a strong link between dying of tuberculosis in the younger or middle working ages. This link to dying of tuberculosis among younger and middle working age, however, is more pronounced in the later period. This result is not unexpected as tuberculosis is known to affect young adults (Farmer, 1997). Age-related

differences for tuberculosis susceptibility reveals peaks in tuberculosis susceptibility amongst adolescents (15-19 years of age) and young adults (age 20-30 years) (Donald et al., 2010).

Table 3-16. Logistic Regression Predicting for Tuberculosis Mortality, Working-age Sample (15-64 years), Hamilton 1881-1882

Independent Variables	**Coeff.**	**Sig.(*p*)**	**Exp(B)**	**S.E.**
Biological Sex				
Males	0.167	0.384		0.192
Females[R]				
Working Age				
Young Working Age (15-29 years)	1.359	0.000	3.891	0.264
Middle Working Age (30-49 years)	0.835	0.001	2.346	0.262
Older Working Age (50-64 years)[R]				
Sample Size	623			
-2 Log likelihood	667.181			

Notes: Coeff. = Logistic Regression Coefficient, Sig. (*p*) = Significance Levels, Exp(B) = Odds Ratio, S.E. = Standard Error, R = Reference Category.

Table 3-17. Logistic Regression Predicting for Tuberculosis Mortality, Working-age Sample (15-64 years), Hamilton 1910–1912

Independent Variables	**Coeff.**	**Sig.(*p*)**	**Exp(B)**	**S.E.**
Biological Sex				
Males	-0.194	0.467		0.267
Females[R]				
Working Age				
Young Working Age (15-29 years)	2.047	0.000	7.746	0.404
Middle Working Age (30-49 years)	1.843	0.000	6.318	0.375
Older Working Age (50-64 years)[R]				
Birthplace				
Canada Born	0.658	0.89		0.387
British Born	0.455	0.260		0.404
Foreign Born[R]				
Occupation				
Industrial	0.311	0.259		0.275
Labourers	-0.28	0.944		0.394
Non-Industrial[R]				
Marital Status				
Never Married	-0.462	0.361		0.506
Married	-0.808	0.87		0.472
Widowed[R]				
Sample Size	825			
-2 Log likelihood	611.067			

Notes: Coeff. = Logistic Regression Coefficient, Sig. (*p*) = Significance Levels, Exp(B) = Odds Ratio, S.E. = Standard Error, R = Reference Category.

HGIS: mortality analysis of tuberculosis, 1910–1912

As shown above, tuberculosis was the leading cause of working-age mortality in Hamilton during the study period, with a strong association for deaths occurring in the young and middle working ages. This association was stronger in the later period. Spatial distribution for tuberculosis mortality by younger and middle working ages are presented in Figure 3-28 and Table 3-18. The spatial distribution of both young and middle working ages continued to show higher frequency of cases in the northeast. Out of all of the tuberculosis cases geocoded, young working age made up 49.32% of the sample and middle working age, 39.73% of the sample. The majority of young working-age cases (51.39%; $n = 37$) were for individuals who lived in the northeast. Likewise, middle working-age cases found in the northeast made up 44.83% ($n = 26$) of the cases for this age group. If the northeast had a higher density, a higher frequency of cases in this quadrant would be expected, with all things being equal. Tuberculosis was common in higher densities areas. Cases in the northeast tended to be clustered towards the inner parts of the quadrant, with no cases occurring on the northeastern limits of the quadrant. This is to be expected, as the eastern limits would be less populated, given the city's eastward growth pattern. While the southeast had the lowest cases overall (16.44%; $n = 24$), there is a clustering pattern similar to the less dense areas at the city's eastern limits. With the northeast accounting for nearly half of tuberculosis cases, other quadrants ranged between 15 and 20 percent.

Tuberculosis thrived throughout North American cities, partially because of overcrowded living conditions, undernutrition, improper ventilation in homes, and poor personal and domestic hygiene (Feldberg, 1995; Sawchuk & Burke, 2000; Vlahov et al., 2004). The airborne nature of tuberculosis allows it to spread where ventilation is poor and people have longer periods of exposure to contaminated air (Long, Njoo, & Hershfield, 1999). Tuberculosis-infected droplets

(from coughing and sneezing of an infected person) can remain suspended in the air for several hours (The Canadian Tuberculosis Committee, 2007). Thus, overcrowded, poorly ventilated homes found in the industrial working-class neighbourhoods of Hamilton during the latter period increased the potential for transmission.

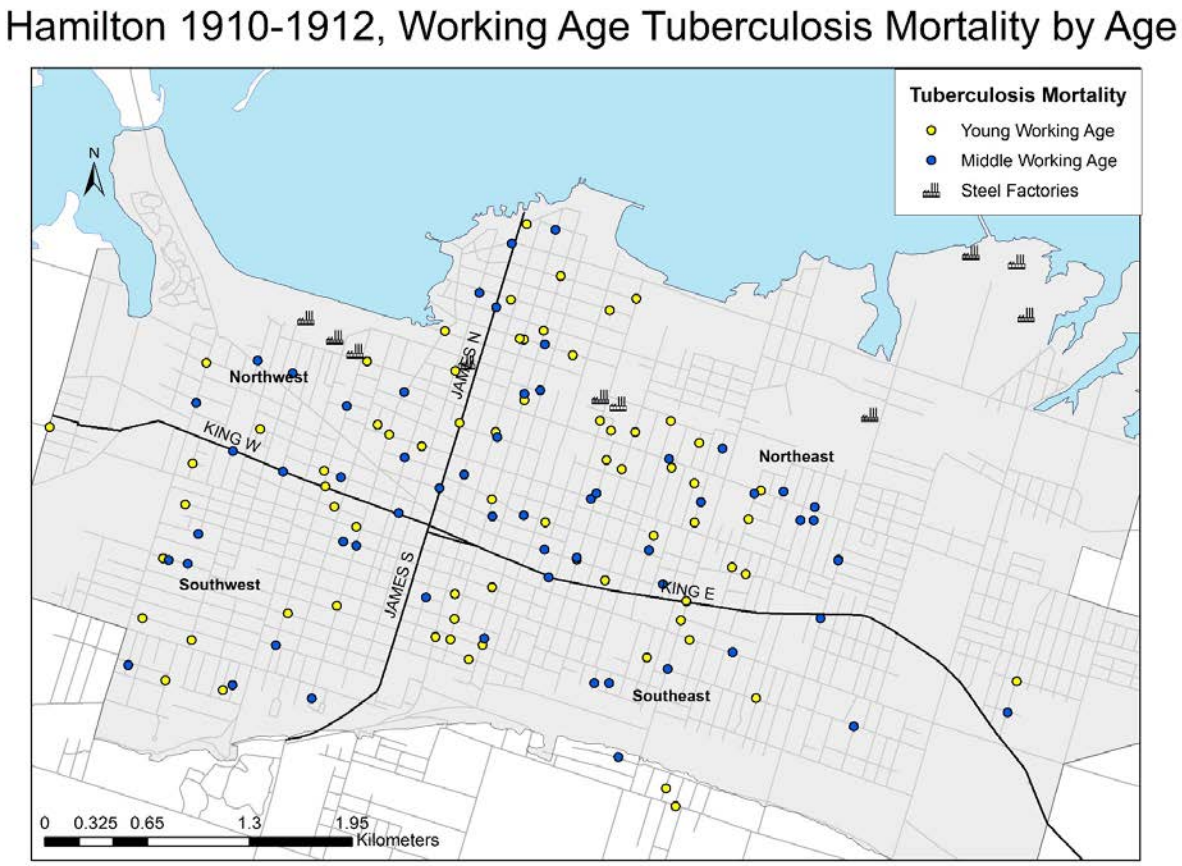


Figure 3-28. Working Age Tuberculosis Mortality by Age, Hamilton 1910-1912

Table 3-18. Working Age Tuberculosis Mortality by Age, Hamilton 1910–1912

City Quadrant	**Tuberculosis Cause of Death**					
	All		Young Working Age		Middle Working Age	
	%	n	%	n	%	n
NW	19.18	28	16.67	12	20.69	12
SW	17.12	25	16.67	12	18.97	11
NE	47.26	69	51.39	37	44.83	26
SE	16.44	24	15.28	11	15.52	9
*Total**	100.00	146	100.00	72	100.00	58

**Total of cases matched during geocoding*

3.4.1.2 Working Age Infectious Non-Respiratory Cause Mortality

Infectious diseases in general are caused by pathogenic microorganisms (bacteria, virus, parasites, or fungi) through either direct or indirect transmission. Direct transmission is usually through the air via inhalation or through physical contact (Meade & Emch, 2010). Transmission through the air can cause respiratory infectious diseases (e.g. pulmonary tuberculosis, whooping cough, influenza). Transmission through physical contact will result in other non-respiratory infectious diseases. Physical contact transmission can occur via sexual transmission (e.g. HIV, syphilis) or via fecal-oral transmission—contaminated food or water is ingested by another person (Meade & Emch, 2010). The fecal-oral route is primarily an indirect route of transmission: for example, it causes water-related infectious diseases (Meade & Emch, 2010). Another form of indirect transmission is through food-borne zoonosis, or the consumption of infected animals or animal by-products (such as zoonotic tuberculosis) (de la Rua-Domenech, 2006; Pritchard, 1988; World Health Organization, n.d.-c). Infectious diseases transmitted indirectly through water and food provide important insights into the health of the local environment, the cultural practices, and the public's health (Brandt & Gardner, 2000; Brown, Inhorn, & Smith, 1996; Cutler & Miller, 2005; Fairchild, Rosner, Colgrove, Bayer, & Fried, 2010; Ferrie & Troesken, 2008; McKeown & Record, 1962). The infectious non-respiratory causes found in Hamilton during both periods help contextualize the impact the environment had on population health.

Logistic regression model: predicting for infectious non-respiratory mortality

The logistic regression model for the 1880–1882 non-respiratory infections (Table 3-19) suggests that, conditional on dying, young working age was a significant predictor. Young working age was the only significant predictor for non-respiratory infections, with over seven

times the odds of dying from this cause (OR = 7.632, $p < 0.001$) than those in the older working-age group. With its high significance value ($p < 0.001$), this model suggests there was a strong link between dying of non-respiratory infections and the younger working ages.

Similarly, the logistic model for the 1910–1912 non-respiratory infections (Table 3-20) suggests that young working age was a significant predictor for deaths due to non-respiratory infections. When compared to the older working-age groups, young working-age individuals had over four times the odds of non-respiratory infection (OR = 4.992, $p = 0.001$). No other independent variables in the model were significant predictors for non-respiratory infections. The results from this model suggest a strong link between dying of non-respiratory infections and young working age. Examining the two models for both periods revealed that the connection between non-respiratory infections and younger working ages was more pronounced in the earlier period.

Table 3-19. Logistic Regression Predicting for Infectious Non-Respiratory Cause Mortality, Working-age Sample (15-64 years), Hamilton 1881-1882

Independent Variables	**Coeff.**	**Sig.(*p*)**	**Exp(B)**	**S.E.**
Biological Sex				
Males	-0.157	0.586		0.288
Females[R]				
Working Age				
Young Working Age (15-29 years)	2.032	0.000	7.632	0.454
Middle Working Age (30-49 years)	0.695	0.163		0.498
Older Working Age (50-64 years)[R]				
Sample Size	623			
-2 Log likelihood	354.644			

Notes: Coeff. = Logistic Regression Coefficient, Sig. (*p*) = Significance Levels, Exp(B) = Odds Ratio, S.E. = Standard Error, R = Reference Category.

Table 3-20. Logistic Regression Predicting for Infectious Non-Respiratory Cause Mortality, Working-age Sample (15-64 years), Hamilton 1910–1912

Independent Variables	**Coeff.**	**Sig.(*p*)**	**Exp(B)**	**S.E.**
Biological Sex				
Males	0.327	0.434		0.418
Females[R]				
Working Age				
Young Working Age (15-29 years)	1.608	0.001	4.992	0.497
Middle Working Age (30-49 years)	0.485	0.315		0.482
Older Working Age (50-64 years)[R]				
Birthplace				
Canada Born	0.323	0.538		0.525
British Born	0.183	0.740		0.551
Foreign Born[R]				
Occupation				
Industrial	0.190	0.642		0.409
Labourers	0.409	0.424		0.512
Non-Industrial[R]				
Marital Status				
Never Married	0.527	0.631		1.097
Married	0.646	0.539		1.052
Widowed[R]				
Sample Size	825			
-2 Log likelihood	327.966			

Notes: Coeff. = Logistic Regression Coefficient, Sig. (*p*) = Significance Levels, Exp(B) = Odds Ratio, S.E. = Standard Error, R = Reference Category.

HGIS: mortality analysis of infectious non-respiratory causes, 1910–1912

The logistic regression results revealed that working ages significantly predicted non-respiratory infections, as younger individuals were more likely than older individuals to die from these infections. Spatial distribution for young working-age cases of non-respiratory infections mortality from 1910–1912 is presented in Figure 3-29 and Table 3-21. The young working-age group made up 21.79% (*n* = 17) of the geocoded sample, with 70.59% of those deaths occurring in the northeast. In comparison, 56.41% (*n* = 44) of all non-respiratory infections occurred in the northeast. While the greatest prevalence of cases occurred in the northeast, the low count did not

lend itself to conclusive clustering; however, most cases occurred closer to the core rather than in the eastern limits of the city.

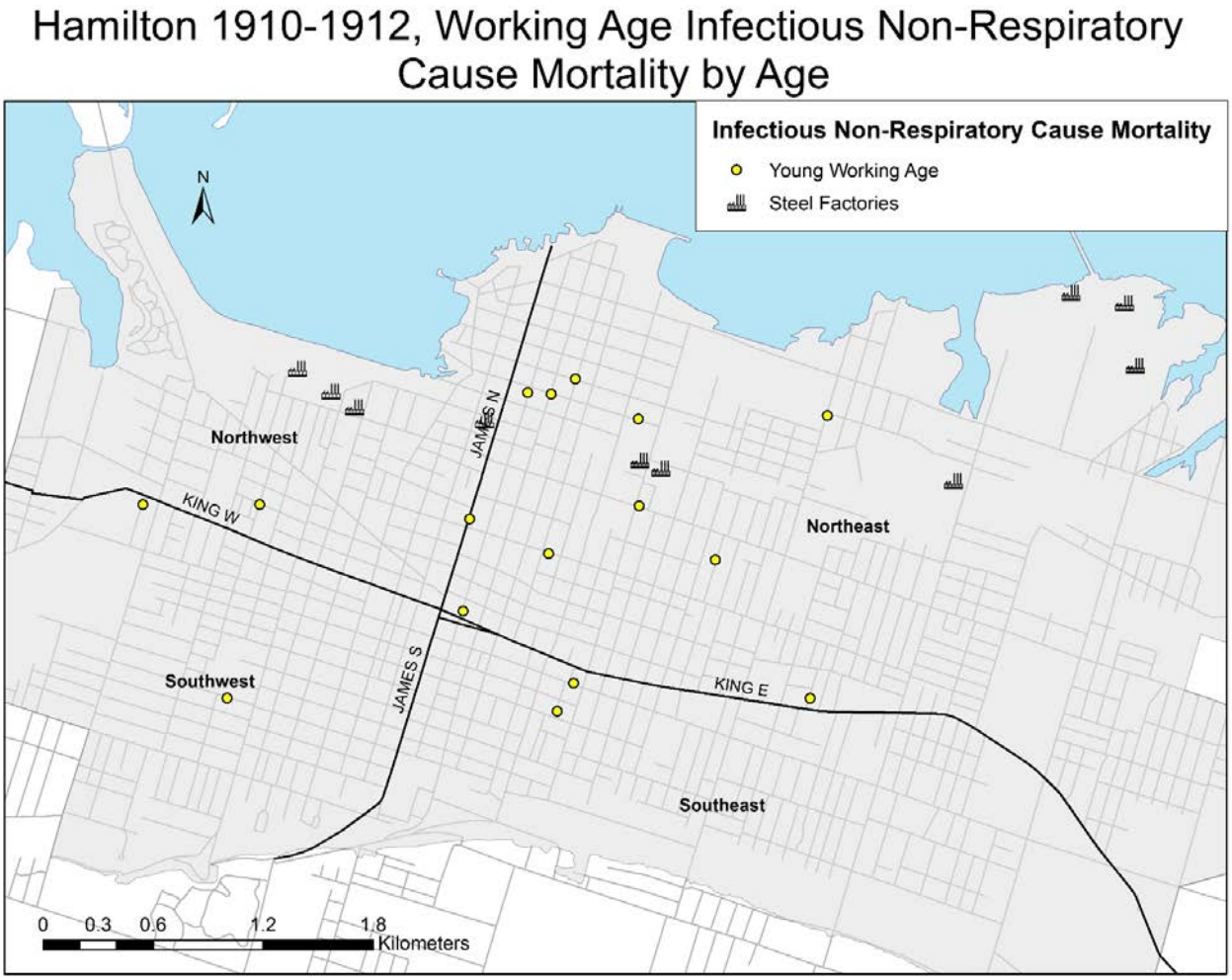


Figure 3-29. Working Age Infectious Non-Respiratory Mortality by Age, Hamilton 1910-1912

Table 3-21. Working Age Infectious Non-Respiratory Mortality by Age, Hamilton 1910–1912

City Quadrant	**Infectious Non-Respiratory Causes of Death**			
	All		Young Working Age	
	%	n	%	n
NW	15.38	12	5.88	1
SW	14.10	11	11.76	2
NE	56.41	44	70.59	12
SE	14.10	11	11.76	2
*Total**	100.00	78	100.00	17

**Total of cases matched during geocoding*

3.4.1.3 Working Age Accidental Cause Mortality

Accidental mortality can be the result of hazards in the social, built, or physical environment. The increase in accidental mortality that occurred over the study period as the city transitioned to heavy industrialization was due to workplace hazards: by the later study period, accidental deaths became the second highest cause of death and showed a significant relationship among working-age males. Industrial accidents, however, were not reported with any consistency before 1910 (Heron, 1988). This study found descriptions of accidental causes (such as the direct cause of the accident or where it took place) were lacking, which makes a direct connection with occupational hazard difficult. The steel plant, however, during the early 20th century was considered a hazardous pace, where employees worked with extremely hot materials and were exposed to toxic chemicals (such as carbon monoxide, methane, nitrogen, oxygen, and pyritic sulphide) (Barreto, Swerdlow, Smith, & Higgins, 1996, 1997; Caplan, 2005; Fitch, 1969; Heron, 1988; Hutchinson et al., 1996; Ludlow & Burke, 2012). Heron (1988) notes that, in 1912, 113 employees at Stelco in Hamilton suffered occupational injuries that required at least a week off work. Relative risks for fatal injuries relate to the noise, heat, dust, fumes, gas, and vapour common to steel plants (Barreto et al., 1996, 1997). Thus, evidence supports the work environment contributed to heightened accidental mortality in Hamilton between 1910 and 1912, in large part due to the emergence of the steel industry at this time.

Logistic regression model: predicting for accidental mortality

The logistic regression model for 1880–1882 accidental cause mortality (Table 3-22) suggests that biological sex was a significant predictor for accident mortality. Males were over seven times more likely to die from an accident (OR = 7.005, $p = 0.002$) than females were. Other independent variables in the model were not significant predictors for accidental cause

death; however the model suggests a significant link ($p = 0.002$) between dying of an accidental cause and being male.

The logistic regression model for 1910–1912 accidental cause mortality (Table 3-23) suggests that biological sex, working age, and birthplace significantly predicted accidental-related mortality. Males had over five times the odds of dying from an accident (OR = 5.200, $p < 0.001$) when compared to females. The young working age group were over six times more likely (OR = 6.110, $p < 0.001$), and the middle working age group over three times more likely (OR = 3.394, $p = 0.002$), to die from an accident than the older working age group. Those born in Canada were significantly less likely to die from an accident (OR = 0.347, $p = 0.004$) than foreign-born individuals. Foreign-born people had over twice the odds of dying from an accidental cause (1/0.347 = 2.88) compared to Canadians. Other independent variables in the model were not significant predictors for accidental cause death. Thus, this model showed a strong link between dying of accidental causes and the male sex, young and middle working age, and foreign birthplaces. Although, in both periods, males were more likely to die from accidental causes, in the later period, other factors, such as age and birthplace, also became associated with accidental-related mortality.

Table 3-22. Logistic Regression Predicting for Accidental Mortality, Working-age Sample (15-64 years), Hamilton 1881-1882

Independent Variables	**Coeff.**	**Sig.(*p*)**	**Exp(B)**	**S.E.**
Biological Sex				
Males	1.947	0.002	7.005	0.637
Females[R]				
Working Age				
Young Working Age (15-29 years)	0.885	0.124		0.576
Middle Working Age (30-49 years)	0.103	0.867		0.618
Older Working Age (50-64 years)[R]				
Sample Size	623			
-2 Log likelihood	161.998			

Notes: Coeff. = Logistic Regression Coefficient, Sig. (*p*) = Significance Levels, Exp(B) = Odds Ratio, S.E. = Standard Error, R = Reference Category.

Table 3-23. Logistic Regression Predicting for Accidental Mortality, Working-age Sample (15-64 years), Hamilton 1910–1912

Independent Variables	**Coeff.**	**Sig.(*p*)**	**Exp(B)**	**S.E.**
Biological Sex				
Males	1.649	0.000	5.200	0.437
Females[R]				
Working Age				
Young Working Age (15-29 years)	1.810	0.000	6.110	0.437
Middle Working Age (30-49 years)	1.222	0.002	3.394	0.386
Older Working Age (50-64 years)[R]				
Birthplace				
Canadian Born	-1.058	0.004	0.347	0.336
British Born	0.024	0.994		0.346
Foreign Born[R]				
Occupation				
Industrial	0.383	0.238		0.324
Labourers	0.419	0.276		0.385
Non-Industrial[R]				
Marital Status				
Never Married	1.078	0.318		1.079
Married	1.002	0.340		1.050
Widowed[R]				
Sample Size	825			
-2 Log likelihood	434.890			

Notes: Coeff. = Logistic Regression Coefficient, Sig. (*p*) = Significance Levels, Exp(B) = Odds Ratio, S.E. = Standard Error, R = Reference Category.

HGIS: mortality analysis of accidental causes, 1910–1912

The spatial distribution for the 1910–1912 logistic regression models predicting accidents is presented below. Addresses for accidental cases were poorly recorded in the record; therefore these findings must be analyzed with caution. Accidents for males had a significant relationship in the logistic regression model. The spatial distribution is presented in Figure 3-30 and Table 3-24. Of the matched cases, males made up 82.43% (n = 61) of the total accidents. The majority of male cases over the three years were matched to addresses in the northeast (70.59%; n = 33). Overall, over half of the cases were matched to addresses occurring in the northeast (56.41%; n = 44).

When selecting for working age, spatial distribution is presented in Figure 3-31 and Table 3-25. Young working and middle working ages made up 81.08% of the cases geocoded (44.59% and 36.49%, respectively). Over half of the cases for each age group were from individuals who resided in the northeast: 54.55% of young working age (n = 18) and 51.85% of middle working age (n = 14). The lowest cases overall (12.16%; n = 9) and for middle working age cases (7.41%; n = 2) were found in the southeast. The lowest cases for the young working age occurred in the southwest (6.06%; n = 2).

The last significant predictor for accidental morality was birthplace (Figure 3-32 and Table 3-25). Of all the cases for accidents (n = 74), British-born people made up 45.95% (n = 34) and foreign-born 14.86% (n = 11) of the geocoded cases. The logistic regression model suggested that the strongest link between birthplace and accidents were for foreign-born individuals. The number of cases for foreign-born individuals is too small to provide any conclusive evidence of spatial patterning. Of the British-born, over half (58.82%; n = 20) of the

accidents occurred among individuals residing in the northeast. The lowest number of cases was found in southwest (2.94%; *n* =1).

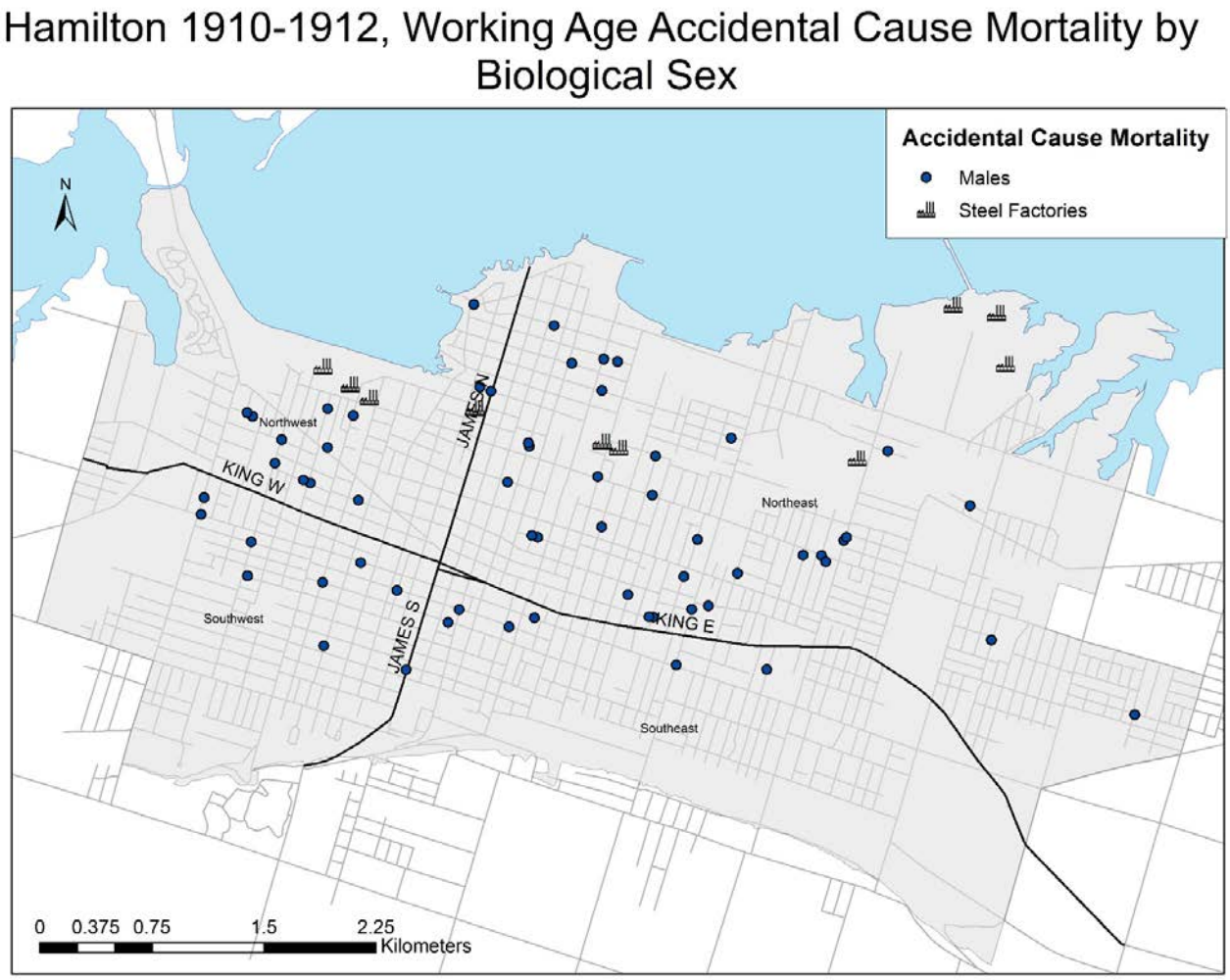


Figure 3-30. Working Age Accidental Cause Mortality by Biological Sex, Hamilton 1910-1912

Table 3-24. Working Age Accidental Mortality by Biological Sex, Hamilton 1910–1912

City Quadrant	Accidental Cause of Death			
	All		Males	
	%	n	%	n
NW	20.27	15	21.31	13
SW	14.86	11	14.75	9
NE	52.70	39	54.10	33
SE	12.16	9	9.84	6
*Total**	100.00	74	100.00	61

**Total of cases matched during geocoding*

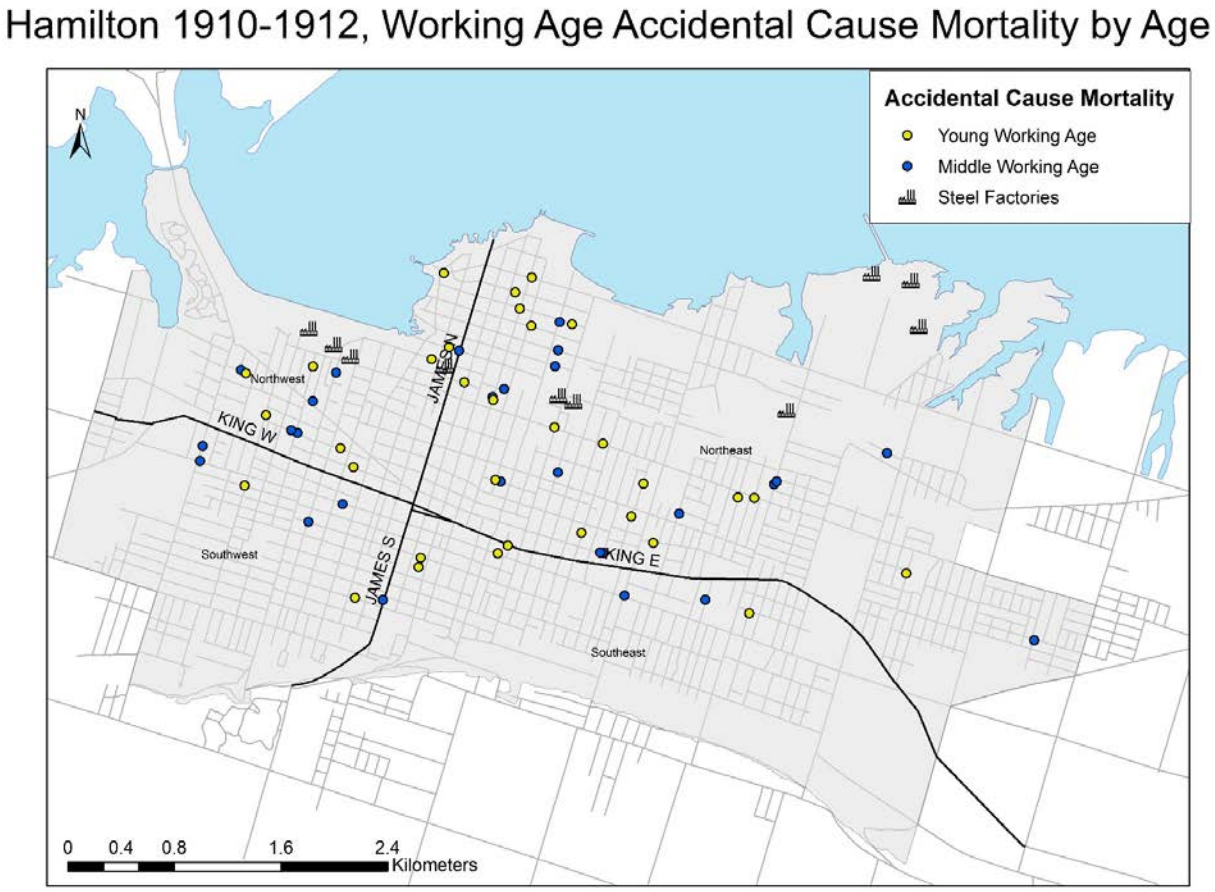


Figure 3-31. Working Age Accidental Cause Mortality by Age, Hamilton 1910-1912

Table 3.25. Working Age Accidental Mortality by Age, Hamilton 1910–1912

City Quadrant	Accidental Cause of Death					
	All		Young Working Age		Middle Working Age	
	%	n	%	n	%	n
NW	20.27	15	24.24	8	22.22	6
SW	14.86	11	6.06	2	18.52	5
NE	52.70	39	54.55	18	51.85	14
SE	12.16	9	15.15	5	7.41	2
*Total**	100.00	74	100.00	33	100.00	27

**Total of cases matched during geocoding*

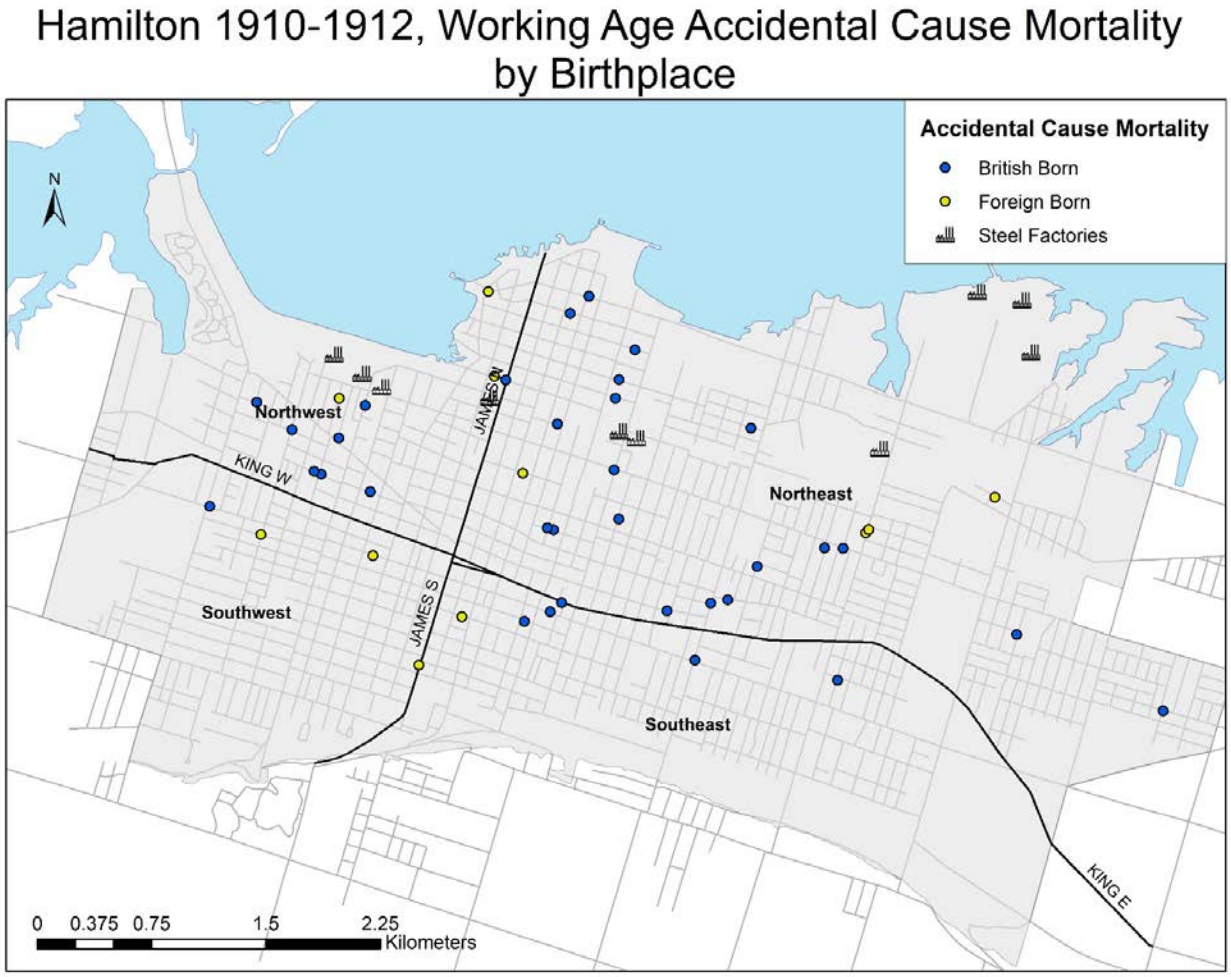


Figure 3-32. Working Age Accidental Cause Mortality by Birthplace, Hamilton 1910-1912

Table 3-26. Working Age Accidental Mortality by Birthplace, Hamilton 1910–1912

City Quadrant	Accidental Cause of Death					
	All		British Born		Foreign Born	
	%	n	%	n		
NW	20.27	15	27.27	3	20.27	15
SW	14.86	11	27.27	3	14.86	11
NE	52.70	39	36.36	4	52.70	39
SE	12.16	9	9.09	1	12.16	9
*Total**	100.00	74	100.00	11	100.00	74

**Total of cases matched during geocoding*

Without adequate recording in the death record of the type and cause of the accident combined with records from the factories, understanding the full extent of accidents related to occupation is not possible. In the United States, at the turn of the 20th century, the poor working conditions of steel plants were reported: deaths due to hot molten steel spills, getting caught in machinery, and explosions were common causes of death in the workplace (United States Department of Labor, n.d.). These reported events were not unlike findings found in the steel plant in Sydney, Nova Scotia, at the turn of the 20th century (Ludlow, 2009; Ludlow & Burke, 2012). Immigrant steel workers have also been found to have an increased risk when compared to non-immigrant workers. This increase in accidental risk among immigrant workers during the early 1900s has been attributed to: long working hours in less than ideal conditions, language barriers in an already noisy work environment, and working in the more hazardous parts of the plant (e.g. blast furnace, coking ovens) (Barreto et al., 1997; Caplan, 2005; Ludlow, 2009; Ludlow & Burke, 2012; Robinson, Kuller, & Perper, 1988). Accidents tended to occur in the early months of a working career due to inexperience, unfamiliarity with dangers and equipment, new working environments, and the natural high spirit of youth (Mekelburg, 1952). Although the young are more likely to be involved in accidents, there would be some protection for younger unskilled working-age males (Gagnon et al., 2011). In Sydney, for example, younger, less experienced males were more likely to work in the labour yards than in the steel plant (Caplan, 2005), where the risk of accidents was not as high. By their mid-twenties, males would have built up their skills and moved into more hazardous sections of the plant (Caplan, 2005; Heron, 1988).

3.4.1.4 Synthesis: Working-age Mortality

The logistic regression results for working-age mortality did support some of the previous descriptive findings observed in this research. In the 1880–1882 working-age sample, young working age significantly predicted tuberculosis and infectious (non-respiratory) disease mortality. In the 1910–1912 working-age sample, young working age significantly predicted tuberculosis, infectious (non-respiratory) disease, and accidental cause mortality. For both periods, male sex was a significant predictor of accidental cause mortality in the working-age sample. Foreign birthplace, in the 1910–1912 working-age mortality sample, significantly predicted for accidental cause mortality. Additionally, the logistic regression results for the working-age mortality sample revealed some predictors not found in the previous quantitative results, mainly that middle working age was a significant predictor for tuberculosis, non-respiratory infections, and accidental mortality.

Spatial examination of the 1910–1912 results revealed a northeast distribution of tuberculosis, non-respiratory infections, and accidents. Tuberculosis is a known disease of poverty, found in areas where overcrowding, poorly constructed homes are prevalent. With young and middle working age being a significant predictor for tuberculosis, these individuals were exposed to such environmental conditions. Non-respiratory infections are also tied to the local environment as well as the food preparation practices. Typhoid fever was one of the most frequent diseases found in this category. Thus, the northeast spatial distribution among young working ages supports spatial inequalities in the urban environment. Finally, the geographical northeast distribution of accidents supports the spatial divisions of working class found in Hamilton. While accidents may not have a direct relationship to the environment, the adverse effects of living in such conditions is potentially linked to one's ability to work effectively: there

is evidence to support that environmental factors can affect mental health and the likelihood of injuries (Howden-Chapman, 2004; Krieger & Higgins, 2002).

The logistic regression results for the working-age sample did not confirm some of the quantitative findings observed previously in this research, namely the chi-square results for cause of death and marital status, suggesting that other variables may have had more influence in predicting for cause of death. Logistic regression models control for the effects of other independent variables to help determine the true importance of an independent variable with respect to the dependent variables. For example, single (never married) revealed a significant relationship with tuberculosis and accidental mortality in the chi-square analysis; however, the logistic regression results showed no significant findings for either mortality models. Thus, never married was not a predictor for tuberculosis and accidental cause mortality. Rather, the relationship is likely due to an age effect, whereby young working age was a significant predictor in both logistic regression models. The chi-square testing independent variables, working age and marital status, further supported the age effect for marital status significance, which observed a higher-than-expected count for single status with respect to young working age (Appendix A). Thus, using a cohesive quantitative analysis method provides a holistic picture of the mortality sample and gives some indication of who might have been at risk.

3.4.2 Infant Mortality Sample

The logistic regression models presented here consider various characteristics in association with the likelihood of infants dying from diarrhoea and respiratory infections compared to all other causes of death among the sample. Based on the chi-square test for independence, independent variables (or predictors of mortality cause) chosen for both the 1880–1882 infant sample and the 1919-1912 sample were infant age and seasonality. Infant mortality

maps were constructed for two of the three major causes of infant death, diarrhoeal cause and infectious respiratory disease, as per the logistic regression model results. For the infant sample, 18.36% of the cases had no spatial attribute, leading to a smaller sample size for mapping cases (Table 3-27).

Table 3-27. Cause of Death Cases Unmatched during Geocoding for the Infant Sample (< 1 year), Hamilton 1910–1912

Major Cause of Death	Number of Cases	Cases Missing Spatial Property (Unmatched)	Percent of Unmatched Cases
Diarrhoeal Cause	155	32	20.65%
Infectious Respiratory Disease	101	15	14.85%
Total	*256*	*47*	*18.36%*

3.4.2.1 Infant Diarrhoeal Cause Mortality

There are various reasons for infant diarrhoea, but it is often associated with an infectious pathogen that affects the intestinal tract. While a shift in different countries has happened over time for infectious disease risk (see the epidemiological transition), diarrhoeal cause of infant death remains one of the leading causes of death for children under five years of age in middle and lower income countries (World Health Organization, 2013). Diarrhoeal causes are predominately water-related, and contracted when contaminated water is ingested (through the washing of food, direct consumption, watering down milk, or unhygienic/unsanitary practices) (Meade & Emch, 2010). The type of disease found in this category has a causal relationship with conditions found in the built environment and are often affiliated with overcrowded poverty-stricken locales where public health infrastructure is lacking, making the population highly susceptible to changes in the physical environment, such as seasonal droughts and flooding (Centers for Disease Control and Prevention, 2011b; Meade & Emch, 2010).

Diarrhoeal causes of infant mortality were a constant risk to Hamilton's infant population in both periods and a manifestation of the human and physical environment.

Logistic regression model: predicting for diarrhoeal cause mortality

The logistic regression model for 1880–1882 diarrhoeal cause mortality (Table 3-28) suggests that infant age and seasonality both significantly predict diarrhoeal-related mortality. Postneonatal infants were over five times more likely to die from a diarrhoea cause (OR = 5.9991, $p < 0.001$) than neonatal infants (infants within their first month of life). Infants overall were 13 times more likely to die in summer of diarrhoea (OR = 13.140, $p < 0.001$) than in winter. They were also six times more likely to die of diarrhoea in autumn (OR = 6.656, $p < 0.001$) than in winter. This model suggests that there was a strong link between diarrhoeal cause mortality and postneonatal infant age, as well dying of diarrhoea during the summer and autumn months. Furthermore, summer seasonal deaths had the strongest link for infants dying from diarrhoea than other independent variables.

Similarly, for the 1910–1912 period, the logistic regression model for diarrhoeal cause mortality (Table 3-29) suggests that infant age and seasonality significantly predict diarrhoeal-related mortality. Postneonatal infants had over 15 times the odds of dying from a diarrhoeal cause (OR = 15.592, $p < 0.001$) when compared to neonatal infants. In the mortality sample, the odds of an infant dying of a diarrhoeal cause in the summer was over four times (OR = 4.367, $p < 0.001$) the odds of dying from diarrhoea in the winter. As well, the odds that death from diarrhoea would occur in the autumn months were over four times higher (OR = 4.136, $p < 0.001$) than in the winter season. The model suggests a strong link between postneonatal infant age and dying of diarrhoea, especially during the summer and autumn months. Postneonatal age had the strongest link for infants dying from diarrhoea compared to the other variables presented

in the model. In the earlier period, the model suggests the strongest link was found between diarrhoea and summer; however, in the later period, the strongest link was found between diarrhoea and postneonatal age.

Table 3-28. Logistic Regression Predicting for Diarrhoeal Cause Mortality, Infant Deaths (< 1 year), Hamilton 1880–1882

Independent Variables	**Coeff.**	**Sig.(*p*)**	**Exp(B)**	**S.E.**
Infant Age				
Postneonatal	1.790	0.000	5.991	0.395
Neonatal[R]				
Seasonality				
Spring	-0.320	0.643		0.690
Summer	2.576	0.000	13.140	0.488
Autumn	1.882	0.000	6.656	0.512
Winter[R]				
Sample Size	513			
-2 Log likelihood	412.823			

Notes: Coeff. = Logistic Regression Coefficient, Sig. (*p*) = Significance Levels, Exp(B) = Odds Ratio, S.E. = Standard Error, R = Reference Category.

Table 3-29. Logistic Regression Predicting for Diarrhoeal Cause Mortality, Infant Deaths (< 1 year), Hamilton 1910–1912

Independent Variables	**Coeff.**	**Sig.(*p*)**	**Exp(B)**	**S.E.**
Infant Age				
Postneonatal	2.741	0.000	15.592	0.428
Neonatal[R]				
Seasonality				
Spring	-1.247	0.039	0.287	0.603
Summer	1.488	0.000	4.367	0.353
Autumn	1.432	0.000	4.136	0.360
Winter[R]				
Sample Size	783			
-2 Log likelihood	608.850			

Notes: Coeff. = Logistic Regression Coefficient, Sig. (*p*) = Significance Levels, Exp(B) = Odds Ratio, S.E. = Standard Error, R = Reference Category.

HGIS: mortality analysis of diarrhoeal causes, 1910–1912

The logistic regression model for diarrhoeal cause mortality predicted a relationship for both seasonality and age. Spatial distribution for summer and autumn are presented in Figure 3-33 and Table 3-30. Summer and autumn make up 89.43% of geocoded diarrhoea cases (51.22% and 38.21%, respectively). Most cases of diarrhoea occurring in summer or autumn were located in the northeast. There were 40 (63.49%) summer cases and 21 (44.68%) autumn cases in the northeast. Similar findings can be found overall, with 56.10% (n = 69) of cases occurring in the northeast. With the northeast having the majority of cases, the range of cases for autumn was quite small (n = 8-11 cases). Summer did show a higher range of cases, with the smallest being 1.59% (n = 1) in the southeast and the second highest cases in the northwest (20.63%; n = 13). There was some weak sporadic clustering throughout the northeast, but there was a cluster occurring across both northeast and northwest quadrants around the northern section of James Street. The connection between seasonality and diarrhoeal infant mortality can be attributed, in part, to poor quality water supply (Sawchuk et al., 2002). Infantile cholera was also known as "summer complaint" due to its association with hot, humid weather (Swedlund, 2010). Contaminated milk caused by poor or nonexistent refrigeration, or cross-contamination of food and beverages with viruses and bacteria (Keusch et al., 2006; Leavitt, 1992; Meade & Emch, 2010) also contributed to infant diarrhoea. Pasteurizing and purifying milk were an attempt to address public health concerns in Canada and the United States (Block, 2009; Czaplicki, 2007; Speake, 2011). Pasteurization of milk, however, was not compulsory in Ontario until 1938 (Speake, 2011).

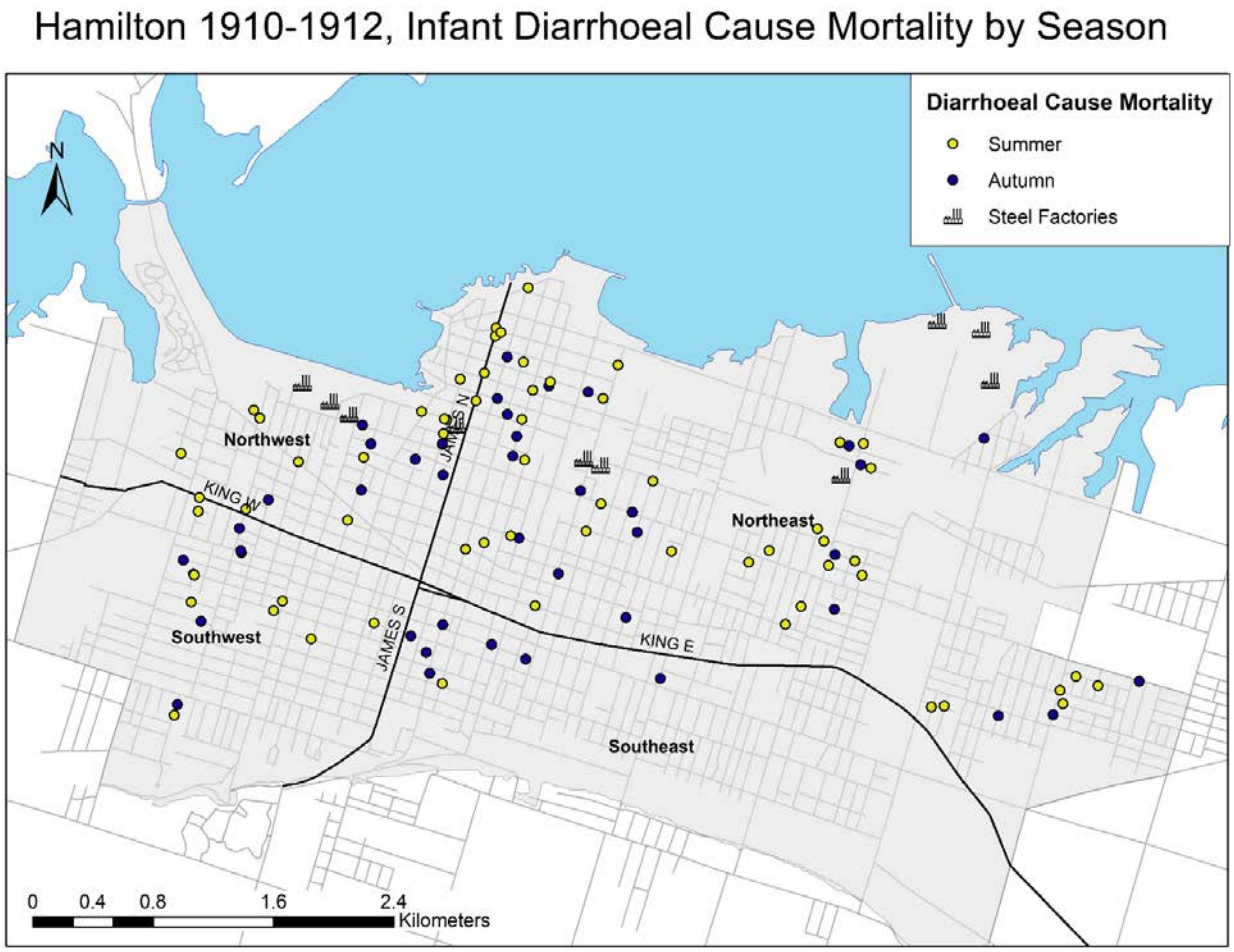


Figure 3-33. Infant Diarrhoeal Cause Mortality by Season, Hamilton 1910-1912

Table 3-30. Infant Mortality by Season, Hamilton 1910–1912

City Quadrant	**Cause of Death**			
	Diarrhoeal Cause		Summer/Autumn	
	%	n	%	n
NW	17.07	21	20.63	13
SW	19.51	24	14.29	9
NE	56.10	69	63.49	40
SE	7.32	9	1.59	1
*Total**	100.00	123	100.00	63

**Total of cases matched during geocoding*

The spatial distributions of postneonatal age and diarrhoea cases are examined in Figure 3-34 and Table 3-31. Postneonatal age made up 36.59% (n = 45) of all geocoded cases of diarrhoea. The majority of neonatal cases were found in the northeast (44.44%; n = 20), and a weak clustering can be found on the western side of this quadrant. Other cases in the northeast are sporadic. Across the other quadrants, cases range from eight to eleven. Overall, however, the least number of cases occurred in the southeast (7.32%; n = 9). Although the southeast only had eight cases geocoded, most of the postneonatal cases in this quadrant cluster near James Street South. Diarrhoea is a known major cause of death occurring beyond the neonate period of infancy (Mulholland, 2007). Neonatal mortality is often due to endogenous causes (those occurring within the body due to genetic make-up), or circumstances before or during birth rather than environmental causes (Reid, 2001). In the United States, around the 1880s, women began to supplement breastmilk with cow's milk shortly after birth and began to wean babies before three months of age (Wolf, 2003). This practice in the early months of life may have caused an increase in deaths in the postneonatal ages. Time of weaning is an important factor to consider because breastfeeding can protect infants from external risks that may cause infectious diseases (Canadian Paediatric Society, 2004).

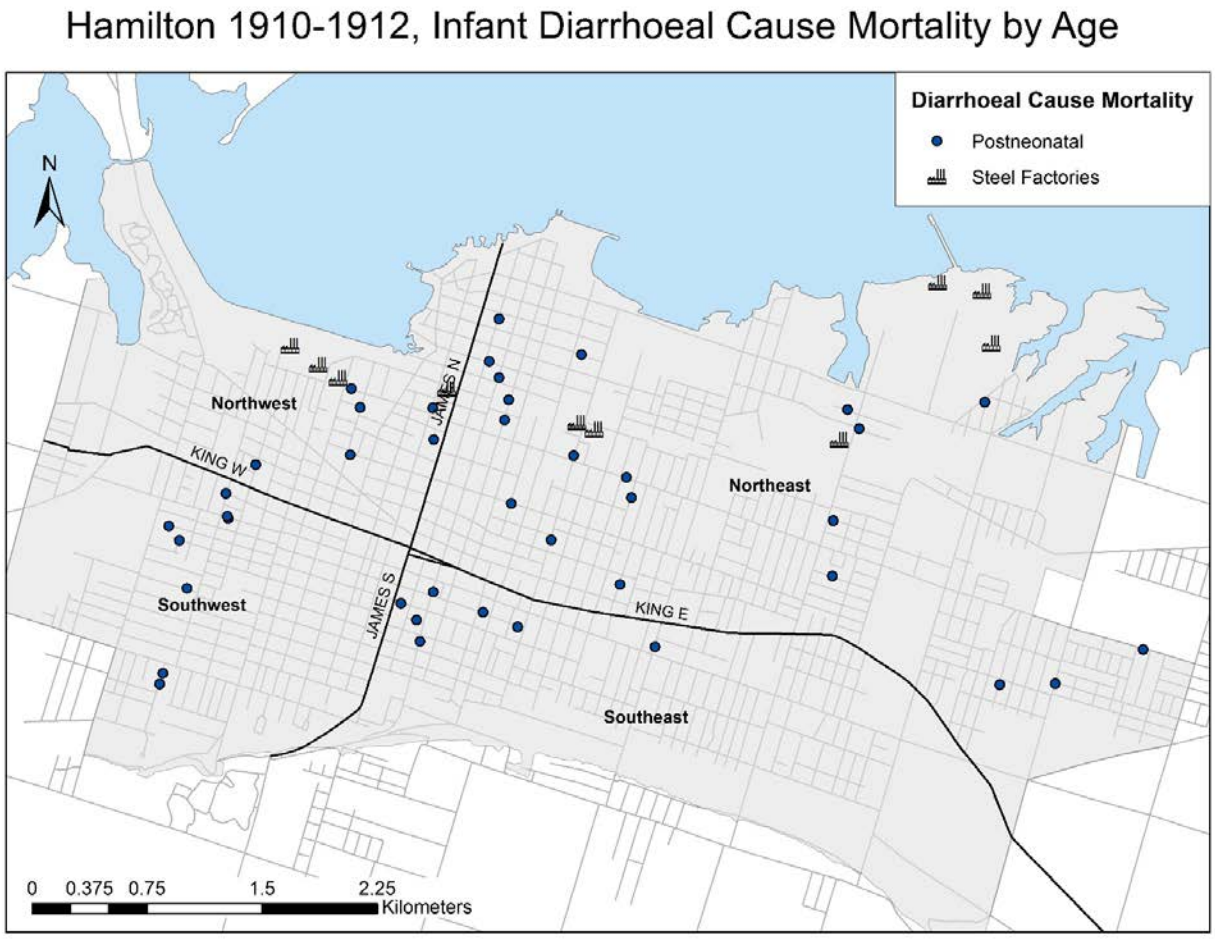


Figure 3-34. Infant Diarrhoeal Cause Mortality by Age, Hamilton 1910-1912

Table 3-31. Infant Diarrhoeal Cause Mortality by Age, Hamilton 1910–1912

City Quadrant	**Cause of Death**			
	Diarrhoeal Cause		Postneonatal Age	
	%	n	%	n
NW	17.07	21	13.33	6
SW	19.51	24	24.44	11
NE	56.10	69	44.44	20
SE	7.32	9	17.78	8
*Total**	100.00	123	100.00	45

**Total of cases matched during geocoding*

3.4.2.2 Infant Infectious Respiratory Cause Mortality

Infectious respiratory diseases are caused by airborne viral and bacterial pathogens. Today, pneumonia is the leading cause of infectious mortality for children under the age of five years (World Health Organization, 2015a). Whooping cough (pertussis), a highly contagious respiratory disease, mostly affects infants and young children, but is especially fatal to infants under one year of age (Centers for Disease Control and Prevention, 2015). Overcrowded conditions, weakened immune systems, non-breastfeeding practices, and poor nutrition are key risk factors of infectious respiratory infant mortality (Cohen, Wilson, & Aiello, 2007; Mercer, 1986; Shulman, 2004; Weiss & McMichael, 2004). Respiratory infectious diseases among infants have also shown a high correlation to environmental factors (Reid, 2002). Such behavioural and environmental risk factors were prevalent in the infant infectious respiratory mortality found in Hamilton at both periods in time.

Logistic regression model: predicting for respiratory infectious mortality

Presented in Table 3-32 is the logistic regression model for 1880–1882 respiratory infections, which suggests that postneonatal infant age and summer months are significant predictors for respiratory-infection-related deaths among infants. Postneonatal infants were over five times more likely to die from respiratory infection (OR = 5.439, $p < 0.001$) than neonatal infants were. Infant mortality during the summer, however, showed significantly lower odds of dying from a respiratory infection (OR = 0.181, $p < 0.001$) than during the winter. In other words, infants were five times more likely (1/0.181 = 5.53) to die from respiratory infection during the winter than in the summer season. The model suggests a strong link between the winter and dying of respiratory infections in the postneonatal infant age.

The logistic model for 1910–1912 respiratory infections mortality is presented in Table 3-33, and suggests that postneonatal infant age and summer and autumn seasonality are significant predictors for infectious respiratory cause mortality. Postneonatal-aged infants had over nine times the odds of dying from respiratory infections (OR = 9.650, $p < 0.001$) than neonatal-aged infants. Infant mortality during the summer and autumn showed significantly lower odds for respiratory infections (OR = 0.150, $p < 0.001$ and OR = 0.060, $p < 0.001$, respectively) than in the winter. In other words, respiratory infections occurred over six times more often in the winter (1/0.150 = 6.67) than in the summer. As with the earlier period, the model for 1910–1912 suggests there is a strong link between dying of respiratory infections during the winter season and postneonatal infant age.

Table 3-32. Logistic Regression Predicting for Infectious Respiratory Cause Mortality, Infant Deaths (< 1 year), Hamilton 1881-1882

Independent Variables	**Coeff.**	**Sig.(*p*)**	**Exp(B)**	**S.E.**
Infant Age				
Postneonatal	1.694	0.000	5.439	0.397
Neonatal[R]				
Seasonality				
Spring	0.336	0.309		
Summer	-1.707	0.000	0.181	0.400
Autumn	-0.919	0.018		
Winter[R]				
Sample Size	513			
-2 Log likelihood	380.901			

Notes: Coeff. = Logistic Regression Coefficient, Sig. (*p*) = Significance Levels, Exp(B) = Odds Ratio, S.E. = Standard Error, R = Reference Category.

Table 3-33. Logistic Regression Predicting for Infectious Respiratory Cause Mortality, Infant Deaths (< 1 year), Hamilton 1910–1912

Independent Variables	**Coeff.**	**Sig.(*p*)**	**Exp(B)**	**S.E.**
Infant Age				
Postnatal	2.267	0.000	9.650	0.385
Neonatal[R]				
Seasonality				
Spring	-0.304	0.311		
Summer	-1.899	0.000	0.150	0.339
Autumn	-1.350	0.000	0.060	0.318
Winter[R]				
Sample Size	783			
-2 Log likelihood	514.038			

Notes: Coeff. = Logistic Regression Coefficient, Sig. (*p*) = Significance Levels, Exp(B) = Odds Ratio, S.E. = Standard Error, R = Reference Category.

HGIS: mortality analysis of infectious respiratory causes, 1910–1912

Seasonality and age also predicted infectious respiratory disease mortality. Cases of respiratory infections occurring in the winter are spatially presented in Figure 3-35 and Table 3-34. Winter made up 34.88% (*n* = 30) of the geocoded cases (*n* = 86). Summer and autumn had lower odds (when compared to winter) of predicting for infectious respiratory cause mortality. The pattern of seasonal flu epidemics occurring during the colder months is consistent with these diseases' typical wintertime seasonal trend (Fisman, 2012; Lofgren, Fefferman, Naumov, Gorski, & Naumova, 2007): overcrowded and poorly ventilated homes coupled with poor diet, weaken immune system, allowed for the spread of such diseases. While overall the northeast presented the highest number of cases (52.33%; *n* = 45), most cases that occurred in the winter were located in the northwest (50.00%; *n* = 15). Three clusters can be found in this quadrant: north by James Street, near the centre to the south, and in the northwest.

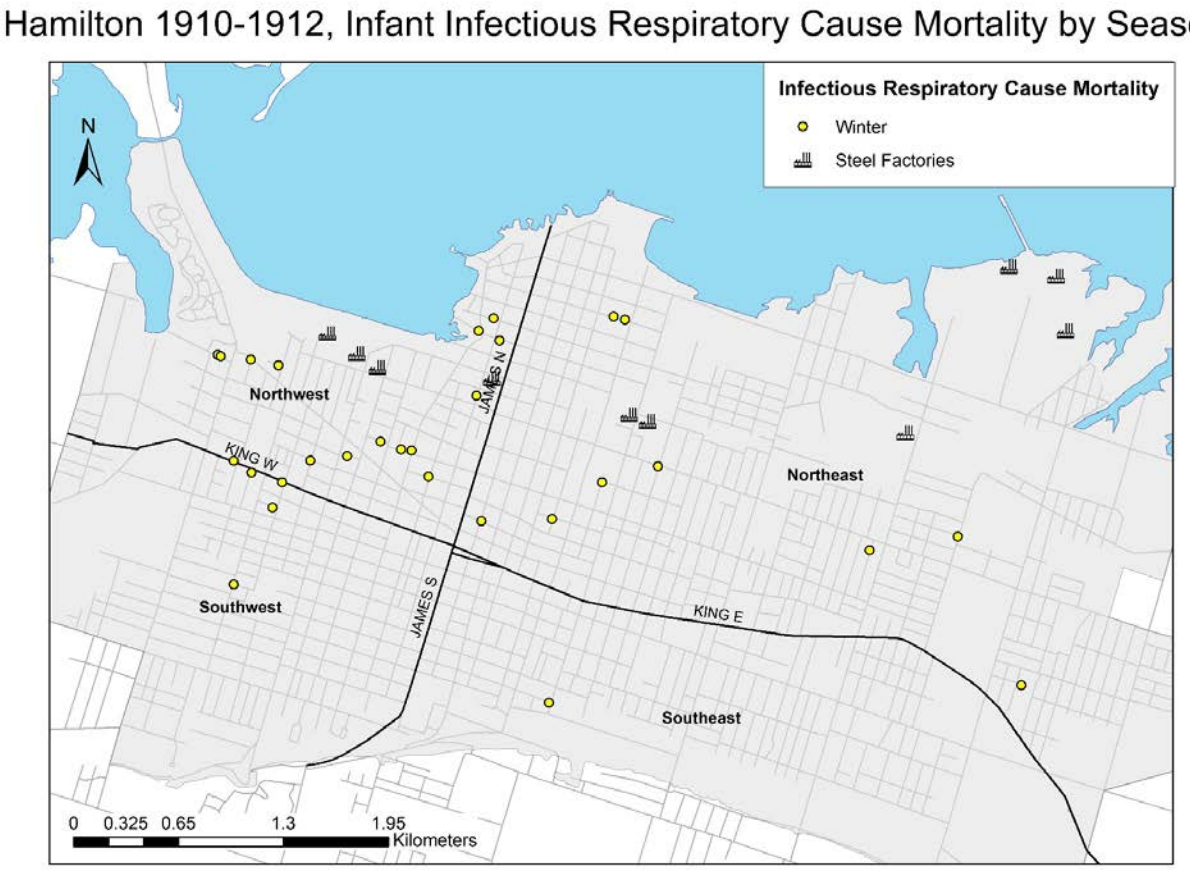


Figure 3-35. Infant Infectious Respiratory Disease by Season, Hamilton 1910-1912

Table 3-34. Infant Infectious Respiratory Disease by Season, Hamilton 1911

City Quadrant	**Cause of Death**			
	Infectious Respiratory Cause		Winter	
	%	n	%	n
NW	32.0	8	50.0	4
SW	8.0	2	12.5	1
NE	52.0	13	25	2
SE	8.0	2	12.5	1
Total	*100*	*25*	*100*	*8*

Spatial distributions were mapped for respiratory infections and age (Figure 3-36 and Table 3-35). Of all the geocoded cases, postneonatal age made up 91.86% (n = 79) of the sample. The majority of these cases occurred in the northeast and the northwest (53.16% and 34.18%, respectively). Clustering is less prevalent than the examination of winter cases. Most cases occur above King Street, reinforce the north/south divide, whereby both southern quadrants revealed only five cases each. The highest risk of complications for seasonal flu patterns occurs in children under the age of two years (World Health Organization, 2014a), and pneumonia in particular is known to have a postneonatal age association (Mulholland, 2007). Respiratory infections, such as pneumonia and bronchitis, are associated with infants who have been artificially fed at two months of age (Reid, 2002).

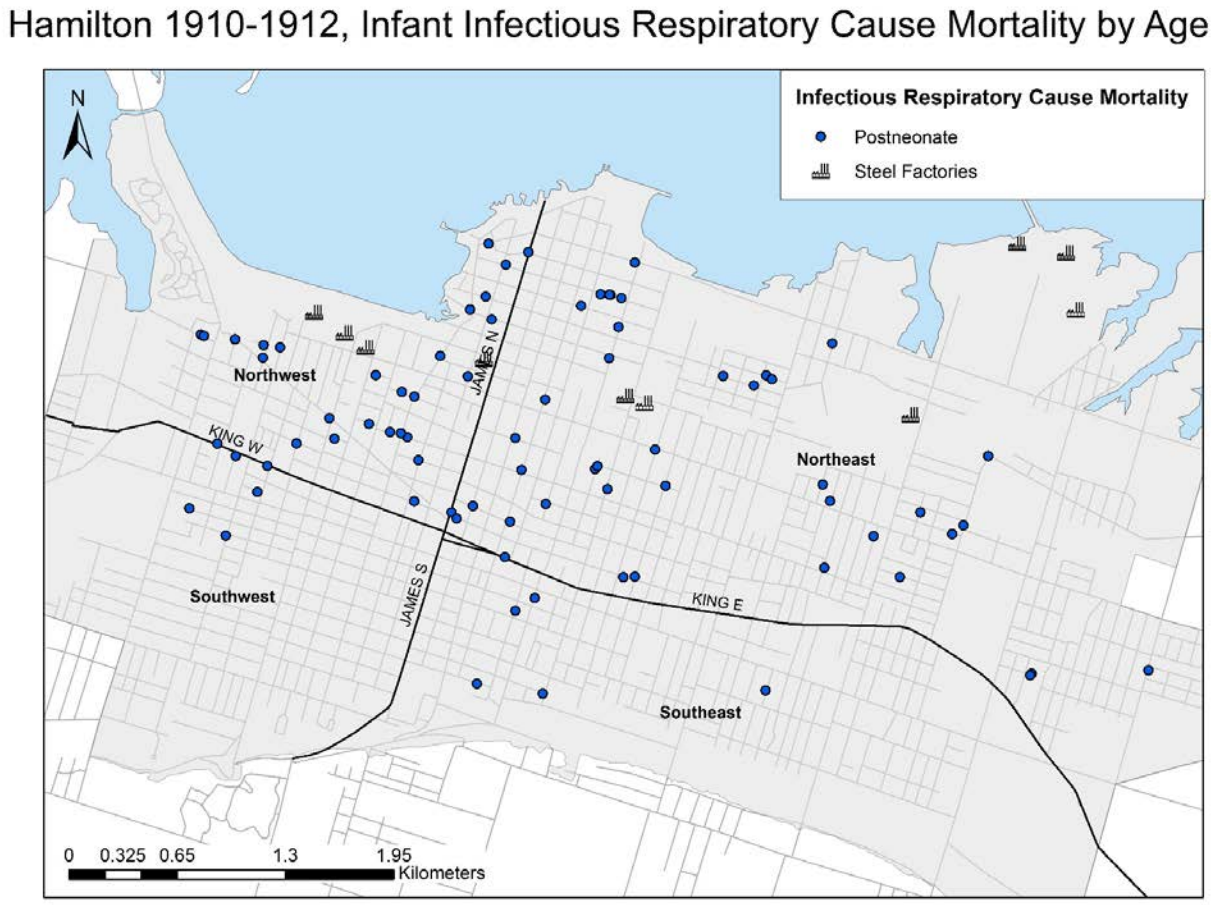


Figure 3-36. Infant Infectious Respiratory Disease Mortality by Age, Hamilton 1910-1912

Table 3-35. Infant Infectious Respiratory Disease Mortality by Age, Hamilton 1911

City Quadrant	**Cause of Death**			
	Infectious Respiratory		Postneonatal Age	
	%	n	%	n
NW	32.0	8	33.3	8
SW	8.0	2	4.7	1
NE	52.0	13	54.7	13
SE	8.0	2	8.3	2
Total	*100*	*25*	*100*	*24*

3.4.2.4 Synthesis: Infant Mortality

The logistic regression results for infant mortality did reflect some of the previous quantitative findings observed in this research. For both periods, 1880–1882 and 1910–1912, infant age and seasonality significantly predicted both diarrhoea and respiratory infections. Again, it is important to reiterate that logistic regression models control for the effects of other independent variables to assist in determining the true importance of an independent variable with respect to the dependent variables. For example, at both periods, the chi-square analysis statistically supported a link between diarrhoea mortality and infant age, as well as diarrhoea mortality and seasonality. Yet, the logistic regression model determined the true importance of these independent variables (age and season) at each given period. For the 1880–1882 infant sample, summer had the highest odds ratio, suggesting the likelihood that summer had a higher association with diarrhoea than the other independent variables, such as age, used in the model had. For the 1910–1912 infant sample, postneonatal age had the highest odds ratio, suggesting the likelihood that age had a higher association with diarrhoea than the other independent variables, such as the season, used in the model.

The relationship between weaning age, seasonality, and infectious disease is tied to the environment and behaviours. The predominate northeast spatial pattern for diarrhoea and northern pattern for respiratory infections help identify variations in the urban environment. Infant diarrhoea is highly associated with poor sanitation, poor water quality, the lack of breastfeeding/early weaning, and the quality of milk supply (Ferrie & Troesken, 2008; Sawchuk et al., 2002; Vaid et al., 2007). In the United States, infant mortality due to infectious respiratory diseases showed a higher peak in the winter months where unhealthy conditions and poverty persisted (Sakamoto-Momiyama, 1978).

Supporting evidence also points to behavioural practices' effect on infant mortality. While age is likely a reflection of the common causes associated with neonates and postneonates, some evidence suggests that infants artificially fed or hand fed from a very young age had an increased risk of death from both diarrhoeal and infectious diseases (Reid, 2001, 2002). A study conducted by Reid (2002) on 20th century infant feeding practices and neonatal mortality in Derbyshire, England, found that infants who were artificially fed at the age of one month were nearly 70% more likely to die postneonatally. Whether the choice to artificially feed was caused by sibling competition, feeding methods, or maternal depletion, infants would have been greatly at risk for exposure to gastro-intestinal pathogens (Reid, 2002). Currently, it is suggested that exclusive breastfeeding for the first six months of life is a best practice (World Health Organization, 2011).

3.5 Qualitative Inquiry of Health in Hamilton, 1880–1882 and 1910–1912

A variety of historical documents were accessed to provide a qualitative investigation of life in Hamilton as it pertained to health concerns. There are no annual health reports for the initial years of this study; however city council minutes from that time present information on urban development and health issues (City of Hamilton Council, 1878-1882). By the later period, the city had its own board of health and reported annually on health issues facing the population (Bowman & City of Hamilton, 1912). As well, a number of images located at the Local and History Archives in Hamilton illustrate the extent of the health concerns faced (Lamb, 2014-2015; Local History and Archives, n.d.). While much of the textual information was used in describing the background of Hamilton's history, this section will present key explanations found in these documents and analyze the images to illustrate the conditions that would have

affected the population, supporting that a socioeconomic shift towards industrialization likely exacerbated health inequities in Hamilton.

3.5.1 Descriptive Text Analysis: State of Urban Health

The rapid rise in industrial activity in Hamilton increased spatial class segregation and, at a time when urban areas were unhealthy environments, led to a constant struggle for the city to provide its population with basic necessities like housing, water supply, and adequate sewerage systems. Even prior to industrialization, Hamilton had chronic public health issues pertaining to inadequate infrastructure and unhealthy practices of the population. Documents from the study period emphasize these issues, raise concerns about the health of the population, and state that specific populations were likely at higher risk for disease susceptibility.

Information obtained from the city council minutes raised concerns about problems of sewage and water contamination, specifically noting the east end not having a functioning sewer system in place. As noted in the 1880 minutes, cost was an inhibiting factor for completing construction of the system (City of Hamilton Council, 1878-1882). Thus, in the east end of Hamilton in the 1880s, nightsoil collection remained a common method for removal of human excrement from residences, whereby men were hired to go around the city to collect waste from privies and water closets (City of Hamilton Council, 1878-1882). While the west end sewer was nearly completed by 1879, the Sewer Committee stressed the importance of completing the city's sewer system as they found that many residents would tap into the sewer without permission (City of Hamilton Council, 1878-1882). The committee requested a by-law to forbid persons from tapping or connecting with any common sewer without permission to prevent damages to the pipe (City of Hamilton Council, 1878-1882). Although illegal tapping into the sewer system was a concern, the size of the sewer pipes was also becoming a larger issue: in 1882,

the Sewer Committee noted that the current sewer pipes were too small to allow for the necessary amount of water to flush out the pipes (City of Hamilton Council, 1878-1882), suggesting that the city was beginning to outgrow the sewer system, but cost continued to hinder the construction of larger sewer mains. The inadequately sized system could cause poor outflow, system backup, and sewage overflow. When such systems are not improved or maintained, the bursting of pipes and the potential sewage overflow can increase water contamination (Deere, Stevens, Davison, Helm, & Dufour, 2001). The use of privies was another environmental concern in Hamilton: without connections to the sewer or water systems, the potential for cross-contamination with a household's water supply could have increased the risk of exposure to particular infectious pathogens.

In the earlier period, concerns regarding the water supply were also raised in the city council minutes. Although the Board of Water Works reported positively in that the city had 'fairly good' water access for the population—attributed to the construction of the water pumping engines and reservoir—some areas of the city continued to receive water by wagons and carts (City of Hamilton Council, 1878-1882). Which areas of the city were still receiving water in this manner was not discussed, however. Stagnant water and problems with drainage were also reported in the council minutes. Two causes for the stagnant water problem were reported: the lack of proper infrastructure for the water supply and residents' practices of discarding water on the city streets (City of Hamilton Council, 1878-1882). Additionally, wealthier areas may have been supplied water-system access or access to the water main may have been financially easier to access. The Board of Water Works in the 1880 minutes noted the need to construct water mains and a hydrant to supply several "first-class" residences being built around Herkimer and Markland streets, and to extend to homes where the homeowner had the

necessary pipe to hook up to the main line (City of Hamilton Council, 1878-1882). Onus was on the homeowner to supply the pipe, therefore, accessing the main would likely have been financially prohibitive for poorer residents, even if they lived in an area with a water main.

Public health concerns were also raised in provincial health reports and locally in the city council minutes, some of which align with the issues described above on urban infrastructure. The Provincial Board of Health's 1884 report on public health concerns, presented to the Hamilton Literary Association (The Legislative Assembly of Ontario, 1884), detailed issues related to sewer gases in homes caused by improper drainage and the occurrence of diarrhoea caused by poor food handling, contamination of water, unhealthy cow stables, unclean milk cans, and sewer seeping from privies into wells (The Legislative Assembly of Ontario, 1884). The Board of Health expressed the need for preventative measures at multiple levels: individual (personal cleanliness and cleanliness of clothes), household (cleanliness and ventilation), private grounds (filth in yards), and in public sanitation (removal of excreta and the adoption all necessary sanitary precautions) (The Legislative Assembly of Ontario, 1884). The concerns raised during this period may have prompted healthy city initiatives to prevent infectious disease.

Although the reports from the earlier period reveal awareness of the issues that affected the health of Hamilton's population and indicate that some areas lagged in adequate infrastructure, similar issues persisted during the later period. The rapid population influx due to the industrial activity may have exacerbated these health inequities. Reports from the later period state that there continued to be a lack of sewerage and water supply infrastructure, mostly in the industrial districts of the city (Bowman & City of Hamilton, 1912; Roberts & City of Hamilton, 1910). The 1909-1910 Board of Health reported on the need for better enforcement

concerning issues with the construction, cleaning, and disinfecting of dry earth closets (Roberts & City of Hamilton, 1910). Throughout America and England in the 19th century, the dry earth closet system (using dry earth to absorb human excrement) was seen as an improvement to the cesspools and privies in use by those without water and sewerage supplies (Sipe, 1988). This improvement, however, was mostly aesthetic: the dry earth closet deodorized but did not disinfect, drain, dilute, or dispose excreta. Thus, they still presented a risk of contamination with water supply (Knight, 1910; Province of Ontario, 1883; Sedgwick, 1902; The Legislative Assembly of Ontario, 1884). The report notes that individuals were "… forced to rely on this primitive method for disposal of excreta" (Roberts & City of Hamilton, 1910, p. 21) due to the lack of sewerage in the rapidly growing manufacturing district.

The 1909-1910 Board of Health report noted significant issues with the waste systems at the time. Sanitary inspections of premises found 132 defective sewer connections and 960 offensive privy vaults (outhouses, pit toilets) (Roberts & City of Hamilton, 1910). Additionally, 686 notices were served to abolish privy vaults; 211 to have privy vaults cleaned; and 130 regarding defective sewer connections (Roberts & City of Hamilton, 1910). Contractors were issued permits to abolish privy vaults (466) and to require houses be connected to the sewer system (483) (Roberts & City of Hamilton, 1910). The 1911-1912 report notes that there was a total of 18,260 houses in the city (Bowman & City of Hamilton, 1912). Of the 20,271 sanitary inspections conducted during 1911-1912, 706 (3.87% of total households) inspections found offensive privy vaults and water closets; 240 (1.31% of total households) inspections found unsanitary and overcrowded houses; and 330 (1.81% of total households) inspections found choked and defective sewer connections. Notices were also served to 141 households (0.77% of total households) for unsanitary defective water closets, 428 (2.34% of total households) for

manure accumulation, and 187 (1.02% of total households) for stagnant water on lots. In addition, 693 orders (3.80% of total households) given to abolish privy vaults and water closets. These efforts were considered to be inadequate by the health community. The medical health officer concluded "that a drastic wholesale condemnation [destruction] of all premises lacking in some essential features of sanitation" was required because of the unsanitary nature of these dwellings (Bowman & City of Hamilton, 1912, p. 23).

The percentages presented above seem low compared to the total number of households; however, annual health reports suggest that these problems persisted mostly in the industrial working-class sections of the city (Bowman & City of Hamilton, 1912; Roberts & City of Hamilton, 1910). As such, these issues are likely clustered in the northeastern section of Hamilton. The homes found in the industrial section of Hamilton were reported to have been overcrowded and unhygienic (Roberts & City of Hamilton, 1910). The medical officer for the year 1911-1912, Bowman, reported that the inspection of homes in sections of the city where there was an influx of artisans and labourers, showed that these areas lacked sufficient accommodation to support the demand (Bowman & City of Hamilton, 1912). These inspections identified a lack of sanitary practices that may have greatly increased health risks to the population. For example, the water supply, especially water that came from private wells, which are especially susceptible to contamination (such as through sewer overflows) (Centers for Disease Control and Prevention, 2011a). Additionally, overcrowding in these areas along with the poor infrastructure would have likely increased exposure to infectious pathogens.

In the later period, concerns related to infectious disease control and prevention were raised in the heath reports. The 1909-1910 annual report speaks to the progress of public health in the city, but acknowledges that the rapid population increase was detrimental to infant health

(Roberts & City of Hamilton, 1910). The city blamed the high infant health risk on feeding practices related to a wide variety of factors, including the poor quality of cow's milk, "the maternity problem" (the lack of education for maternal and paternal responsibilities), poverty, housing, the environment, and social life (relationships, behaviours, and actions) (Roberts & City of Hamilton, 1910). Roberts, the medical health officer for 1909-1910, suggested creating Women Health visitors, who would visit poor homes, instruct women on care and feeding of children, distribute literature on sanitary matters, and aid in cleanliness and health practices (Roberts & City of Hamilton, 1910). Ongoing progress during this period was made by providing better quality milk to the population through increased dairy inspections (Bowman & City of Hamilton, 1912; Roberts & City of Hamilton, 1910); however, even with these attempts, unequal access to pasteurized milk remained an ongoing concern in urban cities. Bowman also provided recommendations designed to relieve the congestion found in the industrial districts: providing municipal housing, developing rapid transit to decentralize industry, preparing and enforcing a housing by-law, maintaining sanitary conditions of homes through supervision, and encouraging the wealthy class invest in charitable organizations for the city (Bowman & City of Hamilton, 1912).

During both periods in time, the lack of urban infrastructure was seen as having a detrimental effect on the health of the population. By the later period however, the health concerns were more pronounced as evident by the city's inability to support its rapidly growing industrial class via proper urban development. This inability intensified health inequity. These reports, especially in the later period, indicate that environmental and behavioural causal factors may have influenced the mortality patterns found in the previous mortality analyses.

3.5.2 Image Analysis

Various images were collected from the Local History and Archives in Hamilton. Permission to use photos from the periods of this study was confirmed by one of the archivists as they are within the public domain and property of the Archives (Hamilton Public Library, n.d.; Lamb, 2014-2015). This section will provide an analysis of the images collected to examine the growth, industrialization, and the public health issues of Hamilton.

The eastern outer limits of Hamilton prior to industrialization were mostly agricultural land, allowing for industrial expansion east, as shown by the following map and accompanying image (Figures 3-37 and 3-38). The image faces northeast, as indicated by locational markers (it was taken from the escarpment, west of the street labelled 'Wentworth', showing inlets along the shoreline and Hamilton beach in the horizon). This area illustrates that the east was sparsely populated: there does not appear to be any urban growth or signs of industry, but instead the landscape is laden with farming plots and bluffs of trees.

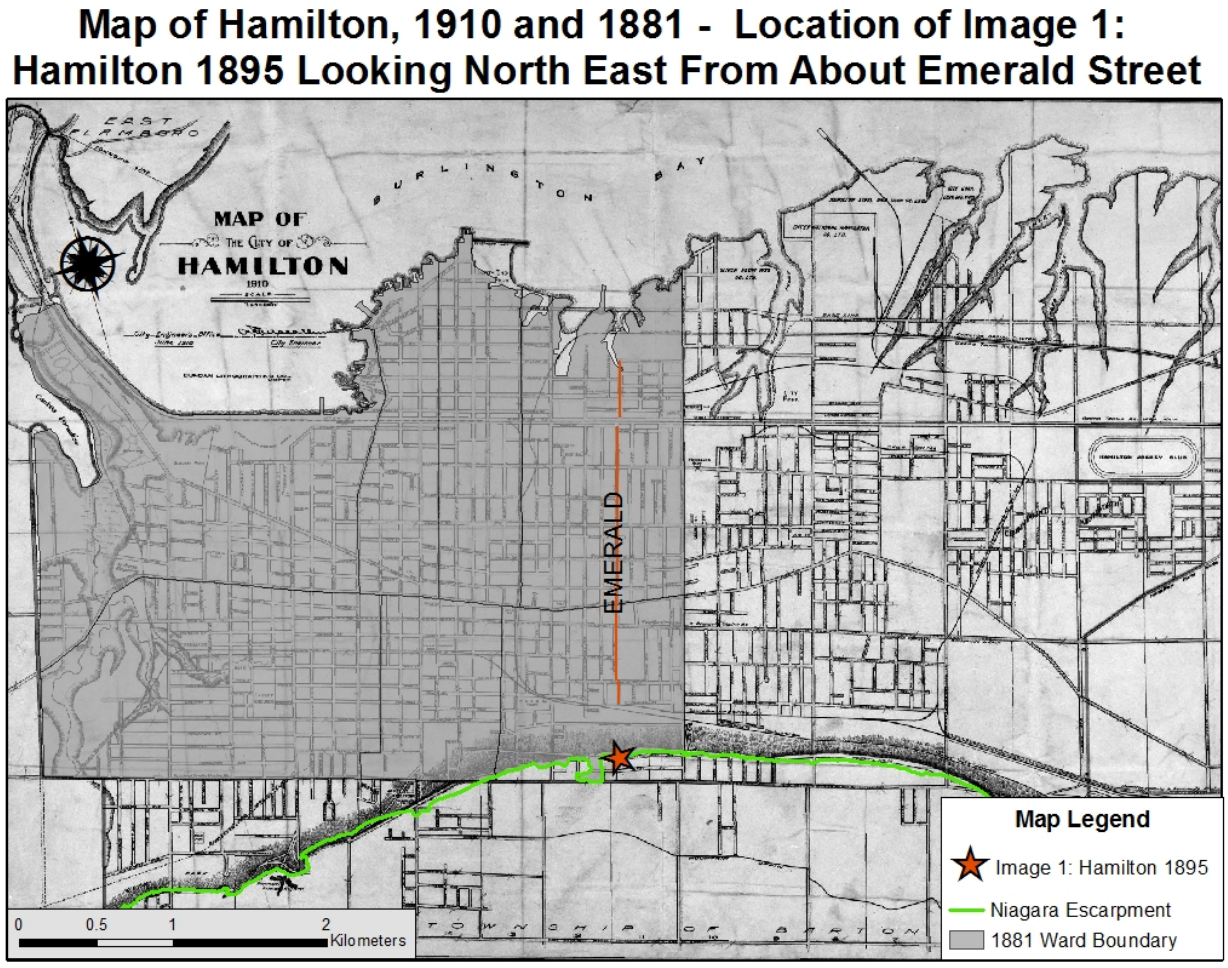
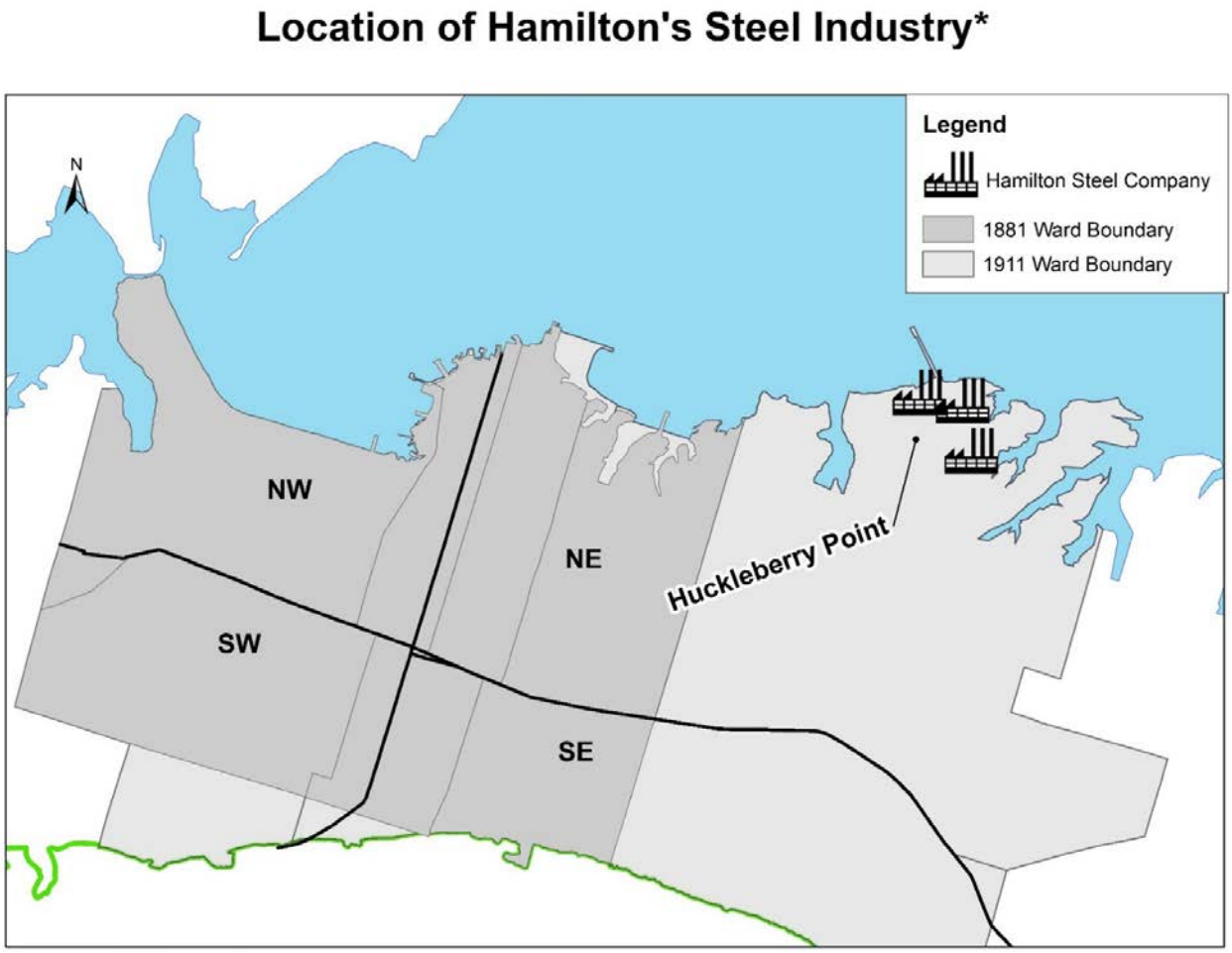


Figure 3-37. Map of Hamilton 1910 – 1881 and Location of Image Looking Northeast from About Emerald Street

Images courtesy of the Hamilton Public Library, Local History & Archives

Figure 3-38. Hamilton 1895 - Looking Northeast from About Emerald Street

The firing of the first blast furnace in 1895 marked Hamilton's transition towards a predominately industrial economy; thus, the first foundry would have been an important aspect in the city's industrial history and urban geography (Figure 3-39). The drawing of Hamilton's first foundry, Hamilton Blast Furnace Company, shown below, reveals the humble beginnings of an industry that would lead Hamilton's economic prosperity in the 20th century (Figure 3-40). As the image illustrates, Hamilton's first foundry was a small independent operation owned by McQuesten and Co. as titled under 'Hamilton Iron Works' in the image. This first foundry and other small independent iron works amalgamated to form The Hamilton Steel and Iron Company, which became, by 1910, the Steel Company of Canada (Stelco) (Heron, 1988). This



*Hamilton's first foundry was in the same location as its predecessor, Hamilton Steel Company, which amalgamated with neighbouring companies to become the Steel Company of Canada in 1910.

Figure 3-39. Location of Hamilton's Steel Industry

first foundry at "Huckleberry Point" became the location of Stelco's works (Library and Archives Canada, n.d.). An aerial image of an industrial area of Hamilton circa 1910 shows Huckleberry Point as the geographical location of industry (Figure 3-41). The image is centred mostly on the International Harvester Corporation in the near left part of the image and the Stelco to the right front and back of the image. There are a number of houses situated in front of International Harvester, these are possibly company housing, but may also be owner-built. Within a 15 year span (1895-1910), the northeastern section of Hamilton altered immensely from an agricultural landscape to an industrial landscape. This rapid industrialization

heavily imped the city's ability to supply adequate public health infrastructure to its growing population.

Images courtesy of the Hamilton Public Library, Local History & Archives

Figure 3-40. Hamilton's First Foundry, 1889

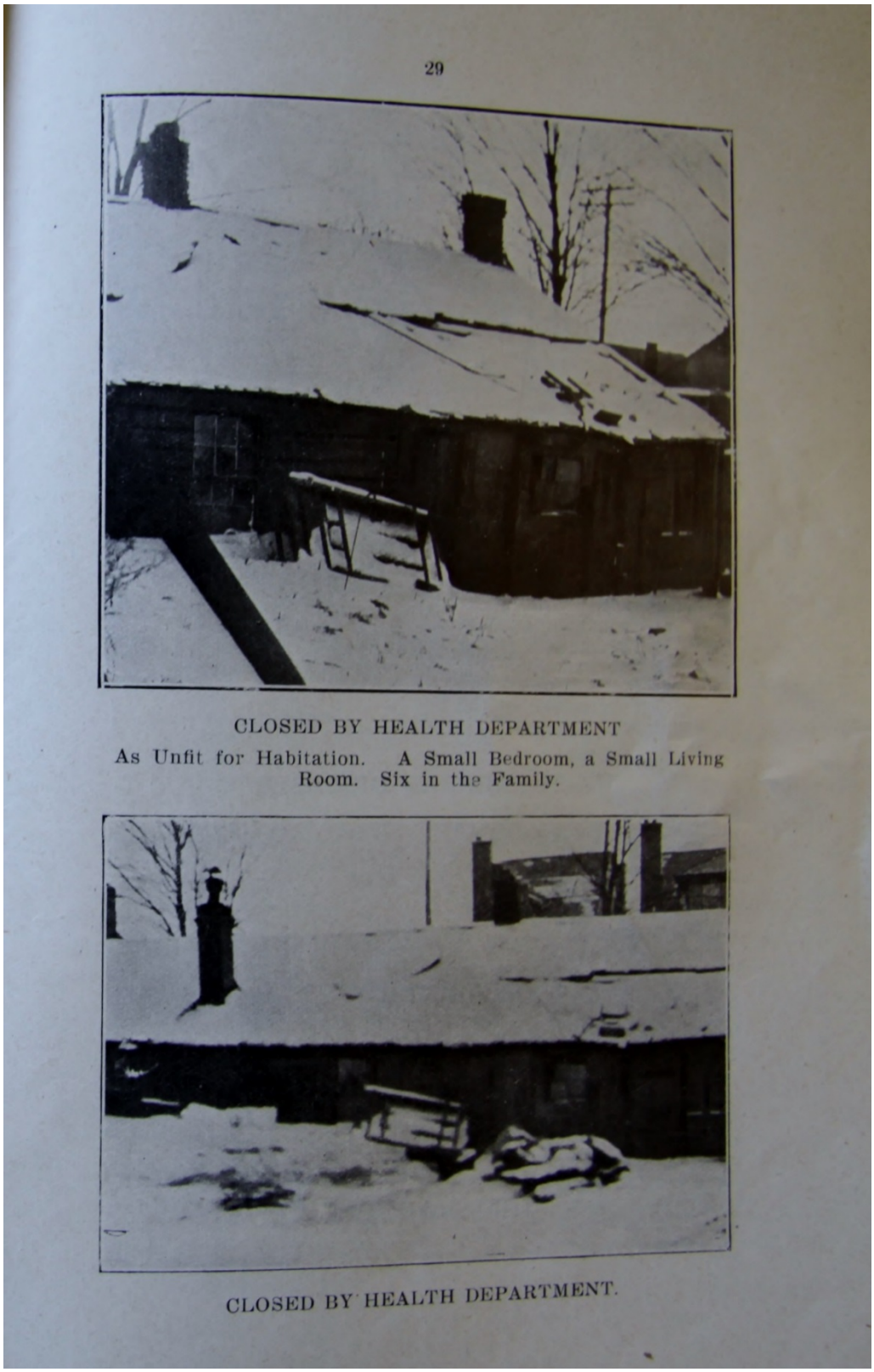
Images courtesy of the Hamilton Public Library, Local History & Archives

Figure 3-41. Industrial Hamilton, circa 1910s (facing northeast), International Harvesters Corporation (front left) and Stelco (Back right)

Housing shortages in the industrial working-class neighbourhoods of Hamilton created subpar living conditions. The Board of Health inspected homes and, in the industrial sector, closed them because of the state of the living conditions (Figures 3-42 and 3-43) (Roberts & City of Hamilton, 1910). Figure 3-42, taken from the 1910 health report, provides details of a small one bedroom house and number of persons living in it (a family of six) to support the overcrowded nature described in said reports (Roberts & City of Hamilton, 1910). The image in Figure 3-43 showed the dismal conditions in a basement occupied by a family of twelve. The caption underneath the photo points to the formation of icicles on the roof and ice on the floor, and indicates that the plumbing was faulty. Although the ice and icicles may indicate defective plumbing, they also indicate the lack of heating that allowed such ice to form in the home. In addition, the image also depicts the overall unsanitary nature of the room. While the images

presented in the health reports are likely more extreme scenarios, textual descriptions of the time note that overcrowding, poor housing, and inadequate water and sewer systems were common features of Hamilton's urban environment, and furthermore features that had a spatial pattern.

Another public health issue illustrated through imagery was the presence of unsanitary cow barns. These barns presented a heightened risk for the consumption of low quality raw milk (Figure 3-44) (Roberts & City of Hamilton, 1910). The comparison used by Roberts, in Figure 3-45, suggest that both stables were used for milk production (note also the large udder on the unsanitary cow, which suggests that the cow was ready to be milked). Both dairy and beef cattle were being tested for bovine tuberculosis and beef tagged as *condemned* due to tuberculosis was identified in carcasses (Figure 3-45 and Figure 3-46) (Roberts & City of Hamilton, 1910). The term "certified milk" in Figure 3-44 suggests a trend towards implementing higher quality milk for consumption; however where unsanitary cow barns remained, lower quality dairy and beef products would be available and likely at a more affordable cost than products that were certified. A milk cart in Hamilton, circa 1910s, was used to deliver what was termed *pure milk* to the city (Figure 3-47) (Roberts & City of Hamilton, 1910), suggesting the continued use of terms like *pure* and *certified*.



29

CLOSED BY HEALTH DEPARTMENT

As Unfit for Habitation. A Small Bedroom, a Small Living Room. Six in the Family.

CLOSED BY HEALTH DEPARTMENT.

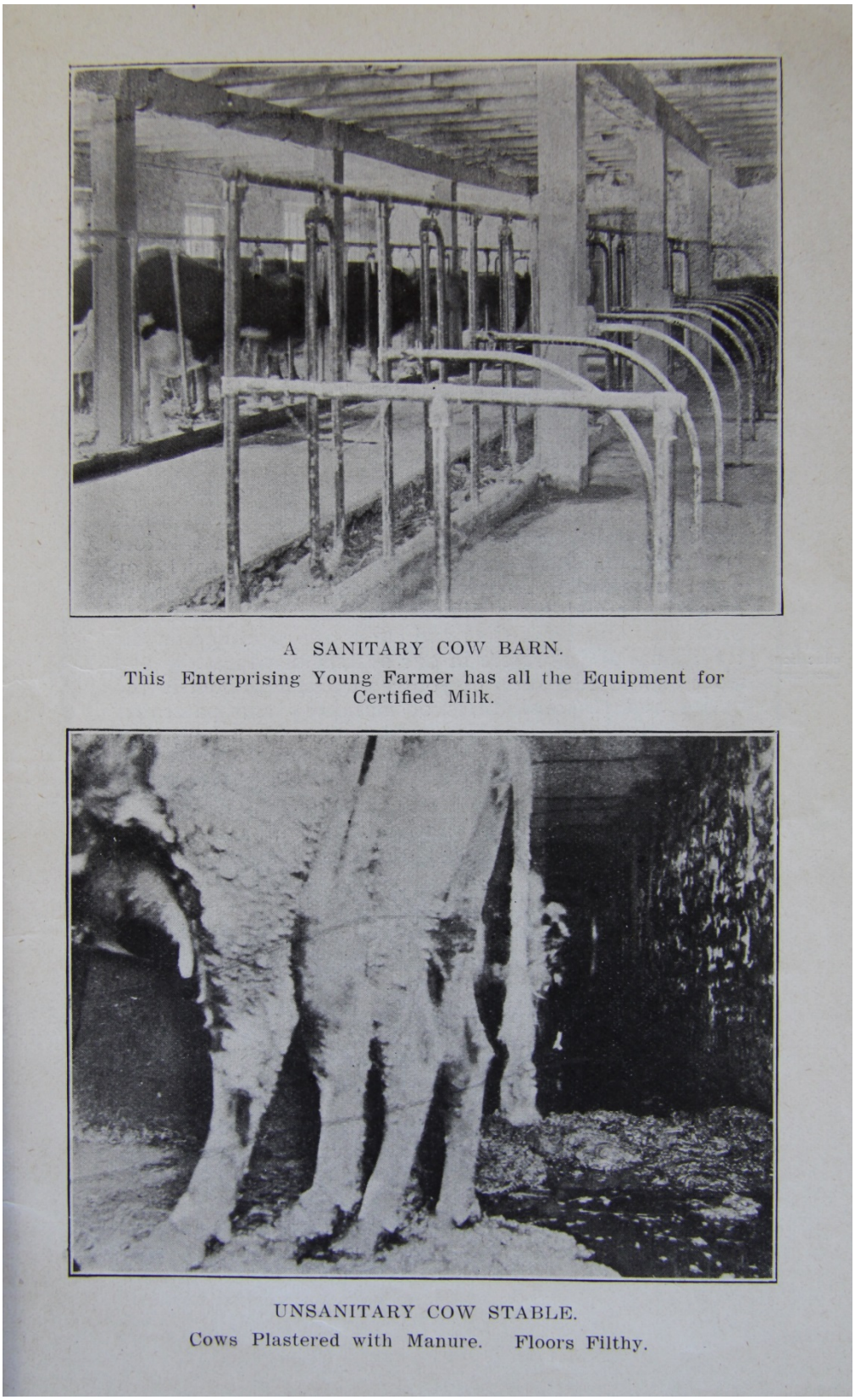
Images courtesy of the Hamilton Public Library, Local History & Archives

Figure 3-42. Hamilton 1910 Board of Health–Condemned Home (Roberts & City of Hamilton, 1910)

A Family of Twelve Occupied this Basement and Two Small Rooms. Note Icicles from Roof and Ice on Floor. Plumbing Bad.

Images courtesy of the Hamilton Public Library, Local History & Archives

Figure 3-43. Hamilton 1910 Board of Health–Condemned Home (Roberts & City of Hamilton, 1910)



A SANITARY COW BARN.

This Enterprising Young Farmer has all the Equipment for Certified Milk.

UNSANITARY COW STABLE.

Cows Plastered with Manure. Floors Filthy.

Images courtesy of the Hamilton Public Library, Local History & Archives

Figure 3-44. Sanitary Concerns about Cow Stables (Roberts & City of Hamilton, 1910)

Images courtesy of the Hamilton Public Library, Local History & Archives

Figure 3-45. Sanitary Concerns about Cow Stables (Roberts & City of Hamilton, 1910)

CARCASS OF BEEF.
Plura Very Badly Affected with Tuberculosis.

Images courtesy of the Hamilton Public Library, Local History & Archives

Figure 3-46. Condemned Beef Carcass due Infection of Bovine Tuberculosis (Roberts & City of Hamilton, 1910)

Images courtesy of the Hamilton Public Library, Local History & Archives

Figure 3-47. Milk Cart in Hamilton (circa 1910s), Frank Woods Collection

3.6 Conclusions

The results presented provided information on demography and mortality for the city of Hamilton at two periods of time: 1880–1882 and 1910–1912. As well, a further in-depth analysis examined the characteristics of the mortality samples, a spatialization of these characteristics, and the purported health issues the people of Hamilton faced. The iterative process of analysis expanded on the descriptive statistics to build the story of life and death.

Demographic variations shown here were likely a reflection of the changing socioeconomic structure of Hamilton and the natural progression of urban growth. The rapid population growth and subsequent changes to occupational structure suggest the transition to

heavy industrialization was swift. This swift transition would have had substantial implications to the state of urban health if Hamilton was not equipped to handle the influx of people and necessary development. The demographic analysis set the stage to begin examining the mortality profile of the city over time. The mortality analysis moved from high level descriptive towards understanding key independent variables that inform more in-depth inferential statistics (logistic regression), providing insights into the individual influences that led to the mortality outcomes. Overall, crude mortality rates were declining, however shifts were occurring in what was causing death, such as the increase in accidents in the working ages and nutritional causes amongst infants. Further chi-square tests revealed correlations between major causes of death and independent factors such as age, sex, birthplace, occupation marital status, and season. The approach taken to narrow down the causes of death allows for multiple causes of death to be examined in order to respond to the purpose of the study. These results present the first component for the mixed methods analysis by providing the elements required to build a profile of life and death in the city of Hamilton.

Although limitations in the availability of ward-level demographics impeded the HGIS, the distribution of deaths reveal a pattern that was further supported by health reports and infer spatial health inequities in industrial districts. This inference towards spatial health inequities, even in light of the limitations in the data, suggests that the north and, more specifically, the population northeast was by then at heightened risk with respect to infectious disease and accidental causes. The qualitative inquiry into Hamilton supports the initial hypothesis that the urban environment was not prepared to handle the growing populace and these conditions were unevenly distributed across the city. Integration of methods enhanced evidence that there were likely multi-level factors (e.g. individual, environmental, and habitual) at play.

CHAPTER 4
DISCUSSION

4.1 Introduction

The explanatory sequential model for mixed-method analysis has a strong quantitative orientation supplemented by additional methods for a more in-depth explanation into the research problem, in other words, to build upon the story presented in the research. The purpose of the discussion, then, is to expand on the information and concepts found in this research to provide further explanation of the changes in urban mortality that occurred over time. In doing so, the discussion is presented first by reintroduction to the healthy cities perspective and human health ecology as the fundamental theoretical foundations of this research, which is then followed by a discussion of each respective research question.

4.2 Healthy Cities: Discussion of the Findings

Studies of the community environment have a longstanding history of linking the social sciences and public health perspectives to allow for movement beyond the traditional individual disease-centred approach towards a more complex systems approach (Diez Roux, 2011; Meade & Emch, 2010; Sampson, 2003). Theories within health ecology align with conceptual approaches to healthy communities because the broader theoretical lens of human ecology is grounded in spatial organization and the human-environment relationship (Ashton, 2009; Meade & Emch, 2010; World Health Organization, n.d.-b). Thus, the healthy community environment, or, rather, the lack thereof, can have a great impact on health disparities and patterns in mortality, placing importance on the need for a geographical lens.

Variations in morbidity, mortality, and health-related behaviours that occur in small areas often carry two plausible explanations: compositional or contextual (MacIntyre & Ellaway, 2003). Compositional explanations examine the differences observed in the population that lives in a particular area (MacIntyre & Ellaway, 2003; Macintyre, Ellaway, & Cummins, 2002; Meade & Emch, 2010). On the other hand, contextual explanations examine both the area effects that occur within a small area and the local physical and social environments (MacIntyre & Ellaway, 2003; Macintyre et al., 2002; Meade & Emch, 2010). Contextual causal variations are likely the result of variations in the built and physical environment, while compositional causal variations are likely the result of differences that occur in the social and behavioural environment. The combination of both contextual and compositional causal explanations enables the use of a meaningful framework to identify the presence of multiple levels of organization in an area and the factors that influence health outcomes (Diez Roux, 2011).

Because of cities' heterogeneous natures, the growth of a city can exacerbate inequalities between areas and people and lead to health inequities. Examining urban population health using mortality and mortality odds ratio, coupled with health reports, presents an opportunity to understand the risks that led to health outcomes. The mortality odds ratio characterizes who was dying, which, when combined with GIS and health reports, reveals where people in the sample lived prior to dying, and gives insights into environmental and behavioural factors of the urban area and population. This next section discusses the findings by research questions to support the overarching purpose of this research.

4.2.1 Research Question 1: What are the socio-demographic profiles in 1881 and 1911 Hamilton, and how did the profile change over time?

Prior to examining mortality patterns in a population, it is important to understand the population at risk to better assess potential compositional variations. The composition of the population is an embedded component of this research. Thus, in order to better understand any spatial differences in the mortality patterns, key demographics were identified of the population in Hamilton during the study period, primarily by analyzing data in the published census records (Canada Census and Statistics Office, 1911; Canada Department of Agriculture and Statistics, 1882; Department of Trade and Commerce, 1915).

Over the course of the study period, Hamilton's population grew 128%, with an annual growth rate of 4.27% between 1881 and 1911. This growth was not even throughout, however: the greatest increase occurred between 1901 and 1911 when the population grew 55.79%, with an annually growth rate of 5.5%. During this period, the sex ratio among the working ages also changed from a population structure in 1881 in which working-age females outnumbered males (90.15 males per 100 females) to one in 1911 in which working-age males outnumbered females (108.72 males per 100 females). This reversal was the result of Hamilton's transition into a predominately industrial economy associated with the emergence of the steel industry after the 1890s (Middleton & Walker, 1980), an economy in which men participated in far greater numbers than women.

While industrialization may lead to increased immigration due to the demand for unskilled labour work (see, Crawley, 1988; Gagnon et al., 2011; Hannon, 1984; Zunz, 1982), the foreign-born population in Hamilton, however, did not show a substantial increase between 1881 and 1911, as the data in a special report on the foreign born population in Canada revealed (Department of Trade and Commerce, 1915). Instead, there was only a modest, 2.59% increase

in immigration over the 30-year period in time. The origins of the population changed slightly: between the 1881 and 1911 censuses, the population from the British Isles (English, Scottish, and Irish) decreased while population from other countries increased. This may have been related to increased industrialization, but it may also reflect the ebb and flow of Canadian immigration waves. There was a geographical shift at this time in the amount of people moving to Canada from other parts of Europe (Avery, 1979; Heron, 1988; Heron & Storey, 1985).

The numerical impact of increased immigration, however, may have been offset by the nearness of other communities to Hamilton that might have surplus labour. In particular, Hamilton is located close to Toronto, and several smaller communities that have since been absorbed by the Hamilton regional municipality. This relative location may have provided the city with access to a large, readily-mobile, Canadian-born population in southern Ontario that would have been attracted to Hamilton's growing industrial wealth. Industrial towns or cities are often developed near the source of raw material, creating an agglomeration of industrial activity. This spatial fixity of resources can isolate an industrial centre. Hamilton is not located near the resources needed to produce steel (coal and iron), but it benefited due to its strategic positioning on Lake Ontario and its deep-water harbour. This location allowed for transportation of the necessary resources from the United States and other parts of Canada into Hamilton (Doucet & Weaver, 1984; Middleton & Walker, 1980; Nader, 1976). Being a port city meant that Hamilton could also import other raw materials for production (e.g. rubber, tobacco, textiles), which allowed it to become an industrial giant without being located near required resources. Hamilton was a manufacturing city, not an extractive one. Extractive cities or towns are often dominated by a single industry, and are the product of economic pioneering; thus the city/town is often governed by the company involved in the industry (Porteous, 1970). Manufacturing cities

or towns, on the other hand, are often a product of socioeconomic forces (Porteous, 1970). Hamilton, as a manufacturing city, presents a different sociopolitical dynamic than an extractive city would, as an already well-established city that transitioned towards an industrial economy instead of one that developed based on a particular resource.

Between 1881 and 1911, female occupation shifted (Canada Census and Statistics Office, 1911; Canada Department of Agriculture and Statistics, 1882). In the 1881 census, 66.7% of females worked in domestic and related occupations and the top five occupations made up 98.04% of female occupations. By 1911, however, more diverse opportunities were available to women. Clothing production became the top female occupation but had only 20.73% of the total employment because women worked in a wider range of jobs. The top five occupations made up 64.58 % of female occupations. Females born in Canada made up 70.63% of females working in Hamilton during the 1911 census year.

Male occupations also shifted; however, this difference was not as distinct. The occupational shift among males supported the notion that an economic shift had the potential to change the demographic structure and health of a city. Labourers and related workers remained the top occupation during both periods, but the second-most occupation in 1881 was clothing production (17.35%) while, in 1911, it was metal processing (11.73%). Immigration reports from the Hamilton Agency in 1881 note concerns in obtaining skilled operatives in cotton and wool millworks to meet the demands in the new clothing-based industries in Hamilton (Commissioner of Immigration, 1882). Although the 1881 census did not provide occupation by nativity, the 1911 census showed that there may have been some differences in attaining higher income occupations (Canada Census and Statistics Office, 1911; Canada Department of Agriculture and Statistics, 1882). Analysis of the census revealed that a high percentage of

males working in professional (69.81%) and clerical (64.50%) occupations were born in Canada. Conversely, labourers and related occupations were filled by a high percentage (64.42%) of immigrant males. This research reveals that the onset of the steel industry at the turn of the 20th century caused a shift in demographics in Hamilton.

4.2.2 Research Question 2: Do these socio-demographic patterns reflect the temporal urban and economic development of Hamilton?

Examining the urban and economic development of Hamilton helped identify the mixture of contextual and compositional factors found in health ecology. The unique social fabric of a city influences the construction of its distinct urban structure (Gilliland, 2002). Hamilton, throughout its history, showed spatial population differences—a manifestation of the city's urban development—from early strategic planning, which was boosted by its developers to become Canada's "Steel City". Hamilton continually presented the spatial segregation of its residents. As early as the 1830s, the south and west sections of the city were predominantly home to the wealthy, while the north and east sections housed the destitute and were seen as inferior (Cruikshank & Bouchier, 2004; Doucet & Weaver, 1984; Herring & Korol, 2012).

4.2.2.1 Pattern of urban development: pre-industrialization to industrialization, 1881–1911

Continued urban growth and development occurred as Hamilton transitioned towards steel-dominated industry, even as spatial segregation increased in the city with the rise of working-class neighbourhoods. Hamilton's urban development took a dominant eastward shift, according to Harris and Sendbuehler (1992). Image analysis supported this eastward shift, revealing the path of industrial patterns. In the 1880s, the eastern outer limits of Hamilton were predominately agricultural land (see Figures 5-10 and 5-11). Yet, by the industrial period, the

northeastern portion of the city around Huckleberry Point became the centre of industrial activity in the city (see Figures 5-12 – 5-13). This shift caused a north-south divide in terms of socioeconomics and demographics, as Herring and Korol (2012) found. The open landscape in the east and the proximity to the lake for transport and processing in the north allowed for the expansion of industrialization and working-class neighbourhoods in the northeast (Cruikshank & Bouchier, 2004; Harris & Sendbuehler, 1992; Nader, 1976). Breaking the city into quadrants (SW, SE, NW, NE) revealed the spatiotemporal pattern of urban development that expanded northeast with an industrial agglomeration and urban health patterns.

While Hamilton continued to urbanize prior to the onset of steel manufacturing, this growth went through ebbs and flow related to neighbouring competition in commercial activity and economic downturns in industry. Such ebbs and flows were apparent throughout areas in eastern Canada that were undergoing accelerated industrialization in the 1880s, and these tended to suffer economically at times due to overproduction, business failures, unemployment, and out-migration (Heron & Storey, 1985). By the 1890s, the municipality directed development towards steel manufacturing, and, in 1895, Hamilton transitioned to a heavy manufacturing city and focused its economy on iron and steel production (Middleton & Walker, 1980; Nader, 1976). Thus, modern Hamilton's beginnings in 1895 in iron and steel ignited continued urban development in a northeast fashion. Within a span of 15 years (1895-1910), the eastern boundary of Hamilton expanded towards the northeast and the previous agricultural countryside developed into an agglomeration of industrial activity.

4.2.2.2 Shift in demography: 1881–1911

The location and population were heavily influenced by economics (Doucet & Weaver, 1984). Between 1881 and 1911, the city limits nearly doubled, growing predominately to the

northeast (see Figures 2-4 and 5-12). New wards were created to accommodate the expanding urban industrial sector and growing working-class population. Thus, the social geography and social fabric also transitioned to one governed by heavy industry, namely steel manufacturing.

The height of Hamilton's growth occurred between 1911 and 1913 and continued into 1918, giving the city the title of "Steel City" (Gagan, 1989; Herring & Korol, 2012; Weaver, 1983). The onset of steel manufacturing brought about rapid population growth; however, the growth revealed a shift towards predominately working-age male migrants. The expansive population pyramids at both periods tell two very different stories about what was driving population growth in the city of Hamilton. The changing demographic profile of the 1880s towards the 1910s from predominately young working-age females to predominately young working-age males supports the temporal shift in demography and urban development due to changing structure in economy.

During the 1880s, young single working-age women were likely drawn to Hamilton for employment opportunities, such as domestic servants and dressmakers. The 1881 immigration report notes the high demand for female domestic servants (Commissioner of Immigration, 1882). The Hamilton Immigration Agency in 1881 reported that, in Hamilton, during the past season, over 200 applications for servants were made with many applicants willing to pay a commission to secure domestic servants upon arrival to Canada (Commissioner of Immigration, 1882). In 1881, there were 1,182 servants in the city of Hamilton, the majority of which were females (n = 1,056). Servant occupations ranked the second highest occupation frequency, not far behind the highest occupation group, labourers (n = 1,332). Domestic service, in the 19th century, was strongly associated with women (Sager, 2007). According to Sager (2007, p. 509), for many young immigrants between 1871 and 1931, "… domestic service was an obvious path

of entry into Canada and its labor markets" (p. 509). The rapid population growth related to industrializing steel manufacturing at the turn of the 20th century shifted the occupational path of immigrants and migrants.

Production, equipment operating, and related occupations at both periods of time, 1881 and 1911, made up the largest proportion of male workers: 68.74% and 72.63%, respectively. Nevertheless, the type of industry they worked in shifted. In 1881, 17.79% of production, equipment operators, and related activity were employed in clothing production and textile manufacturing, making up the major occupation for males apart from labourers. The 1882 report from Hamilton's Immigration Agency suggested that the manufacturing industry was growing in the 1880s: the agency expected an increased demand for immigrants in 1883 due to prosperity in agriculture and the "… rapidity with which our manufacturing industries are being developed" (Commissioner of Immigration, 1882, p. 4). In 1911, apart from labourers (which accounted for 36.61% of male occupations), 11.73% of males were employed in metal processing, making it the second largest occupation category for males. The industrialization of the steel industry brought with it the loss of the artisan craftsman. Artisans were highly skilled in a particular craft (e.g. metalworkers), but, as steel processing became a capitalist venture, activities of such crafts became separated; this industrialization enabled mass production. The separation of the elements that made up an artisan's craft allowed large numbers of semi-skilled and unskilled workers to be employed (Stincombe, 1990).

Hamilton's manufacturing sector provided work for 15,163 men and 3,815 women (Canada Census and Statistics Office, 1915). Labourers and unskilled workers have always been employed in foundry work; however, as artisan tasks became specialized, large-scale manufacturing operations made use of unskilled low-wage immigrant labourers (Heron, 1980a).

In Hamilton in 1911, immigrant males made up 64.42% of the males working in labourer occupations compared to 35.5% Canadian-born males. This type of inequality in job attainment existed for immigrant groups throughout Canada at the turn of the 20th century (Avery, 1979; Caplan, 2005; Heron, 1988; Heron & Storey, 1985; Morton, 1998; Morton, 1999). Inequalities in the distribution of wealth as a result of uneven employment opportunities and uneven urban development will manifest in health inequities, especially where infectious diseases are prevalent and working conditions are poor. Understanding this connection is possible by examining mortality as an indicator of population health.

4.2.3 Research Question 3: What are the mortality profiles for 1880–1882 and 1910–1912 periods in Hamilton, are there any observed changes in the profile over time, and are these observations expected?

Mortality as a health outcome in epidemiology can be used to describe and explain the potential distribution of disease between populations, especially when using a mortality odds ratio to determine the characteristics of the population that died of a specific cause. Over time Hamilton's mortality profile, like its demographical profile, changed with the rapid population growth and increased urban development. Such changes are not without risks. Infants are highly susceptible to environmental changes, making infant mortality profiles important indicators of changes in social deprivation (Davies et al., 1999). With the overarching research question in mind, major mortality categories for each sample were developed to better understand the connection between population, behaviour, and environment.

4.2.3.1 Tuberculosis

The social fabric of a population affects the risk factors associated with tuberculosis, so using an urban development lens can help analyze its spread and impact. An urban development

lens allows geographical difference to be understood in relation to urban structure. More specifically, this lens aids in understanding the spread of tuberculosis because it emphasizes the social, economic, and environmental aspects of the urban process. In turn, controlling the spread of tuberculosis requires social, economic, and environmental interventions (Lonnroth et al., 2009). In Hamilton, throughout the period for this study, tuberculosis remained the number one cause of death among the working-age sample, making up 24.72% (1880–1882) and 13.50% (1910–1912) of all causes of death. Cause-specific mortality rates revealed that tuberculosis was higher from 1880–1882 than 1910–1912 (6.90 tuberculosis working age deaths per 1,000 living working age population and 3.08 tuberculosis working age deaths per 1,000 living working age population, respectively).

A number of attributable factors reflect the higher tuberculosis mortality rate in the earlier period: an actual decline in mortality, variation in distribution between different causes, changes in the methods of record-keeping, and changes in health surveillance and public health practices. The Ontario Board of Health was permanently established in 1882, and by 1911 local medical officers were mandated throughout the province (Wherrett, 1977). In addition, Hamilton had established a municipal board of health by the mid-1880s, so, by 1911, medical officers were already established in the city and discussions on the health of the city were well underway (Bowman & City of Hamilton, 1912; City of Hamilton Council, 1878-1882; Harris, n.d.; Roberts & City of Hamilton, 1910).

The decline in tuberculosis in the 20th century has been attributed to improvements in living and social conditions (Vynnycky & Fine, 1999), and the link between unsanitary conditions and disease was becoming clearer during this period (Wherrett, 1977). Rapid industrialization, however, in this period increased the number of residences exhibiting

unsanitary living conditions. While tuberculosis mortality rates declined in Hamilton, more working-age people were dying from tuberculosis. In the earlier period, the odds of a young person dying from tuberculosis compared to an older working age person was 3.89:1. By the later period, this increased to 7.75:1 odds. Similarly, middle-working-age people had 2.35:1 odds of dying from tuberculosis compared to older working ages in the earlier period. By the later period, these odds increased to 6.32:1. Thus, young working-age people had much higher odds than any other working age group of dying from tuberculosis. The higher mortality odds found among the young working ages in this research may be related to the rapid population growth of the young working classes in the overcrowded, unsanitary, industrial northeast section of Hamilton. Additionally, the working conditions in the steel plant likely exacerbated stressors for individuals suffering from tuberculosis. The inhalation of chemicals, such as carbon monoxide, methane, nitrogen, oxygen, and pyritic sulphide, in the poorly ventilated steel plants of the 20th century further induced respiratory sickness (like tuberculosis) and death among working-class men (Ludlow, 2009; Ludlow & Burke, 2012). Factory conditions were known to be poorly ventilated, overheated, smokey, and dirty (Wherrett, 1977).

For most western countries, the industrial revolution in the late 18th century to early 20th century marked a period of economic transition from a predominately agricultural society and rural landscape towards an economy of mass production and urbanization. As a result, cities became plagued with overcrowding and poor living conditions; the increasing population faced numerous health risks, including a variety of infectious diseases. In the 1909–1910 Annual Board of Health, Hamilton's Medical Officer, Roberts, reported on the conditions found in the growing industrial neighbourhoods of Hamilton:

> In our own city, owing to the inadequacy of housing accommodation around the incoming manufacturing establishments, there is a rapidly growing district

> where overcrowding is very much in evidence, and the careless tendencies of this population are intensified by lack of sewerage. (Roberts & City of Hamilton, 1910, p. 21)

Housing shortages in the industrial working-class neighbourhoods of Hamilton created the subpar living conditions Roberts noted. Image analysis used in this research revealed some of the conditions found in the industrial sectors of Hamilton (see Figures 5-15 and 5-16). Large numbers of people living in small shack-like houses increased their susceptibility to tuberculosis, especially if an individual sharing the accommodations was already infected. Hamilton's Board of Health in 1911 continued to report on the city's inability to eradicate the overcrowded tenements because of the influx of migrants seeking employment (Bowman & City of Hamilton, 1912). In other words, the city's physical development was unable to keep pace with the growing population. As well, as Harris and Sendbuehler (1992) noted, the presence of owner-built homes in the working-class neighbourhoods of Hamilton also suggests the city was unable to keep pace.

Poor living conditions were prevalent among Hamilton's working class: medical officers described the conditions as "prevalent oppressive" and "unbearable" in reports on inspections (Bowman & City of Hamilton, 1912; Roberts & City of Hamilton, 1910). In 1911, 67 houses showed overcrowding to a marked degree; for example, a seven-room house was reported to have 45 men residing in it (Bowman & City of Hamilton, 1912). The lack of strict building regulations allowed individuals to build a one- or two-room shack and expand as money permitted (Harris & Sendbuehler, 1992); renting a room to number of individuals was used as a source of income to build additions. Migrants and immigrants, desperate for housing, accepted whatever they could find: "Every available four walls that under ordinary conditions of city growth would never be accused of being part of a home is eagerly seized upon and occupied, not

matter how outrageous the rent" (Bowman & City of Hamilton, 1912, p. 20). These overcrowded and shoddy conditions allowed for tuberculosis and infectious diseases to spread rapidly among the working classes, as shown in the northeast spatial patterns.

4.2.3.2 Infectious non-respiratory causes

Infectious diseases were a persistent risk to Hamilton's population in both periods. In Hamilton, infectious non-respiratory illnesses made up 9.47% (1880–1882) and 6.22% (1910–1912) of all causes of death found in the city. Overall, the cause-specific mortality rate for infectious, non-respiratory disease for the two periods was higher in the 1880–1882 sample than in the 1910–1912 sample (2.65 and 1.42 infectious disease deaths per 1000 living working age population, respectively). Similar to tuberculosis, the following are attributable factors for the higher mortality rate for infectious disease in the earlier period: an actual decline in mortality, variation in distribution between different causes, changing definitions, and changes in the methods of record-keeping. Whatever caused this shift, these illnesses remained a risk for Hamilton's population. For both periods, typhoid fever was the most common infectious non-respiratory disease among the working-age mortality sample, making up 61.02% (1880–1882) and 36.59% (1910–1912) of all infectious non-respiratory causes. The latter period showed somewhat more variation in the types of infectious non-respiratory causes: extrapulmonary tuberculosis (18.29%) was the second major cause of infectious disease death in 1910–1912. Knowledge on the mode of transmission for infectious diseases like typhoid fever and extrapulmonary tuberculosis, and the age-related findings of this research illustrate the impact of the growing urban populace and urban mortality of Hamilton.

Mortality by infectious non-respiratory disease declined in Hamilton over the study period and the causes of death became more variable. The association between working age and

dying from infectious disease also showed slightly lower odds in the later period. The odds of a young working-age person dying from non-respiratory infections compared to an older working age person was 7.63:1. In the later period, the odds were lower to about 5:1. Typhoid fever remained the leading cause in this category, a disease associated with poverty and poor standards of living. Thus, the known socioeconomic gradient to infectious diseases like typhoid and tuberculosis, and the northeast spatial patterns show the unequal urban development within the industrial working-class sections of the city.

Hamilton had ongoing problems with sanitary conditions, water supply, and standard of living. In the earlier period, 1880–1882, there was a general concern for the state of the urban public health infrastructure. Sewers were under construction in both the west end and east end of the city, which should have alleviated some of the risk of infection by water-borne disease. However, the sewer committee reported that both sewer systems in the city were not in accordance with specifications, that the overall distribution system was a failure, and that the city was outgrowing the system in any event (City of Hamilton Council, 1878-1882). Although both sewer systems were underway, in 1879, the west-end sewer was "progressing towards completion," while the east-end sewer was delayed due to construction costs (City of Hamilton Council, 1878-1882). There were also concerns that pipes were already being damaged, as people continued to connect to them without permission (City of Hamilton Council, 1878-1882).

In addition to the problems building the sewer lines, the overflow of sewage from them was also a problem. One complaint noted that the east-end sewer was discharging onto persons' property (City of Hamilton Council, 1878-1882). Additionally, stagnant water and the need for the collection of night soil, or human waste were being reported as issues (City of Hamilton Council, 1878-1882). The city responded to the health crisis problems posed by attempting to

remove the environmental hazards through physical intervention—a marrying of public health and urban planning (Corburn, 2007). By 1910–1912, reports discuss problems with urban development, the practices of a subset of the population, and the availability of lower quality dairy as common hindrances to the population's health. Board of Health reports for 1909-1910 noted that typhoid fever was at its highest reported levels since 1906, with the highest number of cases reported in the newly-added districts of the city (Roberts & City of Hamilton, 1910). These new areas were the rapidly growing and overcrowded manufacturing districts, which held general unsanitary conditions that were further intensified by the "careless tendencies" of the population and lack of sewerage; these problems superinduced the onset of typhoid fever (Roberts & City of Hamilton, 1910).

4.2.3.3 Accidental causes

The association between accidental mortality and industrialization was reinforced in the quantitative results of this study. With the small sample size for accidental deaths in the earlier period, occupation could not reveal any correlation without over-generalizing the classification of occupations in the chi-square results. As well, although the chi-square analyses for the 1910–1912 working-age sample showed young working age, skilled tradesmen, and labourers as independent variables, the logistic regression model revealed sex, age, and birthplace as significant predictors for accidental mortality. The connection between accidental causes of death and industry in a historical context, however, has been shown in Sydney and Glace Bay, Nova Scotia, where accidents killed more working age men than any other cause of death during the early 20th century (Ludlow & Burke, 2012). Pittsburgh had a similar relationship between heightened accidental deaths among workers in the steel plants in the early 1900s (Fitch, 1969).

Even though occupation was not a significant predictor, other independent factors were more significant predictors for accidental mortality. The work environment heightened accidental mortality in Hamilton, during the later period. This is further supported by the continued association of accidents among men, the increase in accidents among young working ages and foreign birthplace, and the spatial northeast distribution of accidental mortality in Hamilton. In the earlier period, the odds of men dying from an accident than of another cause were 7.01:1. This association lessened in the later period, with odds of 5.20:1. The lowered gap in accidents between men and women is consistent with the increase of women working in factories in the 1900s, but also suggests that other variables were more significant predictors in the model. Young working-age people had 6.11:1 odds that they would die in an accident compared to older working-age people. Middle working-age people were over three times more likely to die of an accident than older working-age people. Immigrants were also 2.88 times more likely to die of an accident than Canadian-born people. The heightened risk of accidents in this period reflects the notoriously poor working conditions in steel plants throughout Canada and the United States during the turn of the 20th century. Young, inexperienced workers were likely not familiar with dangers or equipment and immigrant workers had an added language barrier in the already noisy work environment. Thus, early industrialization alters mortality patterns, particularly for lower socioeconomic members of a society.

4.2.3.4 Diarrhoeal causes (infant mortality)

In Hamilton, diarrhoeal causes made up 22.22% (1880–1882) and 19.77% (1910–1912) of all infant causes of death found in Hamilton. Diarrhoeal causes presented the highest cause-specific P-IMR (119.50 infant diarrhoeal cause deaths per 1,000 infants) for 1880-1881. There was a drop in diarrhoeal causes of death by 1910-1911 (87.72 infant diarrhoeal cause deaths per

1,000 infants). The logistic models for both periods found that infant age and seasonality significantly predict diarrhoeal-related mortality.

In both periods, postneonatal infant age showed a strong link to diarrhoeal cause mortality when compared to neonatal infant age. In 1880–1882, postneonatal infants were nearly six times more likely to die from diarrhoea than neonatal infants. By 1910–1912, 15 postneonatal infants were dying from diarrhoeal causes for every one neonatal infant who died from them. Summer and autumn seasonality both presented an increased risk for diarrhoeal-caused mortality. In the earlier period, infants were over 13 times more likely to die from diarrhoea in the summer and six times more likely in the autumn than in the winter; in the later period, infants were over four times more likely to die from diarrhoea in the summer and in the autumn than in the winter.

Ferrie and Troesken (2008) note that, while literature supports the connection between the reduction of infant mortality and the introduction of public water and sewer lines, the efficacy of these interventions varies. In Hamilton, while age and seasonality were linked to infant diarrhoeal causes, there was a temporal flip in the association with the independent factor. This flip in the associated independent factors suggests that, during the earlier period, environmental causes such as seasonality and water quality were more influential than maternal practices, while, in the later period, maternal practices such as the time of weaning and breastfeeding, had some influence in addition to the environmental factors and sanitary practices. This conclusion is supported by the northeastern spatial pattern for infant deaths caused by diarrhoea. Additionally, the increased significance of age in the later period may also reflect the changing socioeconomic gradient of Hamilton with an increasing working class through the

progression of industrial activity. Women in lower-income families often worked outside the home and, therefore, opted for artificial feeding (Wolf, 2003).

The influence of environmental factors on diarrhoeal-related infant mortality in the early period reflects the challenges Hamilton had with its sewers. Reports from the city council between 1880 and 1882 indicate that sewers pipes were clogged during heavy rains, water was overflowing onto people's properties, and sewers were discharging onto properties (City of Hamilton Council, 1878-1882). Concerns were raised about the abundance of privies and cesspools and the need to collect, regularly, nightsoil (City of Hamilton Council, 1878-1882; Harris, n.d.). The Province of Ontario also noted the wells could easily become contaminated by cesspools through porous soils and expressed the need for better sanitary reforms in the disposal of sewage (Province of Ontario, 1883). Thus, Hamilton and the whole province of Ontario were greatly concerned with building an adequate system for water and sewage.

By the later period, 1910–1912, the Hamilton Board of Health became increasingly concerned with maternal education and milk supply. Lack of sewerage and "primitive" methods for disposing of waste were still concerns raised, especially within the industrial districts (Roberts & City of Hamilton, 1910). The annual report for the year 1909-1910 even suggested an increase in inspectors during the summer and early fall to enforce sanitary rules on the construction, cleaning, and disinfection of privy vaults and dry earth closets (Roberts & City of Hamilton, 1910). The high death rate of infants was however, also attributed to the lack of maternal education for women and the lack of cleanliness of cow's milk (Roberts & City of Hamilton, 1910). To help, the Medical Health Officer suggested that Women Health visitors who would instruct women in the care and feeding of children would benefit Hamilton, due to the large and increasing "foreign element" (Roberts & City of Hamilton, 1910). The

uncontrolled supply of milk was actually a larger contributor to infant morbidity (and plausibly infant mortality), and was more the result of an economic disparity rather than a lack of education in parenting.

Unsanitary cow barns, contaminated milk, and poor milk storage were a significant risk to infants in the later period. Image analysis revealed the state of unsanitary cow barns in Hamilton during the later period (see Figures 5-17 and 5-18). This heightened the risk of the consumption of low-quality raw milk. Awareness of the variations in milk quality led to the implementation of a certification process for dairies producing milk for human consumption (Roberts & City of Hamilton, 1910). The term *certified milk* meant that dairies fell within the standards and regulations of local authorities (Block, 2009; Czaplicki, 2007; Speake, 2011). Certified milk, however, was quite expensive for the consumer (Czaplicki, 2007; Speake, 2011). To alleviate some of the cost, families may have turned to watered-down milk: Sawchuk and co-authors (2002) suggest that poorer families in Gibraltar would have purchased watered-down milk because it was cheaper than the pure milk that was available. Including water in the milk increased the potential for contamination; both the water and milk supply contributed to infant diarrhoeal mortality. As infants were weaned or artificial feeding practices were used, infants' susceptibility to tainted milk, water, or food led to weanling diarrhoea (Sawchuk et al., 2002). Thus, the association of infant diarrhoeal cause mortality with age and season support two causal factors: an urban public-health problem at the city level, and a behavioural problem of early weaning time and poor artificial feeding practices of Hamilton's residents.

4.2.3.5 Infectious respiratory (infant mortality)

Infectious respiratory diseases made up 15.20% (1880–1882) and 12.88% (1910–1912) of all infant deaths found in Hamilton. Cause-specific proxy infant mortality rates for the 1880–

1882 and 1910–1912 periods were 81.76 and 57.16 infant deaths from infectious respiratory diseases per 1,000 infants, respectively. Although infectious respiratory mortality declined in the later period, it still persisted as a major cause of infant death. In both periods, age and seasonality were significant predictors for infectious respiratory mortality in the city of Hamilton.

Age significantly predicted death from infectious respiratory diseases. In 1880–1882, postneonatal infants were 5.44 times more likely to die from infectious respiratory causes than neonatal infants were and, in 1910–1912, they were 9.65 times more likely to die than neonatal infants. Respiratory infections for postneonatal infants occurred in the northern, especially northeast, sections of the city. Maternal feeding practices may have caused infant respiratory tract infections, especially for female infants. Reports on weaning age and artificial feeding in Hamilton is scarce; however, the medical officer was concerned with maternal education and infant feeding practices for new immigrant families in the industrial districts of the city (Roberts & City of Hamilton, 1910).

Seasonality also contributed to infant mortality. Infants were over five times (in the earlier period) and six times (in the later period) more likely to die from infectious respiratory diseases in the winter than in the summer. Winter showed slightly more occurrences in the northwest, followed closely with cases northeast. These findings suggest that the houses in these industrial working-class areas were not suitable in the winter. Houses in these neighbourhoods were in some cases condemned because of the unhealthy and overcrowded conditions in them (Figures 5.15 and 5.16). Conditions in basements that were inspected were described as being poorly ventilated, lacking sunlight, and cold and clammy (Bowman & City of Hamilton, 1912). It was noted that, in such inspections, 28 families were only able to afford to live in a single

room used for living, cooking, and sleeping (Bowman & City of Hamilton, 1912). Thus, such housing conditions in the industrial working-class neighbourhoods in Hamilton presented a space where infectious respiratory conditions easily spread to the most susceptible population, infants.

4.2.4 Research Question 4: Are there spatial differences in mortality for the 1910–1912 period in Hamilton, and do the mortality patterns suggest health disparity in relation to socioeconomic organization and urban development?

Hamilton was well underway to becoming a major urban centre for heavy industry by 1910–1912. In both periods, infectious disease was prevalent and presented a major risk to the health of the population. The spatial mortality patterns described in this study closely align with the spatial socioeconomic divide found in Hamilton. Spatial residential patterns reflect multiple facets of society (Artibise, 1975; Beveridge, 2002; Cutchin, 2007; Herring & Korol, 2012; Vaughan et al., 2005; Zunz, 1982), and locational disadvantages predispose people towards poorer health outcomes (Feldman et al., 2009). Even prior to industrialization, Hamilton retained an uneven and unequal social structure and urban development (Katz et al., 1978). This class-based divide became more pronounced as the city transitioned towards industrialization.

At the same point in time, other American and Canadian industrial centres were experiencing similar divides across socioeconomic groups. In Detroit, urban development lagged in eastern sections of the city where working-class immigrants resided (Zunz, 1982). Similarly, working-class neighbourhoods in Montreal lacked the proper sanitation and drinking water that affluent sections were able to obtain (Pelletier et al., 1997). In Winnipeg, non-British immigrants lived in poor quality housing and faced poor economic opportunities (Artibise, 1975). The urban sociological school of thought and their predecessors suggest that heightened industrial activity causes working-class neighbourhoods to develop in close proximity to the

industrial sector (Mann, 1965; Park & Burgess, 1921; United States Federal Housing Administration & Hoyt, 1939). Industrialization was a key component to the increasing socioeconomic segmentation of Hamilton (Harris & Sendbuehler, 1992). In turn, the high rates of infectious diseases were prevalent in the densely populated areas nearby Hamilton's production centres (Gagan, 1989). HGIS mapping from this research further illustrates the spatial health divide found upon Hamilton's industrial landscape.

The majority of tuberculosis cases among working-age people were found in the northeast quadrant. Of these deaths, young working age and middle working age people had the highest number of cases in the northeast. When selecting for infectious non-respiratory cause, young working age cases were again most prominent in the northeast. For accidents, the highest numbers of cases were for young to middle working-age men who were born outside of Canada and lived in the northeast. Infant deaths from diarrhoeal causes continued to reflect the northeast pattern. Infant died more frequently from diarrhoea in the summer and autumn, especially if they lived in the northeast. Cases of infant respiratory infections mostly occurred in the northwest, followed by the northeast. Most postneonatal deaths in were found in the northeast, followed by the northwest. The prevalent northeast pattern found in the HGIS results indicate that possible causal factors led to this spatial divide.

Household services such as piped water and sewerage increased taxes, so residents occasionally petitioned against these basic needs (Harris & Sendbuehler, 1992). Because of the expense of these services to both the city and the residents, city health reports throughout the period of this research suggest class-based differences in these public health provisions (Bowman & City of Hamilton, 1912; City of Hamilton Council, 1878-1882; Harris, n.d.; Roberts & City of Hamilton, 1910). Spatial patterning found in the HGIS results support the mortality

profiles described above. Poorly-ventilated and overcrowded homes in the northeast, like those exhibited in the image analysis, would have increased the risk for tuberculosis found among the working-age sample. As well, the increased migration of young and middle working-age men (single and with families) who resided in the industrial northeast may have influenced the young adult age effect for tuberculosis in the city of Hamilton.

Environmental causes greatly affected the rates of death in Hamilton. The high rates of infectious non-respiratory causes and diarrhoeal causes in Hamilton were attributed to the unsanitary state of the industrial districts as described in the health reports (Bowman & City of Hamilton, 1912; Roberts & City of Hamilton, 1910). The 1910 city health report suggested both the lack of sewerage and the practices of the population were causal factors (Roberts & City of Hamilton, 1910). Infectious respiratory diseases among infants had a dominant north south divide for both overall and, for 1911, HGIS results. Herring and Korol (2012), in their study of the 1918 influenza pandemic, also found a prominent north south divide in the city of Hamilton. Infectious non-respiratory causes tend to be most frequent in low-income areas (Corner et al., 2013; Kelly-Hope et al., 2007). The increase in accidents as cause of death among the working ages is a by-product of industrial development. That age and sex are significant predictors, together with the northeast spatial pattern, further supports that these accidents were related to work-life and safety.

The spatial patterning of mortality in the later period reinforces that health disparities existed in Hamilton and had a pronounced socioeconomic gradient. These spatial observations revealed aspects of the mortality analysis that were not easily attainable without this tool. HGIS presents the ability to visualize death patterns, which can lead to new investigative insights. Understanding the spatial mortality patterns in the context of environmental and behavioural

factors leads to better examination of the social gradient of health. Social hierarchies of advantaged/disadvantaged groups persist worldwide and contribute to divisions based on socioeconomic status, ethnic or religious groups, gender, age, geography, and health (Braveman & Gruskin, 2003). The *social gradient of health* refers to the concept that the higher the social position, the better the health, and therefore concerns the whole population, as everyone has worse health than those above them in the social hierarchy (Marmot, 2006; Marmot, 2012). Wealth distribution in a population can also influence the health of a population. The Whitehall studies of Great Britain have advanced our understanding of the socioeconomic gradient of health inequity in relation to job and financial security, the employment grade differences in health risk behaviours (smoking, diet, and exercise), early life experiences, and material conditions (home ownership, car ownership) (Ferrie, Shipley, Smith, Stansfeld, & Marmot, 2002; Ferrie, Shipley, Stansfeld, Davey Smith, & Marmot, 2003; Marmot, 1993; Marmot et al., 1991). This research extends on this work by adding importance of the environment alongside behaviours to observing spatial health inequities, using mortality as an indicator. As the choices people make are not always due to a lack of education, but also what is and is not available in their environment.

Infant health correlates to the health of the society, and the distribution of resources within that society. According to Haines (2011), there is currently a 50% - 150% penalty in terms of infant mortality for being in the lowest socioeconomic status group relative to the highest in the United States. Pregnant and nursing mothers can act as buffers against environmental insults; however, when mothers' health is compromised by external environmental factors, infants are also placed at risk (Black et al., 2008; Goodman & Armelagos, 1989). The compromise to maternal health is, again, rooted in environmental, economic, and

contextual factors that cause undernutrition, poverty, and increased risk to infectious diseases (Black et al., 2008). Limited economic opportunities for immigrant populations increase potential health risks, such as living in poor quality housing and working in hazardous environments (Blane, 2006; Durkin, 1998). In Canadian labour history, non-British immigrants often worked in unskilled labour positions (Avery, 1979). These undermining health patterns, as Labonte et al. (2005) suggest, are reinforced by the history of social structure, economic relations, and ideological assumptions. Given these connections between health inequity and the social gradient, the change in socioeconomic and urban structure of Hamilton during industrialization likely affected the overall population health, and specific members of the society more so than others.

4.3 Conclusions

Causes of health inequity are multi-faceted, requiring an in-depth look into population, society, and environment. Historical epidemiology can help to flesh out such connections, as history has the unique ability to examine what has already occurred through a comparative or natural experiment (Diamond & Robinson, 2010). This research supports the argument that mortality had a pronounced social gradient manifested by the structure of society and the socioeconomic condition of an industrial centre. The conditions in urban development further support this gradient, with the less advantaged population residing in areas that had inadequate public health infrastructure, which exacerbated the spatial mortality patterns. Spatial differences have been largely attributed to differences in living conditions, social class, and economic opportunities (Emery & McQuillan, 1988). Yet, in current health promotion there continues to be a focus on education as the solution.

CHAPTER 5
CONCLUSIONS

5.1 Summary of the Study

There is a lack of integrative historical work in urban planning and population health, and this is not helped by limited research into how the spatial patterns of health seen today came into being. Historical health research can expose the multi-faceted complex causes of health inequities, and provide insights into the relationship between industrial and urban expansion, on the one hand, and the emergence these inequities, on the other. More historical research in the health and social sciences would provide insights into such complexities. While researchers acknowledge the importance of historical urban planning and health research, there is a gap in actual engagement in the practice (Campbell & Campbell, 2007; Commission on the Social Determinants of Health, 2008; Etches et al., 2006; Filion & Bunting, 2010). The spatial fixity of a city can retain pockets of health disparities long after the forces that generated them have gone. One way to better understand intra-urban disparities in health is by examining the historical context in which they arose by studying the historical nature of the city and its physical and social geography. In doing so, the profound connection between urban development and population health can be explored.

The purpose of this research was to investigate how the changing socioeconomic nature of a society can affect population structure and urban development in an historical Canadian context, and how these factors relate to the variations of spatiotemporal mortality patterns. This relationship was identified through a multidisciplinary mixed-methods approach using human

health ecology as the theoretical foundation. Human health ecology is used in a variety of disciplines focusing on health because it provides a holistic perspective on the causal factors that affect population health, and is the basis behind the current Healthy Cities/Healthy Communities approaches. This research set out to employ a healthy city concept to examine the connection between the urban environment, public health, mortality, and the population at risk—essentially looking at the *Urban Health Penalty*— in an historical Canadian industrial city. The healthy city approach is a process through which a commitment to health continually creates and improves the social and physical environments (World Health Organization, n.d.-b). Thus, this concept builds from human ecology, which moves beyond a behavioural/educational model, towards a comprehension of the interaction of humans, their behaviour, and the built and physical environments in which they live effect health outcomes.

In this dissertation, mixed-methods were used to analyze the levels of health ecological interactions through empirical research that explored the connections between shifting economic activity, urban development, population change, and mortality. Hamilton, Ontario, around the turn of the 20th century presented an opportunity to explore these connections by examining the city at two periods in time: before and during heavy industrial activity. This approach provided a snapshot of life and death before and during industrialization.

Four questions directed the research. Each of these was driven by the purpose of the research: an attempt to understand how changes in the dominant economic activity in a community can affect urban development, population structure, and mortality patterns. With Hamilton as the case, this study focused on answering four specific questions centred on a period of rapid economic change spanning the turn of the twentieth century. First, this study sought to explore how the city's socio-demographic profiles changed between 1881 and 1911. From this

exploration of demographic changes, it attempted to determine whether those socio-demographic patterns reflected the changing urban structure of Hamilton during this period. This study went on to examine the city's mortality profiles for two periods, 1880–1882 and 1910–1912, to identify any major changes over this period. Using the mortality results, this study sought to look for apparent spatial differences over the 1910–1912 period in order to understand whether health disparities reflected noted variations in socioeconomic organization and urban development.

There is a need to incorporate change over time into disciplinary practice. In historical geography, the incorporation of time into spatial research is often plagued by changes that occur over time, such as administrative boundaries (Gregory, 2000) and whether spatial information is consistently recorded in historical documents. The spatial patterns observed in this research could not be reflected through a temporal lens using HGIS techniques due to limitations in the data; however, the combined mixed-methods approach assisted in detailing spatiotemporal health across multiple levels interaction. In turn, temporal inferences could be made on the possible correlation of factors that influenced mortality in Hamilton at two periods in time. Both periods present a city where infectious disease was prevalent, although these diseases showed a decline in the later period (as presented in the mortality rates), and reports from city officials shed light on unhealthy urban conditions that were likely contributory factors. The state of the built environment in terms of public health infrastructure, the newly arriving industrial working class, and their practices were common aspects reported on by Hamilton's health officials. These factors would have contributed to the risk of major causes of mortality found among the working ages and infants, and tie directly to the concepts behind building healthy cities and the human ecology approach. A city can be resistant to change, but a transition in the social and economic

structure of a city will likely affect the demographic and epidemiologic patterns across time and space. Additionally, the added stressors of industrialization can heighten intra-urban variation in both population structure and development, and manifest such stress via overcrowding, class segregation, and the unbalanced presence or quality of urban infrastructure. Thus, urban development and population health have key implications towards understanding cities, their populations, and health disparities.

5.2 Key Findings of the Research

Across two periods, 1880–1882 and 1910–1912, and using a purposive mortality sample, a snapshot of life and death before and at the height of the era of heavy manufacturing (namely steel manufacturing) was provided in this dissertation. While infectious disease dominated the health landscape, the results suggest that industrialization may have influenced the patterns of mortality. Two major findings come from this research: first, that industrialization significantly contributed to the emerging human health ecology risks, likely correlating to the mortality patterns found in the two samples, and, second, that industrialization tended to increase health inequities and disparities both across the population and across space. These findings are subject to limitations in terms of data acquisition.

Analyzing Hamilton's urban development showed that the pre-existing urban form and social fabric preconditioned the city's industrial growth. The economic activity of Hamilton heavily influenced the geography of industry and population (Doucet & Weaver, 1984; Harris & Sendbuehler, 1992; Katz et al., 1978). With the onset of a predominately-industrial economy, Hamilton experienced rapid population growth especially among young and middle working-age males. This influence of economics is apparent where the growing industrial sector continued class division, in terms of space as well as death. From 1881 and 1911, Hamilton's population

grew 128%; most of this growth occurred as the city transitioned its economy towards industrialization (post 1895), and occurred in the working-class population. Geographically, Hamilton grew in a predominately northeast direction, as this area had been sparsely populated previously and provided lake access for processing and transporting materials and products.

As in many cities, this rapid population growth had implications for urban health, because the infrastructure in place was not be adequate for the growing populace and the city was not equipped to keep pace in terms of urban development (Barkin & Gentles, 1990; Condran & Crimmins-Gardner, 1978; Fischler, 2000; Gagan, 1989; Herring & Korol, 2012; Pelletier et al., 1997). The rapid population growth that occurred especially during the first decade of the 20th century created uneven urban development, which created class-based disadvantages. Growth in industry led to uneven access to adequate housing and public health infrastructure such as sewerage and clean water supply. Qualitative inquiry into Hamilton reinforced that the urban environment was not prepared to handle the growing population due to lack of proper urban development, and that this lack of preparation led to inequitable health opportunities. Conditions in the growing industrial neighbourhoods of Hamilton were described in contemporary accounts as having inadequate housing, overcrowded and poorly ventilated homes, and a lack of sewerage and clean water supply (Roberts & City of Hamilton, 1910). The poor conditions in these neighbourhoods and notable residential segregation are key to explaining socioeconomic organization, as the sociogeographic patterns can elucidate disadvantages in neighbourhood health (Acevedo-Garcia, 2000). The contextual factors in Hamilton imply that uneven urban development was present. This spatial disadvantage would have widened the gap in the social gradient of health through an increased exposure to both respiratory and gastro-intestinal pathogens, like those presented in the mortality analysis of this research.

At the turn of the 20th century, infectious diseases were beginning to decline in North America (Armstrong, Conn, & Pinner, 1999), but places undergoing industrialization tended to show a delay in this decline (Hautaniemi et al., 1999). Previous historical studies have used single industry/company towns to show how the process of industrialization caused rapid urbanization and induced changes in the demographic and epidemiologic profiles of these towns (Hautaniemi et al., 1999; Ludlow & Burke, 2012). Such single industry/company towns are a product of economic engineering (Porteous, 1970), presenting a different sociopolitical dynamic than the situation of Hamilton, a well-established city that became industrialized. Hamilton was already burdened by the health insults of urban living, but industrialization produced additional unequal health risks for the rapidly growing working classes. Using multiple logistic regression models, this study found a lower odds of dying in the young working ages of infectious non-respiratory causes, while a higher odds of dying from tuberculosis was found for young and middle working-age in the later period. In the United States, tuberculosis declined by about one third between 1850 and 1890, but this was not a steady decline as social and geographical variations persisted (Feldberg, 1995). Hamilton shows these types of variations: the northeast section of Hamilton had higher rates of tuberculosis death, which was likely caused by the extremely overcrowded and poorly ventilated conditions in these neighbourhoods.

In addition to the change in tuberculosis rates, accidental deaths increased as Hamilton became industrialized. The increase in accidental deaths, which primarily affected young and middle working-age immigrant men, was a consequence of the poor working conditions in heavy manufacturing (Ludlow & Burke, 2012) and reflected the types of people who were most engaged in dangerous factory work. The interrupted tuberculosis decline and increased accidental deaths during the heavy industrial activity in the later period suggests that the increase

in the working-age population and the unhealthy environment they inhabited were causal factors in both causes of death.

Infants are highly susceptible to environmental change, so infant mortality is an important indicator for social deprivation (Davies et al., 1999). Multiple logistic regression models predicting for infant mortality presented a link between postneonatal age for both infant diarrhoeal causes and infectious respiratory causes of death that increased over time, while the relationship to seasonality for both causes decreased, which can be attributed to underlying mechanisms. At the height of industrial activity, age was the strongest predictor for both infant diarrhoeal and infectious respiratory causes, suggesting that maternal practices and environment may have been causative factors. Maternal health plays an imperative role in infant health, especially during the pre-weaning stages, as breastfeeding usually acts as a protective buffer against environmental insults. Thus, when the health of the mother is compromised by external environmental factors, infants are also at risk (Black et al., 2008; Goodman & Armelagos, 1989). This compromised health is rooted in the environmental, economic, and contextual factors that cause undernutrition, poverty, and increase the risk of infectious diseases (Black et al., 2008). These undermining health patterns, as Labonte et al. (2005) suggest, are reinforced by the history of social structure, economic relations, and ideological assumptions.

Industrial economic growth can alter mortality patterns that affect a specific proportion of a population, as was found in Hamilton. HGIS was used to map mortality; the results suggested that increased mortality disparity occurred between social classes partly due to locational effects, thereby supporting the need to move beyond education and identify additional causal factors to inequity, such as the environment. Other studies have mapped mortality in Hamilton to examine spatial disease patterns using techniques other than HGIS and focusing on different disease

patterns and contexts (i.e. not the role of industrialization) (Gagan, 1989; Herring & Korol, 2012). In this research, mortality data for the years 1910–1912, were amenable to mapping in the HGIS and revealed that most deaths occurred in the northeastern quadrant of the city. Careful interpretation of these results was required, but still offered an enriched visualization of the spatial distribution of mortality during heightened industrial activity, which supported the underlying premise: the changing socioeconomic constructs of a society influenced population structure and urban development, and these factors related to the variations of spatiotemporal health patterns.

The indication of mortality inequities presented throughout this dissertation required a further understanding of the social gradient of health, which refers to the concept that the higher the social position, the better the health, and thus the social gradient of health concerns the whole of society (Marmot, 2006; Marmot, 2012). Analyzing historical documents and images, though based on the perceptions of the time, added contextual value to this research by documenting that the risks to health were likely multi-faceted relating to space and place, the environment and the practices of the population residing in a specific area. Hamilton was not a healthy urban environment before industrialization, nor was the city equipped to deal with the rapid development needed to sustain the growing industrial populace.

Contemporary health reports and images described and illustrated the dismal environmental conditions, the poor behaviours of residents, and the use of poor quality sources of dairy as problems affecting the newly arrived industrial populace (Roberts & City of Hamilton, 1910). These reports supported the previous mortality analyses and added breadth to the causal factors of working age and infant mortality. Such descriptions should not be used to blame a specific population, but rather to understand that such practices coupled with uneven

urban development would have created inequitable health outcomes, which thus demonstrates the interplay of such factors. For example, infant diarrhoea, whether current or historic, is highly associated with poor sanitation, poor water quality, lack of breastfeeding, time of weaning, and quality of milk supply (Ferrie & Troesken, 2008; Sawchuk et al., 2002; Vaid et al., 2007).

The social, economic, and environmental fabric that changed in Hamilton with the onset of heavy industry also changed the population, and therefore, the population at risk. Additional research from various cities in the United States from 1880 onward suggests that both the environment and socioeconomic condition are important determinants of mortality differentials (Haines, 2011). Thus, industrialization would have increased the economic divide of the working classes. Hamilton, as it became dominated by industrial activity, revealed an urban form similar to that found in Mann's urban model: neighbourhoods were divided by class, with the wealthier residents living in areas that were more geographically advantageous. Such division in classes are supported in the historical literature on Hamilton and would have manifested a spatial pattern of stressors related to socioeconomic status and health disparity. Thus, industrialization did bring new stressors to Hamilton that created unequal opportunities for the rapidly growing working classes. A holistic health ecological approach is necessary to understanding the disease patterns that occurred in Hamilton before and during the onset of heavy industry in order to analyze the effects of industrialization. Without insights from public health, population health, urban planning, health geography, anthropology, and history, the story of urbanism and population health would not be complete.

5.3 Implications for Practice and Future Research

In addressing the main research questions of this empirical study, this dissertation reinforces the need to employ a multidisciplinary approach to health research that seeks to gain

important insights into the connection between urban economic development and population heath. Mixed-method research designs enable the collection of diverse types of information to give a more complete comprehension of a research problem that cannot be understood by the use of only quantitative or qualitative sources (Creswell, 2014). This style of research caters to understanding multi-level causal pathways found in human ecology. For example, mixed methods use various sources to identify the possible interaction found in the human ecology framework between population, behaviour, and habitat. The quantitative results provided an understanding of the population at risk. Used alone, the information would only give individual-level characteristics. HGIS mapping of mortality and significant predictors of mortality allowed the results to be contextualized through a geographical lens. Documents and images from the historical record provided the necessary support to understand the possible behavioural and environmental patterns that may reflect sociogeographical and urban development aspects to health inequity, such as cultural weaning and breastfeeding practices, supplying water to wealthy areas first, and inadequate housing in the industrial sections. Sequential explanatory mixed methods allowed this research to identify key compositional and contextual factors of the population, their behaviours, and their environment that lead to divisions in class, level of development, and health inequities

The mixed methods approach allows this research to advance the use of historical epidemiology and interdisciplinary methods to study the interconnections between population, environment, and behaviour. Essentially, historical epidemiology is the study of disease patterns over time through the integration of health ecology and, therefore, population health and urban planning are a natural fit to examine these interconnections. Historical epidemiology requires research from many disciplines to acquire a comprehensive view that uses the four domains

described by Swedlund and Donta (2003): historicity, medical history, epidemiology, and molecular biology. Drawing upon knowledge found in public and population health and a variety of social science disciplines can help to provide this comprehensive view. The current lack of integration of history into health research and the segmentation between disciplines, however, discourages integrative approaches to understanding health patterns. Thus, there is an apparent need to bridge disciplinary knowledge.

The past can provide valuable information about current health outcomes. Some health outcomes are tied to specific social structures, economic relations, and ideological assumptions that are unveiled through temporal heath research (Etches et al., 2006; Labonte et al., 2005). Social hierarchies of advantaged and disadvantaged groups persist worldwide, contributing to divisions of socioeconomic status (Braveman & Gruskin, 2003). Although Hamilton today is different than it was at the turn of the 20th century, the industrial sector remained a prominent economic driver into the late part of that century. Historical epidemiology encourages connectivity across disciplines and, when used, contemporary health researchers can establish connections and patterns of health outcomes past and present. When this past and present knowledge is combined, researchers can work towards improving future health outcomes and healthy spaces.

This dissertation also advances knowledge of the complex interplay of the changing population, economy, and mortality at a time when industrialization brought new stressors to Hamilton that created unequal opportunities for the rapidly growing working classes. There have been three prominent transitions used to explain major changes in human society: demographic transition, industrial transition, and epidemiological transition. These transitions were all present in Hamilton during the study period. This dissertation implicitly synthesizes these key

transitions using the theoretical foundations of health ecology to better understand the causal factors in disease patterns that occurred in Hamilton before and during the emergence of heavy industrial activity, supporting a link between the health disparities and economic transitions.

Szreter (2004), in his theoretical work on industrialization and health states that previous notions that economic growth of industrialization was beneficial for health are misleading. The contributions of this dissertation using a health ecology approach to examine a single city before and during industrialization revealed that, historically, infectious diseases declined. The increase in accidental mortality during the industrial period, however, altered the mortality profiles for the working-age sample. Thus, it may not be that infectious disease declines, but that accidents accounted for more deaths in the sample. Infant mortality also declined across the two periods, but remained high by today's standards.

Mortality rates only give a high-level insight into the characteristics of the population that died. Mortality odds ratio (via multinomial logistic regression) identified key characteristics of the sample, and the changes to these characteristics over time. Indeed, the mortality odds ratio supported that inequities occur in the population at risk, and, for most, revealed a heightened association by 1910–1912, suggesting a tie to the rapid industrial transition that occurred in the city. Furthermore, dominant northeast mortality spatial patterns, and health reports on living standards and health behaviours support that the process of industrialization in Hamilton created stressors for the working-class population.

The spatial patterns in this research reinforce the need for proactive urban planning before and during economic transition in order to minimize health risks. Szreter (2004) suggests, from a current policy perspective, the importance of not encouraging or forcing non-industrialized societies to enter into the industrialization process without clear understanding of

the disruption and difficulties that occur with this process, which history can demonstrate. This is suggesting not a discontinuation of industrialization, rather an awareness of potential issues that occur during a major economic transition. Demographic transition theory holds that, while economic development will ultimately lead to declines in mortality, this development is a period of immense growth and instability—likely revealing increased disparities in morbidity—, and thereby creating health inequities that requires careful urban planning. The lessons from the historical inquiry presented in this dissertation add to Szreter's work, by first suggesting that, where an economic transition is to take place, careful planning in urban development is necessary; otherwise the potential for a concentration of health risks will lead to inequitable health outcomes in a population—leading to further urban penalty for the urban poor. Second, by demonstrating the complex causes of health inequity using health ecology, the plausible interplay between the population, habitat, and behaviour are identified which can assist with improving healthy environments in the future—moving beyond health promotion towards multi-faceted causal explanations to underlying health outcomes. By forgetting the epidemiology of the past, lessons will not be learned, which in and of itself constitutes a risk in public health (Webb, 2015) and, moreover, population health.

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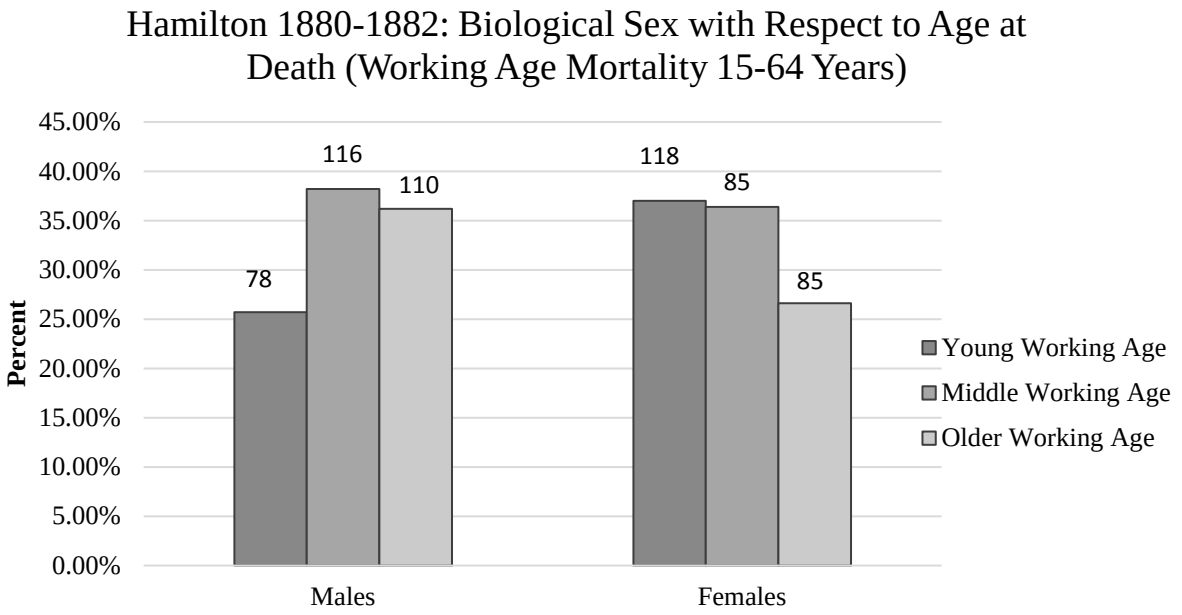
APPENDIX A: CHI-SQUARE RESULTS FOR INDEPENDENT VARIABLES: WORKING-AGE MORTALITY SAMPLE

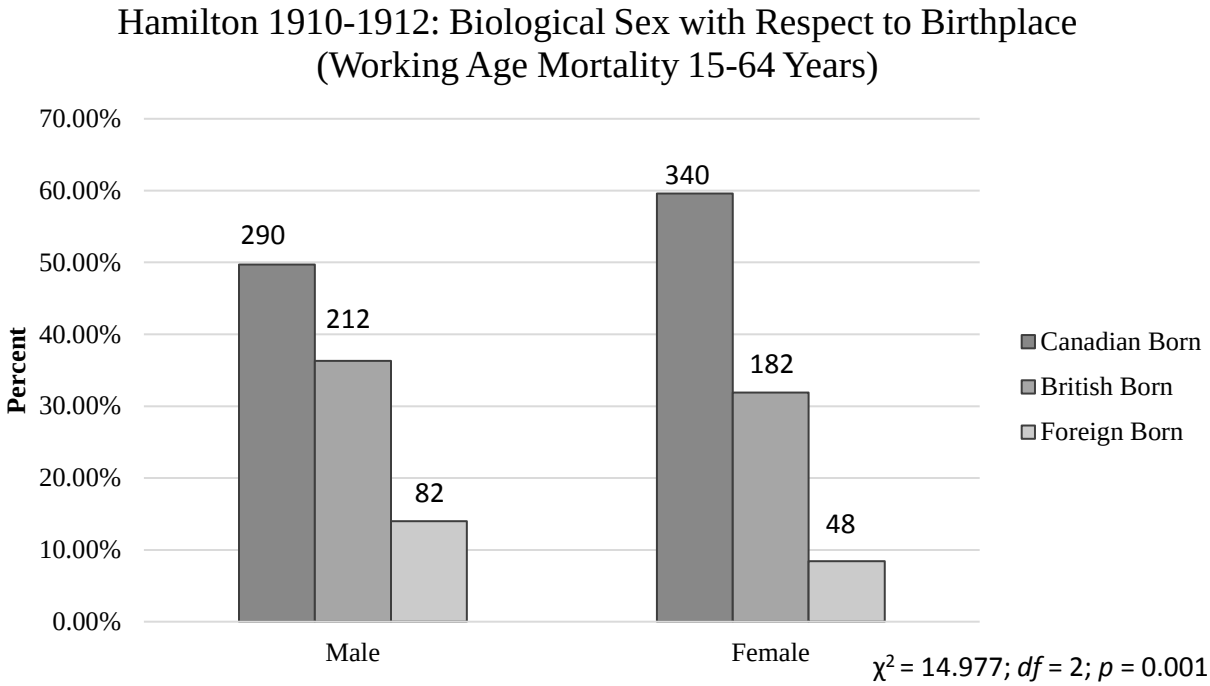
Presented here are the findings for the chi-square test for independent variables. The analysis for biological sex and age at death produced statistically significant results for the 1880–1882 working-age mortality sample ($\chi^2 = 11.014$; df =2; p = 0.004; Figure A-1). Although a statistical relationship was identified, the standardized residual for each cell fell within the critical value (± 1.96), meaning that no anomalous observations were found, or that the number of males and females observed in each age group was similar to that expected by chance. Therefore, while the finding is significant, the relationship is not anomalous to what is expected to occur. The chi-square analysis for biological sex and age at death, however, did not produce statistically significant results for the 1910–1912 working-age mortality sample ($\chi^2 = 0.444$; df =2; p = 0.801).

Chi-square analysis for biological sex and birthplace for the 1880–1882 working-age mortality sample did not produce statistically significant results ($\chi^2 = 1.363$; df = 2; p = 0.441). Thus, the inference that biological sex and birthplace for the 1880–1882 working-age sample are related (dependent) is not supported. Results of the 1910–1912 working-age mortality for sex and birthplace are presented in Figure A-2. The mortality sample showed a relative excess of foreign-born males (*n* = 82; 14.0% of male deaths) than would be expected by chance (*n* = 65.8), but showed a relative deficiency of foreign-born females (n = 48; 8.4% of female deaths) than would be expected by chance (*n* = 64.2). This chi-square analysis for biological sex and birthplace for the 1910–1912 working-age mortality sample produced statistically significant results ($\chi^2 = 14.977$; df = 2; p = 0.001), which support the inference that biological sex and birthplace are related.



$\chi^2 = 11.014$; $df = 2$; $p = 0.004$

Figure A-1. Analysis of Biological Sex with Respect to Age at Death, Hamilton 1880-1882 Working Age Sample



$\chi^2 = 14.977$; $df = 2$; $p = 0.001$

Figure A-2. Analysis of Biological Sex with Respect to Birthplace, Hamilton 1910-1912 Working Age Sample

Results of the 1880–1882 working-age mortality sample for biological sex and occupation are presented here (Figure A-3). The mortality sample showed that more males who died worked in industrial and manual occupations (n = 182; 69.5% of male deaths) than would be expected by chance (n = 125.5), but showed that a lower number of females who died worked in industrial and manual work (n = 11; 7.8% of female deaths) than would be expected by chance (n = 67.5). Alternatively, among the females found in the mortality sample, a higher relative excess was found for household and home duties type work (n = 105; 74.5% of female deaths) than would be expected by chance (n = 37.1). Among the males found in the mortality sample, however, a lower relative deficiency was found for household and home duties (n = 1, 0.4% of males) than would be expected by chance (n = 68.9). Females, who died, also showed a relative deficiency in professional, technical, and managerial occupations (n = 3; 6.7% of female deaths) than would be expected by chance (n = 9.4). The chi-square analysis for biological sex and occupation for the 1880–1882 working-age mortality sample produced statistically high significance results (χ^2 = 272.233; df = 3; $p < 0.001$), supporting the inference that biological sex and occupation are related.

Chi-square results of the 1910–1912 working-age mortality sample for biological sex and occupation are presented here (Figure A-4). A higher relative excess of males who died worked in industrial and manual occupations (n = 437; 72.2% of male deaths) than would be expected by chance (n = 287.4); however, a lower relative deficiency of females who died worked in industrial and manual occupations (n = 29; 7.7% of female deaths) than would be expected by chance (n = 178.6). Working age males who died also displayed a relative excess in clerical, sales, and service occupations (n = 125; 20.7% of males) than would be expected by chance (n = 99.3), but a relative deficiency of females who died worked in clerical, sales, and service (n =36;

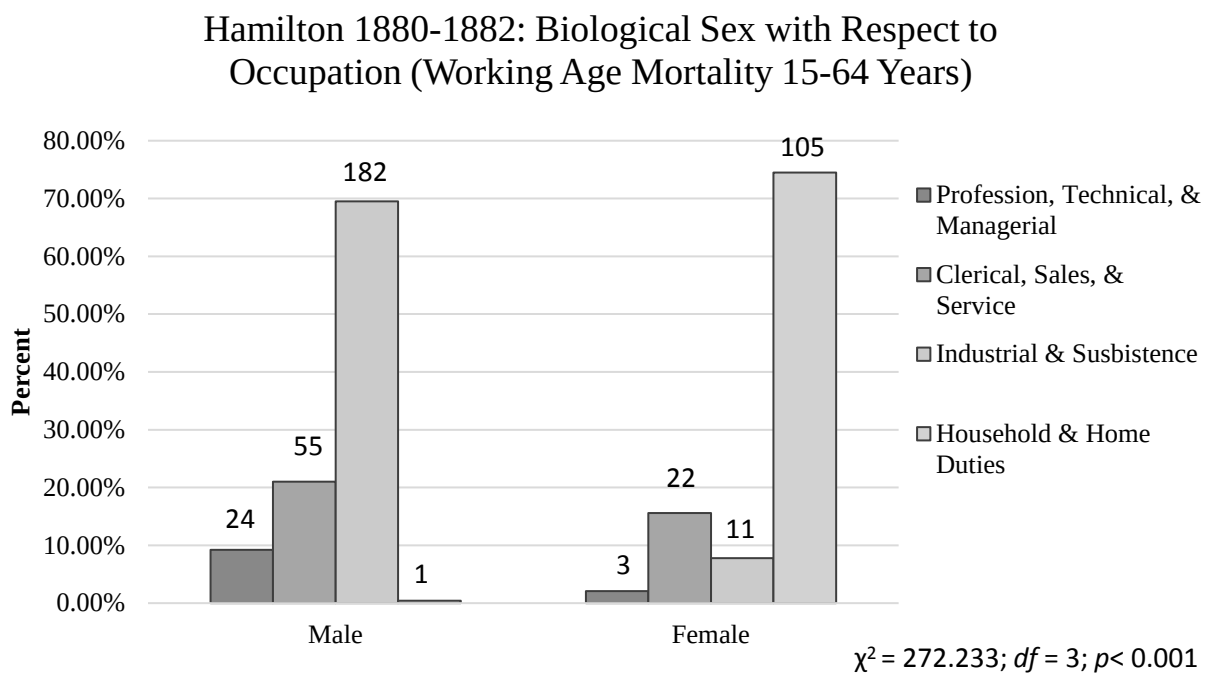


Figure A-3. Analysis of Biological Sex with Respect to Occupation, Hamilton 1880-1882 Working Age Sample

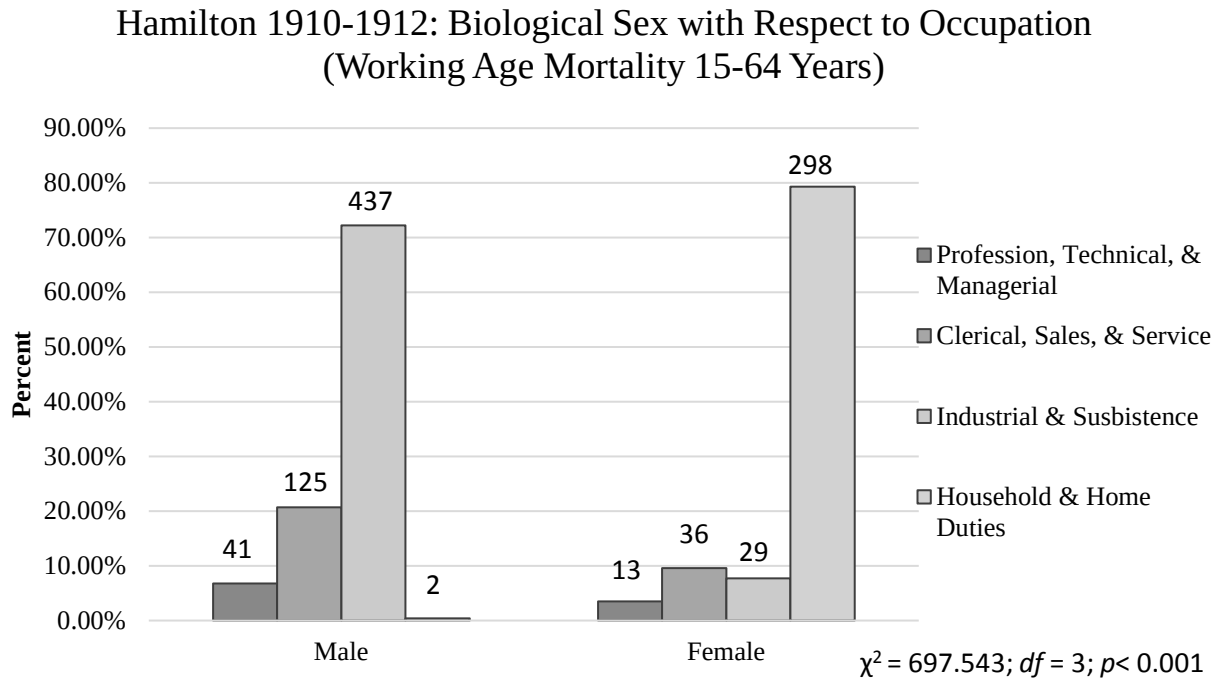


Figure A-4. Analysis of Biological Sex with Respect to Occupation, Hamilton 1910-1912 Working Age Sample

9.6% of female deaths) than expected by chance (*n* = 61.7). Alternatively, females who died presented a higher relative excess in household and home duties (*n* = 298; 79.3% of female deaths) in the mortality record than would be expected by chance (*n* = 115.0), but males who died showed a lower relative deficiency in household and home duties (n = 1; 0.4% of males) than would be expected by chance (n = 185.0). The chi-square analysis for biological sex and occupation for the 1910–1912 working-age mortality sample produced statistically high significance results (χ^2 = 697.543; df = 3; p < 0.001). These results support the inference, then, that biological sex and occupation are related.

The 1910–1912 sample, included marital status and the results of the 1910–1912 working-age mortality sample for biological sex and marital status are presented in Figure A-5. The mortality sample found that a higher number of females who died were widows (*n* = 79; 13.1% of female deaths) than would be expected by chance (*n* = 59.1); conversely, it found that a lower number of males who died were widowers (*n* = 42; 6.6% of male deaths) than would be expected by chance (*n* = 61.9). Chi-square analysis for biological sex and marital status for the 1910–1912 working-age mortality sample produced statistically high significance results (χ^2= 20.350; df = 2; p< 0.001), supporting the inference that biological sex and marital status are related.

The next analysis examined the relationship between age at death and birthplace. Figure A-6 presents the results of the 1880–1882 for age at death and birthplace among the working-age sample. The mortality sample showed a higher relative excess of young working-age deaths for Canadian-born people (n = 124; 63.9% of young working age deaths) than would be expected by chance (*n* = 70.5). As well, the sample showed a lower relative deficiency of young working-age deaths for British-born people (*n* = 53; 27.3% of young working age deaths) than would be

Figure A-5. Analysis of Biological Sex with Respect to Marital Status, Hamilton 1910-1912 Working Age Sample

Figure A-6. Analysis of Age at Death with Respect to Birthplace, Hamilton 1880-1882 Working Age Sample

expected by chance (*n* = 104.6). On the other hand, older working-age deaths showed a relative excess for British-born people (n = 143; 74.1% of older working age deaths) than would be expected by chance (*n* = 104.0), but showed a relative deficiency for Canadian-born people (n = 27, 14.0% of older working age deaths) than would be expected by chance (*n* = 70.2). This chi-square analysis produced statistically high significance results (χ^2= 111.021; df = 4; $p < 0.001$). This test, then, supports the inference that age at death and birthplace is related.

Age at death and birthplace results for the 1910–1912 working-age mortality sample are presented here (Figure A-7). Within the mortality sample, older working-age deaths presented a relative excess in the sample for British-born people (*n* = 394; 40.5% of older working age deaths) than would be expected by chance (*n* = 141.7). Younger working age, alternatively, showed a relative deficiency in the sample for British-born people (*n* = 75, 23.8% of younger working age deaths) than would be expected by chance (*n* = 107.5). Age at death and birthplace chi-square analysis for the 1910–1912 working-age mortality sample produced high statistically significance results (χ^2 = 24.978; df = 4; $p < 0.001$), supporting the inference that age at death and birthplace are related.

Chi-square analysis for age at death and occupation for the 1880–1882 working-age mortality sample did not produce statistically significant results (χ^2 = 11.733; df = 6; p = 0.068). This means that age at death and occupation for the 1880–1882 working-age sample are not related and are, thus, independent. Age at death and occupation for the 1910–1912 working-age mortality, however, did reveal a statistical relationship (χ^2 = 13.706; df = 6; p = 0.033), and the results are presented in Figure A.8. Although a relationship was identified, the standardized residual for each cell fell within the critical value (± 1.96), meaning no anomalous observations

Figure A-7. Analysis of Age at Death with Respect to Birthplace, Hamilton 1910-1912 Working Age Sample

Figure A-8. Analysis of Age at Death with Respect to Occupation, Hamilton 1910-1912 Working Age Sample

were found, or, that the number of young, middle, or older working age observed in each occupational group was similar to that expected by chance.

The 1910–1912 working-age mortality sample for age at death and marital status findings are presented in Figure A-9. The working-age mortality sample found a higher relative excess for single marital status and young working age (n = 208; 64.6% of young working age deaths) than would be expected by chance (n = 97.4). A lower relative deficiency, however, was found for young working age and married (n = 111; 34.5% of young working age deaths) and widow (n = 3; 0.9% of young working age deaths) marital statuses than would be expected by chance (n = 193.0 and n = 31.5, respectively). There was a higher number of observed cases found, however, for middle working age and married marital status (n = 339; 72.6% of middle working age deaths) than would be expected by chance (n = 280.0). On the other hand, there was a lower number of cases for middle working age and single (n = 109; 23.3% of middle working age deaths) and widow (n = 19; 4.1% of middle working age deaths) marital statuses than would be expected by chance (n = 141.3 and n = 45.7, respectively). Furthermore, older working ages in the mortality record exhibited a higher relative excess for widow marital status (n = 99; 22.1% of older working age deaths) than would be expected by chance (n = 43.8), but showed a lower relative deficiency for single marital status (n = 57; 12.8% of older working age deaths) than would be expected by chance (n = 135.3). This chi-square analysis for age at death and marital status for the 1910–1912 working-age mortality sample produced high statistically significant results (χ^2 = 338.580; df = 4; $p < 0.001$) and supported the inference that age at death and marital status are related.

The chi-square analysis for occupation and birthplace for the 1880–1882 working-age mortality sample did not produce statistically significant results (χ^2 = 5.725; df = 6; $p = 0.455$).

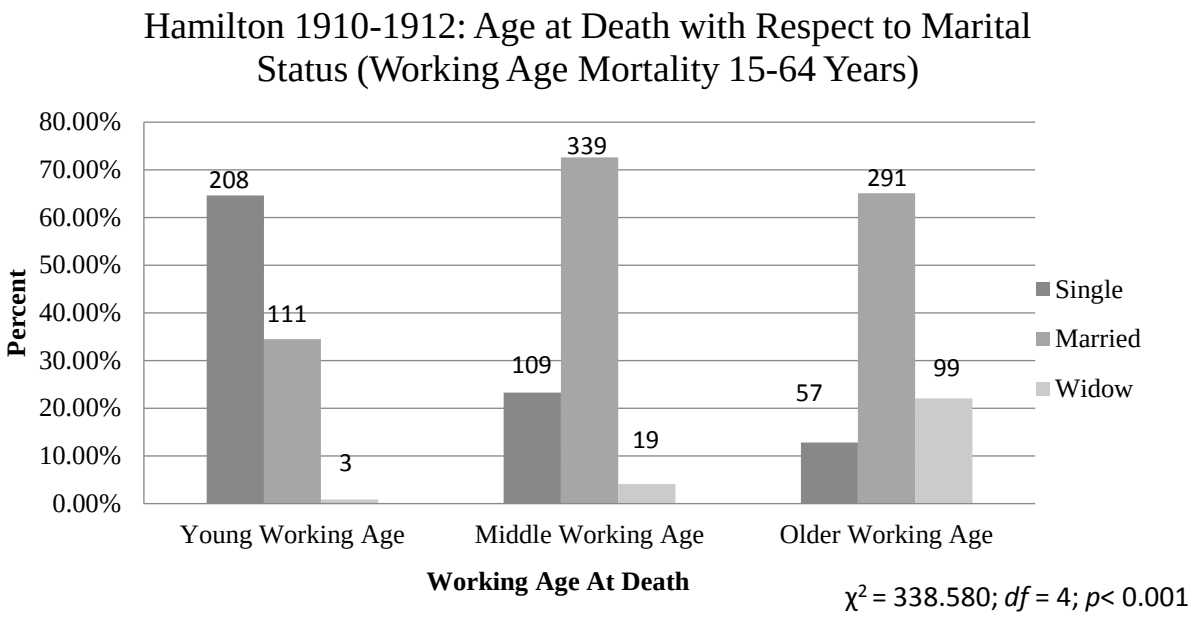


Figure A-9. Analysis of Age at Death with Respect to Marital Status, Hamilton 1910-1912 Working Age Sample

Presented in Figure A-10 are the results of the 1910–1912 working-age mortality sample for occupation and birthplace. Results revealed that, for industrial and manual occupations, there was a relative excess for foreign-born people (n = 65; 16.0% of industrial and manual occupations) in the mortality sample than would be expected by chance (n = 49.0). Alternatively, in the mortality sample, a relative deficiency of foreign-born individuals was found for occupations classified as clerical, sales, and service (n = 8; 12.0% of clerical, sales, and service occupations) than would be expected by chance (n = 16.5). This chi-square analysis (occupation and birthplace) for the 1910–1912 working-age mortality sample produced statistically significant results (χ^2 = 18.467; df = 6; p = 0.005), therefore supporting the inference that occupation and birthplace are related.

Although the chi-square analysis for occupation and marital status produced statistically significant results for the 1910–1912 working-age mortality sample, one cell had an expected

cell count lower than five. This violates the assumption that no cell has an expected count (or frequency) less than five. With this assumption violated, the chi-square distribution for occupation and marital status for the 1910–1912 working-age sample could provide misleading probabilities.

The next chi-square tested the relationship between birthplace and marital status for the 1910–1912 working-age mortality sample (Figure A-11). Results showed that, within the working-age mortality sample, Canadian birthplace had a relative excess for single marital status (n = 221; 37.0% of Canadian birthplace) than would be expected by chance (n = 184.0). British birthplace, on the other hand, showed a relative excess for widowed marital status (n = 46; 12.4% of British birthplace) than would be expected by chance (n = 33.3). There was also a relative deficiency found for single marital status among those identified in the mortality record as British born (n = 83; 22.4% of British birthplace) than would be expected by chance (n = 114.2). Chi-square analysis for birthplace and marital status for the 1910–1912 working-age mortality sample produced statistically significant results ($\chi^2 = 29.639$; df = 4; $p < 0.001$), supporting the inference that there is a relationship between birthplace and marital status.

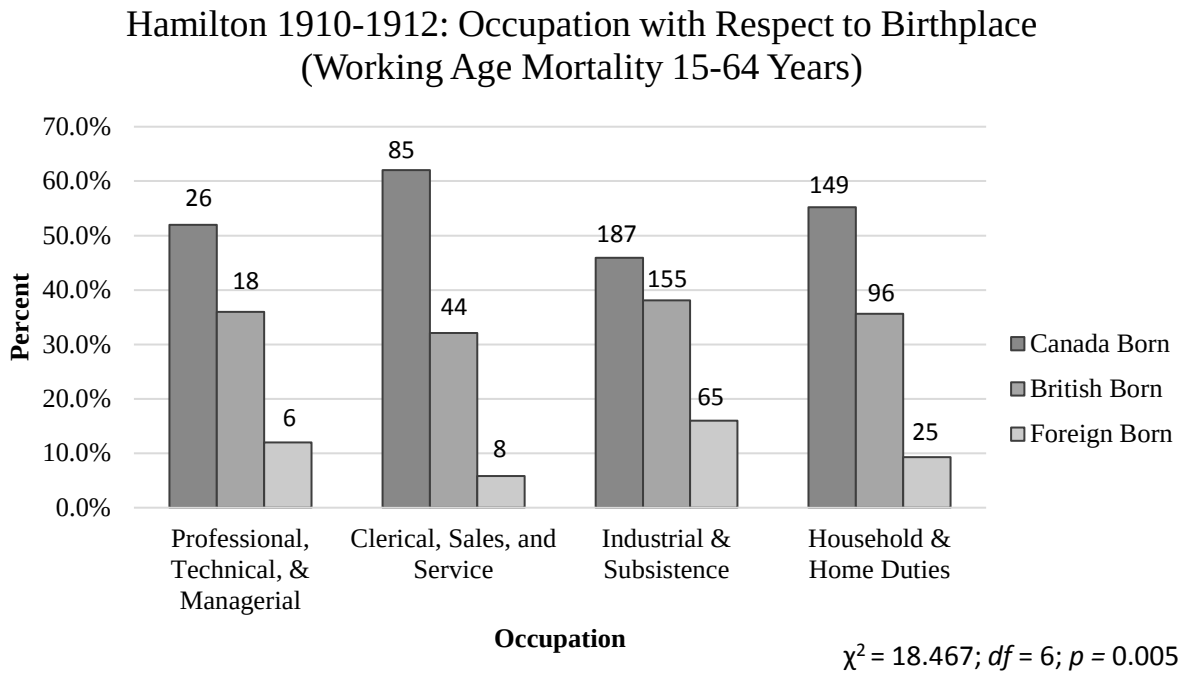


Figure A-10. Analysis of Age at Death with Respect to Birthplace, Hamilton 1910-1912 Working Age Sample

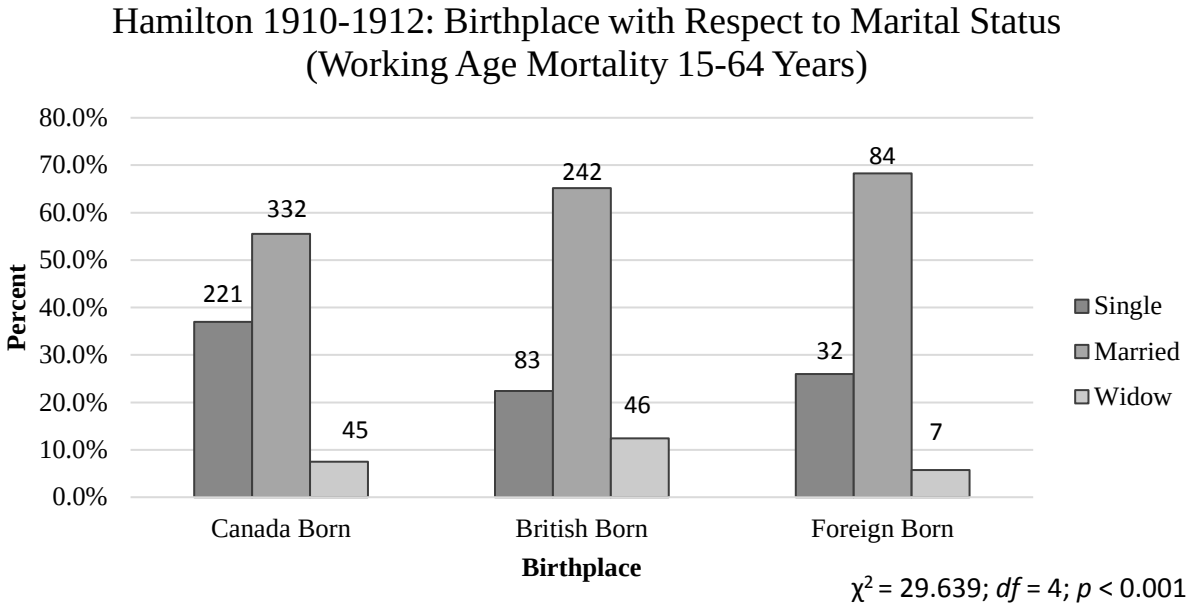


Figure A-11. Analysis of Age at Death with Respect to Birthplace, Hamilton 1910-1912 Working Age Sample

APPENDIX B: CHI-SQUARE RESULTS FOR INDEPENDENT VARIABLES: INFANT MORTALITY SAMPLE

Presented here are the chi-square results for the independent variables for the infant mortality sample. The chi-square analysis for biological sex and age at death produced only slightly statistically significant results for the 1880–1882 infant mortality sample ($\chi^2 = 4.157$; df =1; p = 0.041). Figure B-1 presents the results of this analysis. Although a statistical relationship was identified, the standardized residual for each cell fell within the critical value (± 1.96), meaning no anomalous observations were found, or that the number of males and females observed in each age group was similar to that expected by chance. As well, the chi-square analysis for biological sex and age at death for the 1910–1912 infant mortality sample did not produce statistically significant results ($\chi^2 = 0.035$; df =1; p = 0.851), suggesting there was no relationship between biological sex and age at death in the later period.

The chi-square analysis for biological sex and birthplace also produced only slightly statistically significant results for the 1880–1882 infant mortality sample ($\chi^2 = 1.075$; df =1; p = 0.03). Figure B-2 presents the results of this analysis. Again, a statistical relationship was identified; however, the standardized residual for each cell fell within the critical value (± 1.96), meaning no anomalous observations were found, or that the number of males and females observed in each birthplace group was similar to that expected by chance. As well, the chi-square analysis for biological sex and age at death did not produce statistically significant results for the 1910–1912 infant mortality sample ($\chi^2 = 0.035$; df =1; p = 0.851). For the later period the inference of a relationship between these two independent variables is not supported.

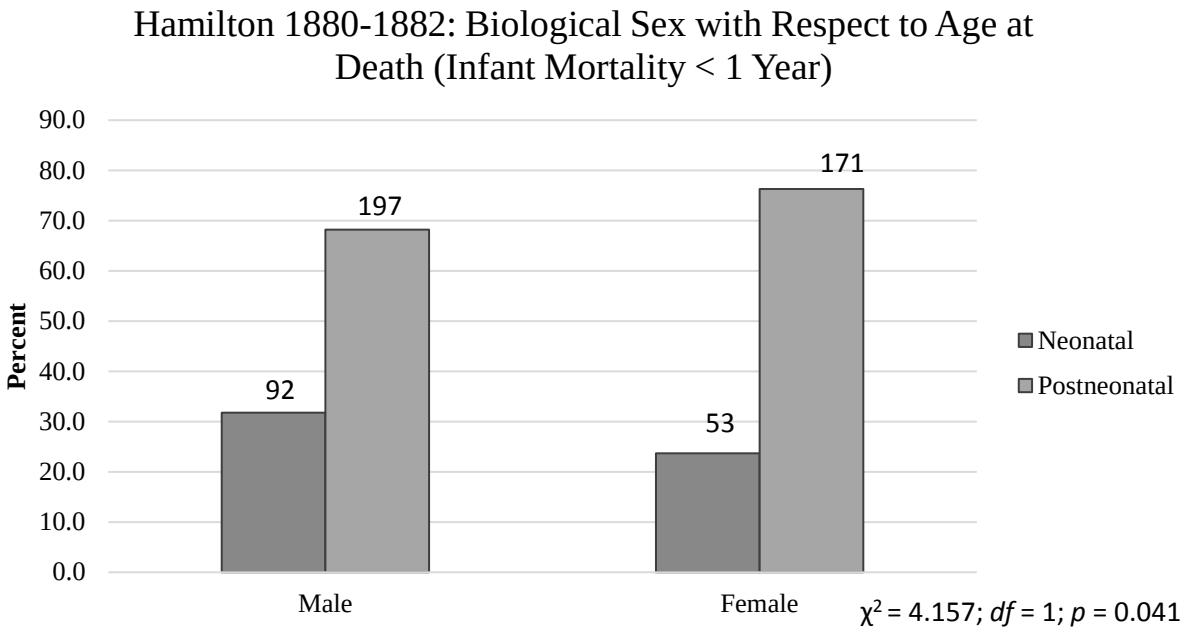


Figure B-1. Analysis of Biological Sex with Respect to Age at Death, Hamilton 1880-1882 Infant Mortality Sample

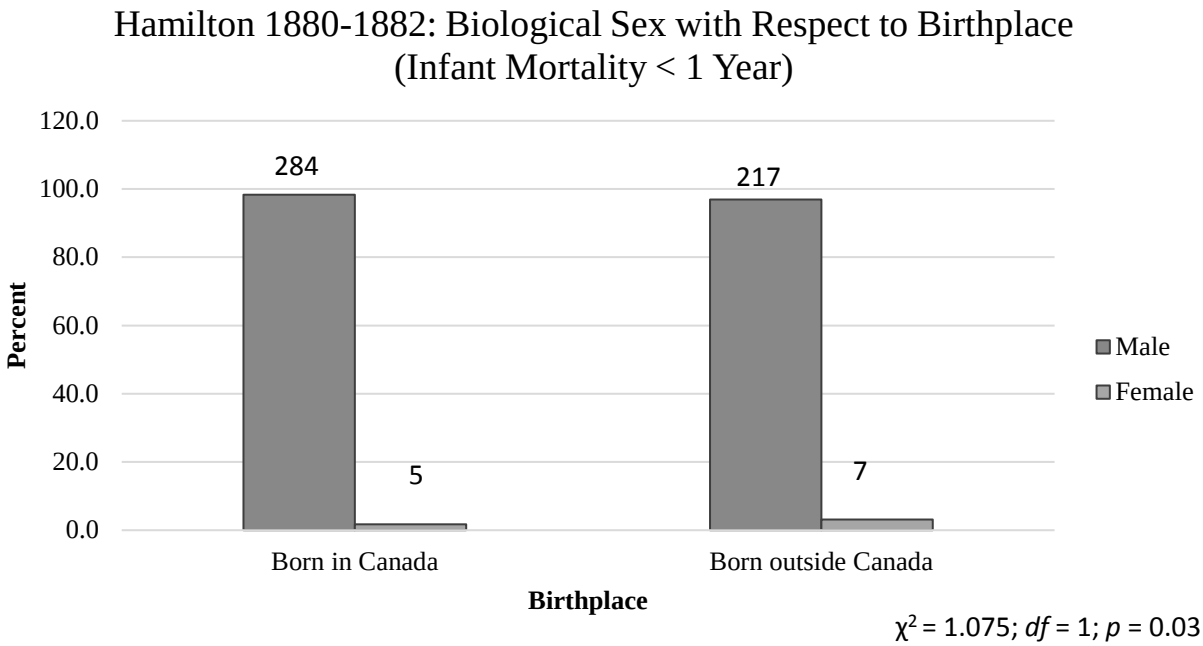


Figure B-2. Analysis of Biological Sex with Respect to Birthplace, Hamilton 1880-1882 Infant Mortality Sample

Results of both the 1880–1882 and 1910–1912 infant mortality sample for biological sex and seasonality did not produce statistically significant results ($\chi^2 = 0.200$; df =3; p = 0.978; $\chi 2$ = 4.747; df =3; p = 0.191, respectively). Overall, biological sex did not reveal any anomalous observation with other predictors than what was to be expected by chance.

Although the chi-square analysis for infant age at death and birthplace produced slightly statistically significant results for the 1880–1882 infant mortality sample, one cell had an expected cell count lower than five, and therefore violated the assumption that no cell has an expected count less than five. With this assumption violated, the chi-square distribution for age at death and birthplace for the 1880–1882 infant mortality sample may provide misleading probabilities.

Chi-square results of the 1910–1912 infant age mortality sample for age at death and birthplace are presented in Figure B-3. The mortality sample showed a relative deficiency of neonatal deaths for infants who were born outside of Canada (n = 0; 0.00% of neonatal deaths) than would be expected by chance (n = 5.7). In other words, none of the neonatal infants found in the death registry were born outside of Canada, yet there is an expectation that at least five of the neonatal infants who died would have been born outside of Canada. This chi-square analysis for age at death and birthplace for the 1910–1912 infant mortality sample produced statistically significant results ($\chi^2 = 9.043$; df = 1; p = 0.003). The inference, then, that the variables age at death and birthplace are related (dependent) is supported.

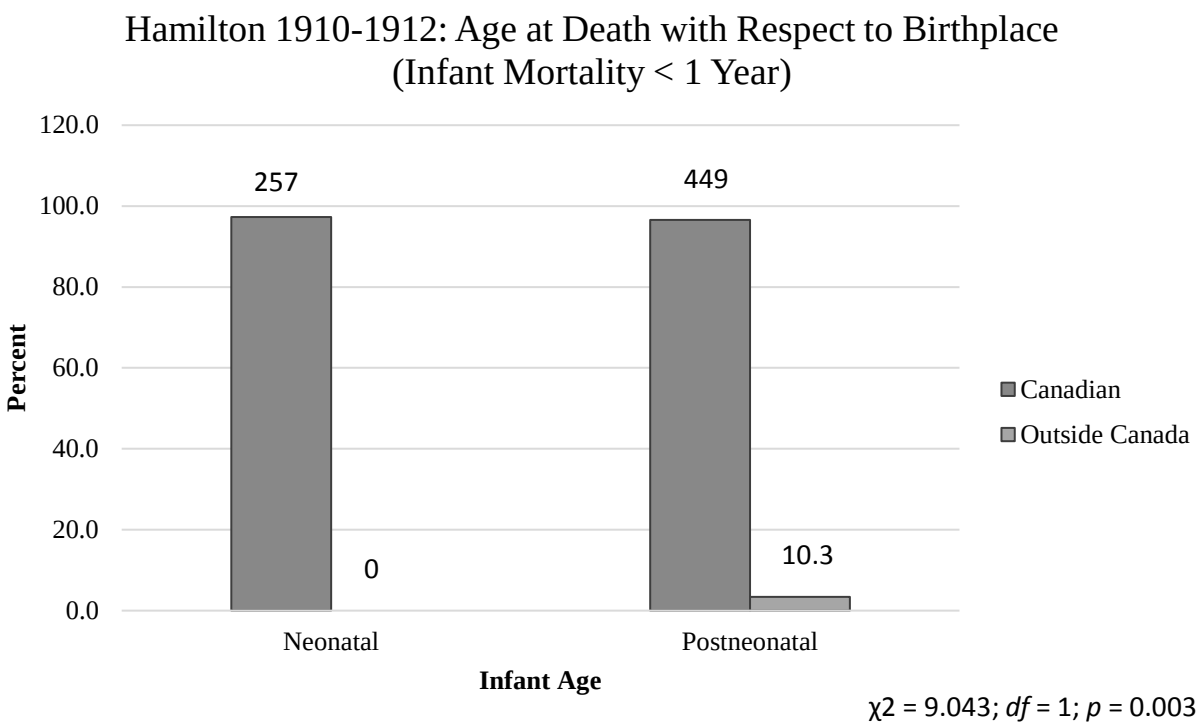


Figure B-3. Analysis of Age at Death with Respect to Birthplace, Hamilton 1910-1912 Infant Sample

Results of the 1880–1882 infant mortality sample for age at death and seasonality are presented in Figure B-4. These findings showed that, within the death registry, neonatal age had a higher number of observed cases found in the winter months (n = 44; 30.30% of neonatal age deaths) and a lower number of observed cases found in the summer months (n = 34; 23.40% of neonatal age deaths) than would be expected by chance (n = 31.1 and n = 50.0, respectively). Postneonatal age also revealed a statistical relationship; however the standardized residual for each cell fell within the critical value (± 1.96), implying no anomalous observations were found, or, that the ages observed in each seasonal group were similar to that expected by chance. The chi-square analysis for age at death and seasonality produced high statistically significant results for the 1880–1882 infant mortality sample (χ^2 = 15.688; df = 3; p = 0.001), supporting the inference of a relationship between the two variables.

The next chi-square results of the 1910–1912 infant mortality sample tested independence between age at death and seasonality (Figure B-5). Within the infant mortality sample, neonatal age was found to have a higher number of cases occurring in the spring months (n = 73.0; 27.70% of neonatal age deaths) and a lower number of cases occurring in summer months (n = 62.0; 23.50% of neonatal age deaths) than would be expected by chance (n = 56.0 and n = 86.0, respectively). As well, postneonatal age revealed a statistical relationship; however the standardized residual for each cell fell within the critical value (± 1.96), and therefore no anomalous observations were found, or the observed cases were similar to that expected by chance. The chi-square analysis for age at death and seasonality did produce high statistically significant results for the 1910–1912 infant mortality sample (χ^2 = 21.710; df = 3; p < 0.001), and, therefore, supports the inference that age at death and seasonality are related.

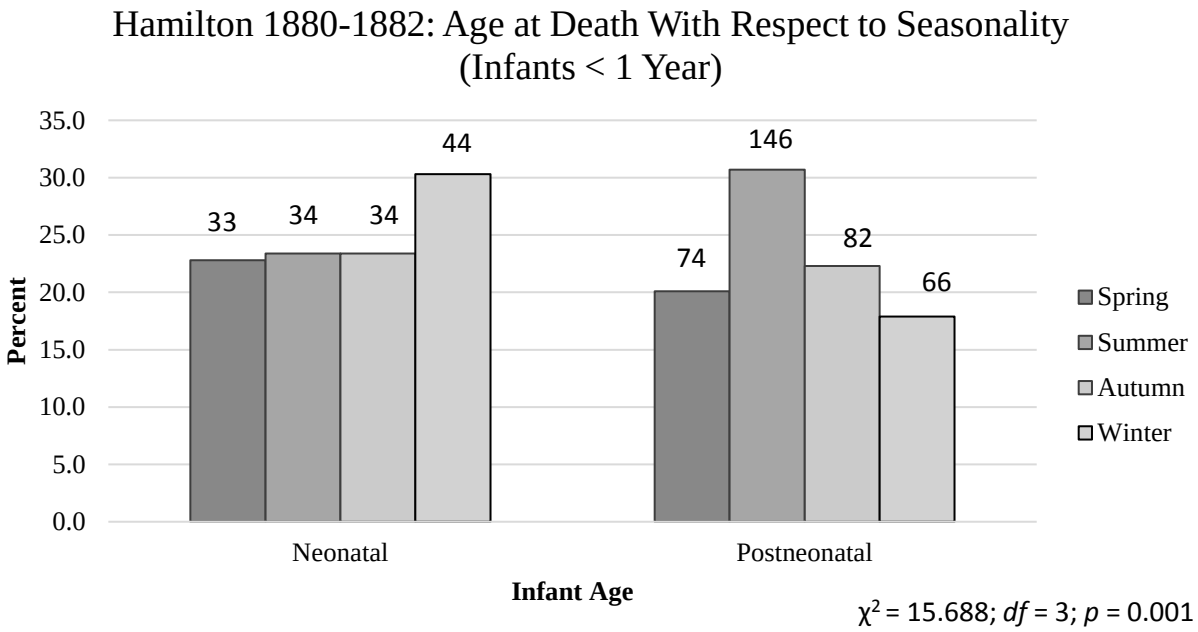


Figure B-4. Analysis of Age at Death with Respect to Seasonality, Hamilton 1880-1882 Infant Mortality Sample

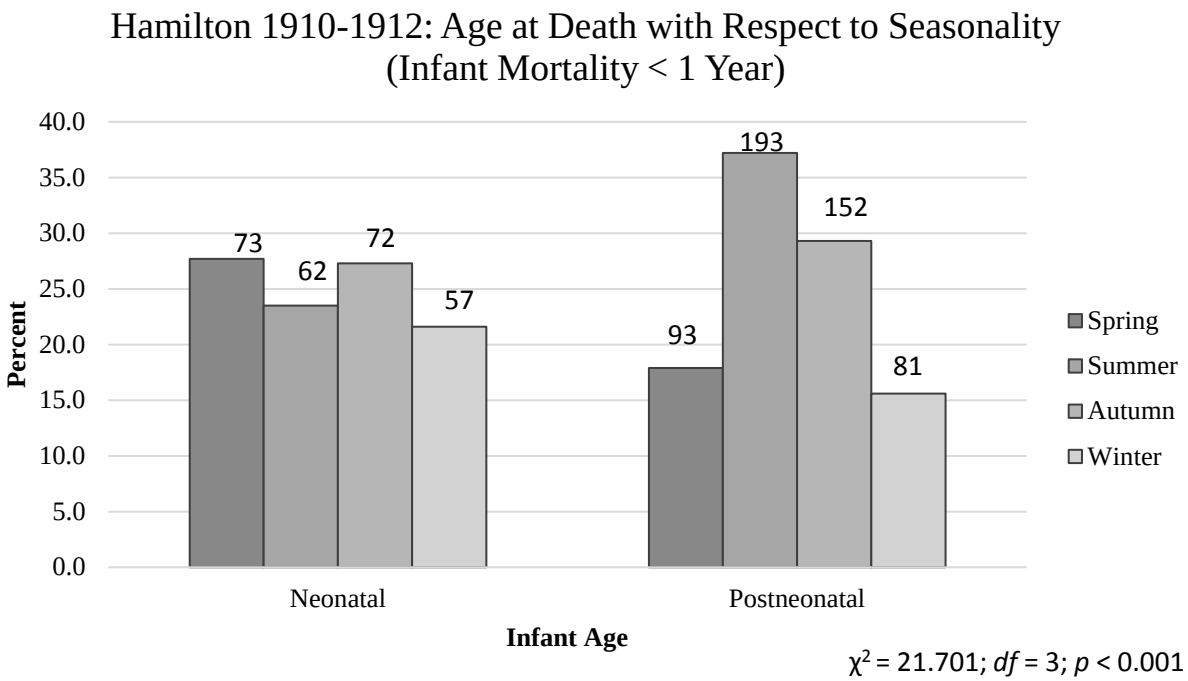


Figure B-5. Analysis of Age at Death with Respect to Seasonality, Hamilton 1910-1912 Infant Mortality Sample

Birthplace and seasonality results for the 1880–1882 infant mortality sample did not produce statistically significant results ($\chi^2 = 7.096$; df = 3; p = 0.185). Although the chi-square analysis for infant birthplace and seasonality produced slightly statistically significant results for the 1910–1912 infant mortality sample, three cells had an expected cell count lower than five, which violated the assumption that no cell has an expected count less than five. This could lead to misleading probabilities for the chi-square distribution for birthplace and seasonality for the 1910–1912 infant mortality sample.