

POLIO MORTALITY PATTERNS IN SOUTHERN ONTARIO (1900-1937)

EXAMINING MORTALITY PATTERNS IN THE EPIDEMIC EMERGENCE OF
POLIOMYELITIS IN SOUTHERN ONTARIO, CANADA (1900-1937)

By

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ABSTRACT

This thesis examines patterns in poliomyelitis mortality in Wentworth and York Counties of southern Ontario, Canada, from 1900 to 1937. This period marked polio's shift from endemic to epidemic status. It was also a time of shifting social, cultural, demographic, and economic patterns. Contemporaries struggled to understand polio's epidemiology, and even today, with the poliovirus on the verge of global eradication, models to explain its changing patterns and impact continue to be revised.

This thesis uses both qualitative and quantitative data collected from a variety of archival sources, including death, birth, and marriage registrations, census records, and newspaper articles, among other records. This information was used to build a geodatabase which forms the basis for analyses of mortality patterns in relation to age and sex, illness duration, seasonality, nativity, birthplace, ethnicity, and religion. Further analyses included family size, birth order, socioeconomic status, and place of residence patterns.

Examined in the context of Wentworth and York Counties in the early 20th century, the results both reveal a local pattern to polio's epidemic emergence and provide a means to test broader hypotheses regarding polio's epidemiological patterns. Specifically, results from this study were compared to the expectations of the intensive-exposure and cross-sex transmission hypotheses proposed by Nielsen and colleagues. Among the most important contributions of this thesis are the results showing a pattern of change over the study period, with two distinct stages. Stage One (1910 to 1927) is characterized by an equal sex ratio and a median known family size of four. Stage Two (1928 to 1937) is characterized by excess male deaths and a median known family size of two. These results link polio mortality patterns to demographic and ecological shifts in the early 20th century and confirm that there is still much to learn from the history of this disease.

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When began this research, I thought the poliovirus would be eradicated by the time I completed my dissertation. Polio is still out there, but as I write this India has now passed 30 months without a new case. Though perhaps not as soon as many had hoped, I think we will see a polio-free world in the not-too-distant future. I dedicate this thesis to all those affected by this disease.

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LIST OF ALL ABBREVIATIONS AND SYMBOLS

GIS	Geographic Information System(s)
IE	Intensive-Exposure
ICM	Infant and Childhood Mortality
ICMR	Infant and Childhood Mortality Rate
IMR	Infant Mortality Rate
SES	Socioeconomic status

DECLARATION OF ACADEMIC ACHIEVEMENT

I declare that the content of the research in this document has been completed by myself, Heather Battles, with recognition of the contributions of Dr. D. Ann Herring, Dr. Tina Moffat, and Dr. Michael Mercier in both the research process and the completion of the thesis.

Chapter 1 Introduction

Poliomyelitis, once known as “infantile paralysis”, emerged as an epidemic disease in developed countries in the late 19th and early 20th centuries. The known history of epidemic polio begins in Scandinavia. There were small outbreaks first in Norway in 1868 and Sweden in 1881, followed by scattered outbreaks throughout Europe from the 1880s and in England from the 1890s (Smallman-Raynor, Cliff, Trevelyan, Nettleton, & Sneddon, 2006). The first significant epidemic in North America occurred in Rutland County, Vermont in 1894 following an outbreak in Boston the previous year (Paul, 1971; Smallman-Raynor et al., 2006). The Rutland epidemic saw 18 deaths out of 132 cases (Smallman-Raynor et al., 2006). In North America, polio was concentrated in the northeast, with the exception of outbreaks in Alabama in 1896 and California in 1896, 1899, and 1901 (Holt & Bartlett, 1908, Batten, 1911, and Lavinder, Freeman, & Frost, 1918, as cited in Smallman-Raynor et al., 2006).

Polio epidemics increased as the 20th century began, both in Europe and North America. Scandinavia saw major epidemics in 1905 (over 2,000 cases) and 1911-13 (nearly 12,500 cases) (Low, 1917 and Bertenius, 1947, as cited in Smallman-Raynor et al., 2006). New York City experienced a major epidemic of 2,500 cases in 1907, with outbreaks in five other states that year and nine states in 1908 (Smallman-Raynor et al., 2006). 1909 saw epidemics in two US states and multiple European countries, including England, Austria, Germany, France, Holland, and western Russia (Bertenius, 1947, as cited in Smallman-Raynor et al., 2006). Canada’s first significant polio epidemics

occurred in Montreal, Quebec in 1909 and Hamilton, Ontario in 1910. Data from family surveys across the urban United States in 1935-1936 indicate that for every 1,000 children born to native white parents between 1911 and 1915, 11 would experience a case of polio before age 25, with 1.58 of these cases being fatal and 1.6-2.4 survivors being left with residual effects (Collins, 1946).

In temperate climates, these epidemics followed a seasonal pattern, which in North America was summer-autumn, shifting to slightly later in the year as latitude increased. Thus in North America polio was known as the “summer plague” and in Sweden as “höstens spoke” (“autumn’s ghost” or “the autumn ghost”) (Axelsson, 2004). In Canada and the northeast United States the height of the polio season usually occurred in August and September (Nathanson & Martin, 1979).

This thesis aims to examine the local expression of polio’s global shift from endemic to epidemic disease. This study examines two counties in southern Ontario, Canada: Wentworth County, which contains the city of Hamilton, and York County, in which the city of Toronto is located. In the early 19th century, both were large, growing cities with outlying suburban and peri-urban areas. Both cities were popular destinations for both immigrants and migrants from other parts of the province and country. Hamilton was an industrial centre and immigrants were attracted by its expanding industrial economy. The population of Hamilton in 1900 was over 50,000, reaching 115,000 by the 1920s and over 155,000 by the 1930s, while Toronto’s population was over 200,000 in 1900 and more than tripled to over 630,000 by the 1930s (Canada, Dominion Bureau of Statistics, 1901-1941). This growth was not steady; Hamilton grew from 114,000 in 1921

to 155,000 in 1931 through immigration, a high birthrate, and declining mortality (Weaver, 1982, p. 137). However, in the 1930s, Hamilton's population growth stagnated due to a falling birthrate and fewer immigrants (Weaver, 1982), even falling 1.4% between 1931 and 1936 (Wood, 1987).

Hamilton's population was largely blue collar working class, with a large number of men employed at the steel and construction industries, and became increasingly so in this period as the city became more industrial and less commercial (Wood, 1987). Offices relocated to Toronto, taking white collar jobs such as clerks and bookkeepers with them (Wood, 1987). Toronto then saw an expansion in its white collar population. Between the 1911 and 1921 census years, the number of white collar workers in Toronto increased 33%, and blue collar workers declined 2.8% (Piva, 1979, p. 15). This meant that only 58.9% of Toronto's workforce was blue collar in 1921, compared to 66.3% in 1911 (Piva, 1979, p. 15). Furthermore, when the Depression hit in 1929, white collar workers were impacted less than blue collar workers. In 1935, 60% per cent of relief recipients in Hamilton were connected with the building trades (Weaver, 1982, p. 131). Other workers kept their jobs but had their hours reduced. Transportation and commercial sectors did better than manufacturing. At the worst of the Depression in 1933, 25% of Hamilton families were receiving relief (Weaver, 1982).

Both Hiebert (1995) and Mercier (2006) found that in the early twentieth century Toronto's residential geography was divided primarily along ethnic and religious lines, with occupation or class playing a secondary role. Hiebert (1995) found that in Toronto in 1931, ethnic groups were twice as clustered as occupational groups. British immigrants

tended to gravitate away from the inner city to the periphery of the city, while East-Europeans clustered in areas with the cheapest housing, particularly the area south of Queen Street and the Junction (Hiebert, 1995). The Jewish community in Toronto was the most segregated, clustered especially in the Ward in 1901 and around Spadina Avenue as of 1931. However, the interrelationship of ethnicity and the labour market meant that ultimately “Toronto’s social geography was characterized by a complex interweaving of class and ethnic patterns” (Hiebert, 1995, p. 64). Residence was often determined in part by proximity to place of employment of the male household head. For example, the Ward was close to garment factories, where many Jews worked, and factory employees wanted to live close to work because they worked long hours and wanted short travel time and the ability to walk to work to avoid cost of the streetcar (Speisman, 1985, p. 109). Similarly, East-European men were employed in large numbers in rail yards and in construction, hence their concentration in areas like the Junction (Hiebert, 1995).

In Hamilton, ethnicity and class were also reflected in the city’s social geography. For example, the wealthy Durand neighbourhood in the city’s southwest remained predominantly British and Protestant until mid-twentieth century, though it began to become more middle-class during the Depression (Manson, 2003). Industrial workers were clustered in the north near the factories. Wood (1987, p. 134) notes that by WWII, working-class areas were divided in part along ethnic lines, by country of origin, language, and religion.

In particular, this thesis seeks to explore the utility of the hygiene hypothesis and newer models, such as Nielsen et al.’s (2001; 2002a) Intensive-Exposure model, for

southern Ontario in the early 20th century. The emergence of a ‘new’ infectious disease may be due to genetic changes in the pathogen or to ecological changes which disrupt the evolved state of equilibrium between pathogen and host (Smallman-Raynor, et al., 2006, p. 11). Lederberg, Shope, & Oaks (1992) outline six areas in which changes will impact the human-microbial equilibrium: human demographics and behavior; technology and industry; economic development and land use; international travel and commerce; microbial adaptation and change; and breakdown of public health measures. The majority of emergent viral diseases may be due to changes such as these, changes which are the result of human action (Morse, 1993). Two of these, changes in human demography and (if “breakdown” is broadened) changes in public health measures, are thought to play a central role in the epidemic emergence of poliomyelitis. Krause (1998) notes that “a complex matrix of social, economic, political, and ecological factors play a major role in the emergence of epidemics due to microbes with which the human population has coexisted for centuries, if not millennia” (p. 20). Luria (1996), however, cautions that the many aspects of life (such as human behavior, urbanization, public health infrastructure breakdown, and poverty) thought to contribute to the emergence and re-emergence of infectious diseases tend to be viewed in isolation from each other, whereas they are more often “interrelated, additive, and synergistic” (p. 60). This is certainly evident in the case of the epidemic emergence of polio, though in this case the usual elements worked together in somewhat unexpected and ironic ways.

Polio was an emerging epidemic disease in a very dynamic period of time in Western industrialized societies that has come to inform much thinking about the

evolution of population health patterns, specifically epidemiologic transition theory. It is thus important to place this study of polio mortality in early 20th-century southern Ontario in the broader health transition context.

Omran's (1971) concept of epidemiologic transition first divided the model into three stages: the Age of Pestilence and Famine, the Age of Receding Pandemics, and the Age of Degenerative and Man-Made Diseases. In the Classical (Western) model, the shift between the first two Ages occurred as Western countries industrialized and modernized in the late 18th and 19th centuries, while the shift between the latter two Ages occurred in the early 20th century (Omran, 1971).

Others have expanded and modified Omran's (1971, 1983) initial formulation of epidemiologic transition. Perhaps the most significant of these contributions to date has been Barrett et al. (1998). Barrett et al. begin with a Paleolithic baseline and outline three successive transitions. The first transition is associated with the advent of agriculture and subsequent increase in infectious disease mortality. The second epidemiologic transition coincides with the Industrial Revolution in mid-19th-century Europe and North America. It is marked by major declines in infectious disease mortality in these regions. Chronic and degenerative diseases, often referred to as "diseases of civilization", instead became the leading causes of human morbidity and mortality. The third transition is characterized by newly emerging infectious diseases (whether because the diseases are actually 'new' to human populations or because of improved detection), re-emerging infectious diseases, and the evolution of antimicrobial resistance.

It was against the backdrop of the second transition that polio ‘emerged’. Barrett et al. (1998) describe the chronic and degenerative diseases such as cancer, diabetes, coronary heart disease, hypertension, allergies, depression and anxiety as the “tradeoffs” for the decline in infectious diseases in industrialized societies (p. 255). Barrett et al. (1998) describe how “some health tradeoffs of the second transition concern the role of industrial technology in the creation of certain artificial environments...” (p. 255). While Barrett et al. (1998) were not speaking explicitly of the hygiene hypothesis, epidemic polio, the “disease of development” (Bunimovich-Mendrazitsky & Stone, 2005), could be considered another of these “unforeseen consequences” (Barrett et al., 1998, p. 255), tying the hygiene hypothesis of polio’s epidemic emergence into epidemiologic transition theory.

In the case of Western industrialized countries, there is general agreement that child mortality rates began to decline between the 1860s and 1880s; Grundy (2005) specifies that for England, mortality rates for older children (ages 5 and over) began to fall in the 1860s, while those for young children (ages 1 to 4) began to decline in the 1880s. Preston and Haines (1991) argue that a lack of available data makes establishing the timing of the decline in the United States difficult, but that their data show that child mortality was declining in that country in the decades before 1900. However, it is the timing of the decline of IM which has received the most attention. McKeown (1976), Szreter (1988), and Grundy (2005) date the start of the decline of IM in England to the beginning of the 20th century, characterized by a sudden drop after 1900. Woods, Watterson, and Woodward (1988; 1989) instead trace the English IM decline back to the

late 1880s and early 1890s when diarrheal diseases are discounted, and Williams and Galley (1995) and Morgan (2002) trace it back even farther, to 1870. Morgan (2002) argues that his study of the town of Preston, England shows a decline in IM began in the 1870s before being interrupted by an increase in diarrheal mortality between 1880 and 1900, when it then began to decline once again.

The Canadian evidence so far appears to fit the pattern seen in England and the US with childhood mortality beginning to decline earlier, in the late 19th century, and IM not showing any significant declines until the start of the 20th century (McInnis 1997). However, the patterns in the Canadian studies so far appear to be more complicated and show a bit of a delay in the declines. Gagan (1981) argues that not only did infant and child mortality (ICM) not decline until 1912 in Ontario, but that “when general mortality rates were decreasing throughout the western world, mortality rates in Ontario, its cities, and in Hamilton increased” (p. 213). Mercier and Boone (2002) also date the decline of IM to 1912, noting for Ottawa that while the first phase of a decline began at the beginning of the 20th century, this was interrupted by epidemics of typhoid and smallpox, thus breaking the decline into two phases.

This study focuses on Wentworth and York Counties, which contain the large urban centres of Hamilton and Toronto, their suburbs, and surrounding rural areas. Their social and geographic diversity, as well as their large populations, make it a useful study area with a relatively large number of polio deaths. Ontario’s relative completeness of death registration records and the availability of other detailed data, such as maps, city directories, and media reports, means that the data collected are among the most reliable

available for this period. Furthermore, the variety of both quantitative and qualitative sources allows a more comprehensive analysis than would be possible for many other places.

The following broad questions guided this research:

- 1) What was the distribution of polio mortality in Wentworth and York Counties in this period:
 - a. Demographically (by age, sex, family size, et cetera)?
 - b. Socioeconomically?
 - c. Geographically?
 - d. Among different cultural, ethnic, or religious groups?
 - e. Among the native- versus foreign-born?
- 2) How do these elements and variables intersect and interact?
- 3) How do these patterns compare with existing models of polio, particularly the predictions of Nielsen et al.'s (2001; 2002a; 2002b) Intensive-Exposure model and cross-sex transmission hypothesis?

This research contributes knowledge about an underrepresented place and time in polio's history, as previous research has tended to focus on epidemics in the United States and the post-World War II period. It addresses the call of Trevelyan, Smallman-Raynor, and Cliff (2005, p. 288) for "analyses at finer geographic scales" and of early polio epidemics to reveal more about the processes which drove the emergence of epidemic polio. It also evaluates the utility and relevance of Nielsen et al.'s models of polio severity to another setting. The epidemiology of polio is now thought to be more complex

than was previously believed (Andrus, 2002), and this thesis seeks to explore that complexity from an anthropological perspective.

My personal interest in polio was sparked by a project for an undergraduate seminar course on epidemics and public health. My aunt caught polio in 1949 and the family was quarantined for two weeks. I interviewed my mother's eldest sister for the project, who recalled that they had thought the younger sister contracted polio on Long Island in the Rideau River near Ottawa, Ontario. The topic of polio spoke to my research interests in children and childhood, disability, and the evolutionary history of human disease ecology. I have continued to do research on different aspects of polio since then. This dissertation project in particular has allowed me to apply my background and training in physical anthropology, medical anthropology, and Canadian history.

Whenever I have spoken about my research on polio, whether to academic colleagues or Rotarians at a fundraising event, invariably someone will mention that they themselves, or an older relative, had polio. I have heard many stories of how terrified parents were of this particular disease; strong memories remain of parental admonitions to avoid swimming in the lake, or eating ice, or any number of other things potentially linked to polio. With the epidemic echo of Post-Polio Syndrome, in which symptoms of muscle weakness and breathing difficulties can resurface decades after the initial infection, many such memories are resurfacing and reigniting interest in this disease which has been banished from Western countries by vaccination.

This dissertation is organized into seven chapters. Following this introduction, Chapter 2 is divided into two main parts. The first covers the background and context of

both polio and the historical study site, Wentworth and York Counties from 1900-1937.

The second part of Chapter 2 lays out the theoretical framework for this study, examining the literature on the emergence of epidemic infectious disease and the theories regarding polio's epidemic emergence. Sanitation and health patterns as well as socioeconomic class receive particular attention.

Chapter 3 is again divided into two parts. The first describes the materials from which the data for this study were gathered and addresses issues of data quality. The second details the methods employed in this study, from the collection of data, to construction of the geodatabase, to data analysis.

Chapter 4 is the first of two chapters presenting and discussing the results of the data analysis. This chapter focuses on patterns of polio mortality and demography in terms of age and sex, illness duration and seasonality, and birthplace, ethnicity, and religion. Chapter 5 investigates family size, birth order, socioeconomic status, and place of residence patterns, including how these interact with the elements examined in Chapter 4. These results are compared with other studies and against the predictions of the various models and hypotheses, particularly Nielsen et al.'s (2001; 2002a; 2002b) Intensive-Exposure model and the cross-sex transmission hypothesis. Explanations for the findings are sought based on existing research of polio studies elsewhere as well as research and knowledge of the local circumstances at the time.

Finally, Chapter 6 explores how the models inform understandings of polio patterns in Wentworth and York Counties between 1900 and 1937, and how evidence from this study can in turn inform models of both polio and health and disease more

broadly. Chapter 7, the conclusion, summarizes the significant findings of this study and makes some general observations, as well as considering avenues for future research and what the anthropology of infectious disease can contribute to work in this area.

Chapter 2 Background and Theoretical Framework

Introduction

This chapter first covers the background and context of both polio and the historical study site, Wentworth and York Counties from 1900-1937. I discuss the general health and sanitation conditions in both Wentworth and York Counties and the history of polio epidemics in the area. I also discuss the consequences of chronic polio for survivors. I then examine the relationship between polio and socioeconomic status, in the context of the early 20th century and the relationship between socioeconomic status and infectious disease risk in infants and children more generally.

Subsequently, I lay out the theoretical framework for this study, examining the literature on the emergence of epidemic infectious disease and the theories regarding polio's epidemic emergence. I examine the hygiene hypothesis, as well as other factors believed to play a role in polio's transition from endemic to epidemic. Finally, I discuss the more recently introduced Intensive-Exposure model, which emphasizes the role of virus transmission within households.

Background

Sanitation and health conditions in Wentworth and York Counties

The early twentieth century was a time of significant changes in infant and child health in the region. While health improved significantly in many ways, there were major challenges, especially in the first two decades. The infant mortality rate did not drop below 135 in Toronto until 1914, and between 1901 and 1921 was only below 100 in

1917 and 1921 (Piva, 1979, p. 114). In fact, at one point IMR actually rose in Toronto from 141.4 in 1902 to 179.7 in 1909 (Piva, 1979, p. 123). Gagan (1981) describes a similar pattern for Hamilton, where deaths from cholera infantum in particular increased between 1905 and 1910. According to Gagan (1981), major change in mortality rates in Hamilton came only after 1920.¹

These mortality rates owe much to the sanitary situation in these areas in the early twentieth century. Both Toronto and Hamilton lacked adequate methods of sewage disposal and suffered problems related to contamination of their water supplies. Figure 2.1 shows a sewage outlet at the foot of Toronto's Yonge Street around 1907. Toronto seems to have addressed sanitary issues earlier than Hamilton (see Table 2.1). After 1911 Toronto no longer dumped raw sewage directly into the Bay, a short distance from the city's water intake (Piva, 1979). This made a major difference in typhoid fever deaths in particular (Piva, 1979, p. 118). In Hamilton, sewage was discharged into the harbour either without any treatment, or with minimal treatment to break up large particles (Wood, 1987). The water was not officially declared to be polluted until 1923, which then ended the practice of cutting ice from the harbour for domestic use (Wood, 1987). Wood (1987) describes how "as late as 1927, the public were still using a 'bathing beach' at the foot of Wentworth Street, despite the presence of nearby sewage outlets both to the east

¹ IMR (the number of deaths in the first year of life per 1,000 live births) is used as an indicator of overall socioeconomic and health conditions in an area or society. A high IMR implies low levels of hygiene and sanitation. Payne (1955) and Paul (1958) investigated the possible relationship between IMR and the appearance of polio epidemics. Paul (1955, p.753-754) identified an IMR of around 60-80 as the "critical infant mortality zone" or "sensitive zone" in which polio was likely to transition from an endemic disease to an epidemic one. Smallman-Raynor et al. (2006, p. 215-221) have investigated this further.

and west” (p. 131). Water quality in Hamilton’s harbour deteriorated further, such that between 1923 and 1947 the harbour’s coliform count increased seven-fold (Wood, 1987).

Year	Event
1883	Testing of drinking water begins.
1888	Introduction of regulations on ice cutting; ice from polluted waters can be sold, but has to be labeled not to come in contact with food.
1890	Completion of first incinerator to burn garbage.
1896	Weekly testing of water quality begins.
c. 1900	By this time, most wells and privies have been eliminated and most buildings in the city connected to water mains and sewers.
1906	Revision of Public Health Act in Ontario includes prohibition of dumping of manure, etc. into waterways.
c. 1912	Island Filtration Plant on Toronto Island begins operation for water treatment.
1912	Ashbridge's Bay Sewage Treatment Plant opens; it decreases raw sewage but is soon overloaded.
1914	Milk pasteurization becomes compulsory in Toronto.
c. 1919	One in three cities, towns, and villages in Ontario has sewerage facilities.
1928	Milk pasteurization becomes compulsory in Hamilton.
c. 1932	Sewers installed in West Hamilton.
1933	New filtration plant built in Hamilton.

Table 2.1 Timeline of Toronto and Hamilton sanitation improvements. Sources: Jardine (1990); Gagan (1992); Benidickson (1999); “Clean water and sewage treatment” (2012).



Figure 2.1 Sewer outlet at the foot of Yonge Street, Toronto ca. 1907. Source: City of Toronto Archives, Fonds 1244 (William James family fonds), Item 641.

Sanitary conditions at the household level were also problematic in many areas.

Weaver describes the situation in one Hamilton neighbourhood:

In the spring of 1936, a team of investigators had combed a twenty-block neighbourhood of older dwellings and low-cost apartments, an area of households burdened with poverty. Located north and east of the central business district, it was a region where nearly two-thirds of the families were on part or full relief. Only one in ten of the houses was occupied by an owner. Apartment dwellings here were carved out of converted houses, rooms above stores or situated in tenements. Eighty per cent of toilet facilities were defined as unsatisfactory; 78 per cent of apartments lacked central heating. Vermin infested over a third of the flats. Houses tended to be in better condition, but lumping together both forms of dwelling, a quarter of the households were lodged in unsatisfactory conditions. [1982, p. 145]

Poor living conditions in the city centre prompted a program of “fresh-air outings” which were covered in the *Hamilton Spectator* (Figures 2.2 and 2.3).



Figure 2.2 Caption reads, "This is no fanciful picture, but a true representation of conditions as they are in a spot not two blocks distant from Hamilton's city hall. In this hen-coop of a yard, with its accumulation of debris and filth, these children and others have their only playground. The others who are not in the picture are out on the street playing. It is for such children as those that the "Tatler's" fresh-air outings are being given. Take a good look at the picture and then send in your subscription." Source: *Hamilton Spectator*, 6 August 1910.

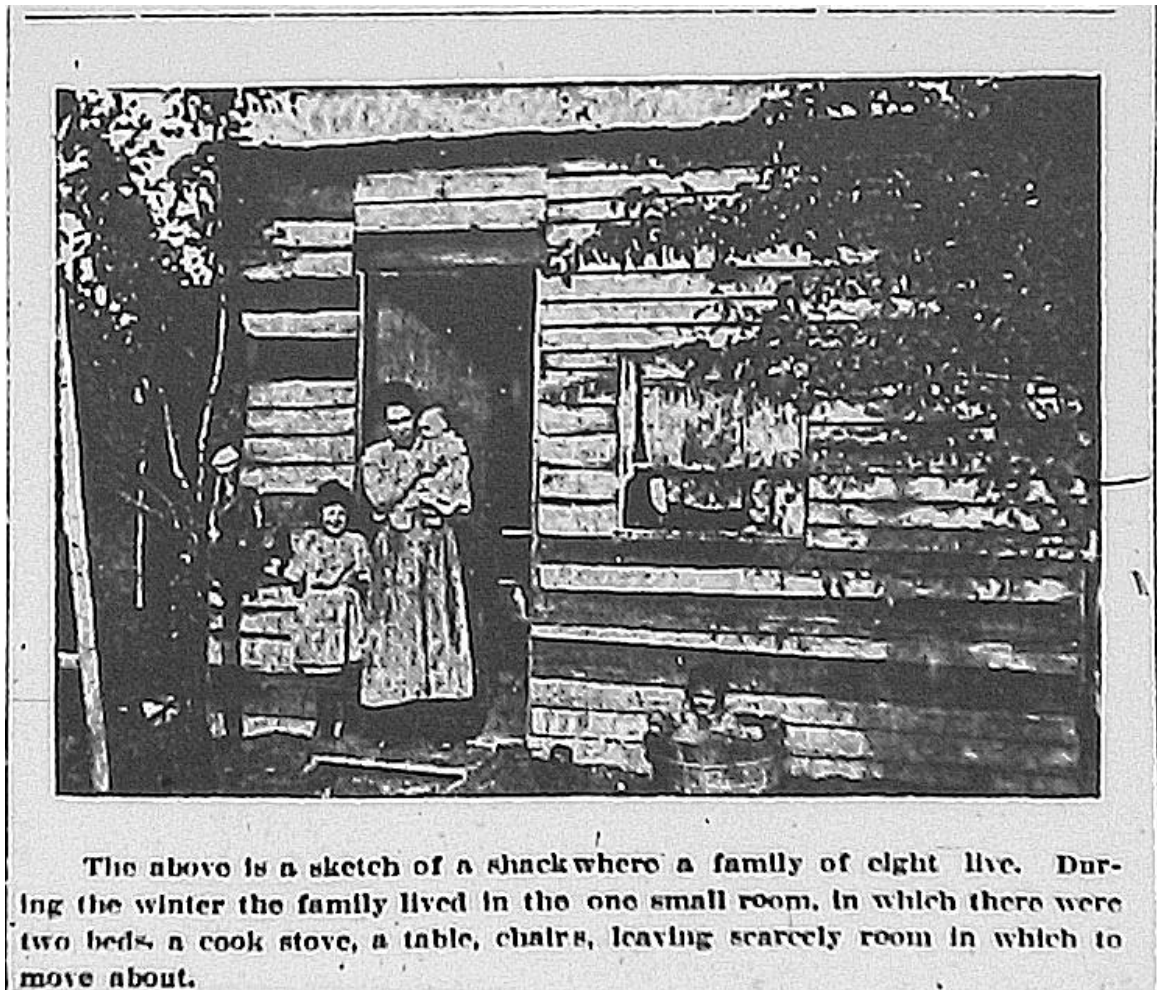


Figure 2.3 A photo of a home in Hamilton accompanying an article urging donations to the “fresh-air outings” fund. Source: *Hamilton Spectator*, 6 August 1910.

According to Wood (1987), conditions on the more rural Mountain were perhaps worse than the most unsanitary parts of the lower city of Hamilton. The Mountain had no sewer system; instead, outhouses were used. However, these were often not properly equipped with tin linings, thus allowing contamination of groundwater. Only a small proportion of the population received piped water; the rest obtained their water from wells. This situation was not dealt with until 1927, after a scarlet fever outbreak.

Toronto's medical health officer Dr. Charles Hastings reported in 1911 that 108 houses in the Ward were unfit for habitation, yet most were occupied (Speisman, 1985, p. 111). Almost one-third of the structures had no plumbing or drainage, with the worst conditions found in rear cottages on laneways (Speisman, 1985, p. 111, citing Hastings, 1911). "Slums" were also identified in Toronto's Corktown and Niagara neighbourhoods (Hastings, 1911, cited in Mercier, 2006). Yet poor sanitation conditions were also identified in the northern suburban fringes of the cities, in working-class "shacktowns" (see Ruggles, 1944 re: Hamilton's Depression-era "Shacktown", and MacDougall, 1990, cited in Mercier, 2006 re: Toronto).

Polio epidemics in Wentworth and York Counties, 1900-1937

Ontario experienced epidemic spikes in polio activity in 1910, 1916, 1922, 1929-1930, 1934, and the largest yet in 1937. The 1910 epidemic affected Hamilton and sparked considerable public concern. A front page headline in the *Toronto Daily Star* declared "CHILDREN ARE ATTACKED BY STRANGE EPIDEMIC" (*Toronto Daily Star*, 17 August 1910, p. 1) while the front page of the local Hamilton newspaper proclaimed "CHILD LIFE IN CITY IS IN DIRE PERIL" and described what was known of the disease at the time:

Infantile paralysis is something like electricity – no person seems to know just exactly what it is. Dr. Roberts says the cause is unknown – that it just comes with the warm weather and is contagious. In a majority of cases it affects children under four years of age, and is considered dangerous. A few cases were reported in the city several years ago, but it cannot be remembered that ever before was there an epidemic of the proportions of the present one.
[*Hamilton Spectator*, 17 August 1910, p. 1]

In the absence of much concrete knowledge about the disease, speculation abounded. The *Toronto Daily Star* carried an article quoting Toronto's medical health officer who proposed that electricity played a role (*Toronto Daily Star*, 18 August, 1910, p. 6). Following another polio death on the evening of August 18th, Hamilton's medical health officer, Dr. Roberts, issued a bulletin on August 20th detailing what was known about polio's epidemiology and epidemic history, including the observation that "unhygienic surroundings have nothing to do with the disease" (*Hamilton Spectator*, 20 August 1910, p. 7). The following week, the front page carried another statement from Dr. Roberts, warning the public about "medical fakirs" offering their services to polio victims (*Hamilton Spectator*, 26 August 1910, p. 1). By September, polio had spread to Toronto and the newspaper carried news of two adult deaths from the disease there (*Toronto Evening Telegram*, 8 September 1910, p. 13). Meanwhile, Toronto's medical health officer continued to deny polio was contagious (Figures 2.4 and 2.5). Media attention faded in October, but polio deaths continued until late November.

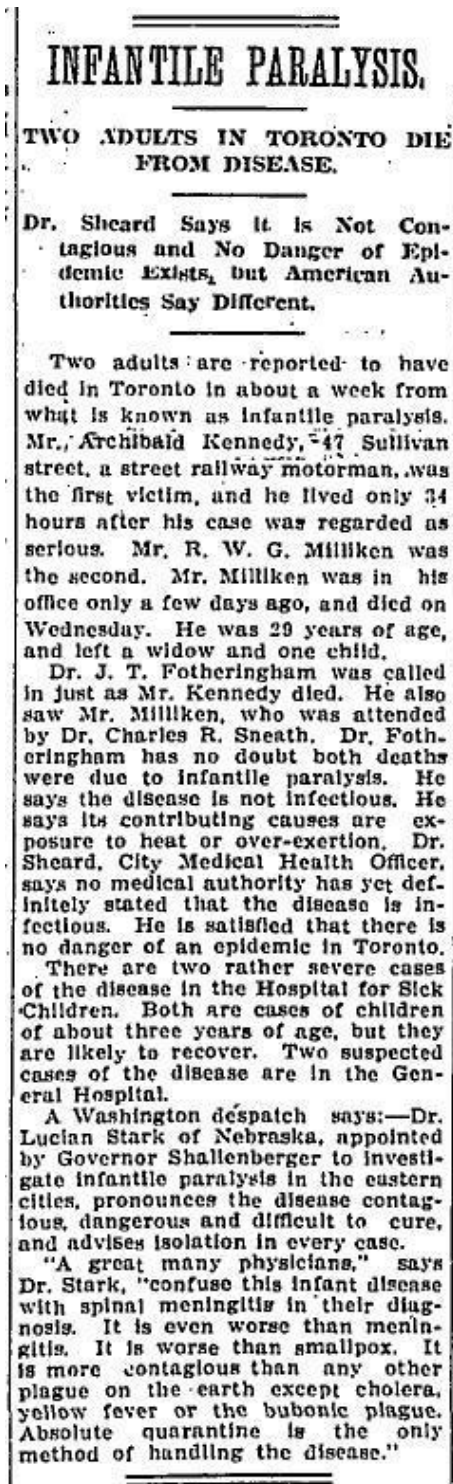


Figure 2.4 *The Globe*, Toronto, 9 September 1910, p. 9.



Figure 2.5 *The Globe*, Toronto, 10 September 1910, p. 13.

Polio continued to reappear every year, though with little media coverage outside of epidemic years. Newspaper coverage ranged from articles from other cities about polio outbreaks there to very specific details about local cases, including names and addresses (Figure 2.6).

INFANTILE PARALYSIS AT DOORS OF TORONTO

Three Cases Have Been Discovered at Todmorden and One at Newmarket, Making Dozen in Ontario.

The dread disease of infantile paralysis, which has caused such consternation in New York, has come close to the doors of Toronto. It was reported yesterday that three cases had been discovered in Todmorden, two on Westwood avenue and one on Gowan avenue. There was also a case from Newmarket. Taking into account reports from Windsor, Ford City, and Walkerville, there are now eleven or twelve victims in the Province.

Three at Todmorden.

It appears that the first of the Todmorden cases, that of two-year-old William Thompson, 18 Gowan avenue, was diagnosed on Friday at the Hospital for Sick Children, College street. The child's mother noticed the boy was not feeling well, and was feverish for a week. Then it was noticed he was not using his arm and could not use his fingers. The second case was that of Dorothy Puppon, 16 Westwood avenue, and it is stated that the lower limbs of the sufferer were so affected that the child could not stand unsupported, although she could still use her arms. The third case is Edna Vall, 56 Westwood avenue. The attacks are said to be mild, but quarantine measures have been taken.

Diagnosed at Hospital.

It appears that after the first Todmorden case was diagnosed at the Hospital for Sick Children, Dr. Hastings, Medical Officer of Health, Toronto, informed the Provincial Board of Health, who in turn acquainted Dr. Eby, St. Clair avenue, who is acting as Medical Officer for York township. The second case was reported on Saturday and the third yesterday morning.

The patient at Newmarket is the six-months-old daughter of Pte. and Mrs. Culley, who recently came to

City on Guard Against Disease

Dr. C. J. Hastings, Medical Officer of Health for the city, asked as to what precautionary measures had been taken to prevent the spread of the disease in view of the Todmorden outbreak, said: "We have got in touch with these cases out there. We have taken the matter up with the Provincial Board, and the cases are being placed under rigid quarantine. We will see that there is no communication from any of these children to the city."

Newmarket from Hamilton. Pte. Culley is now at Camp Borden. The Provincial Health Department was immediately notified and steps taken to prevent the spread of the disease.

Province Acts.

Dr. J. W. S. McCullough, Chief Officer of the Provincial Board of Health, is anxious that the utmost precautionary measures are taken. He declared yesterday that it had been brought to the attention of the board that some doctors in the Province were not reporting cases of infantile paralysis to the medical health officers as the law required. The Board of Health is not going to countenance any such remissness and doctors neglecting this matter in future will be prosecuted in the Police Court.

Some of the Symptoms.

Dr. McCullough, in his recent warning, stated the ordinary quarantine measures that must be taken by medical officers of health, and suggested in addition that wherever an outbreak occurs children should be prohibited from attending large gatherings of any kind, such as Sunday school picnics, moving picture shows, and the like.

He also pointed out that there are many people who develop the disease without suffering anything worse than slight muscular weakness, vomiting, high temperature and headaches, and advised that particular attention should be paid to such symptoms. These cases, while they may not be serious to the ailing one, may be considerably more serious to the public at large, as frequently the disease is not recognized, and the patient acts as a disease carrier, spreading the infection among those with whom he is allowed to come in contact.

Figure 2.6 *The Globe*, Toronto, 25 July 1916, p. 7.

Media coverage also ranged from downplaying the seriousness of the disease to exaggerating it. During the 1916 epidemic, Toronto's medical health officer at the time, Dr. Hastings, called the mention of a polio epidemic "hysteria":

The truth is...the seriousness of the disease is being over-estimated. Infantile paralysis is not nearly so common as one would be led, by the outcry, to believe, nor yet is it, even in the worst of epidemics, so great a cause of death as diphtheria, scarlet fever, measles and tuberculosis.
[*The Globe*, Toronto, 1 September 1916, p. 8]

During the 1922 epidemic, however, one doctor interviewed in the *Hamilton Spectator* described polio as "the most serious of all children's diseases... It has the most terrible effects. [...] I don't want to be considered alarmist, but I do want to emphasize the danger. Let me repeat, infantile paralysis is the worst of children's diseases" (*Hamilton Spectator*, 29 July 1922, p. 14) (for more on the reactions of physicians and the press to polio in Ontario, see Christopher Ruttly's 1990 MA thesis).

In 1937, 2,544 cases were reported across Ontario, with 109 deaths (Ontario Department of Health, 1938, p. 16). Nearly one in three cases in the province occurred in or close to Toronto. However, when adjusted for population size, York County did not have the highest case rate; six other counties had higher rates, with the highest occurring in Halimand County at 229.9 cases per 100,000 population (Ontario Department of Health, 1938, p. 9). Still, York was one of the counties showing a "high" case rate of 130 cases per 100,000 population, while Wentworth was comparatively low at less than 60 cases per 100,000 population (Ontario Department of Health, 1938, p. 9). Toronto had a case rate of 1.2 per 1,000 population in 1937, while many smaller urban centres in York County had comparatively higher case rates, including Mimico at 3.2, East York

Township at 2.7, and New Toronto at 1.6 (Ontario Department of Health, 1938:12).

Geographic variation in cases rates in 1937 did not appear to have a direct relationship to population density, though the authors of the *Report on Poliomyelitis in Ontario, 1937* (Ontario Department of Health, 1938, p. 13) observed that infections radiated out from the urban centres to their surrounding areas.

The artificial respirators known as “iron lungs” were first used on a large scale in Toronto during the 1937 epidemic. One of the Drinker models, invented in 1928, had been acquired by the Hospital for Sick Children in 1930. However, at the start of the 1937 epidemic, it remained the only iron lung in the city (Rutty, 1996). As the polio epidemic worsened in August, there was an immediate need for more respirators. More were ordered, but before they could arrive more patients with paralysis of the chest muscles were arriving (Rutty, 1996). Out of desperation, the hospital began assembling the machines in the building’s basement (Rutty, 1996). These dramatic stories received significant media attention (Figure 2.7). By mid-September, the *Toronto Daily Star* reported that 13 iron lungs were in use in the city, with more ordered (*Toronto Daily Star*, 17 September 1937, p. 1). Sixty-three cases in total were treated with respirators in Toronto; 50% of these were York County residents (Ontario Department of Health, 1938, p. 46). Those treated in respirators were mostly older children and adults (Ontario Department of Health, 1938, p. 46). Of the 63, 40 died, 12 recovered, and 11 were still using the respirators at the time of the report (Ontario Department of Health, 1938, p. 46). “Iron lung” respirators were also in use in Hamilton during the 1937 epidemic.



Figure 2.7 Young boy in a hastily made "wooden lung". Source: *Toronto Daily Star*, 28 August 1937.

In epidemic years, especially 1916 and 1937, control measures included

temporarily closing places where children gathered such as kindergartens, grade schools,

and Sunday schools and banning children from places such as movie theatres. Events such as public picnics were cancelled (*The Globe*, Toronto, 2 August 1922, p. 3).

Whether cases were found in an epidemic year or not, polio patients were quarantined and their houses placarded (Figure 2.8). As Figure 2.8 illustrates, quarantine orders were not always obeyed.



Figure 2.8 A group of children on the steps of a home quarantined for polio at Stanley Barracks in Toronto, August 6, 1947. Source: City of Toronto Archives, Fonds 1266 (Globe and Mail Fonds), Item 117576.

Polio survivors – living with, and dying from, chronic polio

Most of those who survived the acute stage of polio, the ‘polio survivors’, are beyond the scope of this study of polio mortality. However, some discussion of those who lived with chronic polio is needed, as many would die months or years after the initial infection with their deaths directly attributed, at least in part, to polio.

Nielsen, Rostgaard, Juel, Askgaard, and Aaby (2003) found that the most common causes of death in polio survivors in Copenhagen, Denmark were polio sequelae, respiratory tract diseases, gastrointestinal diseases, and suicide. Respiratory polio patients in particular were four times more likely to die in the two decades following their discharge from hospital (Nielsen et al., 2003). Polio survivors also had increased morbidity as measured by subsequent hospitalization, with the highest morbidity seen in those who had respiratory failure during their acute polio illness (Nielsen, Rostgaard, Askgaard, Skinhøj, & Aaby, 2004).

Furthermore, the health care costs associated with chronic polio could be substantial, even overwhelming for some. One polio survivor from Hamilton recalls that after he contracted the disease in 1923 at the age of 18 months, his parents had to sell their family home to help pay for his medical costs (Shorgan, 2004). Thus not only did chronic polio carry its own medical risks, but the impact on a family’s and individual’s socioeconomic status also brought with it additional potential health impacts (see for example the discussion of socioeconomic status and infectious disease risk below).

Polio and socioeconomic class – “the middle class plague”?

Naomi Rogers (1989, 1992) describes the transformation of polio’s image in the United States from a disease of dirt and poverty to one that could attack anyone, especially children from “clean middle-class homes” (1989, p. 490). Other polio historians give similar descriptions; Rutty (1996, p. 277) characterizes the middle class as “polio’s principal target” and Oshinsky (2005, p. 256) calls polio “a disease of cleanliness, striking hardest at the middle class”. As early as 1912, Helen MacMurchy wrote in *MacLean’s Magazine* that “victims of this disease [polio] are not among the poor, or delicate. Often the vigorous and healthy are attacked and those who have comfortable homes and good care” (p. 111). In 1914, the same phenomenon was observed in Ohio (Boudreau, 1914). Boudreau (1914) observed that the Ohio data “demonstrate[d] that the so-called middle classes, or families in moderate circumstances, suffered the most”, but noted that “of course this class comprises a large majority of the population” (p. 14).

Cases of polio among prominent citizens reinforced these observations.

MacMurchy (1912) describes how “the head of one of the largest industrial corporations in Canada was a victim [and so was] a professor in Queen’s College, Kingston” (p. 111). Rutty (1996) similarly describes polio in Canada as “the middle-class plague” which paralyzed and killed “otherwise healthy middle-class children” (p. 278). Yet precisely what is meant by these descriptions is unclear. Did polio have an inverse relationship with class, as the hygiene model suggests, such that the upper socioeconomic classes were disproportionately affected? Or did polio follow a more democratic or “socially neutral” (Mamelund, 2006) pattern, placing all socioeconomic levels equally at risk – with the

upper classes only seeming at increased risk because they usually were less affected by other infectious diseases? Hall (1911) suggests this may have been the case, stating that “this disease affects all classes of society, children of the rich and poor in equal proportion. Many diseases, as we know from experience, affect the poor and not the rich” (p. 116). Or was it some combination of the two patterns, which differed by time and place? It is thus necessary to try to tease out the epidemiological patterns from the social perceptions, while keeping in mind that perceptions nevertheless shaped how data was collected. This raises a further question of how socioeconomic class is being defined, especially “middle class”. The following literature is examined with these questions in mind.

Collins (1946, pp. 347-348) found that the age distribution of polio cases in the urban United States in 1935-1936 varied according to family income level, with higher rates occurring at earlier ages at lower income levels – a pattern similar to other infectious diseases. However, overall the two lowest income brackets (families on relief and those with an annual income of less than \$1,000) had the highest case rates by population, both in the northeast only and in 19 states across the US (Collins, 1946, p. 348). The highest income bracket (families with incomes of \$3,000 or more) had the highest case rate only in the 10-14 age group (Collins 1946, p. 348).

Socioeconomic class in the early 20th century

Sager (2007) examined working-class income in Canada according to the 1901 Census, defining “working-class” as all wage-earners. He found that for men, average ‘blue collar’ earnings were less than ‘white collar’ earnings, though there was a wide range of

variation even within a single occupation; for example, the annual earnings for a machinist in an Ontario engine or railway shop in 1901 ranged from \$350 to \$1,000 (Sager, 2007, p. 349).

Sager (2007) found that in the family cycle, per-person earnings were at their lowest in families with young children. Older adolescents could take on jobs to contribute to the family income (Sager, 2007). In Hamilton in the early 20th century, the majority of working-class children entered paid employment by the age of 14 (Synge, 1979). At this time, children could leave school at age 14; they could leave at age 12 if their parents could show proof their earnings were needed (Synge, 1979). In a sample of six Canadian cities, Baskerville and Sager (1998) also found that nearly one-third of all families with children under age 15 could not survive on the pooled earnings of their wage-earners alone and needed to supplement with resources from the informal economy through such means as bartering or taking on boarders. The period in the family cycle in which unmarried children in their teens and twenties were contributing to the household income was often the only time of relative prosperity for working class households (Synge, 1979).

According to Piva (1979), working-class families were very poor in 1921. Even two incomes were not usually enough to rise above the poverty line. Most building trades workers lived at or below the poverty line between 1901 and 1920, and their economic position declined in that time (Piva 1979, p. 49). Using the 1921 census data on occupations, Piva (1979) found that “with the exception of the running trades on steam

railways, average incomes for blue-collar workers fell substantially below even a conservative estimate of a poverty line” (p. 58).

Still, working class families might not characterize themselves as poor or even working class. A study of people who grew up as working class in Hamilton in the early twentieth century (the children of skilled and unskilled workers and of men with small family businesses employing no non-family labour) found most of the respondents in their study remember their parents thinking of themselves as “middle class”, not “working class” (Synge, 1979).

Socioeconomic class and infant and childhood infectious disease risk

There is evidence to indicate that inequality in infant and childhood mortality between socioeconomic classes increased as the transition to overall lower levels of mortality took place in the late 19th and early 20th centuries. Both Gardarsdóttir (2002) and Ewbank and Preston (1990) attribute this to the upper classes more readily adopting new knowledge and advances in infant care and hygiene. However, Mercier (2006) found that for 1901 Toronto, instead of social class and living conditions, residential grouping by religion was the main factor in differing child mortality rates. Slums with large Jewish populations did not have higher mortality, but largely Catholic areas did. Mercier and Boone (2002) found a similar pattern in Ottawa, in that once cultural factors were controlled for (French versus non-French), economic standing did not account for differences in mortality. However, as economic class rose, the difference in IMRs declined between groups, indicating that better economic position helped reduce the cultural disadvantage of the French Catholics (Mercier & Boone, 2002).

Often lower income meant higher infant mortality. However, sometimes the opposite was true, depending on relative patterns of infant feeding. Lee (2007) suggests that family income in the United States did affect IM in that poorer families were less able to afford good-quality milk, and that in terms of infant diarrheal mortality, market milk quality was a more important factor in IM rates than the domestic environment. Moreover, Lee (2007) also says that poor breastfed infants died from diarrhea more often than wealthy breastfed infants, indicating a role for an unsanitary environment and inadequate refrigeration. In contrast, for Hamilton between 1900 and 1914, Gagan (1981) says cholera infantum affected all classes and was actually higher in the wealthier southern part of the city, probably because of bottle feeding by the middle class. However, Gagan did not consider the role of ethnic factors in Hamilton as Mercier (2006) and Mercier and Boone (2002) did for other cities in Ontario. It is possible that there was a large population with low breastfeeding rates clustered in the south of Hamilton which might also contribute to that discrepancy. For example, the Corktown area, located in the south part of Hamilton, was associated with Irish immigrants, and Preston and Haines (1991) note that Irish immigrants in the US experienced higher levels of mortality than the general population, likely because of infant feeding patterns.

Children of lower socioeconomic status may be especially vulnerable to malnutrition, which in the past has been linked to increased mortality from some infectious diseases (see for example Ulijaszek, 1990). However, such links have been questioned. For example, Hardy (1992) demonstrates that rickets increased the risk of mortality from whooping cough, but notes that rickets was common across social classes,

while whooping cough and measles mortality was much higher among the poor and working classes. Hardy (1992, p. 401) suggests that these class differences in mortality may be due to better childcare practices among the middle and upper classes. Hardy (1992) argues that her data support Aaby's (1988) claim that overcrowding is a more important factor than nutrition in measles severity, and that overcrowding was also an important factor in whooping cough mortality. However, measles and whooping cough themselves affect nutrition and decrease the victim's resistance to other infections (Hardy, 1992). This is important as measles and whooping cough mortality were associated in part with secondary infections and complications such as bronchitis (Hardy, 1992, p. 394). Mamelund (2006) notes that other research has shown that malnutrition does not increase susceptibility to viral infections (Scrimshaw, Taylor, & Gordon, 1959), but certain types of malnutrition do increase susceptibility to bacterial infections (Fox, Hall, & Elveback, 1970). Thus, while malnutrition might not substantially increase susceptibility to the initial disease in cases of viral infections like measles or influenza, it may play a role in susceptibility to the secondary bacterial infections that often complicate such cases.

In regards to two other major childhood diseases of the period, scarlet fever and diphtheria, Hardy (1992) argues that mortality from these diseases was associated more with pathogen virulence than with nutritional or other factors. However, Curtis (2004) argues that scarlet fever mortality was in fact related to nutrition in addition to virulence, finding that children born and nursed in years with food shortages and high food prices in late 19th-century Sweden were more likely to die in the scarlet fever epidemics which occurred two to three years later. Diphtheria in particular was characterized as a

“democratic” disease which affected the rich and poor, hygienic and dirty alike (Hooker & Bashford, 2002). Like polio, it challenged previous conceptions of disease as associated with crowded, polluted places (Hooker & Bashford, 2002).

Mamelund (2006) notes that often there is little distinction made between the chances of being infected by, versus the chances of dying from, a particular disease. While some diseases, such as the 1918 influenza pandemic, might not show any social differences in infection rates, socioeconomic status affects one’s chances of surviving the disease. Mamelund’s (2006) results from an investigation of influenza mortality in Norway in 1918-1919 show that mortality was significantly higher among those in a poor versus wealthy parish and living in smaller apartments. These findings challenge the view of Spanish influenza as a “socially neutral” or “egalitarian” disease in terms of class. A small study of diphtheria patterns in Hamilton, Ontario in the early 20th century similarly found that mortality was higher in wards with higher population densities and lower socioeconomic levels, such as those in the north part of the city (Battles, 2008). See Herring and Korol (2012) for a study of 1918 flu in Hamilton that showed significant differences in influenza mortality in north versus south wards.

Certain medical practices may also contribute to increased risk of infectious disease. Tonsillectomy was popular during the period of polio epidemics in North America, and recent tonsillectomy has been associated with development of the severe bulbar and bulbo-spinal forms of polio (e.g. Barsky & Lauer, 1957; Ravenholt, 1962). Jardine (1990) describes a mass surgery in West Hamilton in the spring of 1919 in which area children had their tonsils removed in a local hall, noting that tonsillectomies were

“very much in vogue in the 1920’s” (p. 69).² Various studies have noted that tonsillectomy was more frequently practiced among higher SES groups (Mertz, 1954; Gutensohn & Cole, 1977; Grob, 2007).

Summary

Wentworth and York Counties in southern Ontario were part of the early epidemic emergence of polio which began in northern Europe and northeastern North America. Both had large and rapidly growing urban areas, with Toronto as a commercial and manufacturing hub and Hamilton more heavily blue collar and industrial. Both experienced major changes in public health in this period, especially in infant and child mortality patterns. The first polio epidemic in the region struck Hamilton in 1910, and Toronto was the centre of Ontario’s worst polio epidemic yet in 1937. As elsewhere, polio was characterized here as a disease of the middle class. However, much of the population in this region, even those who regarded themselves as “middle class”, lived at or below the poverty line, even before the hardships of the Depression. The current literature shows that socioeconomic class impacts morbidity and mortality in a variety of ways. The question remains how socioeconomic circumstances, in combination with other factors, impacted polio patterns in Wentworth and York Counties in this era.

² Jardine (1990) describes the event: “In 1919 a surgery was set up in Buckley’s Hall for the removal of tonsils of local children. According to Bea Austin, who visited the hall with her mother during the mass surgery, several children haemorrhaged during and after the operations. One boy, Elgin Cruickshanks, came near death. Apparently he had worked all day carrying water from a nearby well and was operated on when in a state of near exhaustion. This well-intended exercise, as arranged by the Women’s Institute, seems like a horror story today.

Years later, when the Austin family owned the hall and were doing renovations, blood stains on the wall gave a lurid reminder of the near-slaughter of the innocents” (p. 66).

Theoretical Framework

The hygiene hypothesis

Poliomyelitis is a disease caused by infection with one of the smallest known viruses, an enterovirus in the *Picornaviridae* family of RNA viruses. There are three serotypes of the poliovirus which vary in neurovirulence. Type 1 poliovirus strains are generally the most virulent and type 2 the least virulent. Polio is an enteric infection spread through the fecal-oral route (contaminated water or food, or person-to-person via contaminated hands) or by droplets or aerosols (person-to-person). The virus spreads from the throat or intestines to the lymph nodes and then to the bloodstream. From there it may enter the central nervous system, where it damages motor neurons, causing paralysis and other complications (Smallman-Raynor et al., 2006). Symptoms of polio infection can vary from sore throat and headache to severe and potentially fatal paralysis. There are three clinically recognized forms of poliomyelitis: abortive (minor illness only), non-paralytic (major illness without paralysis), and paralytic (major illness with subsequent paralysis). Between 90-95% of infections are asymptomatic, with 4-8% being the abortive form and only 1-2% becoming major illnesses (Smallman-Raynor et al., 2006). The most severe forms include polio-encephalitis, bulbar poliomyelitis, and spinal respiratory paralysis (Smallman-Raynor et al., 2006). Complications such as pneumonia may also develop and result in death. Polio survivors, especially those with severe paralysis, are also subject to increased mortality in the long term (Nielsen, et al., 2003).

The hygiene hypothesis of polio's epidemic emergence assumes that polio is an ancient disease previously endemic in human populations. Hygiene hypothesis theory was

built largely on studies from the 1950s (Axelsson, 2004). Paul (1971, p. 11, as cited in Smallman-Raynor et al., 2002, p. 14) hypothesized that polio “long contributed to the underlying, and astronomically high, infant mortality rate of urban areas in past times, but was effectively overshadowed by deaths from other infectious agents.” In the theorized endemic era, poliovirus infections would occur in infancy, when individuals were still protected by maternal antibodies. They would then develop their own immunity to the virus, with regular re-infection boosting their immunity into adulthood (Zinkernagel, 2001). The poliovirus “would have established equilibrium in the host population, with the continuous supply of new susceptible infants being born into the population yielding an unbroken chain of infection” (Paul 1971, p. 11).

It has been suggested that an increase in the case:infection ratio with age explains polio’s epidemic emergence (Nathanson & Martin, 1979). However, Nathanson & Martin (1979) dispute this, arguing that the evidence does not support age-specific increases in the case:infection ratio as the explanation, as increasing average age of cases was not necessary correlated with increasing incidence. Bunimovich-Mendrazitky and Stone (2005) also challenge this explanation, arguing that “it does not in itself explain the non-equilibrium epidemic dynamics nor the threshold phenomenon” (p. 313). They suggest instead that epidemics arise when poliovirus transmission among children falls below a critical threshold level (Bunimovich-Mendrazitky & Stone, 2005).

The change in equilibrium which led to the shift to an epidemic pattern has been attributed to improvements in personal hygiene and public sanitation (Nathanson & Martin, 1979; Smallman-Raynor et al., 2006). These improvements delayed exposure to

the poliovirus until past infancy, when children were no longer protected by maternal antibodies, and also resulted in reduced levels of maternal antibodies in mothers as booster infections occurred less often (Nathanson & Martin, 1979; Zinkernagel, 2001). This increased the proportion of the population which was susceptible to polio infection, resulting in periodic epidemics. Decreased breastfeeding in the early 20th century, especially among the middle and upper classes, would also have reduced maternal antibody protection in infants and increased the population of susceptibles (Stewart, 1982).

These improvements in hygiene and sanitation came about as a result of the public health and sanitary movements of the 19th and early 20th centuries which aimed to prevent the spread of water-borne diseases such as cholera and to reduce infant mortality from diarrheal diseases in particular. Polio epidemics then came to be viewed as “the ironic result of a campaign to diminish infant mortality, which all too effectively helped... parents protect their children from germs and dirt” (Rogers, 1992, p. 188). Accordingly, polio’s epidemic emergence has been correlated with declining infant mortality rates (Smallman-Raynor et al., 2006)³. By the 1940s, the emergence of polio epidemics were seen as a sign a country was now ‘modern’ (Báguena, Porras, & Ballester, 2010). Axelsson (2004) too notes that “polio epidemics have been used as a

³ However, correlations between IMR and polio incidence have been difficult to find below the national level. In an examination of 25 Swedish counties between 1909 and 1952, correlation analysis did not find a significant relationship between IMR and polio incidence at the county level (Axelsson, 2004). In fact, Axelsson (2004) notes that some of the first polio epidemics occurred in Stockholm which at that time had one of the highest IMRs in Europe, and by far the highest in Sweden. Still, Axelsson (2004, p. 237) argues that this does not invalidate the hygiene hypothesis; rather, it may demonstrate that IMR is not the most appropriate measure of hygiene and sanitation levels. Haines and Ferrie (2011) similarly note that infant mortality is not always a good measure of living standards and environmental conditions because of variations in breastfeeding patterns, and that instead early childhood mortality may be a better measure of social conditions.

measure to divide how far nations have developed and whether they were civilized or not” (p. 238). In fact, Axelsson (2004, p. 237) argues that the hygiene hypothesis was accepted so readily in part because it explained how countries like Sweden could have epidemics of polio yet still be regarded as modern societies with high standards of hygiene and sanitation.

Thus, polio has come to be known as a “disease of development” (Bunimovich-Mendrazitsky & Stone, 2005) and the hygiene hypothesis remains “the most probable hypothesis” to explain the rise of epidemic polio (Nathanson & Kew, 2010). However, Smallman-Raynor et al. (2006) note that although hygiene and sanitation improvements “are generally believed to have been the main engine behind the transition”, “it is probable that, at different times and in different places, a combination of potential factors influenced the emergence of epidemic poliomyelitis” (p. 17). Bunimovich-Mendrazitsky and Stone (2005) agree, arguing that “there is not single all-encompassing theory that is capable of explaining the history and dynamics of polio epidemics” (p. 302).

So-called “lameness surveys” in the 1970s and 1980s overturned previous assumptions about the relative insignificance of paralytic polio in developing countries (Bernier, 1984). Instead, it became apparent that low rates of paralytic polio in these countries was due to vast underreporting and lack of recognition of the disease (Bernier, 1984; Oforu-Amaah, Kratzer, & Nicholas, 1977). Oforu-Amaah et al. (1977) propose that the incidence of paralytic polio in tropical endemic countries has always been at least as high as in temperate countries during the 20th-century epidemics. They also argue that a shift from endemic to epidemic disease does not necessarily lead to an increase in

incidence. Ofosu-Amaah et al. (1977) suggest that the inverse relationship between IMR and polio rates does not rely on the hygiene hypothesis for explanation, but rather that rising standards of living are associated with better reporting of diseases which would give the appearance of an increase in polio incidence rates. Sabin (1981) also cites incomplete reporting in the tropics as the basis for the ‘old dogma’ of the hygiene hypothesis.

Nielsen et al. (2002) note that “the polio model is often used to explain the epidemiology of diseases of unknown aetiology, suggesting that these may be rare complications of common infections, which have changed age distribution in modern society” (p. 181-182). Some explicitly link their hypotheses to knowledge gained from polio in the 20th century; for example, Tracy, Drescher, Jackson, Kim, and Kono (2010) discuss of the potential role of human enteroviruses in alternately triggering or preventing type 1 diabetes, depending on age of infection. Others may not make a direct link, yet nevertheless the hygiene hypothesis, based on the polio model of increased age at infection, has been extended and applied to allergies such as hay fever (since Strachan, 1989) and various autoimmune diseases, including Hodgkin’s disease, ulcerative colitis, Crohn’s disease, and multiple sclerosis (see Bach, 2002 and Zinkernagel, 2003 for recent reviews of this literature).

Beyond the hygiene hypothesis: other factors in polio’s epidemic emergence

A lower birth rate in a population reduces the number of susceptibles, requiring a higher level of connectivity and transmission in order for a pathogen to remain endemic (Fouchet, Marchandea, Bahi-Jaber, & Pontier, 2007, p. 486). Thus, a reduction in the

birth rate may increase the likelihood of an infectious disease moving from an endemic to an epidemic pattern. And indeed, birth rates in regions where polio first displayed an epidemic pattern had begun to decline in the 19th century (Chenais, 1992; Grundy, 2005). Dauer (1955) argues that the falling birth rate between 1916 and 1945 was a more important factor in changing polio patterns than sanitation levels, at least in Massachusetts. In Canada specifically, birth rates fell rapidly after 1861 (Moore, 1990). In Hamilton, for example, the birth rate was close to 30 births per 1,000 residents in 1920; it dropped to 18 per 1,000 by 1936 (Weaver 1982, p. 140). Polio thus emerged in epidemic form during a period of fertility decline from the late 19th century to 1940 in developed countries; this transition then may have set the stage for the massive outbreaks of the 1950s following the subsequent fertility increase during the post-WWII “baby boom”. As part of the third stage of demographic transition, characterized by both low birth rates and low infant and childhood mortality rates (ICMR), there was not only an actual shift from endemic to epidemic polio (and thus an increased visibility of the disease due to epidemic clustering of severe cases), but increased visibility due to the otherwise lowering ICMR which allowed polio to stand out from the “epidemiological background” (Smallman-Raynor et al., 2006, p. 8).

Nielsen, Wohlfahrt, Melbye, Mølbak, and Aaby (2002b) found that cross-sex transmission of the poliovirus may increase the severity of the disease and that transmission from mother to son may partially explain the higher prevalence among males in all age groups except women’s childbearing years. This pattern is not restricted to polio; both the likelihood of acquiring a viral infection and its severity are higher if the

virus is transmitted from an opposite-sex, rather than same-sex, sibling (Aaby et al., 1986; Aaby, 1991; Aaby, 1992; Pison, Aaby, & Knudson, 1992; Uller 2006). The evidence so far points to an immunological, rather than genetic or cultural, explanation for this phenomenon (Aaby, 1992; Nielsen et al., 2002b; Uller 2006).

Recent research has also demonstrated that the poliovirus uses intestinal microbes for replication and transmission (Kuss et al, 2011). In experiments with mice, depleting the intestinal microbiota with antibiotics made those mice less susceptible to the virus; when infected with the poliovirus, the mortality of untreated mice was twice that of the antibiotic-treated mice (Kuss et al., 2011). It is unknown how this might factor into patterns of mortality in epidemic polio or relate to the epidemic emergence of polio.

Challenging the polio model: the intensive-exposure model

According to the polio model, the severity of the disease increases with age at infection. Advocates of the model reason that since first-born children and those with fewer siblings tend to be infected at older ages, they should have a higher risk of severe polio (Nielsen, Aaby, Wohlfahrt, Mølbak, & Melbye, 2002a). Nielsen et al. (2001; 2002a) have adapted the intensive-exposure model, developed by Aaby (1988) and colleagues (Aaby, Bukh, Lisse, & Smits, 1984) in the context of measles, to polio to explain patterns of differing severity in polio cases. The intensive-exposure model challenges some aspects of the traditional polio model, such as the reliance on age at infection to explain differences in severity. Instead, the intensive-exposure model postulates that intensity of exposure and dose of infection are determinants of the severity of a poliomyelitis infection (Nielsen et al., 2001, 2002a). Laboratory studies with monkeys have demonstrated that higher doses

of the poliovirus increase the risk of severe illness and paralysis (Moore & Kessell, 1943; Sabin, 1951). Nielsen et al. (2002a) examined historical records of hospitalized polio cases in Copenhagen, Denmark and found that severity of poliomyelitis increased with family size and that there was a U-shaped age curve for paralysis patients. Both last-borns under five years old and first-borns over six years old had an increased risk of severe polio, which Nielsen et al. (2002a) argue indicates that children around five years of age were more often bringing infection into the home (as primary cases). Nielsen et al. (2001) found that being infected at home by a sibling leads to more intensive exposure, compared to brief exposure outside the home, and so secondary cases would be more severe than primary cases. It is possible that role of intensive-exposure is related to the route of transmission (droplet vs. fecal-oral), with intensive-exposure being most relevant in the context of respiratory transmission (Nielsen et al., 2002a, p. 185). Nielsen et al. (2002a, p. 185) argue that only the intensive-exposure model explains both the age pattern of polio severity and the impact of crowding factors such as birth order and number of siblings.

Morris, Harrison, Lauder, Telford, and Neary (2012) have made a similar observation about the role of dose in addition to age. While they do not specifically refer to Nielsen et al.'s IE model and only note polio's fecal-oral route of transmission, Morris et al. (2012) observe that "factors that influence the outcome [of contact with an infectious organism] include age at first exposure, dose at first exposure and route of exposure" (p. 4). They argue that an epidemic pattern of spread leads to increased doses of the virus compared to endemic spread, and that this combination better explains polio's

epidemiology than does age alone (Morris et al., 2012). Morris et al. (2012) also note that infectious mononucleosis caused by the Epstein-Barr virus shows a similar pattern to polio.

Summary

Epidemic emergence theory suggests that there are a number of factors which can disrupt the evolved state of equilibrium between microbes and their hosts. Changes in public sanitation and hygiene are chiefly blamed for polio's shift from endemic to epidemic. Changing demographic patterns, particularly a falling birth rate, are also hypothesized to have played a role. The existing dogma of the hygiene hypothesis has recently been challenged by the intensive exposure model. This model does not seek to overturn the hygiene hypothesis, but rather to revise and perhaps complicate it by providing a role for factors such as crowding in the risk of severe polio. Thus the new model may alter our understanding of epidemic polio, reducing its apparent differences with other infectious diseases by considering some of the risk factors, such as crowding, usually associated with lower socioeconomic status.

Chapter 3 Materials and Methods

Introduction

The first part of this chapter describes the materials from which the data for this study were gathered and addresses issues of data quality. The second details the process of data collection from death registrations and outlines the resulting sample of polio deaths. I discuss how various archival records were subsequently matched and their data integrated in the construction of the larger geo-referenced database. I also describe how occupation information was translated into status scores. Finally, I detail the methods of statistical analysis I employed.

Materials

Primary data sources

Data for this sample of polio victims were collected from a number of archival sources. The first among these was the Registered Death Records of the Province of Ontario, MS935, Reels 95-601. Additional sources included the Registered Birth Records and Registered Marriage Records, both for the Province of Ontario and elsewhere in Canada. These death, birth, and marriage records were all publicly available from the Archives of Ontario on microfilm, or digitally for paid subscribers of Ancestry.ca.

The death records contained a variety of information. The exact information requested by the Registrar varied slightly over time, and not all fields were always filled out. In a death registration from 1923, for example, the first portion included name, place of death, sex, racial origin, marital status, age, place and date of birth, occupation,

industry, dates from which and to which employed, length of residence at place of death, in Ontario, and in Canada, parents' names and places of birth, physician's name and address, informant's name, address, and relation to the deceased, place and date of burial, undertaker's name and address, cause of death if no physician attended, and date of death. The second portion, the Medical Certificate of Death, included name of the deceased, date of death, name of parents if an infant, address, dates from which to which medical practitioner attended the deceased, primary cause of death and duration, contributory cause of death and duration, whether an operation preceded death, whether an autopsy was performed, name and address of physician, date of the return, and the date received by the Registrar General. Until 1908, the death registration form included a field for religious denomination. The forms changed slightly again around 1912, 1920, and 1935, for a total of five different forms over the study period (see Appendix A).

Additional sources of data available through Ancestry.ca included census records, border crossing records, passenger lists, World War I attestation papers, and baptism records. City directories were accessed on microfilm in the McMaster University Library Lloyd Reeds Map Collection, and newspaper death notices and articles were accessed both on microfilm at McMaster University's Mills Library and, for Toronto's *Globe* and *Globe and Mail*, online via the *Canada's Heritage from 1844* archive.

Data collection was constrained by the availability of the various sources. During the period of data collection for this study, the Registered Death Records for the Province of Ontario were available only up to and including the year 1937. Registered Birth

Records were available to 1912⁴ and Registered Marriages to 1927. Ontario privacy laws restrict access to these records until a specified period of time has elapsed: 70 years for death records, 80 years for marriage records, and 95 years for birth records. Table 3.1 lists the year range of each primary data source consulted.

Data quality

Any historical data set is subject to issues of accuracy, reliability, completeness, and bias. As this data set was compiled using a number of different historical records, there are many such issues to discuss here.

Ontario's civil registration system began in 1869, and by the 20th century these records were relatively complete, at least in terms of death registrations. Emery (1983) found that Ontario mortality statistics can be assumed to be reasonably complete as of the 1911 census year. Quality also improved significantly over time (Emery, 1983). Williams (1996) found that for Victorian England, cause of death data were most reliable for children and young adults, infectious diseases, and urban areas, and least for deaths in infants and the elderly. This was likely to have been the case in Ontario as well, though overall reliability would have improved over the 1900-1937 study period.

Legibility of these records was also an issue, though more so for the earlier years when the information was hand-written; over time the registrations became overwhelmingly type-written. It was clear as well that the clerks transferring the information into the official books often had a hard time deciphering the physician's

⁴ Birth records for 1913 and 1914 became available during data analysis, and records from those years for two individuals were added to the database.

Materials		Source	Years consulted	Format
Death records		Archives of Ontario	1900-1937	Microfilm
		Ancestry.ca	1900-1937	Digital image
Birth records		Ancestry.ca	1869-1914	Digital image
Marriage records		Ancestry.ca	1869-1927	Digital image
Baptism records		Ancestry.ca	1912	Digital image
Censuses		Ancestry.ca	1861, 1871, 1881, 1891, 1901, 1906, 1911	Digital image
Border crossing records		Ancestry.ca	1909-1930	Digital image
Passenger lists		Ancestry.ca	1907-1930	Digital image
World War I attestation papers		Ancestry.ca	1914-1918	Digital image
Toronto city directories		McMaster University Library Lloyd Reeds Map Collection	1901, 1911, 1917, 1919, 1921, 1922, 1926, 1928, 1930, 1937	Microfilm
Newspaper death notices and articles	Hamilton Spectator	McMaster University Mills Library	1900-1937	Microfilm
	Hamilton Herald	Hamilton Public Library Local History and Archives	1910	Microfilm
	Hamilton Daily Times	Hamilton Public Library Local History and Archives	1910	Microfilm
	Toronto Daily News	McMaster University Mills Library	1900-1919	Microfilm
	Toronto Daily Star	McMaster University Mills Library	1900-1937	Microfilm
	Toronto Evening Telegram	McMaster University Mills Library	1900-1934	Microfilm
	(Daily) Mail and Empire	McMaster University Mills Library	1900-1929; 1929-1936	Microfilm
	Globe	Canada's Heritage from 1844	1900-1936	Digital image
	Globe and Mail	Canada's Heritage from 1844	1936-1937	Digital image

Table 3.1 Primary data sources used for construction of polio death database.

handwriting, given that there were predictable misspellings of terms such as “poliomyelitis”, for example.

As mentioned above, data collection was constrained by the availability of records. During the period of data collection from 2009 to 2010, death registrations were available up to 1937, which included the largest polio epidemic of the study period. However, marriage records and birth records were only available for an early portion of the study period. Also, the 1911 census was the most recent one with individual-level data publicly available. As more records are released in future years, this database can be expanded; however, this thesis is based primarily on the data available as of 2010. Newspaper searches were also constrained by the availability of online archives or microfilm holdings of the McMaster and Hamilton libraries. For example, the *Globe and Mail* newspaper was available in searchable online digitized format, but other newspapers were not and had to be browsed manually, a much more time-consuming process.

There is the question of the accuracy and reliability of the diagnoses of cause of death due to polio. We know that polio is often difficult to diagnose, and may be misdiagnosed. This is illustrated by the newspaper coverage of the case of Marion (called “Murial” or “Muriel” in some newspaper coverage) Mitchell, perhaps the first fatal polio case of the 1910 epidemic centred in Hamilton:

HYDROPHOBIA

Doctors Believe They Have Another Case in Hospital

Suffering, it is believed, with an advanced case of hydrophobia, and exhibiting all the symptoms of the dread disease, little Marion Mitchell, the five-year-old daughter of Mr. and Mrs. Mitchell, 253 Wentworth street north, was taken to the

City Hospital to-day at noon. The discovery of the child's condition was made this morning, when her parents called in Dr. R. Y. Parry. He immediately consulted Dr. Langrill, sen., and the two ordered her immediate removal to the Hospital. They are baffled at the signs shown, and, although they fear she is a victim of the disease, they have been unable to trace it to a bite from a dog.

The child was in a pitiable condition when the police arrived at her home. She was lying on a bed with froth oozing from her mouth, and her father and mother, who were both terribly frightened at the unusual symptoms, were standing over her watching her every movement. The child was unable to speak, but she recognized those about her.

As soon as the girl was admitted to the Hospital the doctors had a nurse make the usual tests. The first course they took was to try and induce her to drink some water. As soon as it touched her lips, however, she seemed to be troubled with the choking sensation apparent when a person has the disease, and she stiffened out and then began to toss about on the bed.

This has led to the general opinion that it is a case of hydrophobia, as Johnny Taylor, the Dundas lad who died in such terrible agony recently, showed practically the same symptoms.

Everything possible has been done to ascertain if the child was bitten, but the only information the doctors can secure is that she had been scratched several times by her pet cat.

For the past few days the child had been acting strangely. She appeared to be seized with attacks of drowsiness, and wanted a pillow and stead off into a corner and stretch herself out on the floor.

The doctors had every precaution taken in placing the child in the ambulance. She was carefully wrapped in blankets, and none of the regular ambulance equipment was used.

Dr. Langrill gave out the following statement to-day at 2 o'clock: "The case is not fully developed, but it looks very much like hydrophobia. The child took ill only last night. There is no sign of any inflamed wound or of any serious wound which has healed. Hydrophobia might have resulted from a scratch from a cat that was infected with hydrophobia. The wounds received from scratches might heal and hydrophobia might not develop for weeks. However, we will likely know by to-night if the child is suffering of that disease. When I first saw her she was apparently dying. Her lungs were filled and she seemed to be unable to clear them. As soon as a successful effort was made at the hospital to clear them she rallied."

[*Hamilton Daily Times*, 2 August 1910, p. 12]

NOT HYDROPHOBIA

An Abscess was Cause of Marion Mitchell's Death

Little Marion Mitchell, aged 4 years and 8 months, daughter of Henry and Mrs. Mitchell, 253 Wentworth street north, who was removed to the City Hospital yesterday, supposedly suffering with an advanced case of hydrophobia, died this morning about 4 o'clock. A thorough examination by the doctors in charge revealed the fact that the death was not due to hydrophobia, but to blood poisoning, which was caused by an abscess, which they believe formed in the throat. The presence of this abscess would account for the frothing at the mouth and the difficulty she evinced in breathing, which first gave the doctors the impression that she was suffering with hydrophobia.

The funeral will be held on Friday afternoon at 2.30, from the above address, to Hamilton Cemetery."

[*Hamilton Daily Times*, 3 August 1910, p. 1]

DIED IN HOSPITAL

Little Murial Mitchell succumbed to Landry's paralysis

Dr. Parry said that Landry's paralysis affected the throat and breathing and was caused by a germ, but the medical profession has not yet been able to ascertain its origin.

[*Hamilton Spectator*, 3 August 1910, p. 1]

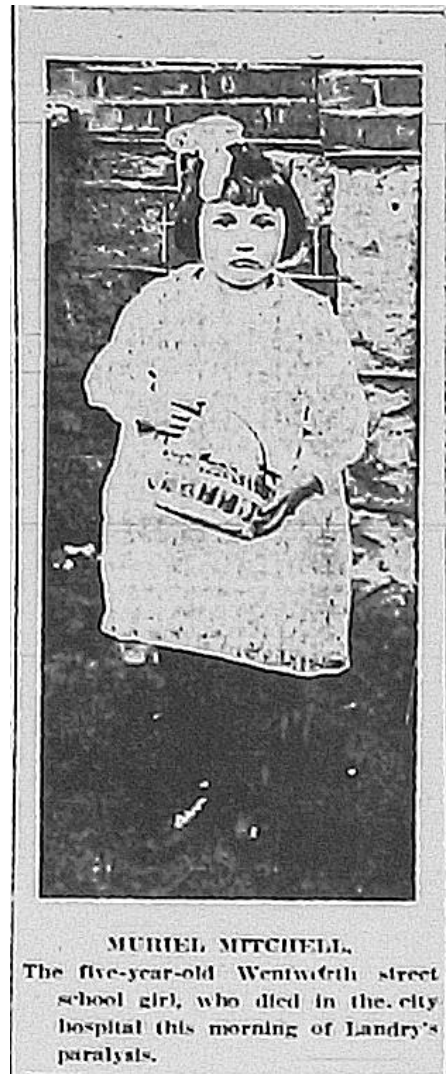


Figure 3.1 Photo of Muriel Mitchell. Source: *Hamilton Spectator*, 3 August 1910, p.1.

Marion Mitchell's death record lists her cause of death as "Landry's Paralysis (Bulbar)" of 48 hours duration, with "Asphyxia" the immediate cause. This sounds very much like bulbar polio.

A week later, the first official polio deaths of the 1910 outbreak in Hamilton were recorded, and two weeks later the outbreak was publically recognized, with officials noting that the first cases had been reported two weeks earlier:

CHILD LIFE IN CITY IS IN DIRE PERIL

Serious Epidemic of Dreaded Infantile Paralysis

Local Doctors Combine to Stamp Out the Disease

Rumors current in the city hall this morning to the effect that there was a serious epidemic of infantile paralysis in the city were confirmed by Dr. Roberts, the medical health officer, and the cat is now out of the bag. That the outbreak is a serious one is admitted, for between 25 and 30 cases have already been discovered, and there are prospects that more will come. It was about two weeks ago that the medical health officer was casually informed by a doctor that he had attended a case of infantile paralysis – the only difference being that the medical man used a Latin word that would bend the press if reproduced here. Later, other doctors informed the officer that they had patients suffering from the same disease, until Dr. Roberts, thoroughly convinced that there was something doing in the epidemic line, started an investigation. The result of that investigation disclosed a startling state of affairs, and he has indefinitely postponed leaving on his holidays until he has taken the necessary preventative measures against the spread of the disease. It was only recently that the provincial health department issued regulations requiring all doctors to report cases of infantile paralysis, and Dr. Roberts has sent notices to the practicing physicians to notify him of all such cases. He stated this morning that the city doctors were supporting him nobly in his crusade, and were doing all in their power to help him fight the epidemic. Dr. Roberts has visited practically all the patients, and in each case has ordered their strict confinement to their homes. The isolation will be strictly and impartially maintained until the epidemic dies out.

[*Hamilton Spectator*, 17 August 1910, p. 1]

The same day, the *Toronto Daily Star* appears to have mentioned Marion Mitchell's death as a case of polio:

CHILDREN ARE ATTACKED BY STRANGE EPIDEMIC

Twenty Cases of Fever and Infantile Paralysis – Once Swept Over the States

Special to The Star

Hamilton, Ont., Aug. 17. - An epidemic of poliomyelitis, or infantile paralysis, a comparatively new disease, which is attracting much interest among medical men the world over, has broken out here.

A score of cases have been reported to the Health Department, and the disease seems to be spreading. It was first noticed three or four weeks ago when a little girl, supposed to be suffering from hydrophobia, was taken to the hospital, where she died. It was later discovered she was a victim of infantile paralysis. The disease generally begins with a high fever and then the patient is suddenly stricken with paralysis.

While most of the cases here are children under four years of age, two or three adults are victims. Some years ago the disease swept over a portion of the States, claiming victims by the hundreds.”

[*Toronto Daily Star*, 17 August 1910, p. 1]

In the early years of the 20th century, there was much variability in the diagnosis of polio and the terms used to describe it (see the following section). Wilbur noted in 1912 that “to enumerate even the names of all the diseases with which epidemic poliomyelitis has been confused would require a long list varying from rachitis to sinus thrombosis” (p. 424). The accuracy and reliability of polio diagnoses, as well as the consistency of the terminology used,⁵ improved over time, as research rapidly progressed and physicians become more familiar with the disease. New medical technology also contributed; in Toronto, for example, the lumbar puncture began to be used to diagnose polio in 1930. Paralytic polio is much more easily diagnosed than the non-paralytic form, with the main symptom of paralytic polio being acute flaccid paralysis (Smallman-Raynor et al. 2006). It seems likely that polio diagnoses were especially reliable in terms of fatal cases, as autopsy was an option. Smallman-Raynor et al. (2006, p. 55) suggest that this is the case, as they argue that we can gain a general idea of the variation in

⁵ There was much initial debate about what the most appropriate name for the disease should be, as “infantile paralysis” lost favour as the disease attacked an increasing number of adults. Alternate terms included variation on “poliomyelitis”, including “epidemic acute meningoencephalopolio myelitis” (Wilbur, 1912, p. 418). Hall (1911) objected to “anterior poliomyelitis”, which later did become the standard, and instead argued in favour of “acute poliomyeloencephalitis”.

reporting of polio morbidity by comparing the number of notified cases of polio to the number of polio deaths.

In terms of sources of bias, certain groups were more likely to be underrepresented in the historical records. Overall, mobile people are more difficult to trace than those who remain in one place (Wrigley 1973, p. 12). Those who were highly mobile (e.g. renters) were more difficult to track down in the city directories; new non-British immigrants were also hard to find in the directories, possibly due to misspellings of their names. Hautaniemi, Anderson, and Swedlund (2000) note that “linkage involving the use of names in historical records is always problematic” (p. 22). It was also difficult to find newspaper death notices from non-British families; possibly, they were posting notifications of death and funeral sources in other, ethnic-specific venues. This is consistent with the findings of other studies. In their study of two Massachusetts towns between 1850 and 1912, Hautaniemi, Anderson, and Swedlund (2000) found that foreign born individuals were underrepresented among linked deaths. Emery (1993, p. 156) notes that as late as 1920 the Ontario birth registrations were significantly incomplete for females and Central European immigrants.

Methods

Collection of initial sample of polio death registrations

Data collection began with a visual search for the terms “infantile paralysis”, “poliomyelitis”, or “polio-” listed as Cause of Death in death registrations from Wentworth and York Counties in southern Ontario in the years 1900 through 1937. No other known specific terms for polio such as “Heine-Medin disease” were encountered,

with the exception of a case of “acute ascending spinal paralysis” in 1910 in a 42-year-old male and the previously mentioned case of “Landry’s paralysis” in 1910. “Acute spinal paralysis”, which could be of “an ascending, rapidly progressive type”, was a term used for polio in the 19th and very early 20th centuries (Boudreau, 1914, pp. 3, 73), but such cases were excluded from this sample because the terms were not specific to polio. However, it must be kept in mind that in the first two decades of the 20th century, “Landry’s paralysis” or “Landry’s type” paralysis was used to refer to acute ascending paralysis (Wilbur, 1912; Williams, 1912), and according to Wilbur (1912), “that most cases of Landry’s paralysis are due to the virus of poliomyelitis seems now well established” (p. 423). While such cases did not meet the criteria for inclusion in this sample, they still may have been polio cases.

Causes of death are not included in the Ancestry.ca search engine, so identifying polio victims involved inspecting every death registration for the study area. Occasionally, note was made of causes of death that were ambiguous and could be polio; only one of these was eventually included in the database after a newspaper report specified the individual as a polio victim. Note was also made of three individuals who had polio listed as a morbid condition not causally related to the immediate cause of death; however, these were not included in the database for analysis, as this study was focused on polio as a cause of death.

All information from the death registrations of polio victims was entered into an Excel spreadsheet. One death registration was excluded from analysis due to lack of data on the death registration.

Outline of initial sample

After the exclusions mentioned above, the study sample consisted of a total of 336 polio victims. Of these, 167 were male and 149 female. 65 were listed in the Wentworth County area, and 271 in York County. Only 16 out of the 336 resided in neither county. 223 were acute cases (either listed as “acute” or having had polio for six months or less at the time of death), 29 were chronic cases (either listed as “chronic” or having had polio more than six months before death), and 84 cases could not be determined as specifically acute or chronic due to lack of information, though were most likely acute cases.⁶

Record linkage and construction of database

The names and other personal information (e.g. date of birth) were then used to locate additional records on each polio victim. These searches were mostly conducted through the basic and advanced search functions on Ancestry.ca (see Figure 3.2); city directories and newspapers were searched separately. These additional records provided information on religion, occupation, previous residences, and number, sexes, and ages of siblings. These details were included in the Excel database file and digital copies of the records were saved.

⁶ I have lumped acute and convalescent cases together under “acute” as opposed to “chronic”, using the six month mark as the dividing line. These terms are defined by varying overlapping durations in the polio literature, with the convalescent phase extending up to two years while the chronic phase might start after one year. In my sample, however, the earliest point at which polio is labeled as “chronic” by a physician reporting the death is at six months duration.

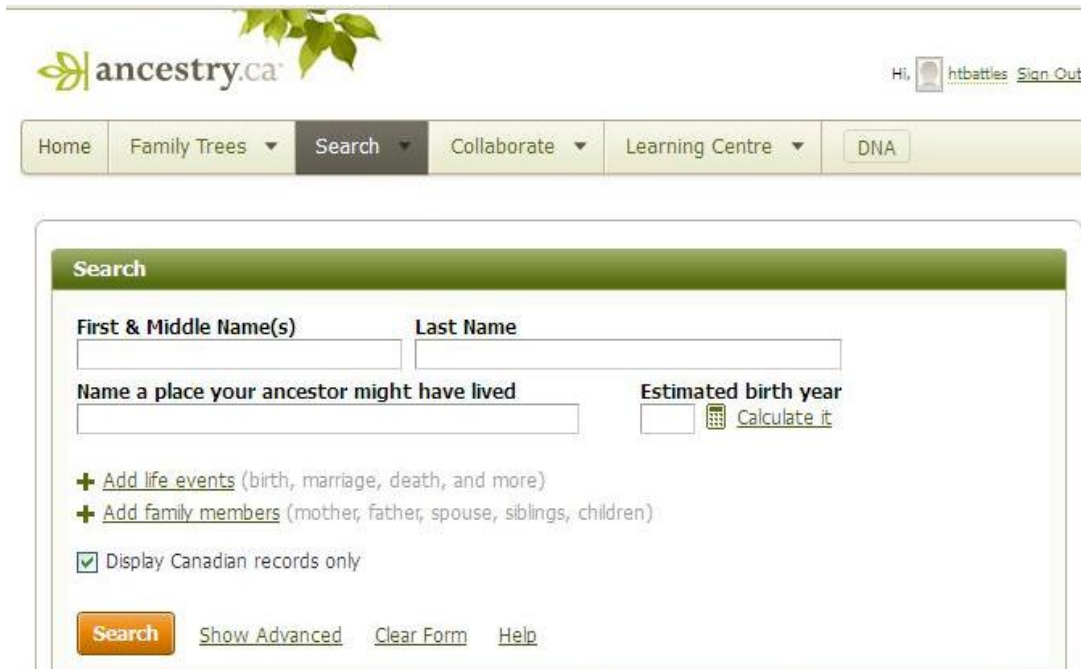
The image shows the top section of the Ancestry.ca website. At the top left is the Ancestry.ca logo with a green leaf icon. To the right of the logo is a user profile section showing 'Hi, htbattles' and a 'Sign Out' link. Below this is a navigation bar with buttons for 'Home', 'Family Trees', 'Search', 'Collaborate', 'Learning Centre', and 'DNA'. The 'Search' button is highlighted. Below the navigation bar is a large search form titled 'Search' in a green header. The form contains several input fields: 'First & Middle Name(s)', 'Last Name', 'Name a place your ancestor might have lived', and 'Estimated birth year'. There are also links for '+ Add life events (birth, marriage, death, and more)', '+ Add family members (mother, father, spouse, siblings, children)', and a checkbox for 'Display Canadian records only'. At the bottom of the form are buttons for 'Search', 'Show Advanced', 'Clear Form', and 'Help'.

Figure 3.2 Basic search form on Ancestry.ca.

To determine the city ward of each polio victim's residence, OntarioRoots.com's Toronto 1911 Census Street Finder tool was used. The ward boundaries in 1911 were the same as in 1901, with the exception of the addition of West Toronto as a new Ward 7 in 1909. This meant that as I assigned addresses for Wards 1-7, I used the boundaries as they were in 1914, before North Toronto became part of Ward 2 and East Toronto joined Ward 1. For Hamilton, ward numbers (Wards 1-8) were assigned using a combination of written descriptions of the 1910 boundaries and a map of Hamilton's ward boundaries for 1920-1930 (unchanged), and current Google maps. For streets along which the ward boundaries run, I assigned addresses to a particular ward based on the side of the street they were on; even numbered addresses in Hamilton fell on the east or north sides, and odd numbered on the west and south, so addresses were assigned to the ward on that side. This does not appear to have affected the results.

GIS: additional data and analysis

Using tools on GeoCoder.ca, the latitudes and longitudes of the residential addresses of the polio victims at the time of their deaths were determined. This was usually only possible for those with a street address, excluding, for example, those rural residents whose addresses were lot and concession numbers. Thus, the GIS (Geographic Information System) sample is primarily urban and suburban. These geographic coordinates were then entered into the Excel database and used to plot the residences of the polio victims in ArcView 3.2, with their associated information from the Excel database attached. ArcView displayed these locations over a modified modern street grid map of the area of southern Ontario covering Wentworth County to the southwest to York County to the northeast.

Drawing upon descriptions of neighbourhoods and their boundaries from a variety of sources (e.g. Harney 1985), these areas were mapped out and drawn as polygons in ArcView. In total, 27 neighbourhoods were mapped out, 14 in Toronto and 13 in Hamilton. Record was made of the polio victims whose residences fell into these different areas and entered into the Excel database. Using secondary sources, these neighbourhoods were classified according to their socioeconomic status as wealthy, middle class, lower middle class, both middle and working class, working class, poor, or mixed (see Appendix F).

The ArcView map was also used to measure distances from the Hamilton and Toronto city centres. For Hamilton (Figure 3.3) the concentric circles radiated out from the intersection of King and James Streets, and for Toronto (Figure 3.4) from King and

Bay Streets. The first circle had a radius of 1.5 km, and each subsequent circle added an additional 1.5 km to the radius, up to a total of seven circles for Hamilton (up to 10.5 km from the city centre) and nine circles for Toronto (up to 13.5 km from the city centre). Each circle was numbered, and the polio victims whose residences fell into each were recorded, with this information entered into Excel.

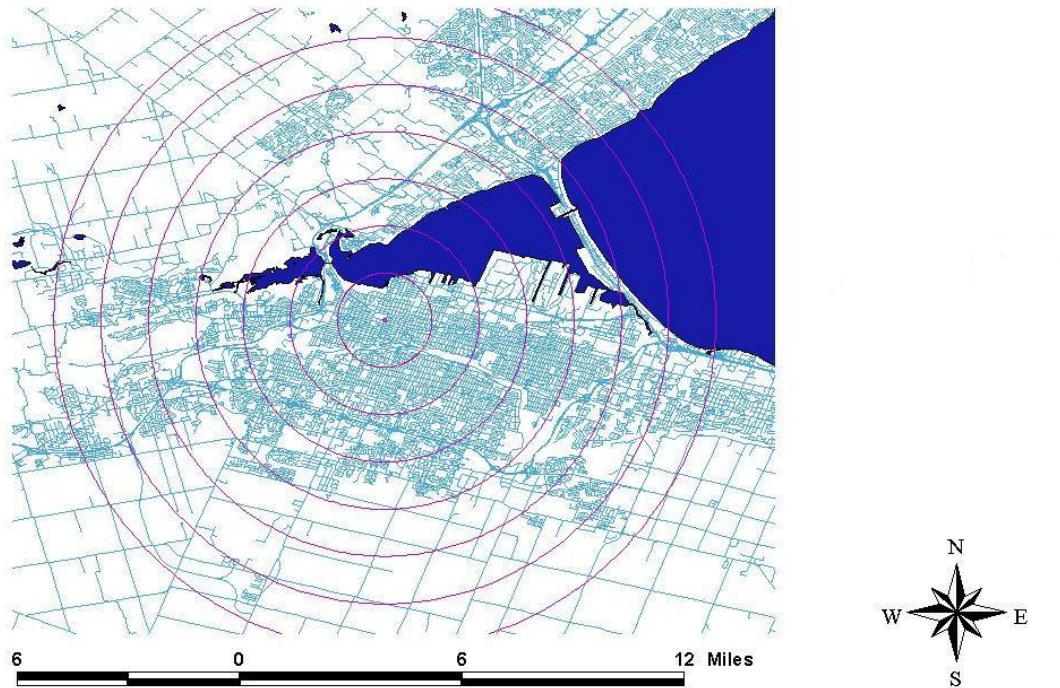


Figure 3.3 ArcView map showing concentric circles radiating from the intersection of King and James Streets in Hamilton.

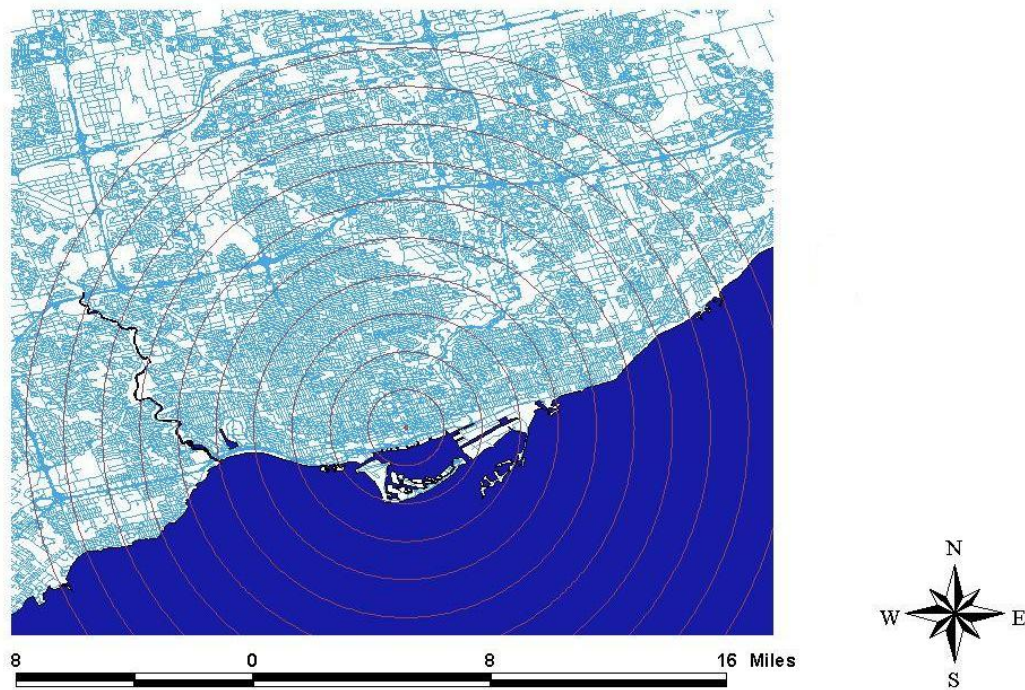


Figure 3.4 ArcView map showing concentric circles radiating from the intersection of Bay and King Streets in Toronto.

Status score coding

Socioeconomic class was coded using the five-point composite score scale from Hauser (1982) (see Table 3.2). Numerical values were assigned to individual polio victims based on father's occupation, or in the absence of data on father's occupation, on the occupation of the adult male victim or adult female victim's husband if married. For victims whose fathers had multiple known occupations over time, the occupation in which the father was engaged for the majority of the victim's early childhood period was selected for scoring, as could best be determined on an individual basis. It would have been preferable to have separate scores for occupation at birth and at death; however, birth records were unavailable for most of the study period.

There are 112 occupation titles listed in Hauser (1982), 38 of which showed up in my sample. Occupations in the sample which were not on Hauser's list ($n = 29$) were scored as close as possible to comparable occupations on the list (see Appendix B). I was able to assign status scores for 201 of the 336 polio victims in my sample (59.8%).

Status score	Category	Example occupations
1	Professional	Dentist, Lawyer
2	Entrepreneurial/Clerical	Clerk, Farmer, Sales manager
3	Skilled labour	Carpenter, Stonemason
4	Semi-skilled labour	Porkpacker, Teamster
5	Unskilled labour	Farm labourer

Table 3.2 Status scores with associated occupational categories and occupations.

For analysis by population, data from the 1921 Census was used. As in the polio sample, the occupations listed were coded according to Hauser's (1982) five-point scale. Data sets included the 1921 Ontario male population over 10 years of age (Canada, Dominion Bureau of Statistics, 1925, table 2), 1921 families by male head of household for Hamilton and Toronto (Canada, Dominion Bureau of Statistics, 1925, table 41), and child population by male head of household for Hamilton and Toronto (Canada, Dominion Bureau of Statistics, 1925, table 41). I was able to assign status scores to 82-87% of these Census data sets.

Statistical analysis

The chi-square test was used to determine statistical significance in sex ratios, using GraphPad's QuickCalc online software. The unpaired t-test was used to test for statistical significance in differences in means for age at death, minimum family or sibship size, or birth order between categories, groups, or time periods, again using

GraphPad's QuickCalc online software. The unpaired t-test (also known as student's test) was used as it compared two different groups of people, as opposed to the paired t-test which compares the same group at different times. Data were converted to z-scores to normalize distributions when necessary. Nonparametric Mann-Whitney U tests were also run on all samples that were subjected to the t-test, using IBM's SPSS Statistics 19 software. T-test results are reported, except in the case of family size in rural versus urban and suburban areas, where the more conservative Mann-Whitney U test produced a different result. In that case, the Mann-Whitney U test results are reported.

One-way ANOVA was used to test for statistical significance of differences in mean minimum family size when Levene's test showed equal variances, with Tukey and Bonferroni post hoc tests applied as needed. When Levene's test showed unequal variances in means for minimum family size or age at death, Welch's ANOVA was employed with Games-Howell post hoc test as needed. Levene's test, one-way ANOVA, Welch's ANOVA, and all post hoc tests were conducted using IBM's SPSS Statistics 19 software.

Where means are reported in the tables and text, the number after the \pm symbol is the standard deviation.

Summary

Through data collection from a variety of archival sources, the geographical tools of GIS, and information provided in secondary historical works, I was able to construct a detailed database of 336 polio victims from the Wentworth and York Counties of southern Ontario spanning the years 1900-1937. The information in this database forms the basis for the

analyses presented in this dissertation. While non-British immigrants and highly mobile groups are likely underrepresented in the additional records, the basic information from the death registrations is far less likely to contain systematic biases.

Chapter 4 Results and Discussion: Patterns of Polio Mortality and Demography

Introduction

In this chapter I examine the basic demographic characteristics of those individuals whose deaths were ascribed to polio in Ontario's Wentworth and York Counties from 1900-1937, including age, sex, nativity, birthplace, ethnicity, and religion. I also examine the illnesses and deaths of these individuals in terms of illness durations, place of death, and seasonality. I show how polio mortality in Wentworth and York in this period follows the known broad patterns of epidemic polio in the 20th century, yet also exhibits some interesting features that depart from or complicate the expected pattern. This analysis reveals a local and fatal character to epidemic polio as it emerged in this region in the context of the demographic transition.

Age and Sex

Age at death

Age at death is one of the most informative features of polio's epidemiology. The predicted pattern, based on previous studies and the hygiene hypothesis, is one of increasing average age at death over time from an initial concentration of deaths in the under-5 age group (Nathanson & Martin, 1979).

Ages at death in this sample ranged from as few as four days to 78 years. For the entire sample of 336 polio deaths, the average age at death was 11.1 ± 12.2 years and the median age at death was 7 years. With the 33 chronic cases removed ($n = 303$), the average age at death was 9.4 ± 9.5 years and the median was 6 years.

For the entire sample of 336, 38.1% ($n = 128$) died before age 5, 21.1% ($n = 71$) were 5-9 years old, 23.2% ($n = 78$) were 10-19 years old, and 17.6% ($n = 59$) were 20 years or older. With the chronic cases removed, 40.6% ($n = 123$) were under age 5, 23.1% ($n = 70$) were 5-9 years old, 22.4% ($n = 68$) were 10-19 years old, and 13.9% ($n = 42$) were 20 years or older. Figure 4.1 shows the ages at death in 5-year age groups for the entire sample and for acute cases (<6 months duration) only.

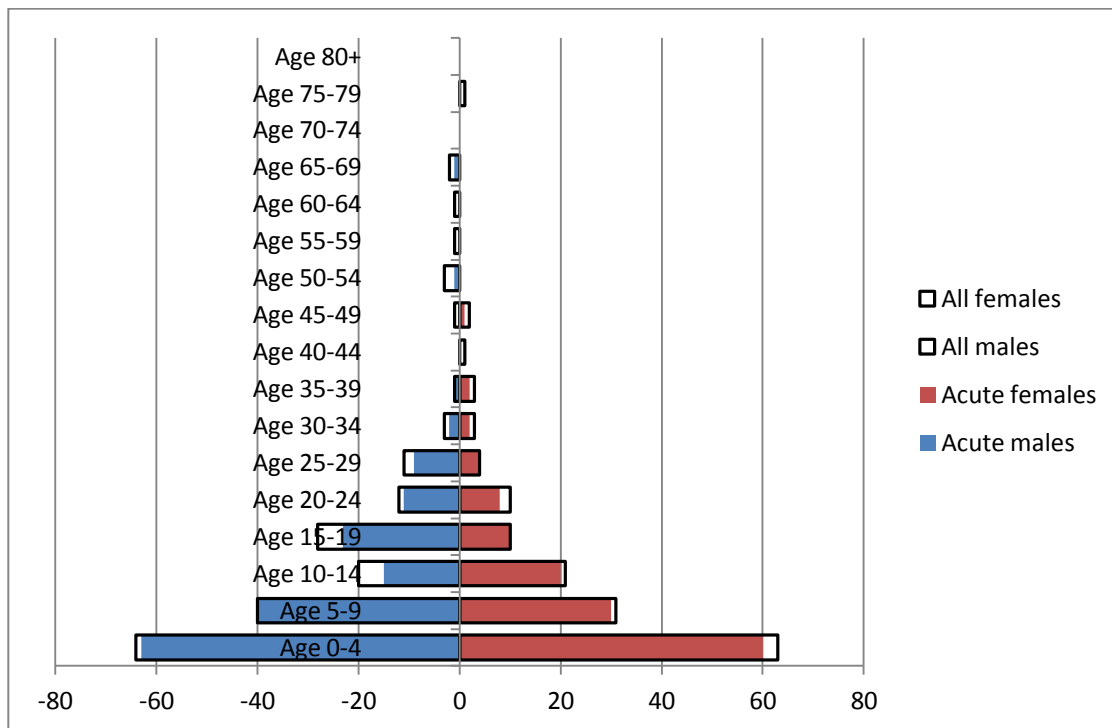


Figure 4.1 Age-specific mortality of all polio deaths ($n = 336$) and acute polio deaths only (<6 months duration, $n = 303$) in Wentworth and York Counties 1900-1937.

When mortality is examined by decade, there is an overall pattern of increasing age at death over time, with some fluctuation (Table 4.1). The lowest median age of 1 year is found in the 1900-1909 period, suggestive of the traditional endemic pattern which gave rise to the term “infantile paralysis” (Pichel, 1950). The highest median age at death, 9 years, is found in the final 1930-1937 period, firmly beyond the under-5 category and indicative of an epidemic pattern (Nathanson & Martin, 1979).

Time period	Median age at death (years)	Average age at death (years)	Number of deaths
1900-1909	1	1.1 ± 0.6	8
1910-1919	4	10.0 ± 11.5	78
1920-1929	4	7.1 ± 9.7	87
1930-1937	9	11.1 ± 7.6	130
All years (1900-1937)	6	9.4 ± 9.5	303

Table 4.1 Median and average ages at death (rounded) by decade for acute polio deaths ($n = 303$).

For the first three decades of the 20th century, more than half of the acute polio deaths in Wentworth/York occurred in children under age 5, 60.5% in the 1900-1919 period and 55.2% in the 1920-1929 decade. In the 1930-1937 period, only 17.7% of polio deaths occurred in children under age 5. In the 1930-1936 period 24.2% were in that age group. Only 11.8% of cases occurred under age 5 in the 1937 epidemic. The greatest shift in the age distribution of polio occurred in the early 1930s: in the 1930-1937 period, for the first time the 5-9 age group has more deaths than the 0-4 age group (Figure 4.2). An unpaired (or independent-samples) t-test (using z-scores to normalize the data) found that the difference between the average age at death for acute cases in 1910-1929 (8.4 ± 10.7) versus 1930-1937 (11.1 ± 7.6) was statistically significant ($t(293) = 2.95, p = 0.01$).

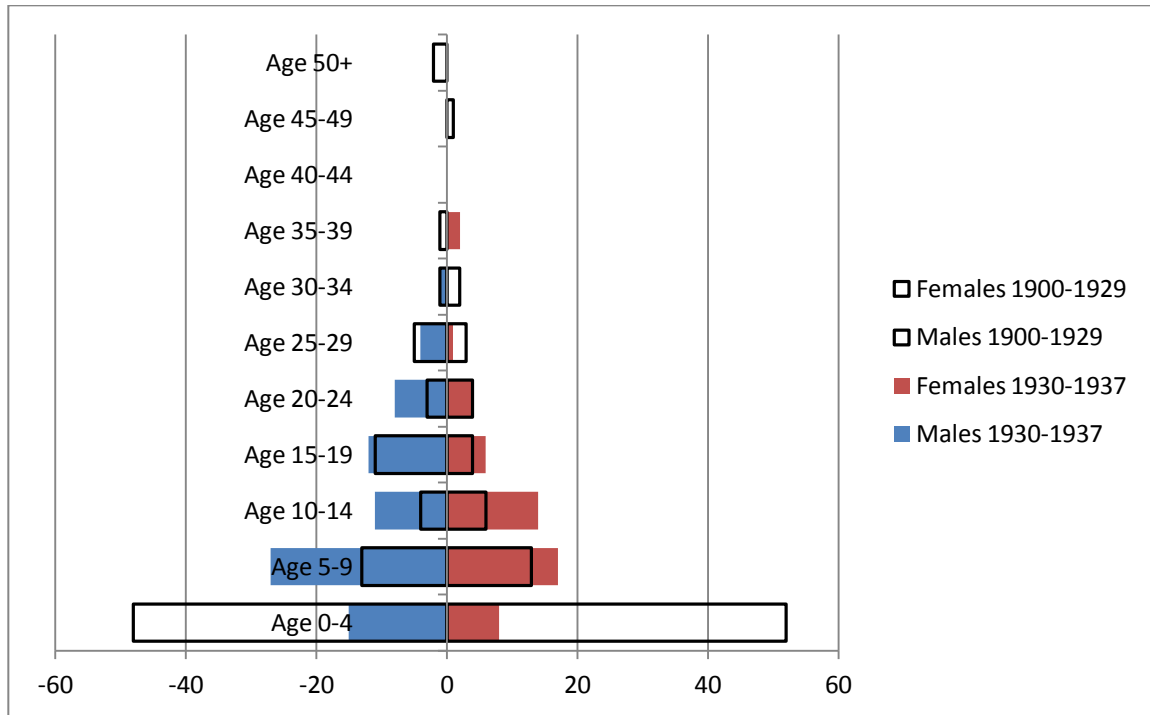


Figure 4.2 Age-specific mortality pyramid for acute polio deaths in Wentworth and York, 1900-1929 ($n = 173$) and 1930-1937 ($n = 130$).

The results for Wentworth/York fall within the range of variation seen for other polio epidemics in northeastern North America and northern Europe (Figure 4.3). Olin (1952) compares case lethality rates in different age groups in Sweden for 1905, 1911-1913, 1925-1924 and 1935-1944. The greatest change is seen in the 0-3 and 3-7 age groups between 1911-1913 and 1925-1934 (Olin, 1952, p. 373, chart 50). This is similar to the Wentworth/York pattern, where the largest shift upward in age groups affected by polio occurred between the 1920s and 1930s.

This upward age shift in proportional polio mortality between 1918 and 1935 has also been noted in the United States. Forsbeck and Luther (1930) date the age shift in Massachusetts to 1918 onwards; Horton and Rubenstein (1948) pinpoint the most marked

decline in polio mortality in the 0-4 age group to the 1920-1930 decade, especially from 1927-1930. For Connecticut, Pichel (1950) observed the largest decline in fatal cases in the 0-4 age group (adjusted for population) between 1926-1930 and 1931-1935. Finally, Gilliam's (1948) data for 20 (mostly northern) US states shows a narrowing of the polio mortality gap in the 1920s between the 0-4 and 5-9 age groups.⁷ As of 1934, the ratios in the 0-4, 5-9, and 10-14 age groups were equal (Gilliam, 1948). As Figure 4.3 shows, the drop in polio mortality is evident between the 1920s and 1930s throughout the northeastern North America region.

⁷ Gilliam (1948) compared the ratio of the polio death rate at a given age to the adjusted polio death rate at all ages.

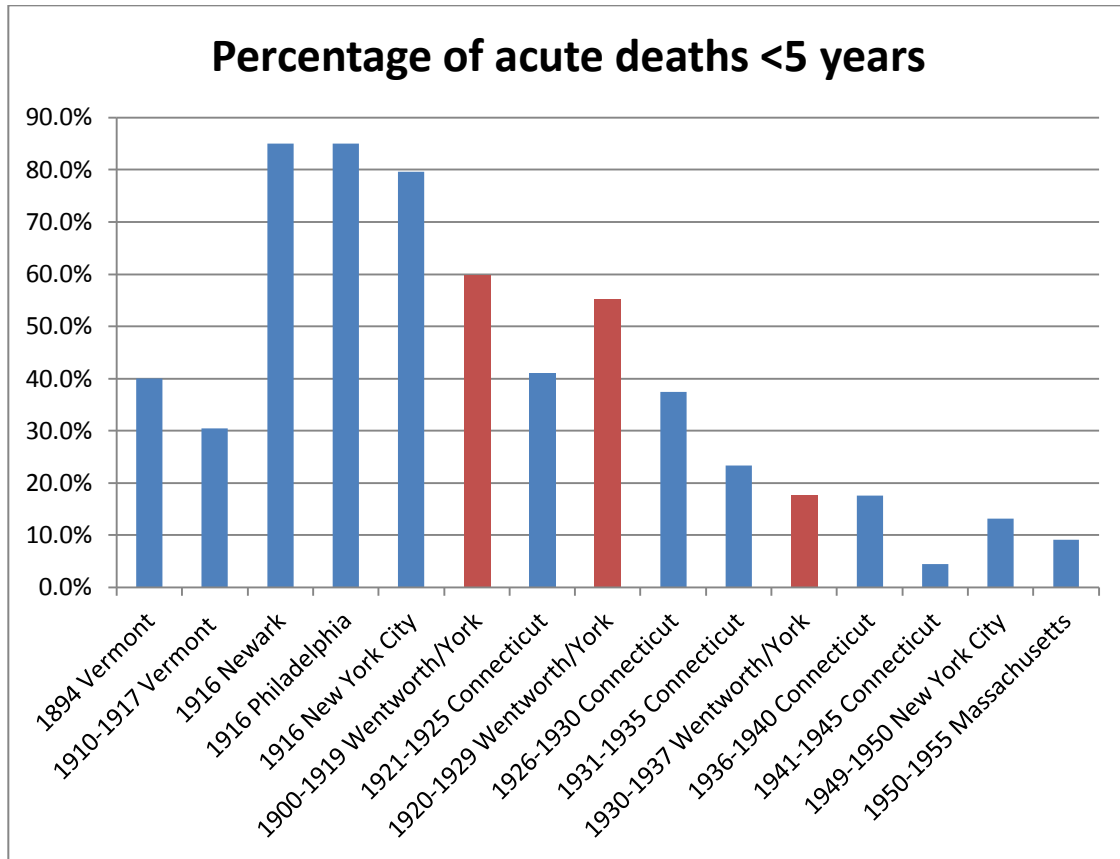


Figure 4.3 Comparison of proportion of acute polio deaths under age five in selected epidemics in northeastern North America, not adjusted for population. Wentworth/York results from this study are highlighted in red. Sources: Abramson & Greenberg (1955), Caverly (1924), Galishoff (1976), “Poliomyelitic Deaths, City of New York, 1916” (nd), Olin (1952), Pichel (1950), “Polio in Philadelphia” (nd), Weinstein (1957).

Similar to the findings of Nielsen et al. (2002a) (see Chapter 2), a U-shaped age curve is seen in the Wentworth/York acute polio deaths, but only in the last study decade, the 1928-1937 period (Figure 4.4). In contrast to Nielsen et al.’s (2002a) results for Copenhagen, Denmark for 1940-1953, which showed a dip at ages 8 to 9 for paralysis cases and at age 4 to 5 in the case-fatality rate, the dip in the 1928-1937 in

Wentworth/York occurs at ages 7 to 8. Why this might be the case is discussed in Chapter 6.

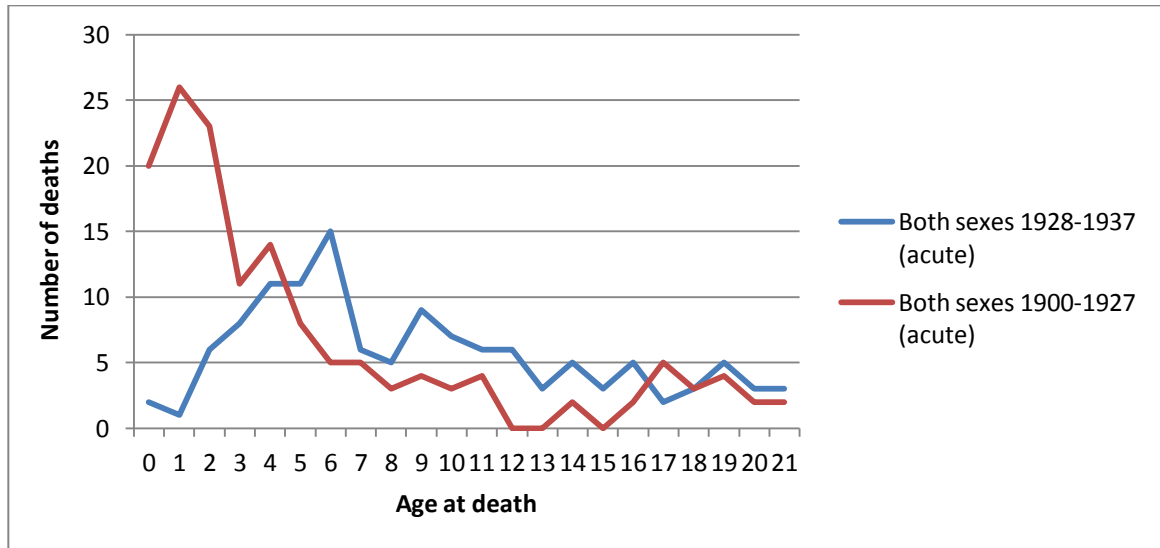


Figure 4.4 Number of deaths for Wentworth/York by age at death for 1900-1927 vs. 1928-1937.

The combination of waning maternal antibody protection and the weaning process is likely to have had a significant impact on the timing of polio deaths in children under two years old. There were 51 deaths in this age group in Wentworth/York, all in the acute category. The youngest died at four days old and the oldest at 11 months. There are relatively few deaths under six months of age ($n = 9$), only 17.6% of the polio deaths occurred under age two. Infants are protected by maternal antibodies transferred via the placenta for three to nine months after birth and by maternal antibodies from breast milk during the period of nursing (Zinkernagel, 2003). Turner's (1950) study found that while 72% of Baltimore infants under three months of age had antibodies to Type 2 poliovirus, by age one this had dropped to only 10%. Similarly, 90% of infants in North Carolina had

neutralizing antibodies to Type 2 poliovirus at birth, but these antibodies were no longer detectable after six months of age (Melnick & Ledinko, 1951). Thus a rise in polio deaths in infants six months of age and over corresponds to a loss of maternal antibody protection. It may also correspond to common weaning timing in urban southern Ontario. According to an article in *Child Welfare News*, just under half of infants in Toronto in 1917 were weaned between eight and nine months of age, considered then to be the “proper time” for weaning (“Breast feeding continues to decline,” 1930). It is not clear, however, what exactly this weaning “time” constituted, as the article also notes that mothers gave babies other foods while continuing to nurse (“Breast feeding continues to decline,” 1930).

It is difficult to examine changes over time due to small sample sizes; however, if the pattern displayed in Figure 4.5 is accurate, it appears that the process of weaning, through which complementary foods were gradually added to the child’s diet, may have become less relevant over time as water and milk supplies became safer (see Chapter 2). This change over time might also be attributed to a loss of polio antibodies in women of reproductive age, leading to an absence of maternal antibodies in infants from birth (and thus no difference in timing of deaths over the first two years of life); however, the studies conducted in the US in the 1940s, showing that infants begin life with full protection, would seem to indicate that this was not the case (Melnick & Ledinko, 1951; Turner et al., 1950).

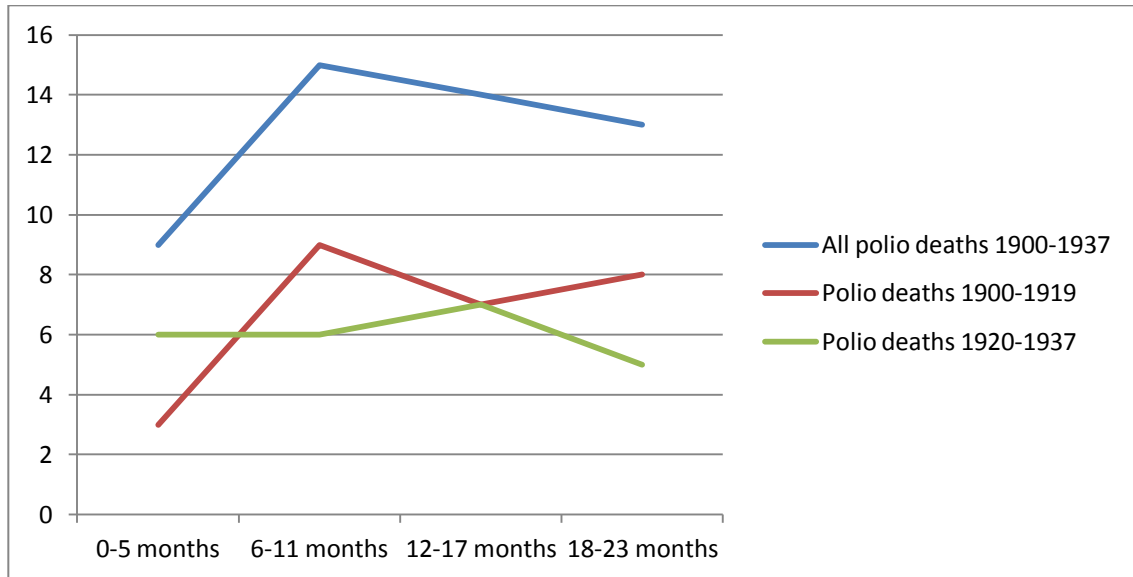


Figure 4.5 Number of acute polio deaths in each age group compared to all acute polio deaths under two years old in Wentworth and York Counties 1900-1937.

Sex: males vs. females

Male deaths from polio have consistently outnumbered those for females in different times and places; the reason or reasons for this discrepancy remain unknown. Out of 336 polio deaths in Wentworth and York, 187 (55.7%) were males and 149 (44.4%) females, resulting in a sex ratio of 1.26. When only acute deaths were considered, there were 166 males and 137 females, yielding a sex ratio of 1.21. These results are slightly lower than the ‘expected’ overall sex ratio of about 1.3 cited in medical textbooks (Mortimer & Cherry, 2004). However, the sex ratio in Wentworth/York changed over time. Among acute deaths, the sex ratio was approximately equal in the first part of the study period (1900-1927 collectively) but was much higher for the last decade (1928-1937) at 1.6 males per female ($\chi^2(1, 142) = 7.21, p = 0.0072$) when compared to an expected ratio of 1.0 (based on the equal sex ratio in the 1900-1927 period).

Figure 4.6 displays cumulative male and female polio deaths over time and illustrates how male polio deaths begin to clearly and consistently outnumber female polio deaths in the final decade of the study period. Looking at the cumulative totals, the sex difference becomes statistically significant with the inclusion of the 1937 epidemic deaths ($\chi^2(1, 336) = 4.298, p = 0.038$).

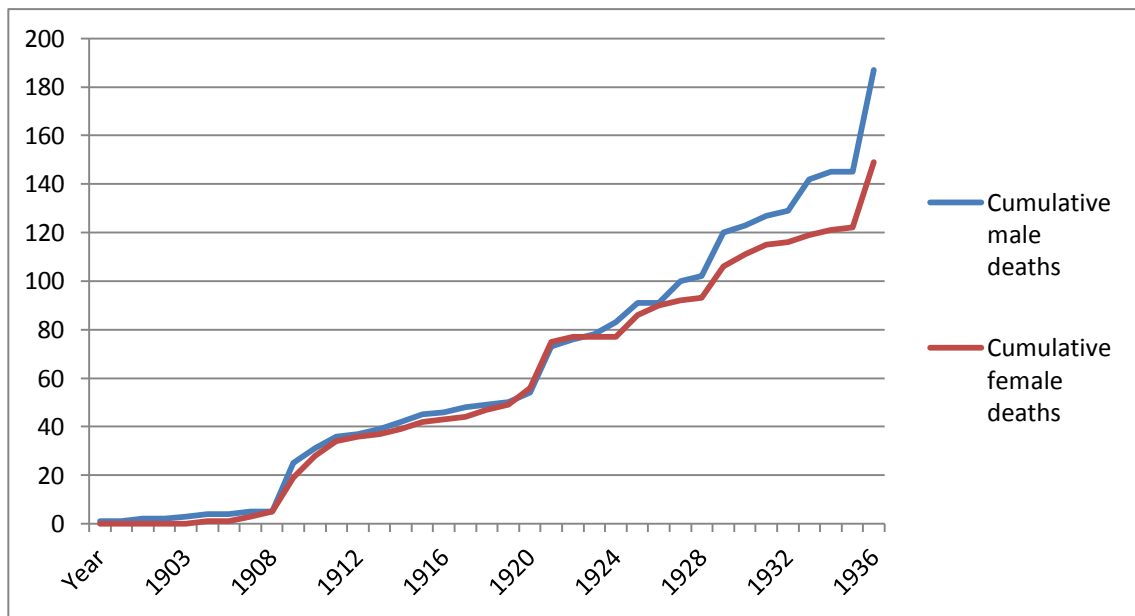


Figure 4.6 Cumulative number of male vs. female polio deaths in Wentworth and York Counties from 1900-1937 ($n = 336$). The sex difference becomes statistically significant as of 1937.

Age and sex

Males and females display moderately different patterns in age at death from polio (see Appendix C for a breakdown by age group and comparison to other studies). In order to further examine male versus female polio mortality over time, particularly in children and

adolescents and adults during the childbearing years,⁸ sex ratios were calculated for the 0-19 and 20-44 age categories (Table 4.2). The excess male mortality in both children and adults is only found in the last decade of the study period and is higher in adults with a sex ratio of 2.0 compared to 1.5 in ages 0-19. The 2.0 sex ratio in 20-44-year-olds in the 1928-1937 period becomes statistically significant ($\chi^2(1, 21) = 4.38, p = 0.036$) with an expected sex ratio of 0.8 or lower (predicting excess female deaths during the childbearing years). Thus, despite some variation in the five-year age groups (see Appendix C), which may be influenced by small sample size, there is a clear shift in the sex ratio over time in both children and adults from relatively equal polio mortality to significantly more polio deaths among males.

	Entire sample	Acute 1900-37	Acute 1900-27	Acute 1928-37
All ages	1.3	1.2	1.0	1.6
Age 0-19	1.2	1.2	1.0	1.5
Age 20-44	1.3	1.4	1.0	2.0

Table 4.2 Sex ratios in polio mortality for entire sample ($n = 336$) and acute deaths ($n = 303$) by age group and time period.

Main findings and discussion

The general predominance of male over female deaths in Wentworth-York conforms to the expected pattern for polio, as does the increase in age at death over time. However, the growing gap between male and female deaths from the mid-1920s does not fit the expected pattern. As these results demonstrate, the overall sex ratio cited for polio masks considerable variability by age and social circumstances, and in morbidity versus

⁸ Previous examinations of polio mortality patterns have noted excess female deaths in the child-bearing years, ages 20-44 (Paffenbarger & Wilson, 1955; Mortimer & Cherry, 2004). Molner & Brody (1959) found more male than female paralytic polio cases in the 20-39 age group, contrary to expectations, in Detroit's 1958 epidemic, but in the post-vaccine era this could be attributed to vaccination patterns.

mortality. The 1.3 ratio also gives an impression of an endogenous (biological) reason for the differences in polio morbidity and mortality between males and females. The sex ratio, however, changes over time in response to social and demographic conditions. Nielsen et al.'s (2002b) cross-sex transmission hypothesis, for instance, links polio's sex ratio to family size.

The lower than expected proportion of females to males dying in the 20-39 age group, representing a portion of the childbearing years for women, may be related to the apparent absence of deaths of women who became infected during pregnancy. Pregnancy is noted as a risk factor for severe polio (Anderson, Anderson, Skaar, & Sandler, 1952; McCord, Alcock, & Hildes, 1955; Siegel & Greenberg, 1956), yet in Wentworth/York only one individual could be identified as having contracted polio during her pregnancy. This information comes from her obituary in the *Hamilton Spectator* (9 March, 1911), not from her death record:

Annie, wife of Herbert Billing, died rather unexpectedly at the family residence 408 Cannon street east, yesterday afternoon, aged 30 years. About seven months ago she suffered an attack of poliomyelitis, but never fully recovered from that attack, and although her death was not altogether unexpected, it came rather suddenly and will be regretted by a wide circle of friends....
Besides her husband, she is survived by three young children, ranging in age from five years down to three weeks...
[p. 11]

Other similar cases may have occurred in Wentworth-York but are undocumented.

Horn's (1955) study of polio mortality rates in Los Angeles County found that they were much higher among pregnant versus non-pregnant women in the 1934-1947 period; however, in the 1948-1952 period polio mortality rates were identical in the two groups. Horn (1955) seems to suggest that this decline in polio mortality among pregnant women

may be attributed to improvements in medical care after 1948. In the 1953 epidemic in Winnipeg, the case-rate for pregnant women was more than double that of males in the same age group (McCord, Alcock, & Hildes, 1955).

While females have been found to have higher overall case-rates in adulthood, Weinstein et al. (1951) and Weinstein (1957) have demonstrated that fatal cases of polio can display a very different pattern. Studies of polio in Massachusetts between 1949 and 1955 found that while the greater number of females in adult cases was in part due to pregnancy, in fatal adult cases males outnumbered females, though only slightly (sex ratio of 1.1). It is also possible that we do not see more reproductive-age female polio deaths in the Wentworth/York sample in part because there simply were not enough susceptible pregnant women in the population to balance or reverse the general trend of excess male deaths for that age group, especially during the 1930s when women had fewer children (McInnis, 2000). Reduced family size may also be behind the overall shift in sex ratio over time in this sample. Nielsen et al. (2002b) found a more equal sex distribution of polio cases with increasing family size, and that this pattern was even stronger among paralytic cases (see Chapter 2 for discussion of the reasons why this might be).

The timing of the upward shift in age at death in polio is associated with a decline in the proportion of the population in the youngest age groups, or an aging of the population, as fertility declined in Ontario, Canada (Appendix D.3) and other Western societies. Within Ontario, this age shift was more pronounced in Wentworth/York than in Ontario as a whole (Appendix D.4), and was particularly marked in Toronto (Appendix D.1). Horton and Rubenstein (1948), Pichel (1950), and Weinstein (1957) observed this

relationship to fertility decline in Massachusetts and Connecticut and determined that the change in population structure could only partially account for polio's upward age shift.

Dauer (1955) also noted a reduction in the proportion of paralytic polio in the under-5 age group in Massachusetts and proposed that the falling birthrate between the two World Wars, with the resulting reduction in family size, may have contributed to the trend.

While Horton and Rubenstein (1948), Pichel (1950), and Dauer (1955) agree that a lower birthrate and consequent 'aging' of the population played a part in the changing epidemiology of polio, they also agree that there were other factors at play. These 'other factors' are, frustratingly, left undetermined.⁹ Pichel (1950) describes the reasons for polio's age shift as "complicated and probably obscure" (p. 337). It is possible, however, that the increasing comprehensiveness of public sanitation measures contributed to the lower proportion of young children contracting fatal cases of polio. Sewer systems, for example, were expanded outside the urban cores during this period; sewage and water systems became more reliable; and regulations were implemented and enforced.

However, Dauer (1955, p. 954) argues that factors such as birth rates, climate, and "other intrinsic and extrinsic factors" need to be studied carefully before attributing polio's age shift solely to the hygiene hypothesis. In any case, the children who contracted and died of polio in the 1920s-1930s were the first generation born after IMRs fell late in the second decade of the century.

⁹ While Horton and Rubenstein (1948) argued that increased reporting of non-paralytic cases accounted for the difference, this issue does not explain the age change when only deaths are examined rather than overall cases (Pichel, 1950).

Although there were few adults among the polio deaths in Wentworth-York, the vast majority are chronic cases; only one contracted the disease before adulthood: Peter MacNabb, who died in 1920 at age 66. He died in Hamilton at the House of Refuge where he lived, having been an “invalid all his life”. His cause of death was listed as “infantile paralysis since babyhood”, with “debility” as a contributing cause. The duration of illness among adults ranged between one and six years before death, including 78-year-old Martha Ritchie, who died in Toronto in 1935 of “myocardial degeneration” (duration seven months), with poliomyelitis as the contributing cause (duration six years). Two men from Hamilton, John L. Brown (1857-1912) and Thomas Crombleholme (1880-1935), both aged 54 at death, had chronic polio for two years. Brown succumbed to “asphyxia” and Crombleholme to “bronchopneumonia”.

Such cases of polio in later adulthood may be anomalies in a disease usually associated with the young. Perhaps these older individuals simply managed to avoid exposure earlier in life, never developed immunity when they were exposed, or failed to acquire booster infections and so their immunity waned. It is also possible that some of these cases were misdiagnosed. John L. Brown’s death registration may have listed “Poliomyelitis Anterior Chronic” as his cause of death, but his obituary in the *Hamilton Spectator* (15 May 1912, p. 16) states that he had suffered from tuberculosis of the bone for the past two years – the same duration as his supposed chronic polio. To give another example, 57-year-old George Pearson’s cause of death in 1923 is listed as “progressive paralysis of poliomyelitis type”, duration nine months, with cardiac paralysis. Progressive paralysis over nine months does not sound like a typical case of polio, and “poliomyelitis

type” is somewhat ambiguous. His death registration states that no autopsy was performed. Similarly, 66-year-old James Crichton’s death in January of 1923 due to “anterior poliomyelitis” with “meningitis” as a contributing cause may also be a case of misdiagnosis, especially as meningitis and poliomyelitis could be easily confused and the death occurred far outside the typical polio season. No autopsy was performed in his case either.

Overall, examination of age and sex patterns in this study and others have demonstrated how polio mortality patterns can differ significantly from the patterns typical for polio morbidity.

Illness Duration and Seasonality

Chronic polio

Of the sample of 336 polio deaths in Wentworth-York, 33 were chronic, while the rest ($n = 303$) were either acute or convalescent cases (with illness durations of less than six months), or had no specific information indicating chronic versus acute polio. The latter were presumed to be “acute”.

In chronic cases, death might not occur until many years after the acute infection, although polio would still be a primary or contributory cause of death. Norman Kemp, for example, contracted polio in Hamilton at the age of three, likely during the 1910 epidemic. He passed away at age 17, with heart disease listed as his primary cause of death and infantile paralysis as a contributory cause. His occupation at the time of his death was listed as “invalid”, and his obituary mentions he had been in poor health:

Norman William Kemp, eldest son of William F. and Mrs. Kemp, passed away yesterday at his parents' residence, 135 Queen street north, aged 17 years. Deceased has not been in good health of late. He had resided in this city practically all his life, and was an adherent of Macnab street Gospel hall, where his many friends will receive the news of his death with sincere regret. Surviving are his parents and one brother, Ross, at home. The funeral will take place on Monday afternoon at 2 o'clock from his parents' residence to Hamilton cemetery.

[*Hamilton Spectator*, 31 October 1924, p. 23]

In another example, Harry Partridge passed away in Hamilton at age 18, with cause of death listed as “infantile paralysis at 5 ½ years” with “deformity of chest due to paralysis of muscles” and “asphxia and myocardial failure”. He likely caught polio during the 1922 epidemic in Hamilton. His obituary simply alludes to “a long illness” (*Hamilton Spectator*, 15 June 1935, p. 10).

Acute illness duration

Information on the duration of illness was available for 181 individuals whose polio lasted less than six months. The average illness duration was 8.6 ± 14.9 days and the median was five days. Since the few illnesses that exceed one or two months (three deaths with illness durations of three to four months and two deaths with illness durations of two months) distort the average, the median is likely the best measure of the usual duration of the acute polio illness. The median duration of physician attendance for acute cases was three days, two days less than the median illness duration.

While acute polio illnesses of between several days to a week were the norm, with medical care generally beginning one or several days into the illness, sometimes death was very sudden. Such was the case in the death of three-year-old Grace Hancock of Toronto (Figure 4.7):

Ten minutes after being admitted to the Hospital for Sick Children, yesterday afternoon, three-and-a-half-year-old Grace Hancock, of 53 Broadview ave., died of what was later decided to be infantile paralysis.

Yesterday morning, apparently well, the child was playing around the house. After lunch she became ill, and at 2.30 the family physician was called to attend her. On his arrival, the child was found to be unconscious, and he had her removed to the hospital. Dr. J. M. Casserley, coroner, ordered an autopsy, which showed acute infantile paralysis. No inquest will be held.

[*Toronto Evening Telegram*, 11 October 1930, p. 21]



Figure 4.7 *Toronto Evening Telegram*, 11 October 1930, p. 21.

The “iron lungs” used during the 1937 epidemic kept many polio patients alive long enough for them to survive the acute illness. However, for most it was not enough:

At Hamilton, where an “iron lung” respirator was used in vain, Alfred Drake, 22, lost his battle with death. He had been in the machine 36 hours, kept alive by the mechanical breathing. His was the fourth death there, while two new cases were admitted to Hamilton General hospital.

[“EPIDEMIC OF POLIOMYELITIS CLAIMS EIGHT MORE LIVES”, *Hamilton Spectator*, 30 August 1937, p. 13]

With nine children in “iron lungs” and six in the celluloid corsets, now being used for more progressed cases, the Hospital for Sick Children to-day reported 15 patients examined, one case of actual paralysis, during the last 24 hours. Despite all efforts to save her, the six-year-old Willowdale girl who was placed in an “iron lung” yesterday, died, having paralysis of the throat, as well as of the chest muscles.

[“SIX-YEAR-OLD GIRL DIES OF PARALYSIS”, *Toronto Daily Star*, 7 October 1937, p. 1]

The “six-year-old Willowdale girl” mentioned above is likely actually eight-year-old Sirkka Tarvainen, who was born in Finland and came to Canada as an infant.

As the report on the 1937 epidemic was being written the following year, a total of 63 polio cases had received treatment in respirators. Of those, 63% had died, 19% had recovered from the respiratory paralysis, and 18% were still in respirators (Ontario Department of Health, 1938, p. 46). Most respirator cases began treatment within the first week of illness, and those who did not survive usually died within the second week (Table 4.3). Hospital physicians admitted afterwards that respirators were of little use in most cases; in the minority of cases in which they were helpful, they mainly prolonged life or relieved symptoms as often as they saved lives (Ontario Department of Health, 1938, p. 47).

Outcome of case	Number of individuals	Average length of time in respirator
Died	40	5 days
Recovered	12	50 days
Still in respirator	11	150 days (at time of report)

Table 4.3 Results and duration of respirator treatment for polio patients during 1937 epidemic in Ontario. Source: Ontario Department of Health (1938, p. 46).

“Death Walks in Summer”¹⁰: seasonality of polio mortality

Acute polio deaths in Wentworth-York display strong seasonality, following the late summer-early autumn pattern typical of epidemic polio in northern North America and northern Europe (Figure 4.8). Chronic polio deaths are more evenly distributed throughout the year, with the highest percentage (18.2%, six of the 33 chronic deaths) occurring in March (Figure 4.8). As Figure 4.8 displays, no chronic polio deaths occurred in the month of September; this may be due to some chronic deaths being misclassified as “acute” when no information to indicate otherwise was available. Forsbeck and Luther’s (1930) examination of reported polio incidence in Massachusetts from 1907-1929 also found a September peak and a slight secondary peak in March. As these are *reported cases* and not deaths, chronic polio deaths do not account for that March bump, suggesting that the small March peak in acute polio deaths in the Wentworth/York sample may not be simply a chance fluctuation resulting from small sample size. However, a satisfactory explanation for the small March bump is elusive; one possibility is a diagnostic confusion with cerebro-spinal meningitis, which peaked in April in Massachusetts (Forsbeck & Luther, 1930).

¹⁰ The title of an article on polio published in *Canadian Magazine* in 1934 (Davies, 1934).

The seasonality observed for polio deaths differs a little from that for polio illness, with the former showing a slightly stronger epidemic peak in illness onset in August and September and epidemic deaths continuing later into the autumn and winter (Figure 4.9). Polio illness onset was calculated from time of death using information on illness duration contained in the death registrations.

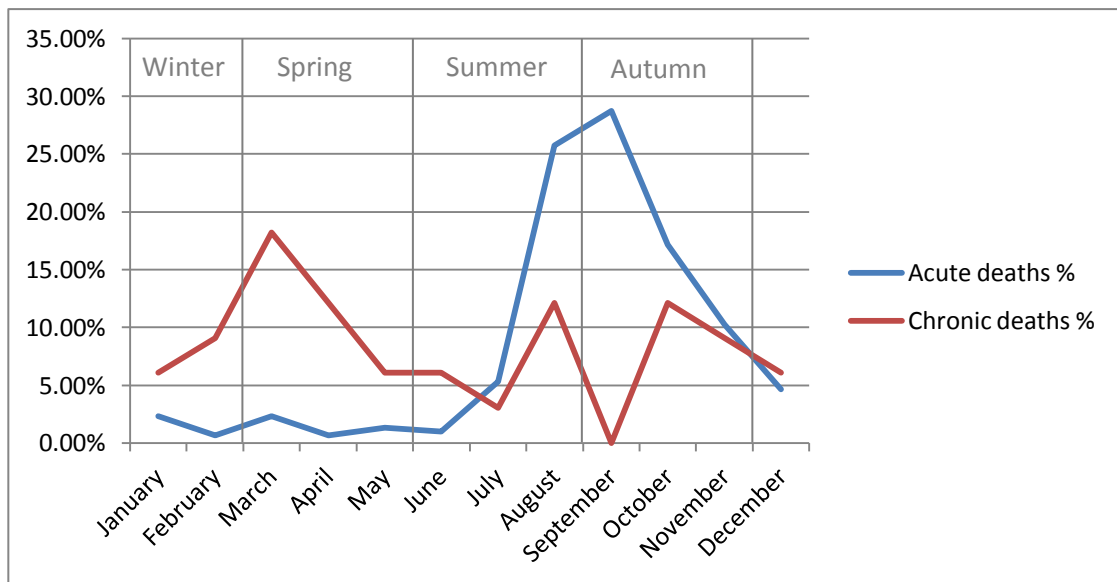


Figure 4.8 Monthly proportions of deaths of acute versus chronic polio cases, Wentworth and York Counties, 1900-1937.

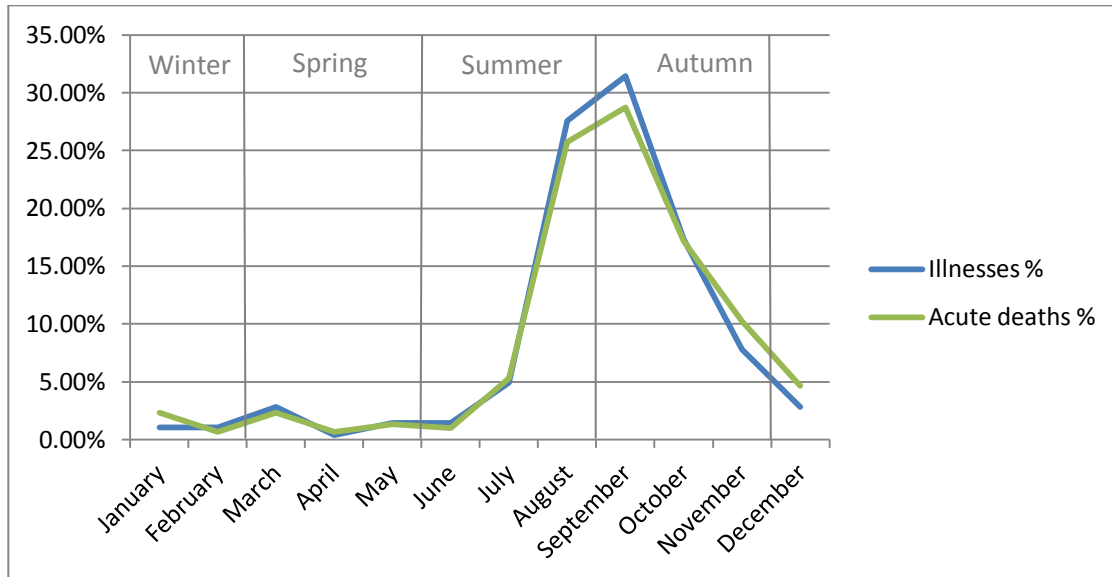


Figure 4.9 Monthly proportion of polio illnesses compared to polio deaths, Wentworth and York Counties, 1900-1937.

The seasonal distribution of chronic polio deaths appears to be related in part to respiratory infections, specifically bronchitis, pneumonia, and broncopneumonia, which were associated with 30.3% of chronic deaths. Polio survivors are known to have increased susceptibility to these infections, in part due to inability to eliminate secretions due to weakness in muscles used for coughing (Bieniek, Fischer, Headley, & Oppenheimer, 2001). This link with respiratory infections was recognized early on; Boudreau (1914), for example, observed in his study of polio in Ohio that “those cases in which a paralysis of the intercostals or diaphragm alone occurs, frequently die of pneumonia” (p. 73). In Wentworth/York, bronchitis, pneumonia, and bronchopneumonia together contributed to 6.6% of acute polio deaths as well, with 30% of these occurring in the month of November. In the six months before and after the polio season (January-

June), these respiratory infection complications contributed to 15.9% of all polio deaths, compared to only 1.0% from July through December.

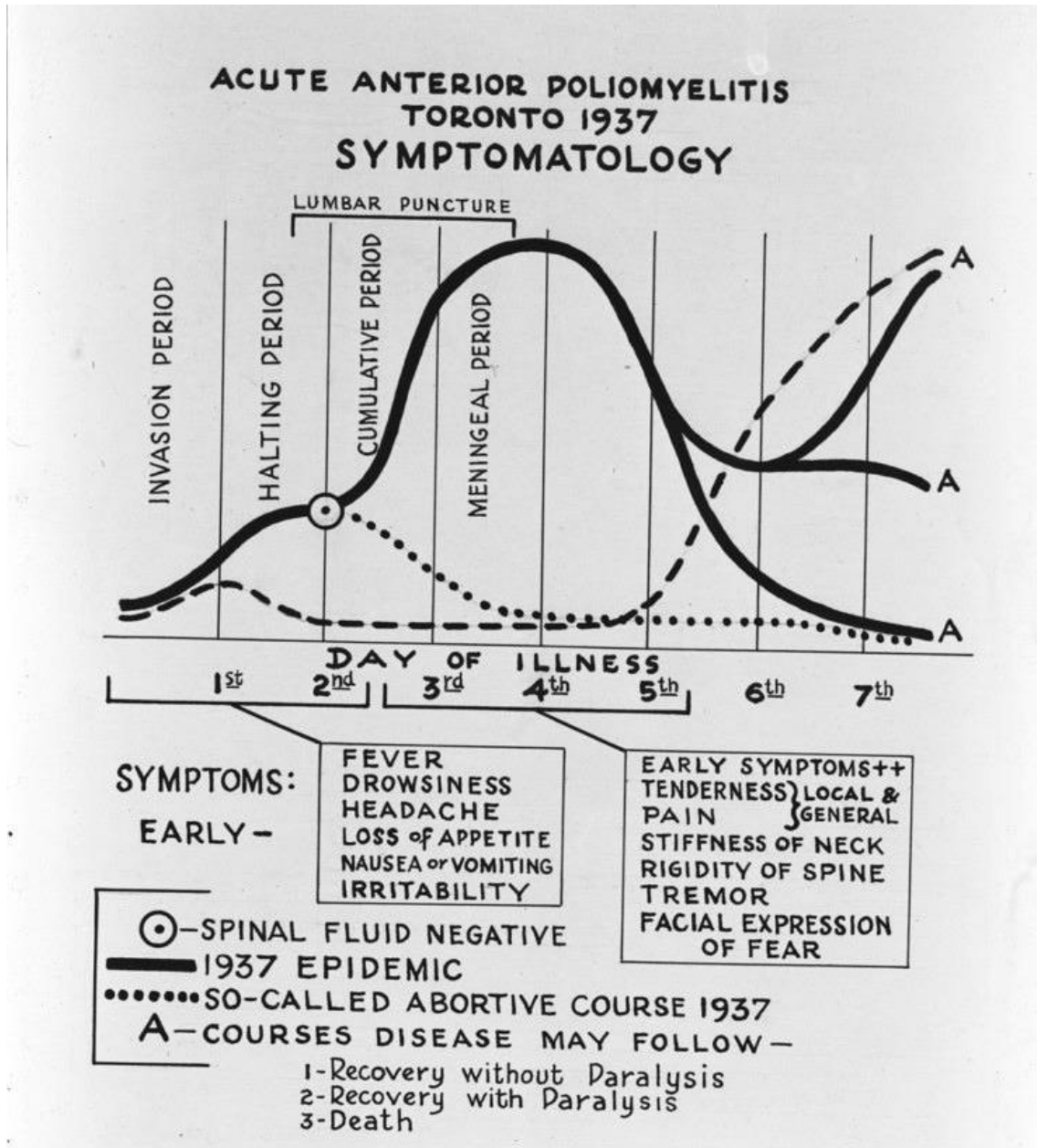


Figure 4.10 Symptomatology of acute anterior poliomyelitis during the 1937 Toronto epidemic. Source: City of Toronto Archives, Fonds 200, Series 372, Subseries 32, Item 876.

Main findings and discussion

Researchers in Toronto studying the 1937 epidemic developed a graph to describe the course of an acute polio illness (Figure 4.10). For the Wentworth/York data, the median difference of two days between illness duration and physician attendance fits with the general tendency for more alarming or specific symptoms to appear in the second or third day in severe cases, as illustrated in the graph. The *Report on Poliomyelitis in Ontario, 1937* states that “among cases who later showed paralysis, symptoms were characteristically observed for the first time later in the illness. This would imply that the onset of polio in paralysed cases was less abrupt, which is borne out by the observations above concerning the relatively small proportion of cases in which onset was described as ‘sudden’” (Ontario Department of Health, 1938, p. 39). Yet as the case of Grace Hancock illustrates, there was significant variability in the expression of polio. The *Report*, however, does note that a physician was more likely to be called within 48 hours of onset in bulbar cases compared to spinal cases during the 1937 epidemic, at 63.4% in bulbar cases compared to 52.7-54.3% in upper spinal and lower spinal cases, respectively (Ontario Department of Health, 1938, p. 40).

The seasonality of polio deaths in Ontario’s Wentworth and York Counties, with a late summer-early fall epidemic peak in August and September, matches the seasonal pattern in infant and child deaths observed elsewhere. Lovett (1908) noted this seasonal pattern early on, saying that polio “offers a striking resemblance to the prevalence of the gastro-intestinal diseases of children which affect children of the same age at much the same time of year” (p. 136). In fact, this was an early clue to the fecal-oral spread of polio

(Lovett, 1908). Swedlund (2010) notes that a home health manual of the 19th century, popular in the northern United States, referred to September as “the month malign” (Hall, 1876, p. 170). This late summer-early autumn pattern of infantile diarrhea (known by various names, including “cholera infantum” and “summer diarrhea”), caused by acute enteric infections, was not recent; it was noted in Britain in the 17th century (Appleby, 1980). However, the timing of the seasonal peak differed by latitude. While in Britain and Sweden the infant diarrhea season was late summer to early autumn, in the more northern United States, the peak commonly occurred in July-August and in the southern United States it was known as the “April-May disease” (Cheney, 1984, p. 563). This matches the seasonal variation by latitude seen in epidemic polio in temperate climates in the 20th century, in which the seasonal concentration of polio increased with distance from the equator (Smallman-Raynor, et al., 2006, pp. 214-215). In the northern hemisphere, the polio season was July-October, and January-April in the southern hemisphere (Smallman-Raynor et al., 2006, p. 207; van Rooyen & Rhodes, 1948). In Canada, the seasonal mortality peak was slightly later than it was in northern U.S. states.

Monachino (2008) found that for the period 1901-1911, diarrheal deaths in Hamilton children under age 12 were highest from July through October, with a peak in August. Typhoid fever was also active in Hamilton from August through October at the beginning of the 20th century (Parker, 2008). Janjua (2009) found that this late summer-early autumn (August-September) spike in infant mortality, dominated by food and waterborne infections, continued in Hamilton until the end of the 1920s. The seasonal polio pattern for Canada was late summer-early autumn as well, slightly later than in the

northern United States (Rutty, 2004). The peak of the polio season in the 1930-1945 period occurred around mid-August in the southern US, four weeks earlier than in the north and west of the country where the peak was generally in mid-September (Collins, 1946). Thus in the southern United States, the polio season did not appear to map onto the infant diarrhea season as closely as it did in the north. However, the timing of the peak is only part of the picture. In Mexico, where epidemic polio rates were comparatively low, there was only moderate seasonal variation, in contrast with the strong seasonal peaks farther north. However, polio peaked in September during the 1922-1949 period (Nettleton, 2002, pp. 142-146; Smallman-Raynor et al., 2006, pp. 210-213).

In Sweden, where polio did follow the same late summer-early autumn seasonal pattern as infant diarrhea, the disease was nicknamed “höstens spoke” (“autumn’s ghost”) because of its association with decomposing fruit and leaves (Axelsson, 2004). In North America, contemporaries associated both infantile diarrhea and polio with the hottest weather of the year and with flies as vectors (Appleby, 1980; Cheney, 1984; Condran & Lentzner, 2004).

This summer mortality pattern was specific to infants and children under two years of age in the 19th century; after age two, wintertime respiratory infections were the greater danger (Condran & Lentzner, 2004). This pattern changed in the early 20th century, all but disappearing by 1920 (Cheney, 1984; Condran & Lentzner, 2004). Summer mortality for one-to-two-year-olds fell first, as the trend towards earlier weaning increased in the late 19th and early 20th centuries and reduced the age at which infants were exposed to the dangers of contamination associated with artificial feeding (Cheney,

1984; Condran & Lentzner, 2004). Diarrheal mortality subsequently declined among infants once sanitary improvements were in place and public health initiatives were underway to educate mothers on childcare and to ensure the safety of the milk supply (Cheney, 1984).

As infant and child summer mortality declined, the impact of polio, the new summer plague, grew.¹¹ Despite the continued use of the term “infantile paralysis”, epidemic polio most often struck those who had survived that dangerous first year of life. The literature on polio’s social history has noted the psychological significance of the “polio season”, as parents worried and restricted potentially dangerous activities such as swimming. However, “polio season” was not only a time of danger and worry; for some, it was also the anniversary of a child’s death from polio. A memorial statement in the local newspaper, alongside the latest death notices, for the one-year-old daughter of a Hamilton motorman demonstrates this:

DOYLE – In ever loving memory of our dear little daughter, Alice Kathleen, who died August 13, 1910.

One year has gone, but still we miss her.

She was a flower too fair for earth,
Lent here but for a while,
God marked her when He gave her birth,
And took her with a smile.

Her father mourns, her mother weeps;
Still, heaven the little treasure keeps.
--Father and mother

[*Hamilton Spectator*, 15 August 1911, p. 8]

¹¹ Seasonal infantile diarrhea and polio have something else in common besides their seasonal pattern and a fecal-oral transmission route: their apparent predilection for previously healthy and well-nourished infants and children (Cheney, 1984; Condran & Lentzner, 2004). Cheney (1984) distinguishes the acute, seasonal diarrheal pattern from chronic diarrhea linked to malnutrition.

Origin and Background: Nativity, Birthplace, Ethnicity, and Religion

Native- versus foreign-born

Birthplace and immigrant status have been associated with variant mortality levels in 19th- and 20th-century North America. Immigrant status variously increased the risk of death from certain diseases and provided protection against others (Leavitt, 1996, p. 37). In Milwaukee in 1890 and 1900, for example, diarrheal deaths were strongly correlated with the percentage of foreign-born people living in each ward (Leavitt, 1996, p. 31). Furthermore, Rogers (1989) reports that there is evidence that native-born children and families suffered a higher proportion of polio cases in the 1916 epidemic in New York City (Doty, n.d.).

Information on birthplace was available for 308 out of 336 (91.7% of the overall sample) of individuals in Wentworth/York whose deaths were ascribed to polio. Thirty were born outside Canada (9.7%), while 278 (90.3%) were native-born. Comparing the polio sample to the 1921 Census, people who died from polio were slightly more likely to be native-born than the general or comparable population (Table 4.4). The nativity status of mothers of polio victims is potentially informative due to the relevance of their immune status. The birth countries for 279 out of 336 mothers of polio victims were known (83.0%). The vast majority, 87.8%, were born in Canada or elsewhere in the British Empire, but only 50.9% in Canada specifically.

Birthplace	Wentworth and York total population (1921 census) %	Wentworth and York population of children ages 7 to 14 (1921 census) %	Polio victims (Wentworth and York) %	Mothers of polio victims (Wentworth and York) %
<i>Canada</i>	63.1	81.1	90.3	50.9
Ontario	60.5		81.2	35.1
<i>British Isles and possessions (except Canada)</i>	28.9	13.9	6.5	36.9
British Isles	28.2		6.5	36.9
England	19.4		3.6	25.8
<i>Foreign/non-British total</i>	8.0	5.0	3.3	12.2
Europe	4.9		1.0	8.2
United States	2.7		2.3	3.9

Table 4.4 Proportion of population by birthplace for polio death sample with known birthplace for Wentworth/York 1900-1937 compared to 1921 Census data for Wentworth/York. Source: Canada (1924, Table 53, Table 110).

Overall, however, there are broadly similar proportions of British Empire-born (Canada included) individuals in both the polio victims and mothers of polio victims compared to the wider population, with slightly more polio victims being native-born compared to the 7-14-year-old population of Wentworth/York.

Ethnicity

Data on ethnicity came from the “race” category in the death registrations and censuses, and I observed that in these records the recorded “race” of the individual was generally based on the “race” of the father. These classifications varied widely and so were only marginally informative. In Canada in the early 20th century, “race”, nationality, and ethnicity were conflated concepts and used interchangeably (Buckner, 2008). For

example, someone of English ancestry born in Canada might be listed under “race” as either “white”, “British”, “English”, or “Canadian”.

Only 77.1% of the polio death records (259/336) contained information on “race”. Not surprisingly, the largest group included those listed as “English” ($n = 109$), followed by “White” ($n = 39$, including 4 also listed as “English” and 1 also listed as “Irish”), “Irish” ($n = 34$), “Scotch” or “Scottish” ($n = 20$), “Canadian” ($n = 16$), and “British” ($n = 8$). There was also 1 “British subject”, 1 “U.E.L.” (presumably identifying a United Empire Loyalist), and 2 listed as “American”. There were 9 individuals listed as “Hebrew” or “Jewish”. The remainder were listed by European country or region (3 Austrian, 1 Dutch, 1 Finnish, 2 French, 1 Galasian, 3 German, 2 Italian, 1 Macedonian, and 4 Polish) or a mix of nationalities (e.g. “German, Scotch, and English”).

For comparison to the 1921 Census, I used only the residents of Wentworth and York counties in the polio death sample ($n = 318$). The comparison cannot be precise because so much of my sample had “race” listed in classification such as “White” or “Canadian”, classifications which the census did not use. 58.6% ($n = 197$) of the polio sample of 336 had data on “race” that corresponded to the census categories.

Overall, the percentages for the so-called races in the polio sample were close to those for the population of the combined counties. The population was overwhelmingly “white” to begin with, at 86.4% British and 12.6% European (99.0% combined) (Canada, 1924). When only those listed “races” that corresponded to census racial categories were included, the Hebrew or Jewish proportions were the same at 4.7% of both the Wentworth/York population in the 1921 Census and in the polio sample. This result does

not support the hypothesis that Jews would have had higher polio mortality due to their comparatively lower infant mortality rates.¹²

Religion

In addition to research associating Jewish populations with lower infant and childhood mortality, studies have also associated high infant and childhood mortality with Roman Catholics, including Toronto (Mercier, 2006). The data for Catholics provide an opportunity to test whether the inverse relationship between IMR and epidemic polio seen at the national level operated at the micro level in Wentworth/York.

Including only those whose religions were explicitly specified in the archival documents seems to lead to an underestimation of the proportion of Jewish individuals in this comparison. If the six other individuals listed by “race” as “Hebrew” are assumed to be Jewish, then the proportion of Jewish people among the polio deaths approximates the proportion of Jewish people in the wider population (Table 4.5). It also underestimates the proportion of Catholic individuals; using information on burial place, polio deaths interred in Catholic cemeteries (Holy Sepulchre in Wentworth County and Mount Hope in York County) and Anglican cemeteries were added to the analysis. This resulted in the identification of an additional 19 Catholic individuals and two more Protestant individuals, included in the proportions shown in Table 4.5.

¹² Mercier (2003, 2006) found low infant and child mortality among the Jews in Toronto in 1901, and similar low mortality patterns have been found in Jewish populations elsewhere in North America and around the world (Schmelz, 1971; Sawchuk, Herring, & Waks, 1985; Condran & Kramarow, 1991; Marks 1994; see Derosas, 2003 for a more comprehensive listing).

Population	Protestants %	Catholics %	Protestant + Catholic mixed family %	Jewish %	Greek Church %	Other %
1921 Census	80.5	11.9	*	4.7	0.8	2.1
Polio deaths	77.9	16.0	1.4	4.2	0.5	0.00

Table 4.5 Comparison of proportion of population by religious affiliation in the 1921 Census and polio deaths Wentworth and York Counties. Source: Canada (1924, Table 38).

* Data not available.

Comparing Protestants and Catholics only, there are proportionally more Catholics in the polio death sample than in the general population, as measured by the 1921 Census (Table 4.5). Protestants comprise 87% of the combined Protestant-Catholic population of Wentworth/York in the 1921 Census versus Catholics at 13%. Among the polio deaths, Protestants comprise 83% of the combined Protestant-Catholic number. This difference between the relative proportions of Protestants versus Catholics in the polio death sample compared to the 1921 Census population is not statistically significant ($\chi^2(1, 200) = 2.83, p = 0.093$). These numbers must be approached with caution, as religion was known for only 63.4% of the total sample of polio deaths, and it is possible that Protestants were more likely to be buried in nondenominational cemeteries and thus be less likely to be included in the religion analyses.

Main findings and discussion

The proportions of ethnic or religious groups among the polio sample were mostly comparable to their relative proportions in the general population. Among individuals

whose deaths were attributed to polio, there was a slightly greater proportion native-born individuals of British background than in the general population by rough comparison.

The hypothesis that there is an inverse relationship between polio mortality and IMR in particular groups (especially among Catholic and Jewish residents) was only partially supported. Jewish residents did not have a higher proportion of polio deaths despite their lower IMR. Additionally, there were (potentially) proportionally more polio deaths among Catholics, who averaged relatively high IMRs. This discrepancy is further evidence that the link between IMR and polio incidence is not as clear at the local level as it is at the national level.

The lack of excess polio deaths among the Wentworth and York's Jewish populations may be related to the fact that all but one of the parents (one mother of the 18 parents of nine victims) were immigrants from regions where polio was endemic (the majority from Russia or Poland, others from Austria, Galacia, and Rumania), if this is representative of the wider Jewish population of the area. Seven out of nine of the Jewish polio victims were themselves native-born, but only one had a mother born in Canada. Among Catholic polio victims in particular, 14 (46.7%) of those whose mothers' birthplaces were known had mothers born in Canada versus 16 (53.3%) born abroad, similar proportions to the overall polio sample, in which 54.8% had mothers born in North America. If these numbers are not simply artifacts of small sample sizes, it may indicate that among Jewish residents, the factors that contributed to their lower infant mortality rates (such as lower fertility and attitudes towards hygiene, as described in Sawchuk, Herring, & Waks, 1985) meant that rather than an increased risk of severe

polio, they faced similar risks of contracting and dying from polio as non-Jewish residents because the parents were largely immigrants from polio-endemic areas and the mothers carried antibodies against the poliovirus.

The earlier epidemic shift in polio in northern Europe, coupled with the predominance of people from that region (either directly or several generations past) in the earlier waves of immigration into eastern North America, especially southern Ontario, where polio epidemics also emerged early on, probably contributed to certain perceptions of the disease, such as the idea that blonde and blue-eyed children were the most susceptible:

REPORT CITY VICTIMS NOT ALL BLONDS
Stratford and London Predominance Is Held Coincidence

Although Stratford, Ont., reports that all children suffering from infantile paralysis there are blond, and London also reports a predominance of patients of that complexion, no such situation exists in Toronto, according to J. H. W. Bower, superintendent of the Hospital for Sick Children.

“I couldn’t speak numerically,” he said, “but I know some of the children suffering from the disease here are decidedly brunette.”

“[P]ure coincidence,” said a Toronto physician, commenting on the Stratford report. “There’s nothing in the pigmentation of a blonde person which would make him or her more susceptible to the disease.”

[*Toronto Daily Star*, 1 September 1937, p. 8]

This stereotype of susceptibility of blonde and blue-eyed children would continue into the 1950s, despite increasing evidence that all groups were at risk (Rogers, 2007).¹³ It is possible, especially in the earlier epidemic years, that victims who did not fit such stereotyping were less likely to be diagnosed with polio. Such stereotyping, in

¹³ There may be some element of genetic susceptibility to polio, as demonstrated by twin and family studies (Addair & Snyder, 1942; Aycok, 1942; Herdon & Jennings, 1951), but what that might mean or have meant at a wider population level remains unestablished.

combination with the general difficulties in diagnosing poliomyelitis, means that the statistical results of studies such as this must be interpreted with caution. Still, fatal cases of polio are considered to be the most reliably diagnosed, combined with an autopsy rate of approximately 25% in this sample.

Summary

The broad patterns observed in the demographic and illness patterns of polio mortality in Wentworth and York Counties between 1900 and 1937 are generally as expected based on previous studies of polio epidemics elsewhere. However, there are some unanticipated results. For the first three decades of the 20th century, the majority of polio deaths in Wentworth and York occurred among children under the age of five. However, in the 1930s there was a major shift, with only a small percentage of deaths in the under-five age group and the vast majority of deaths occurring in older children and adolescents. The increase in age at death over time was expected; the specific timing of that shift, however, is interesting and potentially informative. Possible factors behind this shift include a decreased birthrate, with smaller families, and increasingly reliable and comprehensive public sanitation.

The greater number of male deaths throughout the study period was also expected. However, the variation by age group was somewhat surprising, particularly the lack of excess female deaths during the reproductive years. The increase in excess male deaths in the final decade of the study period was also unanticipated, and happened around the same time as the large shift in the age at death pattern. The lack of excess adult female deaths is not unprecedented; there were also more male than female deaths among adults

over 20 years old in New York City during the 1916 epidemic. Still, the lack of excess adult female deaths does depart from the usual pattern. However, Weinstein et al.'s (1951) and Weinstein's (1957) investigations in Massachusetts demonstrated that polio fatalities can display a very different pattern from overall cases. Thus the polio death sample examined in this study should not be expected to follow patterns observed for polio morbidity.

Individuals whose deaths were attributed to polio were apparently more likely to be native-born than the general populations of Wentworth and York. However, this is difficult to establish definitively given the differing age structures of the native- and foreign-born populations. According to 1911 census data, while around 20% of the total population of Ontario was under the age of 10, that age group made up only 7.4% of the immigrant population in Canada (McInnis, 2000, p. 536). Thus there were fewer immigrant children potentially at risk for polio than would be suggested by the overall proportion of foreign-born in the population. As a result, these results remain inconclusive.

Polio victims were also overwhelmingly "white", British or European, in accordance with the structure of the general population from which they were drawn. The percentage of polio victims listed as Jewish or Hebrew, 4.7%, matches exactly their proportion in the general population in Wentworth and York according to the 1921 Census. There is little evidence for a local-level relationship between infant mortality and polio mortality rates in different ethnic or religious groups. There were slightly more polio deaths than expected among Catholics, but no increased deaths among Jewish

residents. However, other factors such as mother's birthplace may be complicating the picture and making such links difficult to determine at the micro level.

Acute polio illnesses ending in death usually lasted between several days to a week, with medical attention commencing within a day or two of onset. Many cases, however, did not fit this broad pattern, with onset being sudden and severe or else dragging on for weeks before death. The seasonal epidemic peak in August-September was expected, and closely overlaps with the pre-existing seasonal pattern of infant diarrhea which shares a fecal-oral route of transmission. Chronic polio deaths were more evenly distributed throughout the year, with a moderate peak increase in late winter-early spring (March and April). The exact cause of the timing of these chronic deaths is unclear; pneumonia or bronchopneumonia, for example, are no more often listed as a cause of death in the polio deaths in these months than in others.

It is likely that chronic cases were underestimated in this study, due to the assumption that all polio deaths were acute cases unless otherwise indicated. The lack of chronic deaths in September is one indication of this. On the other hand, at least some of the older adult chronic cases may have been misdiagnosed as polio. In any case, it is evident that polio contributed to many more premature deaths than those that occurred during the regular seasonal epidemics. Moreover, their impact would only have increased in the 1940s, 1950s, and beyond as individuals who survived polio during the study period passed away in subsequent years as a result of the lasting damage caused by the disease (see Chapter 2).

In summary, these results are generally in line with existing knowledge of polio's epidemiology and historical patterns; much of where they differ can be explained by the fact that this study is based on a sample of fatal cases only. However, the reasons behind the difference in morbidity versus mortality patterns remain unclear.

Chapter 5 Results and Discussion: Family Size, Birth Order, Socioeconomic Status, and Place of Residence Patterns

Introduction

The collective polio literature of the mid-20th century developed a model centred on age at infection. In this model, risk of severe polio increased with socioeconomic status (SES), because higher SES was associated with older age at infection. Rural residence was also linked to increased polio risk due to older age at infection. Crowding and larger family size were associated with earlier age at infection and thus reduced the risk of severe polio; these factors also varied with SES.

More recently, Nielsen and colleagues (2001) have proposed a model that pays particular attention to interactions between age, family size, and birth order. Nielsen et al.'s (2001; 2002a) Intensive Exposure (IE) model predicts that polio will be more severe (with an increased risk of complications and death) in firstborn children infected at older ages (i.e. over six years old) and later born children infected at younger ages (i.e. under five years old). It predicts secondary cases (other children and adults exposed within the home) will be more severe than the index cases, with five-to-six-year-olds being the most likely index cases (Nielsen et al., 2001).

In this chapter, I present and discuss the results of analyses of family size and birth order, socioeconomic status, and geographic distribution in the Wentworth/York sample of polio deaths. I examine how these patterns vary by age, nativity, and religion, and how they intersect with each other – how geographic distribution varies with socioeconomic status, for example. I consider these findings in light of both the

traditional polio literature and the IE model, and in the final section I address how these results answer four main questions: 1) Does this analysis of status scores support the description of polio in southern Ontario before WWII as a disease of the “middle class”? 2) Is the expected pattern of increasing age at death with increasing socioeconomic status present? 3) What is the relationship between socioeconomic status and family size and birth order? 4) Are the socioeconomic patterns in polio evident spatially in the cities of Hamilton and Toronto?

Family Size and Birth Order

Family/sibship size

I was able to determine a number of minimum siblings for 138/336 individuals who died from polio (41.1% of the total sample). By “minimum”, I mean that I had data to show that an individual had at least that many siblings, possibly more. The minimum number of siblings per individual polio death ranged from 0 to 11. The average of 3.3 ± 2.0 children per family in the polio death sub-sample corresponds to the average of 3.5 children per family found among polio patients during the 1937 epidemic in Ontario (Ontario Department of Health, 1938, p. 19).

Comparing the polio death sub-sample to the general population using data from the 1921 Census, we see that there were proportionally fewer only children in the polio death population (12.3 vs. 33.3, respectively) and more polio deaths of individuals who had at least one living sibling (87.7 vs. 66.7) (Table 5.1). This trend holds for each sibship size (Table 5.1).

Sample	Families reporting children	% with 1 child	% with 2 or more children	% with 3 or more children	% with 4 or more children	% with 5 or more children	% with 6 or more children
Ontario	492,527	33.3	66.7	40.9	24.1	13.6	7.4
Ontario – rural	197,394	29.6	70.4	45.9	28.6	17.1	10.0
Ontario-urban	295,133	35.8	64.2	37.6	21.0	11.2	5.8
Polio deaths	138	12.3	87.7	48.6	34.8	23.2	16.7

Table 5.1 Comparison of percentages of number of children in families reporting children in different samples. Source: Canada, Dominion Bureau of Statistics, 1925, table 22.

In 1921, Hamilton families reporting children had an average of 2.34 children and Toronto families reporting children had an average of 2.35 children (Canada, Dominion Bureau of Statistics, 1925). This compares to an overall average minimum number of children of 2.9 ± 1.5 , with a median of 2, in Hamilton and Toronto families with a polio death ($n = 72$). Therefore, average and median family/sibship size in polio deaths was at least as large as in the general population, and given that those figures are based on minimum numbers of siblings, it is possible that family/sibship size was in fact comparatively larger in the polio death sample. In fact, calculating family size only for those individuals for whom the exact number of siblings was known, both the urban ($n = 33$) and suburban ($n = 15$) samples had an average family size of 3.3 ± 1.6 , with a median of 3 in each. This fits with Nielsen et al.'s (2002a) argument that among polio cases, severity increases with family size (see Chapter 2).

As mentioned in Chapter 4, both the birth rate and average family size were declining during the study period. This trend is visible in the polio mortality sample

(Table 5.2). The difference in mean minimum family size between 1900-1919 and 1920-1937 is statistically significant (unpaired t-test, $t(136) = 2.71$, $p = 0.0077$).

Time period	Average min. number of children in family	Median min. number of children in family	<i>n</i>
1900-1919	3.8±2.2	4	57
1920-1937	2.9±1.8	2	81

Table 5.2 Average and median minimum family sizes of individuals who died of polio in Wentworth/York 1900-1919 versus 1920-1937.

Comparing the two largest religious groups in the sample (Table 5.3), Protestants and Catholics, it appears that the Catholics had a higher average and median minimum number of siblings. However, there is no statistically detectable difference between the two groups, likely because of the small sample of Catholic families (unpaired t-test, $t(96) = 0.96$, $p = 0.34$). A larger sample size would be needed to determine if larger family/sibship size compared to the general population may have led to a higher proportion of polio deaths among Catholics (see Table 4.5, Chapter 4). For now, statistically, family size is the same between the Protestants and Catholics in this sample.

Group	Average min. number of children in family	Median min. number of children in family	<i>n</i>
All	3.3±2.0	2	138
Protestants + Catholics	3.4±2.1	3	98
Protestants	3.4±2.1	3	90
Catholics	4.1±2.0	4	8

Table 5.3 Comparison of minimum number of children in childhood families of Protestant and Catholic polio deaths in Wentworth and York Counties, 1900-1937.

At first glance, family size appears to differ primarily between rural versus urban/suburban populations, and not between urban and suburban groups (Tables 5.4 and 5.5). In an analysis of variance, there were no statistical differences in minimum family

size between these groups in the polio sample (one-way ANOVA, $F(2, 107) = 2.75$, $p = 0.068$) (Table 5.4). The Mann-Whitney U test also found no difference between minimum family sizes in rural versus urban areas ($p = 0.17$). This is also the case if a different measure of the data for family size is used, which is to only include individuals for whom the exact number of siblings was known ($p = 0.15$). Again, combining the urban and suburban samples and comparing that to the rural sample, the Mann-Whitney U test found no significant differences in both minimum family size ($p = 0.20$) and exact family size ($p = 0.15$).

Residential location	Average min. family size	Median min. family size	<i>n</i>
Urban	2.8±1.5	2	73
Suburban	3.0±1.8	2	28
Rural	4.3±3.3	3.5	10

Table 5.4 Comparison of minimum number of children in childhood families of polio deaths by residential location in Wentworth and York Counties, 1900-1937.

Residential location	Average exact family size	Median exact family size	<i>n</i>
Urban	3.3±1.6	3	33
Suburban	3.3±1.6	3	15
Rural	5.3±3.7	4	7

Table 5.5 Comparison of exact known number of children in childhood families of polio deaths by residential location in Wentworth and York Counties, 1900-1937.

Extracting the children of farmers and fruiters ($n = 28$), regardless of their residential locations at death, we find a similarly large family size, with an average minimum family size of 4.3±2.9 and median minimum family size of 3.

Family size varied by SES in the wider population, and so might be expected to similarly vary by status score in this sample of polio deaths; I will examine this in the section on socioeconomic status by status score, below.

Nielsen et al. (2002b) hypothesize that the sex ratio in severe polio cases (e.g. deaths) is linked to family size, with a larger family size associated with a more equal ratio of males and females and smaller families size with excess male deaths. Analysis of acute polio deaths of individuals aged 0-19 supports this hypothesis. To avoid the confounding effect of changing family size over time, the 1910-1927 period (Stage 1, as discussed in Chapter 6) was examined, comparing families with 1 to 2 children with those with 3 or more children. Families with 1 to 2 children had a polio death sex ratio of 2.0 (6 males, 3 females), compared with a sex ratio of only 0.8 in the families with ≥ 3 children (10 males, 12 females). This difference is statistically significant when the 10:12 ratio is compared against an expected ratio of 2.0 (66% males, 34% females) ($\chi^2 (1, 22) = 4.138$, $p = 0.042$).

Birth order

Sufficient data to establish birth order was available for 114 of 336 individuals (33.9% of the overall sample). The average birth order was 2.3 ± 1.7 and the median was 2 (second child). 55 of the 114 (48.2%) were first-borns (including only children). There was no statistically significant difference in average birth order between males <20 years old (2.4 ± 1.9 , $n = 68$) and females <20 years old (2.2 ± 1.5 , $n = 46$) (unpaired t-test, $t (112) = 0.87$, $p = 0.38$), and both sexes had a median birth order of 2. Comparing sex ratios in those with a birth order of 1-2 versus 3 or later (Table 5.6), Nielsen et al. (2002b) predict the sex ratio will be lower (closer to an equal representation of males and females) at higher birth orders. However, a chi-square test found the association not to be statistically

significant ($\chi^2 (1, 82) = 0.25, p = 0.62$). Only those under 20 years old were examined as they were likely to be still living at home; this also allowed comparison with the study conducted by Nielsen et al. (2002b).

Birth order	Sex ratio	<i>Males n</i>	<i>Females n</i>	<i>Total n</i>
1-2	1.4	33	24	57
3+	1.1	13	12	25

Table 5.6 Sex ratios by birth order in Wentworth/York polio deaths under 20 years of age, 1900-1937.

According to the intensive-exposure model for polio suggested by Nielsen et al. (2002b), older individuals (above the mean age in the sample) should be more likely to be of lower birth order (e.g. firstborns) whereas younger individuals (below mean age) should be more likely to be of higher birth order (e.g. lastborns). This is because polio severity is related to intensity of exposure and dose of infection. Cases subsequent to the index cases within a family are more likely to have had more intensive exposure and larger dose of infection, and index cases are more likely to be 5- to 6-year-olds who bring the infection into the family (Nielsen et al., 2002b, p. 304).

Among the acute deaths in this sample, firstborns with younger siblings (excluding only children and those without confirmed younger siblings) appear to have slightly higher average and median ages at death than third-born or later individuals (Table 5.7). The small 0.3 difference in the means, however, is not statistically significant (unpaired t-test, $t (52) = 0.10, p = 0.92$).

Birth order	Average age at death (years)	Median age at death (years)	<i>n</i>
1	13.6±7.9	15	19
3 or later	13.3±11.7	11	35

Table 5.7 Average and median ages at death by birth order for individuals with at least one sibling. Acute polio deaths only.

Age group	Average birth order	Median birth order	<i>n</i>
0-9	3.0±1.6	2.5	30
10-19	2.6±1.9	2.0	21

Table 5.8 Average and median birth order by age group for individuals with at least one sibling. Acute polio deaths only.

Looking at birth order and age another way, I partitioned this sample into two age groups on either side of the mean age at death (9.5 years): 0-9 years and 10-19 years. The 0-9 age group had an average birth order of 3.0 and median birth order of 2.5, which is a slightly higher birth order than observed in the 10-19 age group which had an average birth order of 2.6 with a median of 2 (Table 5.8). This is again consistent with the intensive-exposure model. However, the difference in the means is not statistically significant (unpaired t-test, $t(49) = 0.88$, $p = 0.38$). It is interesting to note that analysis of polio cases during the 1937 Ontario epidemic found no consistent trend in birth order but a tendency for polio patients in larger families to be later-born (higher birth order) (Ontario Department of Health, 1938, p. 19).

Main Findings and Discussion

Sibship size among polio deaths was at least as large as in the general population, and possibly larger, which lends support to the intensive-exposure model. Nearly half of individuals in the sample were firstborns or only children, and the average and median birth order was approximately 2 (second-born) for both sexes. Examinations of the

relationship between birth order and age were consistent with the intensive-exposure model, with older age associated with higher birth order and younger age with lower birth order in individuals with at least one sibling. However, the differences here were fairly slight (e.g. median birth order of 2.5 for ages 0-9 vs. 2.0 for ages 10-19).

Examples of these older firstborn victims include Jack Connell, eldest of four children who died at age 22 in West Flamboro in 1928 and Kenneth Argent, also the eldest of four, who died at age 16 in Hamilton in 1937. Betty Henderson of Hamilton, on the other hand, was the youngest of four when she died in 1928 at the age of six. These three older children were Betty's half-siblings from her father's first marriage (for extended profiles on these individuals, see Appendix E.26, E.27, and E.28).

Comparing these results to the existing literature, Melnick and Ledinko (1951) also looked at sibship size and found that poliovirus antibody levels were lower in children with no or fewer siblings. However, Nielsen et al.'s (2001; 2002a) studies of polio cases in Copenhagen, Denmark between 1919 and 1953 found that polio severity was associated with larger family size. Evidence from this study supporting the predictions of Nielsen et al. (2002a; 2002b) regarding family size and birth order, examined in the preceeding section, will be discussed further in Chapter 6, in light of the intensive-exposure model.

Family size also varies by socioeconomic status and geographic residence, as discussed in the following sections.

Socioeconomic Status by Status Scores

Status scores

As detailed in Chapter 3, the SES of each individual whose death was ascribed to polio was based on father's occupation during the individual's early childhood. In the absence of this information, SES was derived from the occupation of the adult individual him or herself, or in the case of married women, on the occupation of her husband. Occupations were scored according to a five-point scale based on Hauser (1982) (Figure 5.9). To allow comparisons of SES in the polio sample to the general population, the same scale was used to score occupations listed in the 1921 Census.

Status score	Category	Example occupations
1	Professional	Dentist, Lawyer
2	Entrepreneurial/Clerical	Clerk, Farmer, Sales manager
3	Skilled labour	Carpenter, Stonemason
4	Semi-skilled labour	Porkpacker, Teamster
5	Unskilled labour	Farm labourer

Table 5.9 Status scores with associated occupational categories and occupations.

There were 203 polio deaths for whom sufficient information was available for assignment of a status score (60.4% of the overall sample of 336).¹⁴ From largest to smallest, 39.4% ($n = 80$) of polio deaths with status scores fell under status score 3, while 33.0% ($n = 67$) were at status score 2, followed by 14.3% ($n = 29$) at status score 4, 8.4% ($n = 17$) at status score 5, and 4.9% ($n = 10$) at status score 1. The same average status score for all polio deaths, 2.9 ± 1.0 , was found for both Wentworth ($n = 51$) and York ($n = 152$) Counties.

¹⁴ New records continue to be released which provide additional data for this sample, such as birth records for Ontario. Suffice it to say that these results are subject to shift somewhat as additional data becomes available.

Of these 201 individuals with status scores, 21 are classified as chronic polio cases, leaving 182 acute polio deaths.

Comparing status score proportions in the polio sample to 1921 Census data for Hamilton, Toronto, and Ontario as a whole, as expected status score 5 (families of unskilled workers) is underrepresented in polio deaths compared to its proportion in the population (Table 5.10). These trends hold for males only, and for families and children. Status score 3 (skilled labour) dominates with 52.2% of urban polio deaths; however, this is less striking when compared to the distribution of the urban child population (Table 5.10), of which status score 3 made up the largest proportion at 40.7%. This means that most polio deaths occurred among middle class and upper working class families (status scores 2 and 3), in the children (young or adult) of farmers, clerks, carpenters, and mechanics. There were comparatively few deaths among the children of farm labourers, other unskilled labourers, and travellers.¹⁵

¹⁵ The occupation category of “traveller” referred to travelling salesmen. The Wentworth/York polio sample included three individuals with fathers in this category, two with father’s occupation listed as “Traveller” and one as “Com. Traveller”.

Status score	1921 Ontario male pop. >10 yrs old % <i>n</i> =761,411/ 1,173,349 (82.5%)	Wentworth/York polio sample (Ontario residents, by male head) % <i>n</i> =195	1921 Toronto and Hamilton families (by male head) % <i>n</i> =83,592/ 95549 (87.5%)	1921 Toronto and Hamilton child pop. (by male head) % <i>n</i> =143,296/ 164,772 (87.0%)	Polio sample (Toronto and Hamilton residents, by male head) % <i>n</i> =139
1	1.6	3.1	1.5	1.5	6.5
2	38.8	35.4	33.2	28.1	19.6
3	27.1	44.8	38.1	40.7	52.2
4	9.8	10.9	10.7	11.1	13.8
5	22.9	7.3	18.7	18.7	8.0

Table 5.10 Proportions of populations in each status score according to the 1921 Census compared to proportions of polio deaths in Ontario and Toronto and Hamilton combined, 1900-1937. Source: Census of Canada, 1921, Table 2, Table 41. The combined Toronto and Hamilton polio sample excludes children of farmers who moved to the cities.

To illustrate, the following are examples of a polio death at each status score. At status score 1, there is Adam George Ballantyne, the son of a Toronto barrister. At the time of his birth, the family lived in the Annex neighbourhood; at his death in 1928, they were living in the wealthy Forest Hill neighbourhood. Adam was 17 years old and a student when he contracted polio. His father had died just four months previously (see Appendix E.22 for an extended profile). At status score 2, there are two main categories of occupations, farmers and others. Among the children of farmers is Robert Roy Loveless. He was one of nine children in the family, living on a farm in Scarboro. Robert died in 1923 at age 26, though it seems he contracted polio at a much younger age (see Appendix E.7 for an extended profile). Another status score 2 death is Muriel King. Her father, an immigrant from England, worked as an advertiser in a newspaper office. The family lived in North Toronto (see Appendix E.23 for an extended profile).

At status score 3 is Jessie Graham Duncan. Jessie was born in Toronto in 1916 to parents who were immigrants from England. Jessie's father, Private John Duncan, had been a plumber before the war; he died in France the year after Jessie was born. Jessie died in September 1922 during a polio epidemic, at the age of six. At the time of her death she was living with her mother in the Danforth neighbourhood of Toronto (see Appendix E.16 for an extended profile). At status score 4 is Lloyd Alexander Burke. Lloyd's father, John Burke, worked as a stableman and was a widower before he married Lloyd's mother, Winnifred, who worked as a filler. Lloyd died at home in Toronto in September 1922 at one year of age (see Appendix E.15 for an extended profile). Finally, at status score 5 is Bernard Lawrence Roach. His father was a laborer and they lived in

Hamilton's industrial working-class North End neighbourhood. Bernard died in 1934 at the age of two (see Appendix E.24 for an extended profile).

As outlined in Chapter 2, most working class families (status scores 3-5) in southern Ontario at this time fell either below the poverty line or close to it, and relied on the incomes of their older children and from taking on lodgers to make ends meet (Synge, 1979; Piva, 1979; Baskerville & Sager, 1998, as cited in Sager, 2007; Sager, 2007). Throughout the study period, lodgers as a percentage of Toronto households ranged between around 20-37% (Harris, 1996, p. 118-119). In the year 1931, 23.2% of households in Toronto and 20.7% of those in Hamilton had one or more lodgers (Harris, 1996, p. 117, table 5.1). Working class families were especially hard-hit by the Depression and the lodging incidence increased during that time, though it was a strategy available primarily to urban dwellers (Harris, 1996).

Status scores and age

Looking at average and median ages by status score, we can see that age at death increased with socioeconomic status (Table 5.11).

Status score	Median age (years)	Average age (years)	<i>n</i>
1	17.5	15.3 ± 6.6	10
2	12.0	15.1 ± 12.9	59
3	7.0	8.9 ± 7.4	73
4	4.0	7.5 ± 8.7	26
5	2.5	8.2 ± 9.6	14

Table 5.11 Average and median ages at death in acute polio deaths with status scores (*n* = 180) in Wentworth and York Counties 1900-1937.

Small sample sizes mean that most of these differences are not statistically significant. Z scores were used to correct for lack of normal distribution. Levene's test indicated unequal variances ($F = 4.31, p = 0.002$), so a Welch's analysis of variance was performed which found significant variation in mean age at death ($p = 0.003$). Post hoc comparisons with Games-Howell found significantly higher average age at death in status score 2 (15.1 ± 12.9) compared to status score 3 (8.9 ± 7.4) ($p = 0.012$) and status score 4 (7.5 ± 8.7) ($p = 0.017$), the status scores with the largest sample sizes. The status score 2 group, made up of entrepreneurs, clerical workers, farmers, and their children, was significantly older than status score 3, composed of skilled blue-collar workers and their children, and status score 4, composed of semi-skilled blue-collar workers and their children.

Comparing the pre-Depression period to the 1930s, we see age at death increased substantially in the later period in the lower status scores (Table 5.12). The increase in mean age at death between the two periods was statistically significant in status score 3 ($t(71) = 3.05, p = 0.0032$) and status score 4 ($t(24) = 2.26, p = 0.034$), but not in status score 5 ($t(12) = 1.50, p = 0.16$). The lack of a statistically significant increase in age in status score 5 may be due to small sample size, or possibly there actually was little age increase in status score 5 because these families (unskilled workers) still lagged behind their higher status counterparts in terms of living conditions and access to similar standards of hygiene and sanitation. The apparent decrease in mean age at death in the top two status scores (see Table 5.12) was statistically non-significant in both status score 2 (t

(57) = 1.00, $p = 0.32$) and status score 1 ($t(8) = 0.72$, $p = 0.49$). More studies with larger sample sizes would be needed to explore this further.

Status score	Median age at death 1900-1929	Median age at death 1930-1937	Average age at death 1900-1929	Average age at death 1930-1937	1900-1929 n	1930-1937 n
1	18.0	16.0	17.7 \pm 0.6	14.3 \pm 7.8	3	7
2	12.5	11.0	16.4 \pm 14.6	12.9 \pm 9.1	37	22
3	4.0	10.0	6.9 \pm 7.3	12.0 \pm 6.7	45	28
4	2.0	11.0	4.9 \pm 7.3	12.3 \pm 9.2	17	9
5	2.0	16.0	5.9 \pm 9.4	14.0 \pm 8.6	10	4

Table 5.12 Average and median ages at death by status score for the pre-Depression period (1900-1929) and Depression (1930-1937) for the acute polio death sample from Wentworth and York Counties.

Within in the 1900-1929 period, Levene's test indicated unequal variances ($F = 5.78$, $p = 0.000$), so a Welch's analysis of variance was performed which found significant variation in mean age at death among status scores (Table 5.12), $F(4, 35.8) = 6.31$, $p = 0.000$. Post-hoc comparisons using Games-Howell found significant differences between status score 1 (17.7 \pm 0.6) versus status scores 3 (6.9 \pm 7.3) ($p = 0.000$), 4 (4.9 \pm 7.3) ($p = 0.000$), and 5 (5.9 \pm 9.4) ($p = 0.02$), and between status score 2 (16.4 \pm 14.6) versus status scores 3 (6.9 \pm 7.3) ($p = 0.006$) and 4 (4.9 \pm 7.3) ($p = 0.003$). In other words, the statistically significant differences in mean age at death for the 1900-1929 period were found between the upper two status scores and the lower status scores. Within the 1930-1937 period, an analysis of variance found no significant variation in mean age at death among status scores (Table 5.12), $F(4, 65) = 0.15$, $p = 0.96$.

The increase in age at death over time in children assigned to the lower status scores does not appear to have resulted in any major change in the relative distribution of deaths by status score (Table 5.13). One might have expected the increase in age at death

in status scores 3 and 4 would have led to an increase in the percentage of polio deaths in these status scores.

Status score	1900-1929		1930-37		1900-1937	
	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%
1	3	2.7	7	10.0	10	5.5
2	37	33.0	22	31.4	59	32.4
3	45	40.2	28	40.0	73	40.1
4	17	15.2	9	12.9	26	14.3
5	10	8.9	4	5.7	14	7.7
Total	<i>112</i>	<i>100.0</i>	<i>70</i>	<i>100.0</i>	<i>182</i>	<i>100.0</i>

Table 5.13 Proportions of acute polio deaths by status score over time, Wentworth/York 1900-1937.

Status scores and neighbourhoods

When the socioeconomic characteristics of various neighbourhoods in Hamilton and Toronto (see Appendices F and G) are compared with the status scores of the residents who died of polio, the average status scores were generally in line with what was predicted; that is, the average status score of a resident who died of polio matched the predicted average status score of residents based on the general characterization of the neighbourhood (see Table 5.14).

SES of neighbourhood	Predicted status score of neighbourhood	Average status score of polio deaths	Average status score of polio deaths higher, the same, or lower than predicted neighbourhood score	Status score range of polio deaths	<i>n</i>
Wealthy	1-2	2.3 ± 1.2	Lower	1-4	4
Middle class	2-3	2.8 ± 0.8	Same	2-4	6
Upper working class/lower middle class	3	3.0 ± 0.8	Same	2-5	22
Working class	3-4	3.1 ± 1.0	Same	1-5	22
Mixed	2-3 (average)	2.2 ± 0.8	Same	1-3	9

Table 5.14 SES characterization of Hamilton and Toronto neighbourhoods with predicted status scores compared to average status scores of residents who died of polio.

Where the average status score is lower than expected, small sample size may be the cause. However, only the wealthiest neighbourhoods show a striking mismatch between the predicted and observed status scores for individuals who died from polio. This includes a death of a status score 3 individual in Toronto's Rosedale neighbourhood (Florence Cowlin, the 21-year-old stonemason's daughter in 1921), albeit living on the edge of the neighbourhood, and a status score 4 individual in Hamilton's Durand neighbourhood (Mary Packer, the infant daughter of a hostler in 1916) (for more details on these two individuals, see Appendices E.5 and E.6).¹⁶

Also, though the average status scores might match the predicted averages, the total range of status scores of individuals whose deaths were ascribed to polio was often greater than the overall SES characterization of the neighbourhood. For example, the full range of status scores from 1 to 5 was found in neighbourhoods described as "working

¹⁶ The term "hostler" refers either to a groom in a livery stable, taking care of the horses, or to a similar job in a railround roundhouse, caring for the locomotives. In the case of Mary Packer, her father was a hostler in a livery stable.

class” (Table 5.14). Similarly, the individuals mentioned above died in neighbourhoods described as “wealthy”, yet their status scores were well below that level. Perhaps living in neighbourhoods with good sanitation contributed to the delay in exposure to the poliovirus (though Florence Cowlin’s childhood living conditions are unknown). Perhaps they are simply anomalies. In any case, the lack of spatial patterning (see Appendix G) and range of status scores in each type of neighbourhood may be as much a reflection of the heterogeneity of these cities’ social geographies in terms of socioeconomic class by occupation as of patterns of polio mortality risk.

Status scores and nativity

It was possible that nativity analysis was confounded by differences in socioeconomic status between the native- and foreign-born. However, the status scores were virtually identical for native- and foreign-born individuals in the polio sample. Native-born individuals ($n = 181$ with status scores) had an average status score of 2.9 and foreign-born individuals ($n = 15$ with status scores) of 3, with a median of 3 for both.

Status scores and religion

Again, it was possible that religion and socioeconomic status might be conflated.

However, there is, at least, no difference in status scores between Protestants and Catholics; Protestants ($n = 141$ with status scores) have an average status score of 2.9 ± 1.0 and Catholics ($n = 16$ with status scores) of 2.9 ± 0.8 with a median of 3 for both.

There is only one Jewish individual with a status score, and it is a 3 as well.

Status scores and family size

Family size varied by socioeconomic status, which is likely one reason for the variation in polio by SES. In Ontario in 1901, owners and white collar workers had low fertility compared to manual workers; farmers also had high fertility (Gossage & Gauvreau, 2007). However, this is not necessarily reflected in the polio sample. Even when farmers are removed from status score 2, the family size at status score 2 remains high (Table 5.15). Levene's test indicated unequal variances ($F = 4.33$, $p = 0.003$) so a Welch's analysis of variance was performed which found the variation to be statistically non-significant ($p = 0.064$). An analysis of variance with farmers excluded (see Table 5.15) found significant variation in minimum family size ($F(4, 75) = 3.21$, $p = 0.017$) but post-hoc comparisons with Tukey and Bonferroni tests did not identify any significant differences between groups.

Status score	Average min. family size	Median min. family size	<i>n</i> with known family size	% firstborn	<i>n</i> firstborn	<i>n</i> with known birth order
1	4.5±2.7	4	6	50.0	3	6
2 (incl. farmers)	3.0±2.5	3	35	30.3	10	33
2 (w/o farmers)	3.8±2.0	3.5	12	58.3	7	12
3	2.6±1.4	2	44	52.6	20	38
4	3.2±1.5	2.5	14	22.2	2	9
5	4.0±1.4	4.5	4	60.0	3	5

Table 5.15 Family size and proportion firstborn by status scores for Wentworth/York polio deaths 1900-1937.

This lack of difference in family size among status scores in the polio sample, when there was such a difference in the larger population, might indicate that larger

family size was a particular risk at the upper status scores. Normally large families should mean greater likelihood of immunity to the poliovirus in early childhood (Walton & Melnick, 1955). Nielsen et al. (2002a), however, indicate that better sanitary conditions and practices may increase the importance of droplet transmission, in which the Intensive Exposure model comes into play, with family size and crowding emerging as risk factors for severe polio.

However, it is difficult to make sense of these results without examining whether the siblings were younger or older than the individual who died of polio; Walton & Melnick (1955) found that a lack of older siblings resulted in delay in age of infection. Thus family size must be examined in combination with birth order. It seems larger family size presented greater risks to older siblings who had escaped previous exposure, and who were then exposed to the poliovirus by younger siblings in the home, fitting Nielsen et al.'s (2001; 2002a) IE model.¹⁷ There does not seem to be any pattern in the proportion of polio deaths among firstborns that can be attributed to status score (Table 5.15).

Main Findings and Discussion

In this section I address how these results answer the four questions mentioned in the introduction to this chapter: 1) Does this analysis of status scores support the description

¹⁷ Nielsen et al. (2002a) found that the risk of polio was higher among children with siblings than among only children, and that the risk of paralytic polio increased among older children (7-20 years) in concert with an increasing number of siblings but not (actually the reverse) among youngest children (0-6 years) with an increasing number of siblings (p. 183).

of polio in southern Ontario before WWII as a disease of the “middle class”?¹⁸ 2) Is the expected pattern of increasing age at death with increasing socioeconomic status present? 3) What is the relationship between socioeconomic status and family size and birth order? 4) Do the socioeconomic patterns in polio manifest spatially in the cities of Hamilton and Toronto?

Can pre-WWII polio be described as a “middle class” disease, at least in terms of mortality? The answer is both yes and no. Or rather, the answer depends on how one looks at the data. The ‘middle’ status scores (2 and 3), when combined with the potential “middle class” status of farmers, white collar workers, and skilled manual labourers, together account for 72.1% of the polio deaths in the total sample. However, it is debatable whether status score 3 (skilled manual labourers) should be considered “middle class” at this time. Piva (1979) states that prior to WWII people in this group are more properly considered as “working class”, living close to the poverty line. Synge (1979) also observes that working class families in Hamilton often considered themselves “middle class”, despite actually falling into the “working class” category, according to objective criteria such as requiring two incomes and having children leave school early to work. Furthermore, blue-collar workers in southern Ontario were more severely impacted by the Depression than white-collar workers (Hiebert, 1995). Manufacturing jobs declined between the 1921 and 1931 censuses (Hiebert, 1995). If status score 3 individuals are better classified as part of the “working class”, the combined proportion of

¹⁸ Numerous scholars have characterized epidemic polio in this way, including Rutt (1996), Nielsen, Aaby, Wohlfahrt, Mølbak, and Melbye (2002a), and Shell (2005). Others have described epidemic polio as evenly distributed among socioeconomic classes before the availability of a vaccine, such as Colgrove (2006, p. 132), who studied polio in Chicago, USA.

status scores 3 to 5 make up 62.1% of the polio deaths. This gives a picture of polio as more of a ‘working class plague’ than a middle class one, or perhaps rather as more of a democratic disease that affected all segments of society. Whether or not status score 3 should be considered as part of the upper or lower economic group, or as middle or working class, the lowest status scores (4 and 5) clearly formed a much smaller proportion of the polio deaths than they did of the wider population of Wentworth and York Counties.

In contrast, the *Report on Poliomyelitis in Ontario, 1937* found that polio morbidity occurred “with disproportionate frequency in somewhat overcrowded homes of relatively low economic status” (p. 56). Investigators examined the number of persons per household and number of rooms in the households of polio cases relative to the 1931 Census data for Ontario (Ontario Department of Health, 1938, p. 18). They found that households with polio cases in 1937 had more persons per household than the general population and fewer rooms per person (Table 5.16).

	Poliomyelitis Ontario, 1937	Population Ontario, 1931
Average number of persons per household	5.8	4.1
Average number of rooms per house	6.5	6.2
Number of rooms per person	1.1	1.5

Table 5.16 Comparison of number of persons per household and rooms per house in 1937 Ontario polio cases and 1931 Ontario Census population. Source: Ontario Department of Health, 1938, p. 18.

This information on morbidity, taken together with the mortality data presented in this study, suggests that prior to WWII, polio in southern Ontario was not concentrated among middle class families. Since Nielsen et al. have demonstrated that crowding

increases the risk for intensive exposure to the poliovirus, with higher doses of the virus linked to increased polio severity, this indicates that higher SES (associated with increased age at infection) was not the only risk factor for polio death, but that lower SES (associated with crowding) likely also conferred another set of risks. Cockburn (2005) describes this idea of mixed high and low risk, noting that “in poor and unhygienic households you were more likely to be protected against the virus but you were also more likely to meet it” (p. 260).

Furthermore, at least by the 1930s, the middle and working classes were experiencing similar living conditions. First, as a result of the housing boom of the 1920s, there was an oversupply of good quality houses with water and sewage connections, and much of the working class (those who still had employment) were able to benefit from these cheap rental opportunities, such as occurred in the eastern edge of Hamilton (Ward 8) (Doucet & Weaver, 1991). Thus many of them were able to enjoy a more ‘middle class’ standard of living during the Depression, in terms of housing and sanitation. On the other hand, Bordessa & Cameron (1980) note that water quality in Toronto declined between 1912 and 1945, as population growth outstripped the capacity of the sewage works. The authors of a 1939 report to the City of Toronto on the state of its sewage treatment system remarked that “the present contamination of the water supply and the bathing beach waters is serious, and Toronto has been fortunate not to have suffered severely from outbreaks of water-borne diseases” (Berry et al., 1939, pp. 1-2, cited in

Bordessa & Cameron, 1980, p. 131).¹⁹ Filtering and chlorination reduced the risk of waterborne disease, but Bordessa & Cameron (1980, p. 132) also credit medical advances with preventing serious consequences from contaminated drinking water. Such contamination (with poliovirus, for example) would have affected everyone receiving drinking water from the municipal supply. Thus, as the working class gained greater access to amenities such as running water, they would have unwittingly increased their exposure to contamination from such sources (though they may have been at risk before as well, from wells contaminated by nearby privies). These changes do not address the consistency in the proportion of polio deaths at each status score over time, but they may help to explain the upward age shift in the lower status scores over time to match that of the upper status scores (discussed further below).

Rural residence had its own polio severity risks, and farmers are classified as status score 2. Thus the inclusion of farmers and their children (rural residents, or those who grew up in a rural setting) can muddy the picture of SES patterns in the cities. If farmers and their children are excluded from status score analysis in Toronto and Hamilton, status score 3 becomes even more prominent, making up more than half (52.2%) of urban polio deaths compared to only 38.1% of families and 40.7% of children in the combined Hamilton/Toronto 1921 Census population (Canada, Dominion Bureau of Statistics, 1925). Thus in urban southern Ontario before WWII, it seems that skilled blue-collar workers and their families were particularly vulnerable to death from polio.

¹⁹ Keeping in mind that the poliovirus is carried in sewage and could be considered a waterborne disease, and there was a serious polio outbreak in Toronto in 1937, just two years earlier.

Considering information gained from polio studies in which antibody levels could be measured directly adds another dimension to the understanding of these results. In their work on the 1948 North Carolina polio epidemic Melnick and Ledinko (1953) found a difference in polio antibody levels between upper and lower economic groups, but no differences between economic groups for influenza and mumps viruses which they note are spread by respiratory pathways.²⁰ They determined that during the epidemic, polio antibodies (especially type 1) increased more in lower economic groups than in the upper economic groups. They compared poliovirus antibodies to Cocksackie virus antibodies, and found that in contrast, Cocksackie virus antibodies increased to the same extent in both economic groups. Therefore the poliovirus behaved quite differently from many other common viral infections in that lower economic groups were exposed and infected to a greater extent; however, to understand how this led to their smaller proportion of polio deaths, age must be considered.

This leads to the second question of whether we see the expected pattern of increasing age at death with increasing socioeconomic status as measured by status scores. The answer to this, as demonstrated in the results above, is a partial yes. Specifically, the age jump is greatest between status scores 3 and 2 prior to the 1930s. However, isolating the 1930-1937 period, this pattern of increasing age correlated with increasing status score disappeared. Instead, every status score had a comparatively high median and average age at death (medians between 10-16 years and averages 12.0-15.2

²⁰ Melnick and Ledinko (1951) had five SES levels, lumping levels 1-3 together as “upper” and 4-5 together as “lower”. The higher manual jobs and lower white collar jobs were at level 3; this study took place post-WWII, when grouping the skilled manual jobs with the “upper” economic group was perhaps more appropriate (see Piva 1979).

years). This is explained solely by the increase in the average age at death among polio deaths assigned to status scores 3-5; the already higher median and average ages at death in status scores 1 and 2 did not increase, and actually declined slightly.

The timing of this epidemiological shift (around the end of the 1920s and early 1930s) is part of a larger pattern discussed in Chapter 4. There are two very different age-sex patterns in the distribution of polio deaths before and after this shift. Before the 1930s, the majority of polio deaths occurred under five years of age and the sex ratio was equal. After the late 1920s, the majority of polio deaths occurred over five years of age and there was an excess of male deaths. When the disappearance of differences in ages at death between status scores is added to the picture, one obvious interpretation for the epidemiological shift is that improvements to public sanitation and hygiene had sufficient stability and reach to alter polio infection patterns across all social classes.²¹ As Velasquez-Manoff (2012) has argued, “roughly around mid-century, the lower classes in developed nations passed through the epidemiological transition that the upper classes had initiated in the previous century” (p. 295). Sanitary improvements had an especially notable impact on children born after WWI, as indicated by the decline in IMR at this time. Yet, this increase in the ages at which people assigned to the lower status scores died from polio was not combined with any increase in the proportions of the polio deaths found in these categories. In fact, the relative proportion of polio deaths at the lower status scores declined slightly; status score 4 individuals made up 15.2% of the deaths in

²¹ As Mechanic (2002) observes, some interventions to the environment benefit a range of socioeconomic classes relatively equally, as opposed to behavioural interventions. Kunitz (2007) notes, however, that there may still be health and mortality disparities between neighbourhoods of different socioeconomic classes.

the 1900-1929 period and status score 5 individuals 8.9%; by the 1930-1937 period these proportions had declined to 12.9% and 5.7%, respectively.

The polio literature on age and socioeconomic status for the most part shows higher socioeconomic status correlated with older age at polio illness, with some exceptions. Fales and Taback (1952) studied the 1950 Baltimore polio epidemic and showed that the overall attack rates in the lower and upper SES groups were similar; however, the lower SES group was infected instead at younger ages and the upper at older ages. Fales and Taback (1952) described this as “evidence of an infectious process which eventually reaches an entire population group” (p. 50). Among paralytic cases, bulbar cases were much more likely to occur over age 5 compared to the 0-4 age group. Collins (1946) found basically the same thing in the results of a 1935-1936 family survey; he found that the per capita incidence of polio was higher in the lower income groups, but that the age distribution differed; the polio rates for children under 5 years old decreased as income increased.

Similar to Fales and Taback, Collins (1946) said that the high rates of polio at the youngest ages is “presumably due to more contact in the crowded areas of the city” (p. 347) – in other words, population density (and population density and crowding would tend to correlate with SES). Collins (1946) looked at various income levels, including those on relief and found that case rates varied by age and income level; rates were highest in the 0-4 age group in the lowest income levels, in the 5-9 age group at a moderate income level, and in the 10-14 age group at the higher income levels. However, the peak overall attack rates were not much different across age groups and income

levels. The association of increased age with increased risk of severe polio would explain why upper SES groups, first infected at a higher age than lower SES groups, would be more affected by the more severe polio (and death).

Nielsen et al. (2002a), however, found very different results in their examination of polio patterns in Copenhagen, Denmark from 1940-1953. They found that in children hospitalized with polio, those in the higher social classes did not have a higher mean age at illness onset than those in the lower classes (Nielsen et al., 2002a, p. 184). They even found that when looking only at children with paralysis, those from the higher social classes were actually slightly younger than those from the lower classes (Nielsen et al., 2002a, p. 184). When they adjusted for age, gender, and time period, Nielsen et al. (2002a) found no difference between the higher and lower social classes in terms of risk of death from paralytic polio.

What my results indicate is that the socioeconomic discrepancy in age *at death* for polio in southern Ontario had apparently disappeared by the 1930s. This contrasts with the studies that show that polio cases still displayed a pattern of increasing age with increasing socioeconomic status in the 1930s and 1940s. This discrepancy does not appear to have been a matter of a difference between patterns in Ontario versus elsewhere in North America; the authors of the *Report on Poliomyelitis in Ontario, 1937* found that increased economic status was still associated with increase in age in that major epidemic (Ontario Department of Health, 1938).

Socioeconomic status also showed an interesting relationship to family size. In the wider population, family size generally varied with socioeconomic status, decreasing as

SES increased, with the exception of farm families (Gossage & Gauvreau, 2007).

However, this was not the case in the polio death sample. Small sample sizes at some status scores make analysis preliminary, but it seems clear that the polio sample is not simply a reflection of the general population – certain types of families, and certain people within them, were at higher risk of dying from polio than others. In this study, polio deaths were more prevalent among firstborn children and adolescents in larger families, which is especially evident in the upper status scores. These include William Cork, the firstborn of five children of a Toronto silk merchant, who died of polio in 1926 at age 18 while still a student (see Appendix E.9 for an extended profile), and Lillian Treble, firstborn of three or more children of a Toronto physician, who died of polio in 1930 at the age of 22 (see Appendix E.1 for an extended profile). Yet there are many exceptions to the pattern, such as Adam Ballantyne, the youngest of three children of a Toronto barrister, who died in 1928 at the age of 17 (see Appendix E.22 for an extended profile).

Finally, there is no clear-cut answer to the question of whether the socioeconomic status patterns in polio mortality are also evident at the spatial level. There is little spatial patterning of polio deaths, apart from the observation that they decidedly *do not* cluster in the poorest, most crowded areas (see Appendices G.1 and G.2). This is likely due to the overall rarity of polio deaths as well as the mixed socioeconomic makeup of many neighbourhoods. Hiebert (1995) notes that in 1931, Toronto's social geography was complex at the micro scale, and that even at the extremes – the wealthiest and poorest areas of the city – there was socioeconomic mixing. In 1931, 11.9% of households in

wealthy Rosedale were headed by a blue-collar worker (5.9% skilled and 5.0% unskilled) (Hiebert, 1995). When various urban neighbourhoods with known socioeconomic characters are examined, we find that polio deaths in those neighbourhoods had a wider range of status scores than the neighbourhood's general characterization. For example, collectively there were polio deaths in families assigned to status scores 1-4 in the "wealthy" neighbourhoods and among all families assigned to status scores (1-5) in the "working class" neighbourhoods.

Geographic Distribution

Urban, suburban, and rural distribution

Population size and density have long been recognized to play a role in polio's epidemiology. Relatively isolated areas with low population density were especially vulnerable to epidemics, as exposure to the poliovirus was infrequent, resulting in an accumulation of susceptibles. Paul (1971) notes that this pattern was visible in the world's first major epidemics in Scandinavia and Vermont. However, the main difference was often in age of polio onset rather than incidence; rural cases tended to appear in older individuals compared to urban cases (Hoyne & Cotsirilos, 1947; Olin, 1952; Smallman-Raynor et al, 2006).

To examine the effects of population size and density in this study, I partitioned the sample of polio deaths into three categories: urban, suburban, and rural. A residence was classified as urban if it could be assigned a city ward number in Hamilton or Toronto, suburban if it was outside the city proper (e.g. in Dundas near Hamilton or Todmorden

near Toronto) but had a specific street address, and rural if it was the name of a farm or a concession and lot number.

In this sample, 284 out of the 303 acute polio deaths could be classified into one of the three categories: rural ($n = 15$), suburban ($n = 62$), urban ($n = 175$), or non-urban (outside a city but unable to determine whether rural or suburban; $n = 6$). The majority of deaths occurred in an urban (city) setting (67.8%), with rural deaths forming the smallest percentage (5.8%). Comparison to overall population distribution provides an indication of differences in risk of death from polio (Table 5.17). However, these results must be viewed with caution because there is no information on the number of individuals who were born in and spent at least part of their childhood in rural areas but later moved to a suburban or urban area, where they died.

Category	Polio deaths 1900-1937 %	1921 population (Wentworth/York) %	1921 population <10 years old (Wentworth/York) %
Rural	5.8	17.3	21.1
Suburban (Census: towns and villages)	24.0	3.3	*
Non-urban (undetermined)	2.4	*	*
Urban (cities)	67.8	79.4	*

Table 5.17 Comparison of proportions of rural, suburban, and urban residents in Wentworth and York Counties in acute polio deaths and 1921 Census. Source: 1921 Census of Canada, Tables 38, 90.

*Data not available

Age and urban, suburban, and rural distribution

According to the polio literature discussed above, age at death should be higher in rural areas than in suburban areas, which would in turn be higher than in urban areas. Table

5.18 shows the average and median ages at death for each type of area. Levene's test indicated unequal variances ($F = 8.68$, $p = 0.000$), so a Welch's analysis of variance was performed, revealing statistically significant variation in age at death by location ($p = 0.01$). However, post hoc comparison with Games-Howell did not find statistically significant differences, although the p value for urban versus suburban mean age at death approached significance ($p = 0.051$). An unpaired t-test comparing only the urban and rural data did find a statistically significant difference in average age at death between those two groups ($t(188) = 4.51$, $p = <0.0001$). An unpaired t-test comparing the rural sample with a combined urban and suburban sample (average age at death 8.3 ± 8.0 , median = 6 years) also found a statistically significant difference in average age at death ($t(250) = 4.26$, $p = <0.0001$).

Geographic classification	Average (years)	Median (years)	N
Urban	7.5±7.8	5	175
Suburban	10.4±8.3	9	62
Rural	18.2±17.0	16	15

Table 5.18 Average and median ages of acute polio deaths for Hamilton and Toronto and their surrounding areas, 1900-1937.

Age and distance from city centre

Another way to examine the effects of location and population density is to look at residential distance from the city centre rather than by comparing suburban versus urban categories. Such an approach avoids the problems associated with defining urban and suburban categories on the basis of political boundaries (see Jindrich, 2012). Annexations in the late 19th and early 20th centuries had expanded cities like Toronto considerably, bringing suburban and even rural areas within the city boundaries (Jindrich, 2012).

I expected that the age at which people died from polio would increase with distance from city centre, as population density (both in terms of residence and contacts) would generally decline from a high at the city centre moving outwards into the fringe and suburbs. This pattern was confirmed by the analysis of the residential addresses of polio deaths using ArcView (Figure 5.1 and Table 5.19). Here I am focusing on of the City of Toronto and its surrounding area due to its larger geographical size and sample size of polio deaths. This discussion does not include rural farm residences, as lot/concession addresses were not plotted in ArcView.

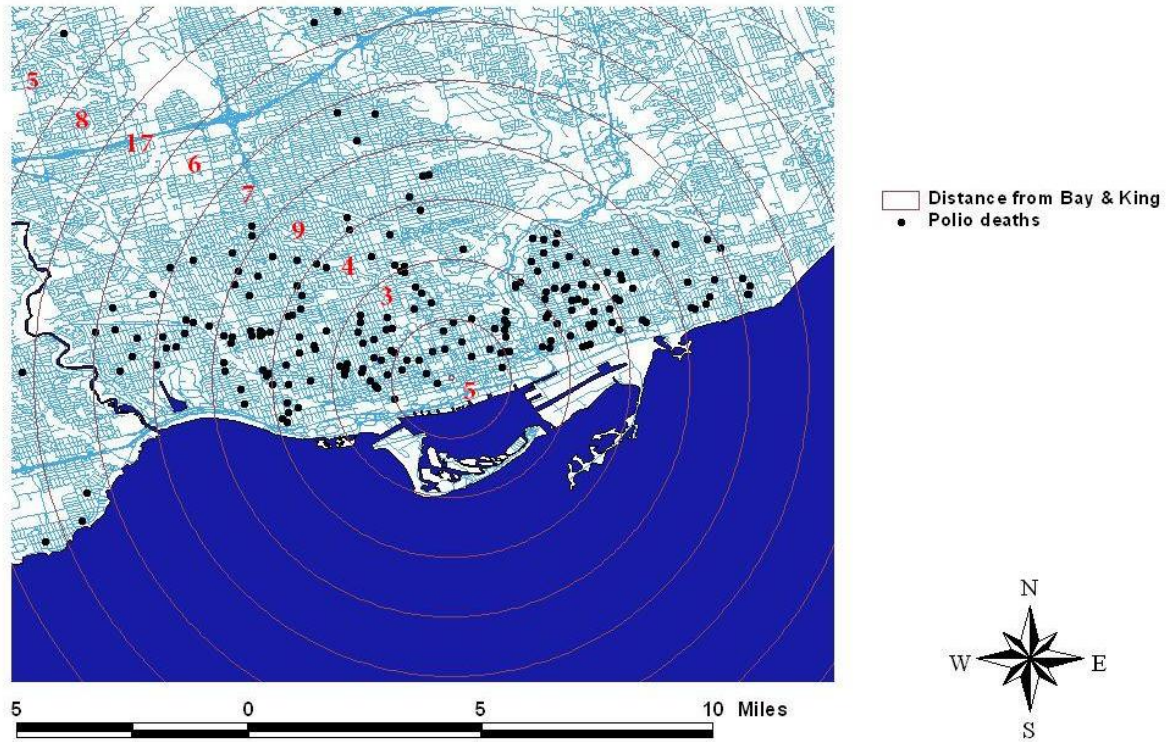


Figure 5.1 Map of acute polio death for Toronto and its surrounding area, 1900-1937. Distance from city centre in 1.5 km intervals. Numbers in red show median ages at death for polio deaths in that circle.

Figure 5.1 shows that the age at death for residents less than 4.5 km from the city centre was consistently lower than for those living farther away. Because sample size dwindles in the outer circles, I partitioned the results into two groups of circles: 1 to 3 (0-4.5 km) and 4 to 9 (4.51 km and above) (Table 5.17). When grouped, the difference in the mean ages at death is statistically significant (unpaired t-test, $t(191) = 2.99$, $p = 0.0031$).

Distance from city centre(s)	Average (years)	Median (years)	<i>n</i>
Circles 1-3 (0-4.5 km)	6.7±7.0	4	114
Circles 4-9 (4.51-13.5 km)	9.8±7.3	9	80

Table 5.19 Average and median ages of acute polio deaths for Toronto and its surrounding area, 1900-1937.

The spread of built-up residential areas over time in Toronto might introduce bias into the analysis, as those who lived in newer residences farther from the city centre would have lived there later in the study period (e.g. the 1930s) when average ages at death from polio were higher. However, the following analysis (Table 5.20) divides the polio deaths into two time periods and confirms that in both instances age at death was higher farther from the city. An unpaired t-test determined that in 1900-1919, there was no difference in mean ages at death by distance from the city centre ($t(43) = 1.01, p = 0.32$), possibly due to small sample size. On the other hand, the difference in age at death was statistically significant for the 1920-1937 period ($t(146) = 2.35, p = 0.020$).

Distance from city centre(s)	1900-1919			1920-1937		
	Avg. (yrs)	Median (yrs)	<i>n</i>	Avg. (yrs)	Median (yrs)	<i>n</i>
Circles 1-3 (0-4.5 km)	5.2±8.3	2	36	7.3±6.3	5	77
Circles 4-9 (4.51-13.5km)	8.2±7.3	4	9	10.0±7.4	9	71

Table 5.20 Average and median ages of acute polio deaths for Toronto and its surrounding area, 1900-1919 vs.1920-1937.

Main Findings and Discussion

There were few polio deaths in rural areas, especially compared to the overall rural population of Wentworth and York Counties as of the 1921 Census and most of all compared to the rural population under 10 years old. However, migration of youth and adults from farms to towns and cities likely means these numbers, based on residence at

death, underestimate the rural contribution to polio mortality. It is likely that many people who grew up in rural areas, and thus escaped early exposure to the poliovirus, would later die of polio in an urban area. Also, there were somewhat fewer polio deaths than expected in the cities compared to their overall populations, though these still made up the majority of polio deaths. Suburban deaths stand out in comparison to the population of towns and villages in the 1921 Census, with 24% of polio deaths compared to 3.3% of the census population (Table 5.17). However, it necessary to note that the 1921 Census gives only a rough gauge of the demographic composition of the population of Wentworth and York: it represents a snapshot of one point in time over a dynamic period of sanitary-social change, whereas the polio sample spans 38 years. While this analysis suggests that suburban families might have been at a slightly higher risk of polio death relative to urban and rural families, city dwellers still made up the vast majority of polio deaths in this period. More detailed data on the age structure of the suburban population and change over time might help to better evaluate this observation.

In terms of age at death, analysis using both rural/suburban/urban classifications and distance from city centre corroborate earlier studies that have shown an inverse relationship between age of polio illness and death, and population density.

Summary

The recurring theme of this chapter is that these many variables (age, sex, family size, birth order, SES, population density) are intimately interrelated. No single variable alone, nor a simple combination of variables, explains polio mortality patterns and their changes over time. Family size mattered, but primarily in conjunction with birth order and age –

as noticed by contemporaries in their research on poliovirus immunity levels (Walton & Melnick, 1955) and theorized in the IE model by Nielsen et al. (2001; 2002a). Family size, furthermore, may be a factor in differences in sex ratios, with more male deaths found in smaller sibships (Nielsen et al., 2002b). Additionally, status scores as a measure of SES follow certain expected patterns (such as increased age at death with increased SES) but not others. For instance, we do not see a clear pattern of increased deaths concomitant with increased SES; actually, polio deaths seem to have been disproportionately prevalent among the skilled blue collar group, status score 3, in Wentworth/York in this period. There is no obvious explanation for this; comparing the spatial distribution of status score 3 deaths to all mapped deaths reveals no immediate clue (see Appendix H). However, we might speculate that families in status score 3 represent the nexus of the traditional polio model and the IE model, with the higher risks of severe polio associated with both (see further discussion in Chapter 6).

The inclusion of rural families at status score 2 introduces some problems in terms of confounding variables, since rural residency in childhood is a recognized risk factor for severity of polio infection due to delayed age at exposure. Even at higher population densities (suburban and urban areas), age at death increased with distance from the city centre. This could correspond with SES, though that was not evident in this time period. Suburban polio deaths were disproportionately high relative to the census population, yet the majority of polio deaths still occurred in the cities. While it is possible that there were differences in probability of diagnosis with polio between rural and urban areas, at least in the early period, the rural areas of Wentworth and York were not particularly remote

compared to other parts of the province. Furthermore, especially in the later study period, serious cases were sent to city hospitals. By the third decade of the study period, the majority of the polio deaths in this study occurred in a hospital, and during the 1937 epidemic, 97% of deaths in this study occurred in a hospital.

Together, these analyses present a complex picture of polio mortality in southern Ontario before WWII – a period of transition, both in terms of polio's emergence and the wider epidemiological and social shifts that accompanied it. While certain socio-demographic circumstances created a higher risk of polio death, such that the type of person most likely to die was a firstborn, native-born, Protestant, young adult male who grew up in a farm family (and so on), still there are numerous individuals in this sample who do not fit this profile. One example is Ruby Chrysdale, who died in Toronto in 1915, one month shy of her second birthday. She was the daughter of a driver, with six older siblings, though it is unclear how many of them survived infancy and early childhood. They were a Catholic family, with children born nearly every year between 1906 and 1913. Ruby was born just as Toronto's new water filtration plant went into operation and milk pasteurization became mandatory. Her maternal grandfather and aunt also lived with them in their home in downtown Toronto (see Appendix E.29 for an extended profile). Another example is 5-year-old Mark Vangloff, the son of immigrant parents from Macedonia. His father was a fur dresser, and they too lived in downtown Toronto. Mark died in September 1930 at the Hospital for Sick Children, from bronchopneumonia complicating bulbar polio (see Appendix E.30 for an extended profile).

By the third decade of the 20th century, people from families living in lower socioeconomic circumstances were dying of polio at similar ages to those from more affluent families. Yet, the overall proportions of polio deaths at the various SES levels did not change. Improved public sanitation operated in combination with social and demographic patterns of falling birthrates, smaller families, and a smaller proportion of the population under age five.

How these results compare and contribute to efforts to understand and model polio's epidemic emergence and mortality patterns will be discussed in more detail in Chapter 6.

Chapter 6 Explaining Severe Polio and Epidemic Emergence

Introduction

As detailed in Chapter 2, changes in hygiene and sanitation improvements are recognized as the major drivers of polio's shift from endemic to epidemic. The hygiene hypothesis plays a key role in both the polio model and the intensive-exposure (IE) model, as discussed in Chapters 2, 4, and 5 and compared in Table 6.1 below. However, it is also recognized that many other factors were at play and that no one theory can explain polio's epidemic patterns in all places and times (Bunimovich-Mendrazitsky & Stone, 2005).²²

The polio model often seems to exist in isolation, rarely discussed in relation to larger models. However, it is very similar to patterns observed and modeled for many infectious diseases affecting humans and other mammals which focus on increasing severity with age of infection and point to transmission dynamics as key to determining disease impact (Anderson & May, 1991; Coleman et al., 2001; Bunimovich-Mendrazitsky & Stone, 2005; Fouchet et al., 2006; Fouchet et al., 2007). Fouchet et al. (2006) for example look at population fragmentation and its effect on disease transmission in rabbit populations. In another, related paper, Fouchet et al. (2007) use a model to look at the effect of population fragmentation on the emergence of new pathologies, linking it to Zinkernagel's (2001; 2003) hypothesis that improved hygiene and sanitation reduced transmission of the poliovirus and that a decrease in maternal antibodies played a critical

²² Smallman-Raynor et al. (2006) also note, "it is probable that, at different times and in different places, a combination of potential factors influenced the emergence of epidemic poliomyelitis", even as improvements in hygiene and sanitation represent the main driver in the process (p. 17).

role in polio's shift from endemic to epidemic mode. In their 2007 paper, Fouchet et al. argued that reduction in the transmission rate leads to more severe disease.

	Polio model	Intensive-exposure model
Why incidence of severe polio increased:	Average age at first infection increased due to improved hygiene/sanitation.	Improved sanitation increased the importance of droplet spread, with severity related to intensity of exposure to the virus.
Also applied to:	Autoimmune diseases (e.g. type 1 diabetes being caused by infection with enterovirus).	Measles (IE model was adapted from model for measles).
Predicts more cases in:	<ul style="list-style-type: none"> - Older ages - Higher SES groups - Rural areas 	<ul style="list-style-type: none"> - Firstborns >6 yrs old and laterborns <5 years old - Secondary cases in household - Crowded households
Shortcomings and weaknesses:	<ul style="list-style-type: none"> - Focus on increased prevalence in age-specific groups does not account for threshold phenomenon where epidemics are produced when transmission of the virus falls below a certain level. - Change in age distribution can also be explained (at least in part) by fertility decline (smaller proportion of the population in youngest age groups, smaller family size, leading to reduced transmission of virus). 	<ul style="list-style-type: none"> - Adapted from model developed for measles, which has a droplet mode of spread, whereas poliovirus has two modes of spread (droplet and fecal-oral).
Key references:	Paul (1971); Nathanson & Martin (1979)	Nielsen et al. (2001; 2002a)

Table 6.1 The polio model and intensive-exposure model compared.

The IE model expands the polio model, and other age-based models, by hypothesizing that the severity and impact of the pathogen are also determined by the nature and circumstances of the exposure to (and infection by) the pathogen (e.g. intensity and dose), not only by the fact of its delay. Gregory et al.'s (2012) study of the patterns of

mortality in a recent polio outbreak in the Democratic Republic of Congo has provided further support for the IE model, finding higher polio mortality with smaller house sizes (two rooms or less).

New research has added more fine detail to the debate and continues to expand our knowledge of what made certain people and places more or less vulnerable to severe polio. Such research includes the finding of Kuss et al. (2011) that intestinal bacteria increased the infectivity of the poliovirus and of Perng et al. (2012) that restricted intrauterine growth, which can result from prenatal malnutrition, was associated with decreased polio incidence. These studies provide further clues about polio's generally inverse relationship with socioeconomic status and go beyond the hygiene hypothesis. For example, people of lower socioeconomic status might have had a lower risk of developing severe polio not only due to earlier exposure to the poliovirus, but also because pregnant women had a greater likelihood of maternal malnutrition that would affect the development of the fetal immune system.

In this chapter I discuss the ways these models inform understandings of polio patterns in Wentworth/York in the study period under scrutiny (1900 to 1937), and consider areas in which they fail to account for observations revealed through this analysis. I then examine ways in which the current models can be revised in light of these findings, both in regards to polio and to infectious diseases more generally.

How the models inform understandings of polio patterns in Wentworth and York Counties between 1900 and 1937

The polio model and hygiene hypothesis

The traditional polio model explains the increase in average age at death from 1900 to 1937 seen in Wentworth and York Counties and the age gradient by status score evident in the early period (1900-1927). It also explains the higher average ages at death in the rural areas. Under the polio model, the polio epidemics in Wentworth and York can be interpreted as the result of ecological change created by sanitation and hygiene measures (e.g. chlorination of the water supply, expansion of sewer systems) aimed at preventing the spread of other infectious diseases. Although the term “ecology” is not used in the polio model literature, sanitation improvements could be considered as an ecological driver behind the epidemics and their changing patterns. The polio model places the experience of southern Ontario in the context of a wider, global phenomenon in the early 20th century in which industrialized and Western settler societies saw polio epidemics arise in the wake of public health movements which successfully reduced infant mortality rates. In countries like the UK and the US, earlier sanitary measures based on infrastructure such as sewers and water lines and regulations such as mandatory pasteurization of the milk supply gave way by the 1920s to “a burgeoning consumer society that... fetishized cleanliness to a near pathological degree” (Valasquez-Manoff, 2012, p. 295), all of which cumulatively served to decrease human contact with microbes. While such measures were very successful in reducing the impact of other diseases such as cholera and typhoid fever, this decreased contact allowed the accumulation of people lacking immunity to the poliovirus, leading to epidemic outbreaks of that disease.

The polio model falls short, however, in explaining variability in several features of the epidemics at different times and places. For instance, it does not account for the way the age gradient by status score observed in Wentworth/York is not sustained for the entire study period. It also does not explain the disproportionately large number of deaths in the middle status score (skilled manual workers) without a similarly disproportionately large number of deaths in the upper status scores. Furthermore, the polio model does not provide an explanation for differences in death rates between males and females, with the proportion of male deaths increasing over time; neither does it provide insight into why there might have been a change in the sex ratio of acute polio deaths over time (from 1.0 in 1900-1927 to 1.6 in 1928-1937). Another shortcoming of the polio model is that it does not explicitly consider population dynamics (such as the declining birth rate that occurred while polio emerged and became epidemic), nor account for differences between transmission routes (fecal-oral vs. droplet). As such, while the polio model can help to explain the epidemic emergence of polio in southern Ontario and its very general patterns in the context of social and ecological changes that were occurring in many regions at that time, other models are required to understand the specific, local patterns that emerged in Wentworth/York and that characterized polio's impact there. As Paul (1971, p. 84) admits, changes in the age at onset and death can only provide a partial explanation for polio's epidemic shift.

The Intensive-Exposure model

The intensive-exposure model can provide clues to why status score 3 (skilled manual workers) had disproportionately more polio deaths than higher and lower socioeconomic

groups. In their discussion of the IE model, Nielsen et al. (2002a) hypothesize that droplet spread became a more important mode of transmission for poliovirus as sanitation improved and prevented the typical fecal-oral route of spread. Racaniello (2006) also notes that in developed countries with high standards of sanitation, the poliovirus has respiratory route transmission in addition to fecal-oral route transmission. This shift in the route of transmission adds an additional layer of complexity and provides a role for crowding in the increase in polio severity. In interpreting the unexpectedly high prevalence of polio among status score 3 individuals in the Wentworth/York study sample, the polio model would suggest that these individuals experienced sufficient sanitation levels to avoid fecal-oral transmission of the poliovirus earlier in life. The IE model, on the other hand, suggests that their households were sufficiently crowded to augment the likelihood of intense droplet transmission of the poliovirus during an epidemic. Thus, families in status score 3 may represent the intersection of the traditional model and the IE model, in which case the IE model is an important contribution to the understanding of the dynamics of polio infection and mortality in Wentworth/York.

The IE model may also provide further insight into the low number of polio deaths among rural residents: rural residents were generally isolated from both droplet and fecal-oral transmission of the poliovirus in their early years, which explains their typically higher age at death, but their larger family sizes meant that individuals were more likely to receive a higher dose of poliovirus upon later exposure.

Again, the IE model makes sense of the evidence that the family sizes of individuals who died of polio were likely at least as large, if not larger, than those in the

general population.²³ The polio model, conversely, generally predicts smaller than average family sizes as risk factors for polio. The IE model also explains the high number of firstborns but underrepresentation of singleton children in the Wentworth/York polio mortality sample.

Finally, Nielsen et al. (2002a) found a U-shaped age distribution in polio severity for hospitalized cases in Copenhagen, Denmark for 1940-1953. They found that paralysis cases were lowest among 8- to 9-year-olds, while the case-fatality rate was lowest in the 4-5 year age group (Nielsen et al., 2002a). Nielsen et al. (2002a) hypothesize that the 4- to 5-year-old age group was particularly important for polio transmission, with kindergarten and first-year schoolchildren being most likely to become index cases, contracting the poliovirus outside the home and subsequently infecting family members. Nielsen et al. (2002a) had access to hospital records and so could examine aspects such as case-fatality rates by age, which were not available for the Wentworth/York sample, as it was restricted to polio deaths rather than all cases. However, the raw numbers do appear to show a U-shaped age curve in the final decade of the study period (1928-1937), with a low dip in deaths at ages 7-8 (see Chapter 4). More work is needed to examine this further, but a cursory investigation of school attendance shows that, according to the 1931 Census (Canada, Dominion Bureau of Statistics, 1937), attendance rates were relatively low before age 7, with compulsory attendance in Ontario beginning at age 8.²⁴ In 1931,

²³ However, as this was a period of changing birthrates and family sizes, this is difficult to establish with certainty, and may not have been the case throughout the entire study period.

²⁴ According to Murdoch C. MacLean, the author of the 1931 Census “Illiteracy and School Attendance” study (Canada, Dominion Bureau of Statistics, 1937), school attendance before age 7 was not only unnecessary but also potentially harmful. MacLean writes that “attendance at the very tender ages of 5 and 6 robs [the average child] of two carefree years of childhood” (p. 90) and that “the parent who sends the

only 11.29% of 5-year-olds attended school for any length of time, and only 53.13% of 6-year-olds who spent an average of approximately one less month in school than older children (Canada, Dominion Bureau of Statistics, 1937, p. 91). Thus, at that time, the 7-8 age group was the first in which the vast majority had entered school and was attending as often as most of the older students. This suggests that it was the 7- to 8-year-olds in Wentworth/York who may have played a particularly important role in poliovirus transmission and thus, according to the IE model, they would have been more likely to be index cases with a lower likelihood of severe illness and death. In the terminology of the field of disease ecology this age group could be considered ‘key hosts’ or ‘superspreaders’ (Hawley & Altizer, 2011).

Of course, polio was a summertime disease, which calls into question the idea of age at school entry as playing an important role in its epidemiology. However, factors such as incubation period and timing of the summer holiday would have to be taken into account. Nielsen et al. (2001; 2002a) do not mention the timing of either the typical polio season in Copenhagen in the 1940-1953 period or the school summer break. To speculate, perhaps the answer lies in the social interactions of this particular age group, which may have been too old to be kept at home but too young to be out working (and thus interacting more with adults); this age group may have spent much of their summer time interacting with other similarly susceptible children, facilitating the spread of the poliovirus.

child to school too young...is culpable” (p. 91). Indeed, the percentage of 5-year-olds in school declined between 1921 and 1931, and MacLean declares this trend “to the good” (p. 97).

While not related to theories of polio's epidemic emergence, Nielsen et al's (2002b) cross-sex transmission hypothesis of polio severity, which seeks to explain sex ratio differences in polio epidemics, has much in common with the IE model. Both hypotheses were adapted from measles studies and focus on details of transmission patterns. Both also seek to explain variation in polio infection severity and highlight the roles of family size and birth order. Nielsen et al. (2002b) hypothesize that transmission of the poliovirus between members of the opposite sex increases polio severity and contributes to excess male cases. The hypothesis rests on the idea that boys have a greater likelihood of being infected by their mothers, rather than their fathers, because mothers have been found to play a greater role in disease transmission in other viral infections, such as those caused by rhinoviruses (see Fox, Cooney, & Hall, 1975). Nielsen et al. (2002b) argue that this cross-sex transmission hypothesis is the most likely explanation for patterns they observed in their study of hospitalized polio patients in Copenhagen from 1919-1953, and that it is compatible with the existing polio literature. In that study, Nielsen et al. (2002b) found that the proportion of female polio patients increased with family size and with birth order; in other words, higher family size and birth order were associated with a more equal sex ratio among polio patients. They also found that when two cases occurred in the same household, they were usually of the same sex and that tertiary cases were more severe when the secondary case (the presumed source of the infection) was of the opposite sex (Nielsen et al., 2002b). Nielsen et al. (2002b) suggest that in larger families, girls would be more likely to be infected by a brother.

This cross-sex transmission hypothesis explains the equal sex ratio observed in the early period of my study (1900-1927) and the growing predominance of males in the later period (1928-1937); the hypothesis is also in line with the general pattern of decreasing family size observed in my sample. Thus my study supports the cross-sex transmission hypothesis, although the mortality sample did not allow for the fine-grained level of detail available to Nielsen et al. (2002b). The cross-sex transmission hypothesis again calls attention to the important role of demographic and social factors in shaping polio patterns, as they interacted with as-yet-undetermined biological factors (Nielsen et al., 2002b).

What remains unexplained after the polio and IE models are applied?

Age at death increased over time among individuals assigned to the lower status scores, to the point where average and median ages at death were equal across status scores.

However, neither the traditional polio model nor the IE model suggests why this would have occurred without an accompanying change in the proportion of deaths in these lower status scores. This finding needs further investigation and explanation.

Both models focus on specific elements or factors that account for a substantial proportion of the patterns seen in epidemic polio across time and space. The IE model is an important step that incorporates greater complexity into polio modeling, at least in terms of considering multiple interacting factors (age, sex, family size, birth order, SES, etc.) that shift over time. The model has been extremely useful in interpreting the patterns seen in Wentworth and York between 1900 and 1937. Yet it still cannot capture all the possible variables, or necessarily explain all patterns.

How evidence from this study can inform models

Informing models of polio

One of the contributions of this study is to provide further evidence against which to test Nielsen and colleagues' recently introduced IE model and cross-sex transmission hypothesis. The results of this study do generally support both. In regards to the cross-sex transmission hypothesis, this study showed a statistically significant decrease in minimum family size among the polio deaths between 1900-1919 and 1920-1937 and a statistically significant excess number of male deaths by 1937. And while many of the other results are not statistically significant, they do trend in the direction anticipated by the IE model.

The results of this study can also be compared against those of Nielsen et al. (2001; 2002a) for Denmark, upon which the IE model was tested. For the 1940-1953 period, Nielsen et al. (2002a) found that polio mortality did not vary by SES, but paralytic polio was slightly more likely in the three highest SES groups, compared to the two lowest. In comparison, the results from the Wentworth/York mortality sample do show some variation, though not the clear gradient of disproportionate deaths with increased SES that the polio model predicts. This suggests that epidemic polio may not have a universal SES pattern but instead vary somewhat over time and according to local conditions.

Looking collectively at the results reported in Chapters 4 and 5, a two-stage pattern emerges (Figures 6.2 and 6.3). The first stage begins with the epidemic year of 1910 and continues through to the late 1920s, where the second stage begins, ending with the 1937 epidemic (and the last year of the study period) (Table 6.2). For the purposes of

statistical testing, the two stages are divided more precisely as 1910-1927 and 1928-1937 (Table 6.3). Stage 1 is characterized by a predominance of deaths under the age of 5, an equal sex ratio, larger family sizes, and higher ages at death in higher status scores. Stage 2 is characterized by a predominance of deaths above age 5, excess male deaths, and a U-shaped age curve that dips at ages 7 to 8. As discussed previously, the two-stage pattern is likely associated with multiple factors during a period of rapidly changing socio-ecological conditions, characterized by advances in public sanitation and hygiene, a declining birth rate, and smaller family sizes.

	Stage 1 (1910 to mid-1920s)	Stage 2 (late 1920s-1930s)
Age at death	Majority <5 years of age Median = 4 years	Majority >5 years of age Median = 9 years
Sex ratio	Equal	Excess male deaths
Family size	Larger	Smaller
Age gradient by SES?	Yes	No

Table 6.2 Generalized two-stage model of changing patterns of epidemic polio mortality in Wentworth and York Counties, 1910-1937.

	Stage 1 (1910-1927)	Stage 2 (1928-1937)
Age at death	Majority <5 years of age Median = 4 years	Majority >5 years of age Median = 9 years
U-shaped age curve?	No	Yes (dip at ages 7-8)
Sex ratio (ages 0-19)*	1.0 (equal)	1.5 (excess males)
Family size (number of children)**	Average = 3.7±2.7 Median = 4	Average = 2.5±2.2 Median = 2
Age gradient by SES?	Yes	No

Table 6.6.3 Specific two-stage model of changing patterns of epidemic polio mortality in Wentworth and York Counties, 1910-1937.

* Difference is statistically significant (χ^2 (1, 121) = 5.17, p = 0.023).

**Difference in averages is statistically significant (unpaired t-test, t (46) = 2.57, p = 0.0014).

This two-stage pattern demonstrates the interconnectedness of various aspects of epidemic polio mortality, such as the link between family size and older ages at infection, and family size and sex ratio. It indicates that the process of polio's epidemic emergence was not simply a matter of increasing age and increasingly large epidemics. The first pattern was evident during the early period (1910 to mid-1920s), despite the small sample size of polio deaths available for study. Important observations can emerge from studies of small samples, even though it may sometimes be necessary to sacrifice the statistical significance that comes with the analysis of large, aggregated samples.

Informing broader models

In terms of broader models of infectious diseases, the IE model and the support for it found in this study provides further evidence for the need to consider multiple routes of disease transmission when studying any pathogen. They also indicate that those who have attempted to adapt the traditional polio model to explain patterns in other diseases (see Chapter 2) might need to consider the implications of new models of polio's emergence and shifting epidemiology.

Summary

In his review of current knowledge of poliovirus pathogenesis, Racaniello (2006) suggests that further research on this topic could be used “to establish paradigms that will help us better understand other virus diseases” (p. 14). This indicates the importance of continued research on polio despite the approach of global eradication. Research using historical records will become increasingly important, as experimental research using

virulent poliovirus strains will be restricted once eradication is achieved (Racaniello, 2006).

The continued revisions of models of polio epidemics many decades after the introduction of the vaccines demonstrate the difficulty of incorporating multiple interacting and shifting variables. Smith (2007) comments on the challenges of working with this type of complexity, asking, “Multidimensional, multi-causal, multi-variable – how do you judge a moving biosystem?” (p. 7). Blalock (1979) noted this as one of the major problems facing historical demographic research in the social sciences, arguing that “reality is sufficiently complex that we will need theories that contain upwards of fifty variables if we wish to disentangle the effects of numerous exogenous and endogenous variables on the diversity of dependent variables that interest us” (p. 881). A model like Worthman’s bioecocultural model of child development, adapted for polio, may be of use in highlighting the interplay of multiple factors (see Figure 6.1 below).

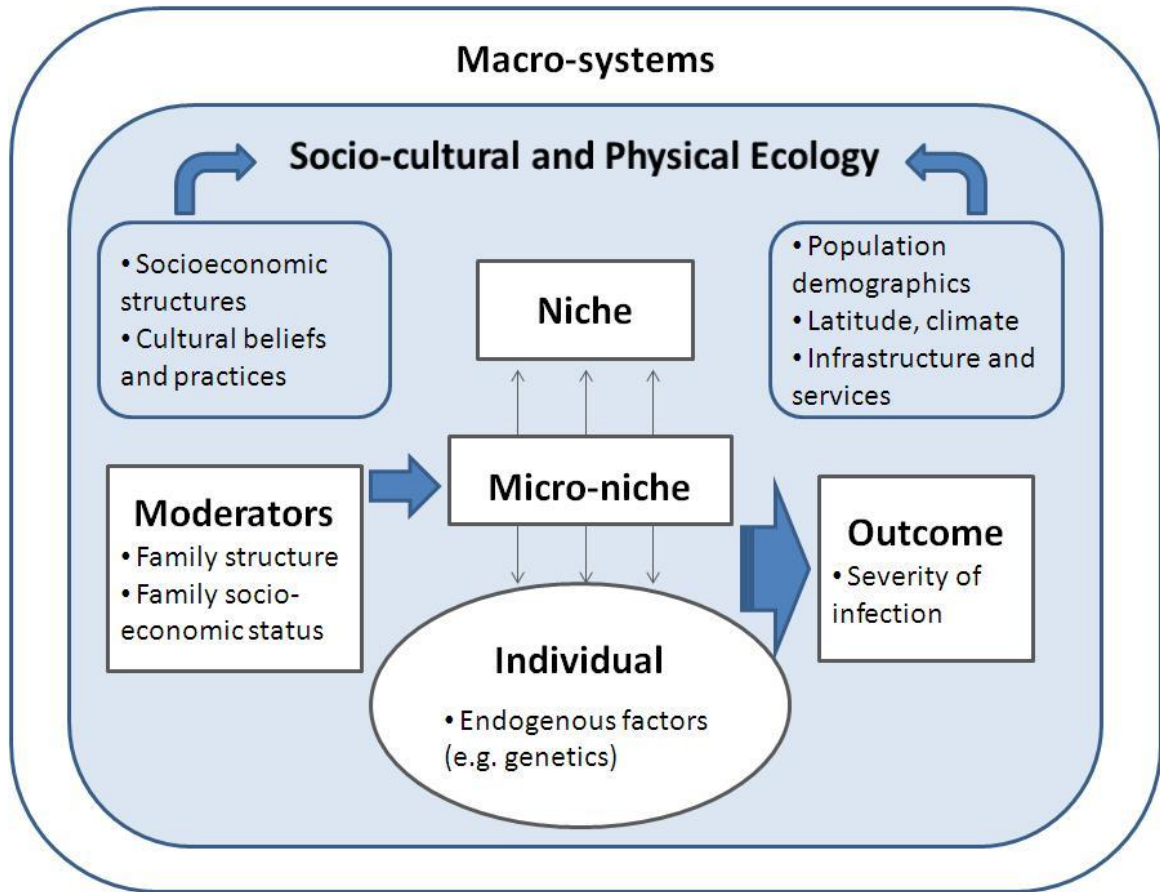


Figure 6.1 Bioecocultural diagram of factors affecting polio's patterns and severity. Based on diagram in Worthman (2010, figure 4, p. 556).

This study of polio mortality has highlighted some of these variables in the particular ecological and historical context of early 20th-century southern Ontario, adding to the effort to better understand the complexity of this disease and its place in the larger system.

Chapter 7 Conclusion

This study of polio mortality in southern Ontario's Wentworth and York Counties during the period of epidemic emergence before World War II has examined a range of demographic, social, cultural, and economic factors which influenced the patterns of this disease. It has demonstrated that these patterns shifted significantly over the course of the study period (1900-1937) as factors in the biological, ecological, and cultural environment changed. Moreover, it has applied and tested the Intensive-Exposure and cross-sex transmission hypotheses as presented by Nielsen and colleagues (2001; 2002a; 2002b). While this research has found much support for these hypotheses, it is also clear that much about polio remains to be understood.

A number of observations have arisen from this work. This study has hinted at the death toll from polio that did not get recorded in the case-fatality statistics of the epidemics because the deaths, although directly attributed to the disease, took place months or years later. Furthermore, the historical research beyond the death registrations provided a glimpse into the personal circumstances and repercussions of individual polio deaths. Additionally, this thesis has shown that it is not enough to simply say that "the middle class" was particularly hard hit by polio, without specifying who is included in that "middle class" and what their living conditions were like at the time.

Applying basic GIS analysis to these data was found to be useful in some ways far more than others. Spatial patterns in polio mortality may not be as informative as those of other diseases or in other locations for several reasons: the fact that polio was symptomatic in less than 10% of cases, and fatal in only 1-2%; the social geography of

Toronto in this period was fairly heterogeneous; and migration from rural to urban areas and movement within Toronto and Hamilton were both very common in this period.

Finding patterns, especially ones that change over time, in such a dynamic and complex context is a major challenge for historical GIS studies in general. This is particularly true for a disease like polio for the reasons mentioned above.

Future research can build on this work by expanding the sample size and thus increasing the statistical power of the data. Each year, additional birth and marriage records are released for this time period, which can significantly increase the amount of data available for the individuals in this study. Marriage records, for example, contain information on birthplace and occupation of both the individuals being married and of their own parents. Birth records allow determination of family sizes and also provide such information as the occupation of the father at the time of the birth and residential address at birth. Furthermore, at this time I would also like to expand the study sample and compare the findings of this study to other counties in Ontario. This would provide an opportunity both to test whether the patterns found in Wentworth and York Counties hold true elsewhere in the province, and to expand the sample size and increase the statistical power of the study.

Another question that this study did not explicitly address but became a topic of interest as data analysis took shape is the possible role of changing personal circumstances placing individuals and families at risk of severe polio infection. For instance, in the cases where individuals with reduced exposure to the poliovirus earlier in their lives due to their high socioeconomic status or rural residence later faced poorer

living conditions or moved to an urban area. Such otherwise advantageous earlier living conditions could have set the stage for greater likelihood of a severe case of polio if their living conditions later worsened – such as was the case for many families during the Depression years. As an example of such changes in living conditions, families often would take on boarders, increasing crowding in the household. This might add a further element to the hygiene hypothesis and bring us back in a way to the traditional medical anthropological focus on factors such as breakdown in sanitary health conditions and crowding that increases the risk of infections. This may be evident in polio outbreaks today; Gregory et al. (2012) found that use of well water during a water shortage was associated with higher polio mortality during the September 2010 – January 2011 polio outbreak in the Democratic Republic of Congo. While it is difficult to test this given the historical data currently available, the continued annual release of Ontario birth registrations in particular as well as the future release of additional national censuses should provide the means of comparing individuals' living conditions at time of birth versus at death.

Even as the poliovirus nears global eradication, research continues to expand into new areas. Studies of the interaction of the poliovirus with intestinal microbiota (Kuss et al., 2011) and of the effects of intrauterine growth conditions on later polio infection outcome (Perng, Cnattigus, Iliadou, & Villamor, 2012) demonstrate how much remains to be learned about the often paradoxical dynamics of poliovirus-host interactions. The hygiene hypothesis and various models clearly only capture part of what is becoming an increasing complex picture, not only in terms of polio but about human immune ecology

in general. A dominant theme of writing on the third epidemiological transition and the emergence and re-emergence of diseases is that the driving force behind this is increased contact between humans and pathogenic microbes due to ecological change (Barrett, Kuzawa, McDade, & Armelagos, 1998). Yet, according to the hygiene hypothesis, the epidemic emergence of polio was a consequence of change that *decreased* human contact with pathogenic microbes. Thus the study of polio's emergence can contribute to existing critiques of the model of epidemiological transition and, as Barrett et al. (1998) suggest, to efforts to expand the model.

A perspective from the anthropology of infectious disease has much to contribute to endeavors to address this complexity. Anthropological work has long brought together evidence from multiple disciplines to understand complex phenomena. The study of emerging infectious disease must embrace complexity (Levins, 1995), which medical anthropologist Paul Farmer (1996) notes must include social complexity. An anthropological perspective can offer broader theoretical models, like Worthman's (2010) bioecocultural model, which bring together not only a diverse range of factors but also operate on multiple levels, from the individual to the local to the global. It can ground theory about population-level phenomena, like the hygiene hypothesis, in the lived realities of individuals, families, and communities, "the traditional microlevel foci of anthropology" (Stonich, 1996, p. 80). In so doing, it has the potential to significantly expand our knowledge and understanding of diseases like polio and their impact on individuals and populations, which even after decades of study still remain somewhat of a mystery.

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Schedule D.

DEATHS

County of ⁽²⁾ York Division of ⁽¹⁾ York Township

Surname First		Surname First		Surname First	
McMicking, Irma		Leeks, Louise Emily		Todd, Mary G.	
Sex, and Race.		Female		Female	
Date of Death.		Sept 8th 1912		Sept. 8th. 1912	
Date of Birth.		June 24th. 1912		July 23rd 1911	
Age and Place of Birth.		2nd. 13 yrs York Twp.		14 mos. 12 Gallow Ave	
Place of Death, City, Town, Village, or Township and Locality, if known, give name, and any previous place of residence.		1711 Gallow Ave York Township		114 Gledhill Ave	
Occupation.		036129		036130	
Single, Widowed, or Married.		036131		036131	
Full Name of Father.		McMicking, J. Howard		Leeks, Alfred	
Birthplace of Father.		Canada		Twelve	
Maiden Name of Mother.		Dawson, Mabel		Coates, Naomi	
Birthplace of Mother.		Canada		Ontario	
Name of Physician who attended Deceased.		Dr. James Dow		Dr. Demary	
Certified by		L. Scruton.		Alfred Leeks	
Address		1841 Dundas St.		114 Gledhill Ave	
Date		Sept. 6th 1912		Sept. 9th. 1912	
Medical Certificate of Death.		Medical Certificate of Death.		Medical Certificate of Death.	
I hereby certify that I attended the deceased.		I hereby certify that I attended the deceased.		I hereby certify that I attended the deceased.	
Name.		McMicking, Irma		Leeks, Louise Emily	
From		June 24th. 1912		Aug. 21st. 1912	
To		Sept. 6th. 1912		Sept. 7th. 1912	
That I last saw this person alive on		Sept. 1st. 1912		Sept. 7th. 1912	
That the Death occurred on		Sept. 6th. 1912		Sept. 8th. 1912	
CAUSE OF DEATH.		Marasmus		Acute Infantile Paralysis	
Primary.		Two months		36 hours	
Duration.		Anaemia		above	
Immediate.		36 hours		two days	
Physician's name.		James Dow		A. F. Demary	
Address.		175 St. John St.		2 Ryall Ave.	
Date.		Sept. 6th. 1912		Sept. 9th. 1912	
Remarks.		Regd. Sept. 7th. 1912		Regd. Sept. 10th. 1912	
I hereby certify the foregoing to be the true and correct entries of all Deaths returned to me for the quarter ending day of		A.D. 1912		A.D. 1912	

*H.K.—This reference number given for use in Form A, to assist in transcribing.

Figure A.2 Example of a death registration form from 1908-1912 study period. Archives of Ontario, Series MS935, Reel 182, via Ancestry.ca. Ontario, Canada, Deaths, 1869-1938 and Deaths Overseas, 1939-1947 [database on-line]. Provo, UT, USA: Ancestry.com Operations Inc, 2010.

218 DEATHS

County of YORK Division of SCARBORO.

SURNAME of Deceased	Farquharson	Dark	Terranova
Christian Name	William Stewart	William	Lucia
Sex	Male	Male	Female
Age	28 yrs	89 yrs	4 yrs
Date of Death	Nov, 19, 1919	Nov, 30, 1919	Dec, 1, 1919
Place of Birth	Glace, Ont	England	Toronto
Place of Death, City, Town, Village, or Concession and Lot	Agincourt	Lot 27, Con 3.	Stop 45 Kingston Road
Place of Burial	Agincourt	Washington Cemetery	St Johns Cemetery
Occupation	Journalist	Retired	
Single, Married or Widowed	Single	Married	037405
Name of Father	William Farquharson		Frank Terranova
Maternal Name of Mother	Annie Couts 037403	037404	Dollie Jtniri
Cause of Death, if known	Tuberculosis		
Name of Physician who attended Deceased	Dr A.W. Farquharson	Dr Walters	Dr Young
Name of Informant	A.W. Farquharson	John T. Dark	S. Irongo
Address	Agincourt	Agincourt	West Hill
Date of Return	Nov, 19, 1919	Nov, 30, 1919	Dec, 1, 1919
Physician's Return of Death	Farquharson ✓	Dark ✓	Physician's Return of Death
Christian Name	William Stewart	William	Terranova ✓
Date of Death	Nov, 19, 1919	Nov, 30, 1919	Dec, 1, 1919
DISEASE CAUSING DEATH	Tuberculosis 26	Organic Heart Disease 19	Infantile Paralysis 63
Duration	6 Years	Years	Three months
Immediate Cause of Death	Exhaustion	Heart Failure	Exhaustion
Duration	Gradual		Immediate
Physician's Name	W. Farquharson	W.R. Walters	Thos A. Young
Address	Agincourt	1502 Danforth Ave	Scarboro
Date of Return	Nov, 19, 1919	Dec, 1, 1919	Dec, 1, 1919
Remarks			

I hereby certify the foregoing to be the true and correct entries of all Deaths returned to me for the quarter ending Dec, 31st 1919
 Given under my hand, this 15th day of January A.D. 1920
 Christian Registrar of Scarboro
 W.D. Annis

Figure A.3 Example of a death registration form from 1913-1919 study period. Archives of Ontario, Series MS935, Reel 260, via Ancestry.ca. Ontario, Canada, Deaths, 1869-1938 and Deaths Overseas, 1939-1947 [database on-line]. Provo, UT, USA: Ancestry.com Operations Inc, 2010.

THIS FORM MUST BE FILED FORTHWITH WITH THE DIVISION REGISTRAR OF THE DIVISION IN WHICH THE DEATH OCCURRED BEFORE A BURIAL PERMIT CAN BE ISSUED.
WRITE PLAINLY WITH UNFADING INK. THIS IS A PERMANENT RECORD.

Every item of information should be carefully supplied.
AGE should be stated EXACTLY. PHYSICIANS should STATE CAUSE OF DEATH in plain terms, so that it may be properly classified. Their statements of DEGREE OF DEATH, and RACIAL ORIGIN will be described by stating to what people or race the deceased person belonged, whether English, Scotch, Irish, French, German, etc. The terms "American" or "Canadian" should not be used, as they express citizenship but not a race or people.

FORM 6 PROVINCE OF ONTARIO 5055 005893

CERTIFICATE OF REGISTRATION OF DEATH

1. PLACE OF DEATH: County of York Township of Toronto
If in City, Town or Village (Name) Street (Name) House No. _____

2. NAME OF DECEASED: Heather John (Name)
Residence: 79 Mimico Ave (Usual place of abode)
Mimico, Ont

3. Sex: Female 4. Racial origin: English 5. Single, Married, Widowed, or Divorced, state which: Single

6. BIRTHPLACE: Mimico, Ont (Province or country)

7. DATE OF BIRTH: _____ (Month) _____ (Day) _____ (Year)

8. AGE OF DECEASED: 9 Years _____ Months _____ Days If less than one day old _____ hrs. or _____ min.

9. OCCUPATION OF DECEASED: _____ (Trade or occupation or kind of work)

10. LENGTH OF RESIDENCE (in years and months):
(a) At place of death: 1 day (b) In province: _____
(c) In Canada (if an immigrant): _____

11. Name of father: Charles L. Heather (Province or country)
12. Birthplace of father: Ontario
13. Maiden name of mother: Rosina Hendry (Province or country)
14. Birthplace of mother: Ontario

15. Name of Informant: Charles L. Heather
Address: 79 Mimico Ave, Mimico
Relation to Deceased: Wife

16. DATE OF DEATH: August 20 1930
17. I HEREBY CERTIFY that I attended deceased from August 19 1930 to August 20 1930 and last saw him/her alive on August 20 1930
The CAUSE OF DEATH was as follows:
Acute Anterior Poliomyelitis (Bulbar type)
(duration of) _____ yrs. _____ mos. 5 days
CONTRIBUTORY CAUSE (Secondary): Refusal of food
(duration of) _____ yrs. _____ mos. _____ days

18. Where was disease contracted if not at place of death? ?
Did an operation precede death? No Date of _____
Reason for operation: _____
Was there an autopsy? No
(Signed) Geo. Caldwell M.D.
Address: Hospital for Sick Children
Date: August 20 1930
(Month) (Day) (Year)

19. Place of Burial: Park Lawn Cemetery Date of Burial: August 21/1930
20. Name of Undertaker: Ross Ghera Address: 2353 Bloor St. W. Toronto
Filed at _____ (Hour) _____ m. this _____ day of _____ (Month) 1930
DIVISION REGISTRAR CITY OF TORONTO

For use by Division Registrar only:
Burial Permit was issued by: _____ Name: Geo. Harper Address: 1066 Denison Date: Aug 21st 1930
POWER OF ATTORNEY

Figure A.4 Example of a death registration form from 1920-1934 study period. Archives of Ontario, Series MS935, Reel 382, via Ancestry.ca. Ontario, Canada, Deaths, 1869-1938 and Deaths Overseas, 1939-1947 [database on-line]. Provo, UT, USA: Ancestry.com Operations Inc, 2010.

FORM 1

THIS form is placed in an envelope, marked "Deaths Statistics—Free, penalty for improper use \$300," and properly addressed and sent through the mail "FREE" 239

5122

005921

PROVINCE OF ONTARIO—CERTIFICATE OF REGISTRATION OF DEATH

1. PLACE (County or District of) York Township of Hospital for Sick Children House No. 239

DEATH 114 City, Town or Village (Name) Street Hospital for Sick Children (If death occurred in a hospital or institution, give the name instead of street and number)

2. LENGTH OF STAY (in years, months and days) (a) In City, Town or Township where death occurred. (b) In Province. (c) In Canada (if immigrant)

3. NAME OF DECEASED MULLIN (Family name) JANE (Given name or names) RNN

RESIDENCE No. 376 Street BRUNSWICK City, Town, Village or Township Toronto Province Ontario

4. Sex FEMALE 5. Nationality (Citizenship) Canadian 6. Racial Origin ENG 7. Single, Married, Widowed or Divorced (circle the word) SINGLE

8. BIRTHPLACE Canada (Province or Country)

9. DATE OF BIRTH (Month) (Day) (Year)

10. AGE in Years 6 Months 6 Days None If less than one day old hrs. or min.

11. Trade, profession or kind of work as spinner, tea master, office clerk, etc. None

12. Kind of industry or business, as settler, mill, lumbering, bank, etc.

13. Date deceased last worked at this occupation

14. Total yrs. spent in this occupation

15. If married give name of wife or husband of deceased

16. NAME Archibald Mullin

17. BIRTHPLACE Canada (Province or Country)

18. MAIDEN NAME Alexina Linkerton

19. BIRTHPLACE United States (Province or Country)

20. Person giving information sign here: S. A. Mullin Address 376 BRUNSWICK AVE Relationship to deceased Father

21. Place of Burial, Cremation or Removal Mount Pleasant Date of burial or removal Aug 31-36

22. Undertaker Wm. J. 3055 Clair W. (Name and address)

MEDICAL CERTIFICATE OF DEATH

23. DATE OF DEATH August 30, 1936 (Month) (Day) (Year)

24. I HEREBY CERTIFY that I attended deceased from: 19__ to 19__ and last saw him/her alive on 19__

CAUSE OF DEATH

I acute Rho-Encephalitis

Give disease, injury or complication which caused death, and the mode of dying, such as heart failure, asphyxia, asbestosis, etc. due to

II World conditions, if any, giving rise to immediate cause (stated in order proceeding backwards from immediate cause).

Other world conditions (if important) contributing to death but not causally related to immediate cause.

25. If a woman, was the death associated with pregnancy? Yes

26. Was there a surgical operation? No Date of operation 19__ State findings Was there an autopsy? Yes

27. If death was due to external causes (violence) fill in also the following:— Accident, suicide or homicide? Yes Date of injury 19__ Manner of injury Stab (State which) Nature of injury Flow autopsied Specify whether injury occurred in industry, in home, or in public place

Signed by Dr. J. M. W. 3055 Clair W. Address 177 Denby Ave. Date August 30 1936

28. Division Registrar's Record Number JAMES W. L. 1936

29. Filed 19__

POWER OF ATTORNEY

THIS FORM MUST BE FILED FORTHWITH WITH THE DIVISION REGISTRAR OF THE DIVISION IN WHICH THE DEATH OCCURRED BEFORE A BURIAL PERMIT CAN BE ISSUED

WRITE PLAINLY WITH UNFADING INK THIS IS A PERMANENT RECORD

Every item of information should be carefully supplied. (See reverse side for instructions.)

Figure A.5 Example of a death registration form from 1935-1937 study period. Archives of Ontario, Series MS935, Reel 537, via Ancestry.ca. Ontario, Canada, Deaths, 1869-1938 and Deaths Overseas, 1939-1947 [database on-line]. Provo, UT, USA: Ancestry.com Operations Inc, 2010.

Appendix B : List of Occupations and Associated Status Scores

Occupation title	Composite status score (Hauser 1982)	My score	All scores
Clergy	1		1
Commission Merchant	1		1
Gentleman	1		1
Lawyer	1		1
Merchant	1		1
Physician	1		1
Bank Manager		2	2
Civil engineer		2	2
Provincial officer		2	2
Customs officer		2	2
Agent	2		2
Bookkeeper	2		2
Broker	2		2
Contractor		2	2
Builder	2		2
Chemist	2		2
Clerk	2		2
Dentist	2		2
Druggist	2		2
Dry Goods/Fancy	2		2
Farmer	2		2
Fruitgrower/fuiter		2	2
Grocer	2		2
Hotel Keeper	2		2
Innkeeper	2		2
Jeweler	2		2
Liquor Dealer	2		2
Manufacturer	2		2
Sales Agent	2		2
Salesman	2		2
Stockkeeper		2	2
Storekeeper	2		2
Student	2		2
Tavern Keeper	2		2
Teacher	2		2
Tabacconist	2		2

Victualer	2		2
Counter man (similar to waiter)		4	4
Electrician		3	3
Blacksmith		3	3
Welder		3	3
Solderer		3	3
Metal worker/iron worker		3	3
Baker	3		3
Barber	3		3
Boat Captain	3		3
Boiler	3		3
Bookbinder	3		3
Brewer	3		3
Bricklayer	3		3
Brickmaker	3		3
Brickmason	3		3
Butcher	3		3
Cab Maker (cabinet maker)	3		3
Carpenter	3		3
Carriage maker	3		3
Tobacco worker/tobacco roller		3	3
Cigar Maker	3		3
Conductor	3		3
Confectioner	3		3
Cooper	3		3
Cordwainer	3		3
Dealer	3		3
Dyer	3		3
Engineer	3		3
Gas Fitter	3		3
Glass Blower	3		3
Saddle Maker	3		3
Hatter	3		3
Joiner	3		3
Machinist	3		3
Mechanic		3	3
Mason	3		3
Moulder	3		3

Toolmaker		3	3
Nail Maker	3		3
Operator	3		3
Painter	3		3
Paper Hanger	3		3
Clothes repairer		3	3
Pattern Maker	3		3
Peddler	3		3
Piano Maker	3		3
Plasterer	3		3
Plumber	3		3
Printer	3		3
Puddler	3		3
Saddler	3		3
Ship Carpenter	3		3
Shipwright	3		3
Shoemaker	3		3
Stonecutter	3		3
Stonemason	3		3
Tailor	3		3
Tanner	3		3
Tinsmith	3		3
Turner	3		3
Typesetter	3		3
Upholsterer	3		3
Weaver	3		3
Wheelwright	3		3
Fur dresser		3	3
Telephone repair man		3	3
Lineman (telephone company)		3	3
Press hand/Pressman		3	3
Policeman		3	3
Fireman		3	3
Shipper		4	4
Pork packer		4	4
Caddie (messenger or errand boy)		4	4
Mail collector/mail carrier		4	4
Bartender	4		4
Boatman	4		4

Carman	4		4
Motorman		4	4
Carter	4		4
Chair Factory	4		4
Coachman	4		4
Driver/chauffeur		4	4
Ferryman	4		4
Fisherman	4		4
Furnaceman	4		4
Gardener	4		4
Hostler [groom/stableman]	4		4
Mariner	4		4
Porter	4		4
Quarryman	4		4
Railroad Worker	4		4
Soldier		4	4
Sailor	4		4
Seaman	4		4
Servant	4		4
Teamster	4		4
Waiter	4		4
Watchman	4		4
Foreman (electric company)		3	3
Locomotive foreman		3	3
Trainman		4	4
Yardman	4		4
Drayman	5		5
Farm Laborer	5		5
Lab Man	5		5
Laborer	5		5
Waterman	5		5

Table B.1 List of occupations and associated status scores. Includes all occupations from Hauser (1982) as well as those found in the polio sample.

Appendix C : Age and Sex Patterns

Males make up a higher proportion of polio deaths in every age group except 10-14 years, with the greatest gap between the sexes in the 25-29 year old age group (Appendix C.1).²⁵

The 15-19 age group forms a larger proportion of the male acute deaths (13.9%) than in the females (7.3%), while in the females the 0-4 and 10-14 age groups each form a greater proportion of acute deaths (43.8% and 14.6%, respectively) in comparison to those groups in the males (38.0% and 9.0%, respectively; see Appendix C.2).

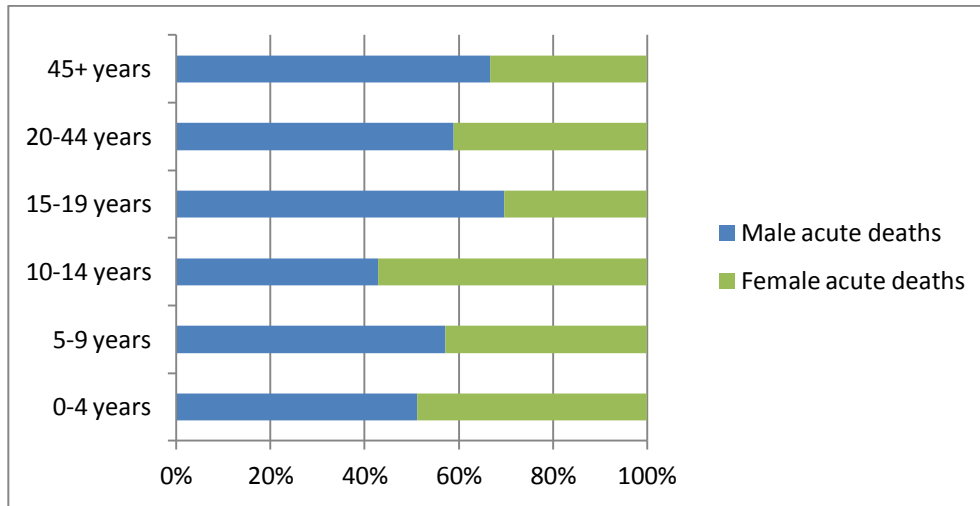


Figure C.1 Proportion of polio deaths by sex for acute polio deaths in Wentworth and York Counties 1900-1937 ($n = 303$).

²⁵ If the 20-44 age group is broken down into 5-year age groups, females outnumber males in the 30-34 and 35-39 age groups. However, the number of deaths in these groups is very small. For ages 30-39, there are 4 males and 6 females total, with only 3 males and 5 females in non-chronic deaths.

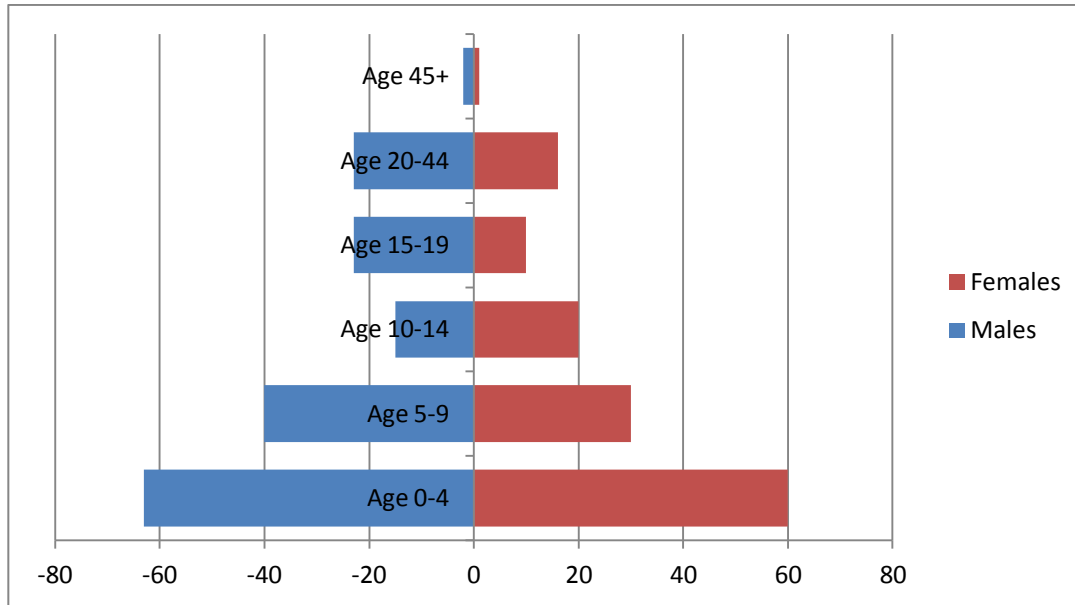


Figure C.2 Age-specific mortality pyramid for acute polio deaths ($n = 303$) in Wentworth and York Counties 1900-1937.

Polio deaths from New York City for 1916 and 1940-41 show similarities to Wentworth-York (Appendix C.3). All three samples show excess male mortality in most age groups, but by differing degrees. This variation in sex ratio by age has been noted elsewhere; for example, Forsbeck and Luther (1930) found that the sex ratio in polio cases in Massachusetts was highest during adolescence (ages 14-18). The sex ratio is similarly high in Wentworth/York in the 15-19 age group.

However, polio morbidity patterns are not necessarily reflected in mortality. In examining polio deaths specifically, Weinstein (1957) found an equal sex ratio in victims under age 15 in Massachusetts, despite there being more males in total cases in this age group. He also found more male deaths among those 16 years and over, despite females

predominating in cases (Weinstein, 1957). This issue of morbidity versus mortality patterns in age and sex is discussed further in Chapter 4.

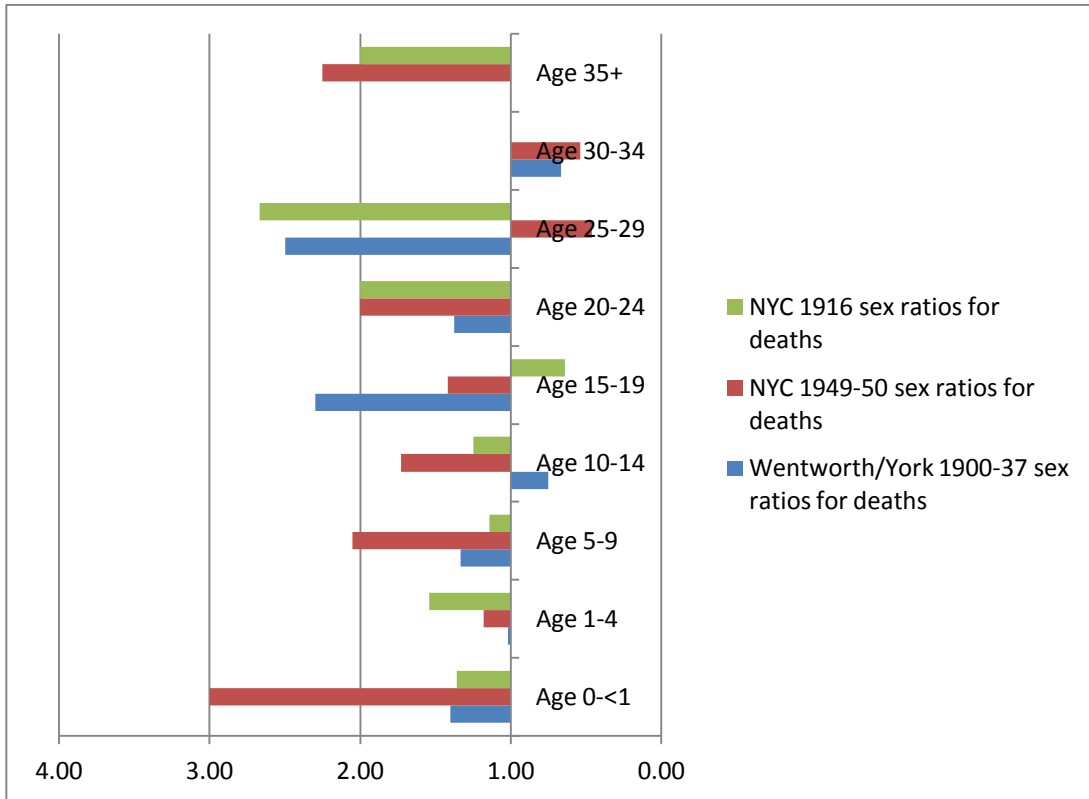


Figure C.3 Comparison of sex ratios in the acute polio sample from this study ($n = 303$) and the sex ratios from New York City polio epidemics in 1916 (“Poliomyelitic Deaths, City of New York, 1916”) and 1949-1950 (Abramson & Greenberg, 1955).

Appendix D : Population Age Structure According to Censuses

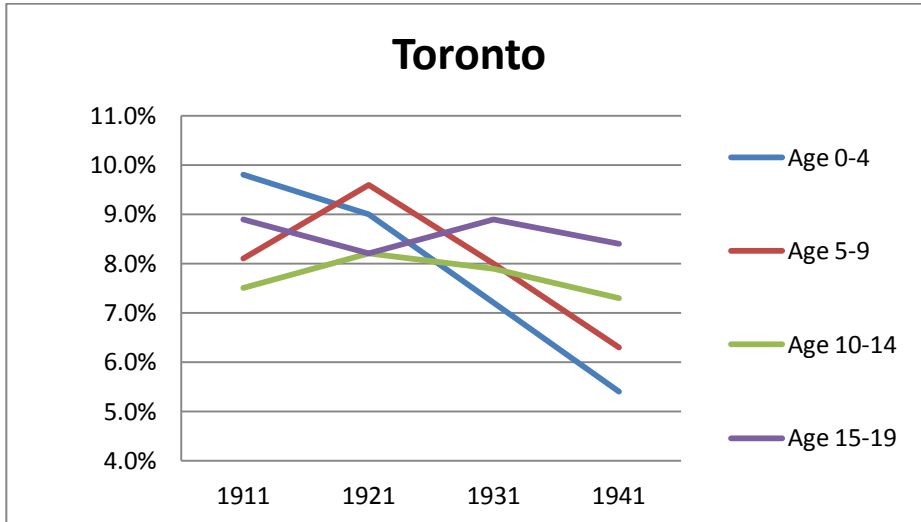


Figure D.1 Proportion of total population for Toronto by five-year age groups, 1911-1941. Source: Canada (1925; 1933; 1935; 1946).

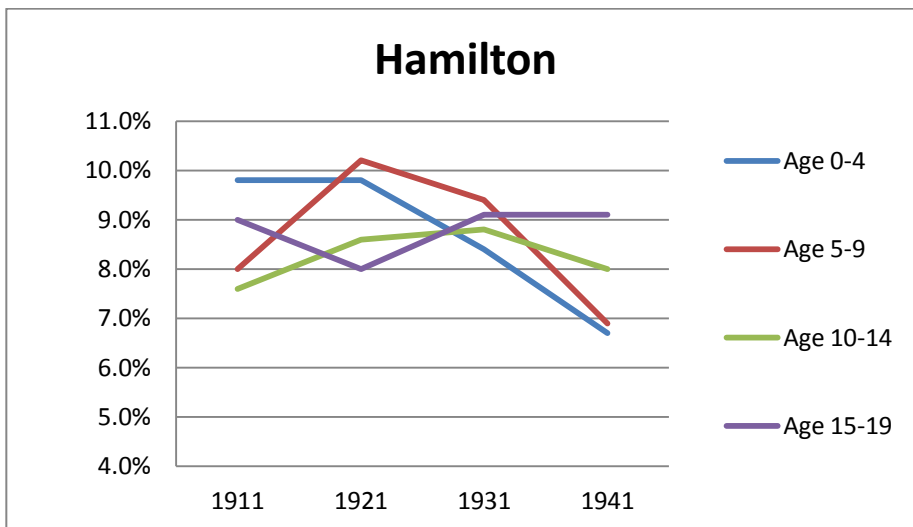


Figure D.2 Proportion of total population for Hamilton by five-year age groups, 1911-1941. Source: Canada (1925; 1933; 1935; 1946).

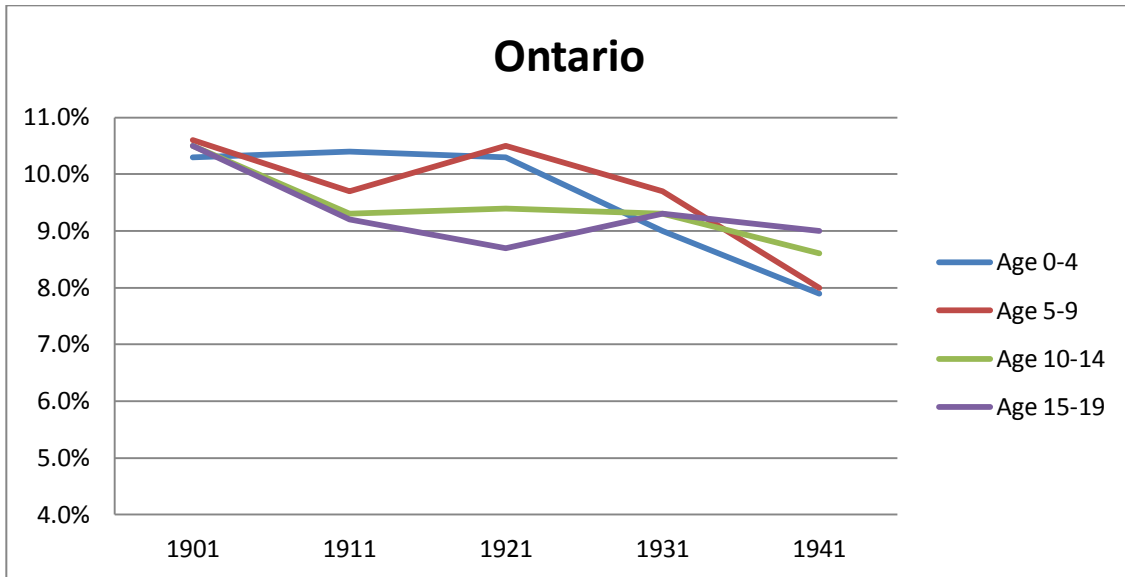


Figure D.3 Proportion of total population of Ontario by five-year age groups, 1901-1941. Source: Canada (1935; 1944).

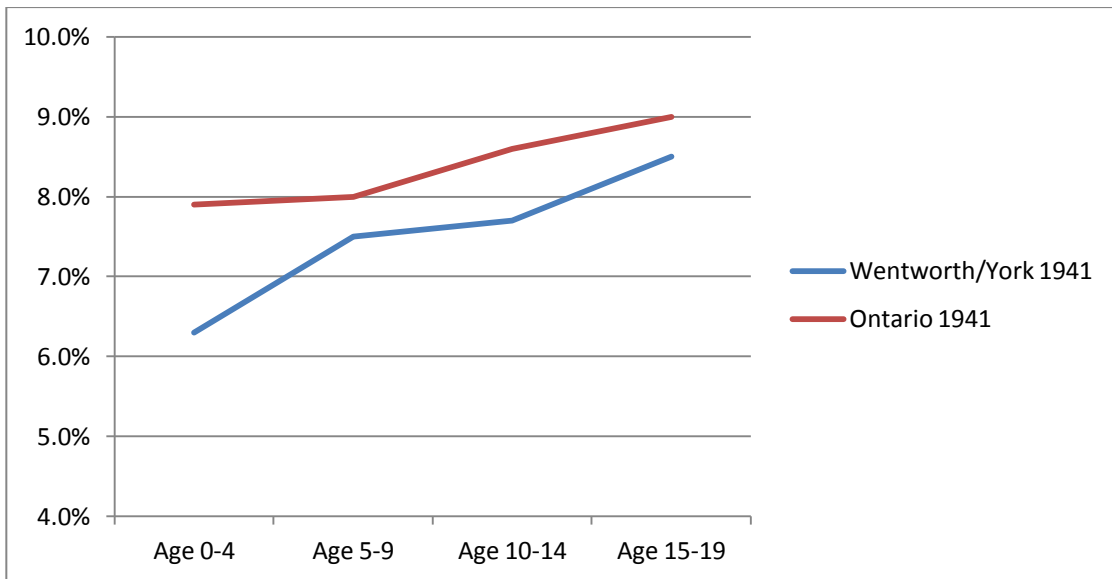


Figure D.4 Proportion of total population by five-year age groups in Wentworth and York Counties vs. Ontario, 1941. Source: Canada (1944).

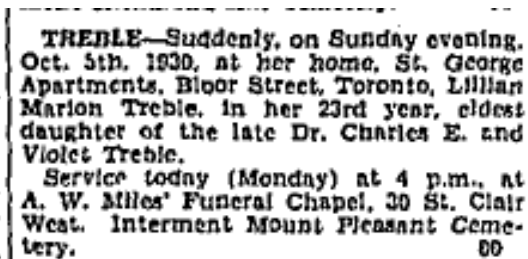
Appendix E : Profiles

“All generalizations about the social determinants of health and disease begin in the lived reality of particular people and places. It is to that reality that our masterful images must be compared, and against which they must be judged” (Kunitz, 2007, p. 185).

The profiles below represent approximately one-third of the study sample of 336 individuals whose deaths were ascribed to polio in Wentworth and York Counties, 1900-1937.

Appendix E.1: Lillian Marion Treble

Lillian was born in 1908 in Toronto. Her father, Charles E. Treble, was a physician. In her earliest years, her family lived at 207 Palmerston Avenue in Toronto’s Ward 5. She was the eldest daughter, with at least two younger sisters. Lillian died in October 1930 at age 22, after an acute polio illness of eight days. At the time of her death, Lillian lived in the St George Apartments, at St George and Bloor Streets in Toronto’s Annex neighbourhood (Ward 4). She was buried in Mount Pleasant Cemetery. Her death notice appeared in Toronto’s *Globe* newspaper:



TREBLE—Suddenly, on Sunday evening, Oct. 5th, 1930, at her home, St. George Apartments, Bloor Street, Toronto, Lillian Marion Treble, in her 23rd year, eldest daughter of the late Dr. Charles E. and Violet Treble.
Service today (Monday) at 4 p.m., at A. W. Miles' Funeral Chapel, 30 St. Clair West. Interment Mount Pleasant Cemetery. 00

Figure E.1 Lillian Treble’s death notice (*The Globe*, 6 October 1930, p. 14).

There is some discrepancy over whether her father predeceased her; the *Globe* death notice refers to him as “the late Dr. Charles E.”, yet he is listed as the informant on Lillian’s death record.

Appendix E.2: Arthur W. Sweeney

Arthur's father, Hugh C. Sweeney developed his own roofing business (H.C. Sweeney Roofing Co.) and was an alderman in Hamilton from 1903-1907 (Crozier, 1992). Arthur was born in 1907, the fourth child and first boy after three girls. Arthur died at age 3 years, 8 months in September 1910, in the midst of Hamilton's first significant polio outbreak. At the time of his death, he and his family were living at 395 Aberdeen Avenue, in Hamilton's Kirkendall/Lock Street neighbourhood (Ward 3; see Figure E.2). Hugh was appointed district immigration inspector in 1911 (Crozier, 1992).



Figure E.2 395 Aberdeen Ave. as it appears in 2012.

The report of Arthur's death made the front page of the *Hamilton Spectator*:

FROM POLIOMYELITIS

Young Son of Ex-Ald. Sweeney Succumbed to the Disease

The sympathy of a very wide circle of friends will be extended to the ex-Ald. Hugh and Mrs. Sweeney in the death, at the family residence, last night, of their four-year-old son, Arthur Winters Sweeney. The child was in his usual health until a few days ago, when he became ill with poliomyelitis, which, in spite of all that could be done for him, proved fatal. The little fellow was a particularly bright boy, and was much beloved by the friends of the family, who will regret to learn of his death. The funeral took place from the home of his parents, 395 Aberdeen avenue, this afternoon, to Holy Sepulcher cemetery.
[19 September, 1910, p. 1]

The entry for Hugh in the Dictionary of Hamilton Biography (Crozier, 1992)

mentions that Hugh had three daughters, and that they outlived him; no mention is made of Arthur.

Appendix E.3: Guy C. Flintoff

Born in 1883 to native-born parents John Flintoff and Rose Ann Lonsdale, Guy was the lastborn of seven living siblings (eight siblings total). Guy grew up in this rural farm family in the Darlington area of Durham (census district 59). Sometime after 1901, he left the Durham area and became a labourer in Toronto.

Guy died at Toronto General Hospital in September 1910 at the age of 27, after an illness of four days with “acute ascending anterior poliomyelitis”. According to Guy’s father’s death record, his father died in 1914, only four years after Guy, at age 79.

Appendix E.4: Barbara Bertram

According to the Dictionary of Hamilton Biography Vol. III 1925-1939 (Filer, 1992), Barbara’s grandfather Henry Bertram was a mayor of Dundas and owned a manufacturing

business, “John Bertram and Sons”, which made tool and farm machinery. His son (also named Henry), Barbara’s father, was also a mayor of Dundas and “an engineer and financier” who eventually was given control of the family company (Filer, 1992).

Barbara’s parents married in 1921 and Barbara was born in 1925, the second of four daughters. She attended public school in Dundas and Sunday school of St. Paul’s United Church (*Hamilton Spectator*, 13 August 1937, p. 10). She was a relative of Dr. Thomas Bertram, the medical officer of health for Dundas (*Hamilton Spectator*, 13 August 1937, p. 10). At the time of her death in August 1937 at age 12, Barbara’s family’s usual residence was at 32 Cross Street in Dundas. However, they had a summer residence at Port Maitland, and it was believed that she contracted the disease there (*Hamilton Spectator*, 12 August 1937, p. 1). She was ill when she came back from Port Maitland and was immediately taken to the hospital (*Hamilton Spectator*, 13 August 1937, p. 10). The *Hamilton Spectator*’s front page carried a report of Barbara’s case as well as another case in the city:

The condition of the young Miss Bertram is given as critical. She has not yet reached the stage where it can be determined to what extent, if any, paralysis will follow.

Both are being kept isolated in special rooms, and are being treated with serum, it was learned from Dr. Walter F. Langrill, general superintendent of the hospital.

[12 August 1937, p. 1]

Barbara died the following day at Hamilton General Hospital of “polio-encephalitis”, with aspiration bronchopneumonia listed as a comorbid condition, and was buried in Dundas’s Grove Cemetery.

Appendix E.5: Florence Isabel Cowlin

Florence was born in England in 1900. She immigrated to Canada with her family in 1907, including at least one sibling, an older brother. Florence's father, Walter H. Cowlin, was a stonemason; he apparently died in 1919. At the time of her death, she lived with her mother at 46 Collier Street at the western edge of the upscale Rosedale neighbourhood of Toronto. Goad's Atlas of the City of Toronto and Suburbs (1924, Vol.1, Plate 33) show 46 Collier St. as a wooden structure in a row of fairly narrow attached houses (Figure E.3). The same structure is shown as far back as the 1890 edition of Goad's Atlas.

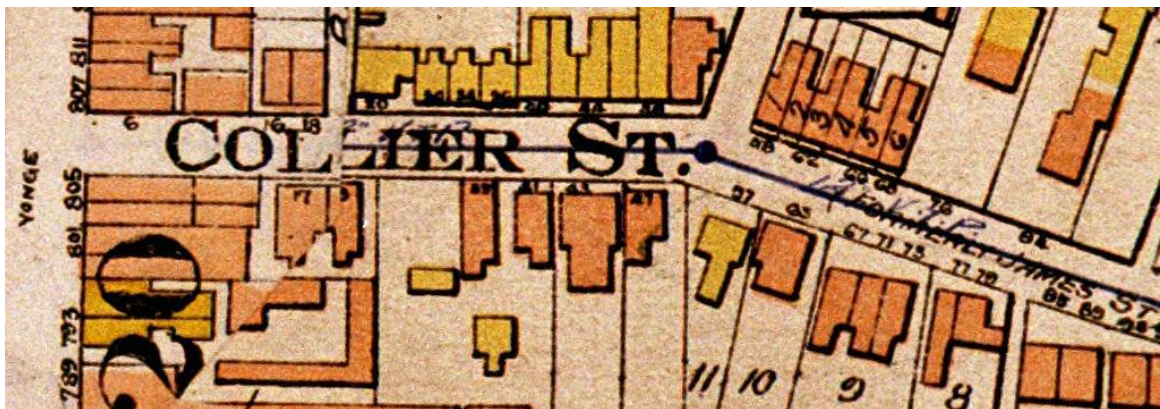


Figure E.3 Section of Collier St., from Atlas of the City of Toronto and Suburbs (Goad, 1924, Vol.1, Plate 33). Source: City of Toronto Archives.

Florence died of “Polio Encephalitis” in December 1921 at Toronto General Hospital at the age of 21 and was buried in Mount Pleasant Cemetery. Her death notice was posted in Toronto's *Globe* newspaper (Figure E.4).

COWLIN—Suddenly, at Toronto, on Sunday, Dec. 4th, 1921, Florence Isabel, dearly beloved daughter of Mrs. Isabel Cowlin, of 46 Collier st., aged 21 years.

Funeral service will be held at the Church of the Redeemer, corner of Bloor and Avenue road, on Thursday, Dec. the 8th, at 2.30 p.m., thence to Mount Pleasant Cemetery.

Figure E.4 Death notice for Isabel Cowlin (*The Globe*, 6 December 1921, p. 12).

Appendix E.6: Mary Packer

Both of Mary's parents were English immigrants, her father, William John Packer, arriving in 1903 and her mother, Eva Knight, in 1906. As of the 1911 Census, they had one son. Mary was born in 1915. The family lived at 196 Bay Street South, in Hamilton's relatively well-off Durand neighbourhood. Mary's father was a hostler in a livery stable. She died at home in January 1916 of "infantile paralysis", less than a month before her first birthday and two months after the initial illness. She was buried in Hamilton Cemetery.

Appendix E.7: Robert Wilfred Roy Loveless

Robert, or Roy as his death notice called him, was born in 1896 to a farm family in Scarboro, the third child and first son. His family would eventually grow to nine children in total, including five younger brothers and one younger sister who died in infancy in 1902. Robert died in February 1923 at age 26 of "infantile paralysis" with "heart failure" as a contributing cause; on his death record his occupation is listed as "cripple from youth", indicating that he had contracted polio at a young age. His death notice appeared in Toronto's *Globe* newspaper (Figure E.5).

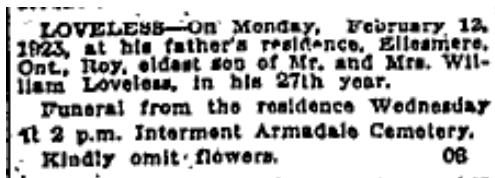


Figure E.5 Death notice of Roy Loveless (*The Globe*, 14 February 1923, p. 12).

Appendix E.8: John E. Bennett

John was born in Port Hope, Ontario around 1914. Little information was found about his life, except that he contracted polio three years before his death. John died at Toronto's Home for Incurable Children in April 1918 at age 4, with poliomyelitis listed as ultimate cause of death and cardiac failure the immediate cause.

Appendix E.9: William Andrew Cork

William, or "Billy", was born in Toronto in 1907, the first child in a family which would later include a total of five children (three younger sisters and one younger brother for William). As of 1911, William and his family lived at 429 Brunswick (Ward 4) in Toronto. His father was a silk merchant. Around 1924, the family moved to 524 St Clair Avenue (Ward 6) in Toronto. William died at home in January 1926 at the age of 18 after an illness of "acute anterior poliomyelitis" lasting two days, according to his death record. At the time of his death, he was a student. The *Toronto Evening Telegram* (2 January 1926, p. 19) reported that his 12-year-old brother Campbell had a case of polio at the same time. He was buried in Mount Pleasant Cemetery.

Appendix E.10: Thomas Francis O'Hara

Thomas was born in 1913. His father was born in Ontario and his mother was from Ireland; they married in 1903 and had at least one child before Thomas. Thomas's father was a farmer and Thomas was working as a labourer in the steel industry.

The report of his death in the newspaper provides details of his final days:

Ancaster Youth Passes Suddenly

Believed to Be Victim of Infantile Paralysis – Widely Mourned

It has been reported that Thomas O'Hara, 20-year-old son of Mr. and Mrs. Thomas O'Hara, Ancaster, died of infantile paralysis last evening, shortly after being admitted to the General Hospital. Young O'Hara, who resided with his parents, Mr. and Mrs. Thomas O'Hara, on the highway about a mile and a half out of Ancaster, first complained of feeling ill on Wednesday, but despite his illness attended a baseball game in Guelph on Wednesday evening. In the absence of Dr. J. H. Roderick, of Ancaster, Dr. Ronald Kettle, of Hamilton, was called to the young man yesterday afternoon, and diagnosed the case as infantile paralysis of a very severe type, and he was immediately sent to the hospital.

The deceased was popular in the community where he lived, and his passing will occasion widespread regret. Besides his parents, a sister, May, and two other brothers, John and Joseph, survive. The funeral will be held privately. [*Hamilton Spectator*, 25 August 1934, p. 8]

Appendix E.11: John McWilliams

John died at Toronto's Home for Incurables in March 1904 at age 17. The only family mentioned in the death notice in the *Toronto Daily News* (21 March 1904, p. 3) is his mother, "the late Lillian McWilliams".

Appendix E.12: Arthur Russell Percy Steeds

Arthur Russell Percy Steeds, known as Russell, was born in Toronto in 1923, four years into his parents' marriage. When Russell was born, his father, an immigrant from England, was employed as a motorman. Russell was a choir member at St. Paul's Anglican Church and attended Pauline public school (*Toronto Daily Star*, 13 August 1937, p. 2). He was a music student and "obtained four honors in his piano work and two first-class honors in musical theory at the Toronto Conservatory of Music" (*Toronto Daily Star*, 13 August 1937, p. 2). Two months before his death at age 13, he was recommended for high school entrance and had plans to enroll at Bloor Collegiate in the fall.

Russell died at Riverdale Isolation Hospital during the major 1937 epidemic in Ontario. He was summering at a farm near Acton, Ontario when he suddenly became ill and was rushed back to Toronto (*Toronto Daily Star*, 13 August 1937, p. 2). At the time of his death, his family's usual residence was at 57 Wallace Avenue in Toronto (Ward 6). His father was then a police constable, and he had one younger brother, Raymond. He was buried at Prospect Cemetery.

Appendix E.13: Margaret Jewell

"Maggie" Jewell was born in 1893 in the Ontario's Victoria County to native-born parents. Her father, Benjamin Jewell, worked as a mechanic. As of 1901, she was still living in the census district of Victoria South with her family, including a younger brother and sister.

Maggie contracted polio in September 1910 at the age of 17. She was sick for three days before her case progressed to paralysis of the diaphragm and intestinal muscles. At the time of her death, she was working as a clerk and living at 16 Elm Street in "The Ward" neighbourhood of Toronto (Ward 2), possibly at the YWCA (Goad's Atlases for 1903 and 1913 show only two large buildings, the YWCA at 18 Elm Street and St. George's Hall, run by a charity in aid of British immigrants, at 14 Elm Street, with no space between them). Her funeral was held in Manilla, Ontario, in the Victoria County area her family was from (*The Globe*, 21 September 1910, p. 8).

Appendix E.14: Lena Puccini

Besides Maggie Jewell, Lena was the only other resident of The Ward neighbourhood to die of polio between 1900 and 1937.

Lena was born in Toronto in 1918 to Italian Catholic immigrant parents. She died at Riverdale Isolation Hospital in September 1937 of “polio-encephalo-myelitis” during the major Ontario polio epidemic. At the time of her death, she was 19 years old, unmarried, and living with her family at 93 Elm Street, on the corner of Elm and Chestnut streets and across from the Poor House (Goad, 1924); her death record listed her occupation as “at home”. She was buried at Mount Hope Cemetery.

Appendix E.15: Lloyd Alexander Burke

Lloyd’s father, John Burke, was an immigrant from Ireland and a Presbyterian. He worked as a stableman and was a widower before he married Lloyd’s mother, Winnifred Jackson, in 1917. Winnifred was an Anglican immigrant from England and worked as a filler. Lloyd was born in Toronto in April of 1921.

Lloyd died at home, 14 Pape Avenue in Toronto (Ward 1), in September 1922 at age 1 year, 5 months. He was buried at Prospect Cemetery.

Appendix E.16: Jessie Graham Duncan

Jessie was born in Toronto in 1916, in the midst of World War I. Her parents were immigrants from England, her father a Presbyterian and her mother a Methodist. Jessie’s father, Private John Duncan, had been a plumber before the war; he died in France the year after Jessie was born.

Jessie died in September 1922 at the Hospital for Sick Children in Toronto, during a polio epidemic, at the age of 6. Her death record lists her cause of death as “Anterior Poliomyelitis of the Bulbar type”. At the time of her death she was an only daughter living with her mother in the Danforth neighbourhood of Toronto at 544 Jones Avenue, a wooden, attached house next to a public school (Goad, 1924, Plate 109). She was buried at St John’s Cemetery, Norway.

Appendix E.17: Lorna Eileen Ferguson

Lorna was born in Toronto in 1913 or 1914 to native-born parents. Her father was a carpenter before the war; he was killed in action at Passchendale in October 1917.

Lorna died in August 1922 at age 8. She died at home at 82 Hastings Avenue in Toronto (Ward 1), in a narrow, detached wooden house (Goad, 1924, Plate 106) which still exists today, where she had been living with her mother and at least one older sibling. She was buried at St John’s Cemetery, Norway.

Appendix E.18: Jeanette Haberman Donnenfield

Jeanette was born in Romania around 1890 to Jewish parents, Wolfe and Clara Haberman. She immigrated to Canada as a teenager, around 1904. She married Joseph Donnenfield in 1906 at age 17 and had seven children, five boys and two girls. She may have been living in the United States at the time of the 1920 United States Census.

Jeanette died at Toronto General Hospital in October 1934 at age 44. She had been a survivor with chronic polio for about a year. At the time of her death she was living at 298 Deloraine Avenue in the Bedford Park area north of the city of Toronto and

her death record listed her occupation as “housewife”. She was buried at Roselawn Cemetery.

Appendix E.19: Joseph Frederick Dunning

Joseph was born in 1906 on a farm (Lot 20, Concession 6) in Ontario’s King Township, to native-born parents who had married in 1904. His father was then a farmer; sometime between 1909 and 1911 the family left the farm and he became a teamster in Aurora. He reported \$1,000 in earnings for 1910. Joseph was the second of four children; he had an older brother (born 1905), younger sister (born 1909), and a baby brother (born May 1911). Joseph’s father’s sister also lived with the family in Aurora, according to the 1911 Census.

Joseph died at his home on Larmont Street in the town of Aurora, Ontario in October 1911 at 5 years of age. His baby brother Arnold, 5 months old, died the same day of “gastric enteritis” and “exhaustion”; perhaps it was coincidence, or perhaps it was contracted from the same contaminated source (e.g. water) which carried the poliovirus. Perhaps one or both cases were misdiagnosed.

Appendix E.20: Frances Elizabeth Furlong

Frances was born in Toronto in 1918. Her parents, John Lawrence Furlong, a solderer (silver worker), and Elizabeth Donohue, a stenographer, both Catholics from Ontario, married in June 1912. Frances was their third child, but apparently the only one to survive beyond infancy. The first, a boy, was stillborn in September 1912. In June 1913, a second child, a girl, died of heart failure due to “congenital pulmonary stenosis” at only 10 days

old. At that time, the family was living in an attached brick house at 1006 Bathurst Street in Toronto (Ward 4) (Goad, 1913, Plate 32).

Frances died in 1922 at the Hospital for Sick Children, after an illness lasting four days. At the time of her death at age 4, she and her parents were living at 125 Bathurst Street in Toronto (Ward 4), across the street from St. Mary's Roman Catholic Church and a school. This was a large brick building (Goad, 1924, Plate 101), constructed in 1869 as a private home for a prominent Toronto merchant, John Mulvey; it was sold to the Roman Catholic Parish of St. Mary in 1909, who added an addition in 1910, and used for multiple purposes (Doors Open Toronto, 2009). Frances's father worked there as a caretaker.

Frances was buried at St. Michael's Cemetery in Toronto, in the Donohue family plot with her siblings and later her mother who committed suicide two years after Frances's death.

Appendix E.21: Tunis Family: Leah and John E. Tunis

The Tunis family was one of West Flamboro's original settler families. Rees Tunis, a United Empire Loyalist, came to the area in 1790 and was given a land grant in West Flamboro (S.S. No. 5 Centennial Committee, 1948). He bought more property in the area, and operated a mill in Ancaster. His two sons (John and William) inherited parts of his property and raised large families there (S.S. No. 5 Centennial Committee, 1948). One of their descendents, John E. Tunis (grandson of Rees's eldest son John), was born in West Flamboro in 1881. John's father Jacob was a farmer; John himself worked as a mail clerk. John E. Tunis died at age 28 at Hamilton's City Hospital in September 1910, during the

large polio outbreak in the Hamilton area that year. He was unmarried. The *Hamilton Spectator* reported on his funeral service:

The funeral of the late John E. Tunis took place from the family residence, Greensville, on Sunday afternoon to the Grove cemetery, Dundas, and was largely attended. The funeral was under Masonic auspices, the members of Valley Lodge, Dundas, and Dufferin Lodge, West Flamboro, of which deceased was a member, turning out in large numbers. The funeral services were conducted by Rev. Mr. Bowers, of Capetown, and Bros. W. J. Stutt and Alfred Jones, of Dufferin Lodge. The following members of the craft acted as pall-bearers: - Wesley Green, Harvey Betzner, Edgar Morden, D. Heslop, W. Surerus, and W. Taylor.
[22 September 1910, p. 2]

During that same outbreak, Leah Tunis contracted polio as well. She was one year old at the time, the daughter of John E.'s younger brother Charles Roy. Charles Roy Tunis was a farmer at the time of his marriage to Ethyl Evelyn Simon in 1905. Their first child, Earl Roy, was born in 1907, and Charles Roy's occupation by that time and for the following several years was listed variously as "delivery man", "clerk", and "driver" for a clothing store. Earl Roy's 1907 birth record gives the family's residence as Lot 2, Concession 2 in Barton, and Leah's 1909 birth record simply lists the family's address as "Bartonville".

Leah survived the acute stage of infantile paralysis, but died six months later in April 1911 after developing broncho pneumonia. By that time, the family was living at 27 Woodbine Crescent in Hamilton's Strathcona neighbourhood (Ward 4). The 1911 Census lists Ethyl's 23-year-old sister Emma as one of the residents of that small house.

Appendix E.22: Adam George Ballantyne

Both of Adam's parents were natives of Ontario. They married in 1897, and had two children, a boy and a girl, before Adam was born in 1911. Adam's father was a barrister

in Toronto. At the time of Adam's birth, the family was living at 118 Walmer Road in the Annex neighbourhood.

In May of 1928, at the age of 63, Adam's father died at Toronto General Hospital after an operation on his gall bladder and appendix. In October of that year, Adam contracted polio and died at home, at the age of 17, after an illness of two weeks. At the time of his death, Adam was a student and the family was living at 34 Dunvegan Road in Toronto's upscale Forest Hill neighbourhood. He was buried at Mount Pleasant Cemetery, where his father had been laid to rest just months before.

Appendix E.23: Muriel Dora King

Muriel was born in England in 1897 to parents. She immigrated to Canada in 1906 with her mother, her father having come to Canada the previous year.

She died in September 1911 at her home at 9 Earl Street in North Toronto of respiratory paralysis due to polio infection. At the time of her death, she was 14 years old and attending school. She had two younger siblings, a brother and a sister, and her father was working as an advertiser in a newspaper office.

Appendix E.24: Bernard Lawrence Roach

Bernard was born in 1931 to Hamilton-born Irish Catholic parents. Bernard's father George worked as a labourer and the family lived in Hamilton's industrial working-class North End neighbourhood. Bernard died in September 1934 at the age of two, just two months shy of his third birthday. He was buried in Hamilton's Holy Sepulchre Cemetery.

Appendix E.25: Joseph William Warren

Joseph's father had been born in New Zealand and his mother in England. His mother immigrated to Canada in 1905, and his father in 1906; they were married in Liverpool, England in 1909. Joseph was born in Toronto the following year.

His family appeared to move relatively frequently. Over the years they had several addresses in the general Toronto area, including 189 Logan Avenue (Ward 1) at the time of Joseph's birth in 1910, 28 Sutton Avenue (Ward 2) in 1911, and 14 Power Street in 1914 (Corktown neighbourhood). His father worked as an insurance agent at the time of the 1911 Census, but on his 1914 Attestation Paper his occupation was listed as dry goods man. At the time of his death from pulmonary tuberculosis in 1932 his occupation was listed as window dresser; according to his death record, they had been living at 230 Ashworth Avenue for the past ten months.

At the time of his death in September 1937 at age 27, Joseph was living with his mother at 207 Lauder Avenue, just a few blocks from Prospect Cemetery, and working as a driver. He died at Toronto General Hospital, during the major polio epidemic that hit Ontario that year. He was buried at Prospect Cemetery.

Appendix E.26: John "Jack" Connell

John or "Jack" was born in West Flamboro in 1905. His father Thomas was a farmer, and both of Jack's parents had themselves been born in West Flamboro. He had three younger siblings, two sisters and a brother.

Jack died at his home in the 2nd concession, West Flamboro (Dundas) in August 1928. He was 22 years old, unmarried, and working as a farmer. He was buried in Dundas's Grove Cemetery.

Appendix E.27: Kenneth Russell Argent

Kenneth was born in Hamilton in 1920. His parents were immigrants from England, and his father worked as a carpenter.

Kenneth died in August 1937 at the age of 16. His official cause of death was listed as “Acute Anterior Poliomyelitis of brain stem and cord”. At the time of his death he was living with his family at 219 George Street in Hamilton’s Strathcona neighbourhood (Figure E.6) and working as a clerk. The *Hamilton Spectator* carried the following death notice:

Second victim of infantile paralysis in Hamilton, Kenneth Russell Argent, who died at the General Hospital Saturday following several days’ illness, was a member of Salvation Army No. 1, the junior band and the Sunday school. He was the eldest son of Thomas Argent, 219 George street, and the late Mrs. Argent. Besides his father he leaves two brothers, David and Lloyd, and one sister, Eileen, all at home. Funeral private to-day from funeral chapel of J. H. Robinson & Co., Ltd. Interment in Hamilton cemetery.
[16 August 1937, p. 22]



Figure E.6 219 George St. as it appears in 2012.

Appendix E.28: Betty Eleanor Henderson

Betty's father, a provincial officer in the Customs House Department, was widowed with three children before he married Betty's mother in 1920. Betty was born the following year. The family lived in the VanWagner's Beach area of Hamilton and Betty attended VanWagner's Beach public school (*Hamilton Spectator*, 25 June 1928, p. 23).

Betty died in 1928 at Hamilton's General Hospital. She was six years old. One of her obituaries states, "left to mourn her loss, besides her parents, are one brother, Gordon, and two sisters, Reta May and Mamie, at home, and her two grandmothers..." (*Hamilton Spectator*, 25 June 1928, p. 23). She was buried in Hamilton Cemetery.

Appendix E.29: Ruby Elizabeth Chrysdale

Ruby was born in Toronto in around January 1914 to native-born parents. Her father's occupation was listed variously as shipper, driver, collector, and teamster. Ruby had six older siblings, though it is unclear how many of those survived infancy and early childhood. They were a Catholic family, with children born every nearly every year between 1906 and 1913. According to the 1911 Census, her maternal grandfather and aunt also lived with them in their home at 210 Richmond Street West in downtown Toronto (Ward 4).

Ruby died at home in December 1915 at the age of 1 year, 11 months. Her cause of death was listed as "Polio Encephalitis".

Appendix E.30: Mark Vangloff

Mark was born in Toronto in 1924, the son of immigrant parents from Macedonia. His father was a fur dresser, and the family lived at 91 Sydenham in downtown Toronto (Ward 2).

Mark died in September 1930 at the Hospital for Sick Children, from bronchopneumonia complicating bulbar polio. He was five years old. Mark was buried in the Necropolis Cemetery.

Appendix E.30: Howard Gladstone Binkley

Howard was born in 1917 to native-born parents. His father, Ewart Gladstone Binkley, was a barrister. At the time of his death he was a student and living with his family at 48

Undermount Avenue in Hamilton's Kirkendall/Locke neighbourhood (Ward 3; see Figure E.7).



Figure E.7 48 Undermount Ave. as it appears in 2012.

Howard died during the province-wide polio epidemic of 1937. As his death notice notes, funerals could not be public due to the epidemic situation:

BINKLEY – At the Hamilton General Hospital, on Sunday, August 22m 1937, Howard Gladstone Binkley, in his 21st year, beloved and only son of Mr. and Mrs. E. G. Binkley, 48 Undermount avenue. Funeral strictly private (of necessity), from chapel of Blachford and Wray, Main street and West avenue, Tuesday afternoon. Internment in Woodland cemetery.
[*Hamilton Spectator*, 23 August 1937, p. 14]

Appendix E.31: John Lynwood Church

John was born in Toronto in 1922. His parents had married the previous year; his mother was born in Toronto, and his father was from London, England and worked as a telephone repairman.

At the age of seven, after an illness of six days, John died at the Hospital for Sick Children on September 22, 1930. He was an only child, and at the time of his death he and his family were living in Toronto's Cabbagetown neighbourhood at 153 Winchester Street – a narrow, semi-detached brick house at the eastern edge by the Don River, next to Riverdale Park and the Necropolis Cemetery (Goad, 1924, Plate 27). John was buried in that cemetery. The *Toronto Daily Star* reported on his death:

INFANTILE PARALYSIS TAKES 2 MORE VICTIMS

Death Toll Here Is Seven, Since Beginning of Epidemic

Two more deaths from infantile paralysis, reported to the M.O.H. to-day, brought the number in Toronto from that disease during the present outbreak to seven.

The latest victims are two boys, one nine months old and the other seven years old, who died in the Hospital for Sick Children. The death rate from the disease in Toronto had been a little more than five per cent of the cases reported. Cases reported this month total 69.

In the case of one death the victim was ready for burial before the department was notified of the case of infantile paralysis. Dr. Pequenat pointed out that this only stresses the importance of parents advising their doctor as soon as a child shows any symptoms of the disease, which are, mainly, sore throat and shoulders, slight nasal discharge, nausea and peevishness.

[23 September 1930, p. 1]

The nine-month-old boy referred to in the article would be Robert Collier, who lived on the other side of Toronto near the intersection of Dufferin and College Streets. However, a few weeks later on October 14, another Cabbagetown boy, eight-year-old Arthur

Martin, who lived at the other end of the block from John at 339 Carlton Street, across from Riverdale Park (Goad, 1924, Plate 28), also died of polio at the Hospital for Sick Children.

Appendix F : Toronto and Hamilton Neighbourhoods and SES

City	Neighbourhood	SES character	Sources for neighbourhood boundaries and SES characteristics
Toronto	Rosedale	Wealthy	Harris (1984); Mercier (2006); Solomon (2007); Hayes (2008); Whitzman (2009)
	Forest Hill	Wealthy	Harris (1984); Solomon (2007); Hayes (2008)
	The Beaches	Middle class	Harris (1984); Solomon (2007)
	Danforth	Lower middle class	Harris (1984); Lindstrom-Best (1985); Mercier (2006)
	West Toronto Junction	Working class	Zucchi (1985); Hayes (2008)
	The Junction	Working class	Harney (1985); Mercier (2006); Solomon (2007)
	Dovercourt	Working class	Mercier (2006)
	Brockton Village	Working class	Mercier (2006); Hayes (2008)
	The Ward	Working class	Speisman (1985); Mercier (2006); Solomon (2007); Hayes (2008)
	Cabbagetown	Mixed	Harris (1984); Kelly (1984); Careless (1985); Mercier (2006); Hayes (2008)
	The Annex	Mixed	Harris (1984); Lemon (1986); Mercier (2006); Solomon (2007); Hayes (2008); Whitzman (2009)
	Parkdale	Mixed	Harris (1984); Mercier (2006); Hayes (2008); Whitzman (2009)
Hamilton	Durand	Wealthy	Manson (2003); Sagaert (2008-2010)
	St Clair	Middle class	Sagaert (2008-2010)
	Corktown	Upper working/lower middle class	Manson (2003); Sagaert (2008-2010)
	Lansdale/Gibson	Working class	Sagaert (2008-2010)
	Keith	Working class	Sagaert (2008-2010)
	North Stipley	Working class	Sagaert (2008-2010)
	North End	Working class	Manson (2006); Sagaert (2008-2010)

Table F.1 List of neighbourhoods in Toronto and Hamilton for which general SES characteristics were determined.

Appendix G : Toronto and Hamilton Neighbourhoods and Polio Deaths

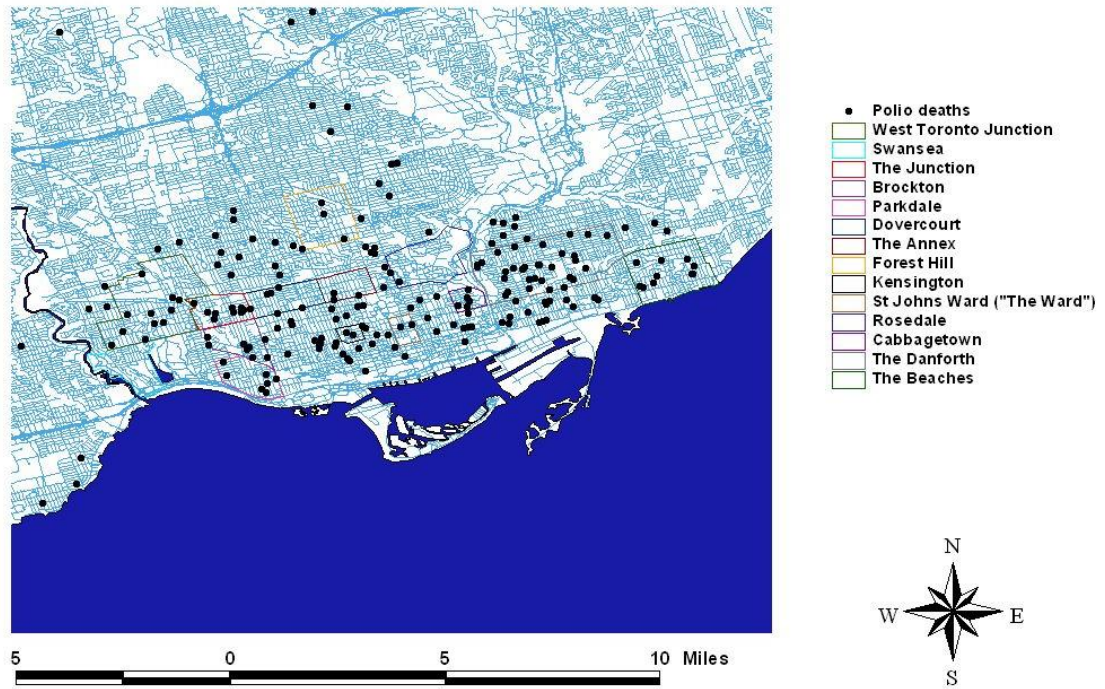


Figure G.1 Map of Toronto neighbourhoods, with plotted residences of individuals who died of polio from 1900-1937.

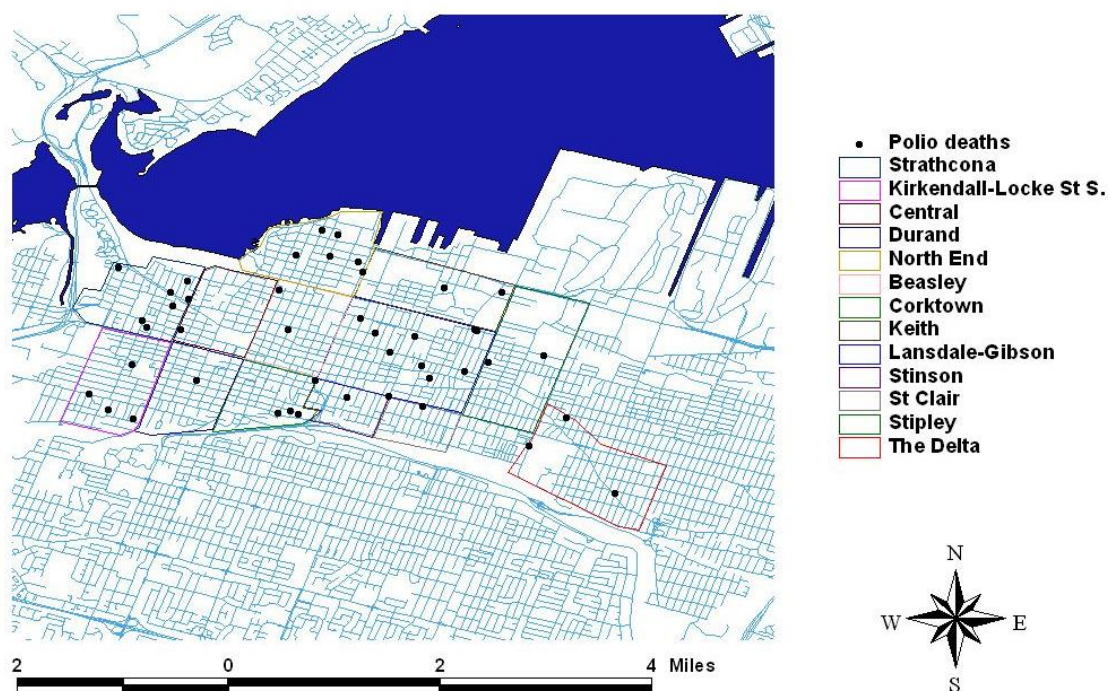


Figure G.2 Map of Hamilton neighbourhoods, with plotted residences of individuals who died of polio from 1900-1937.

Appendix H : Toronto- and Hamilton-Area Polio Deaths

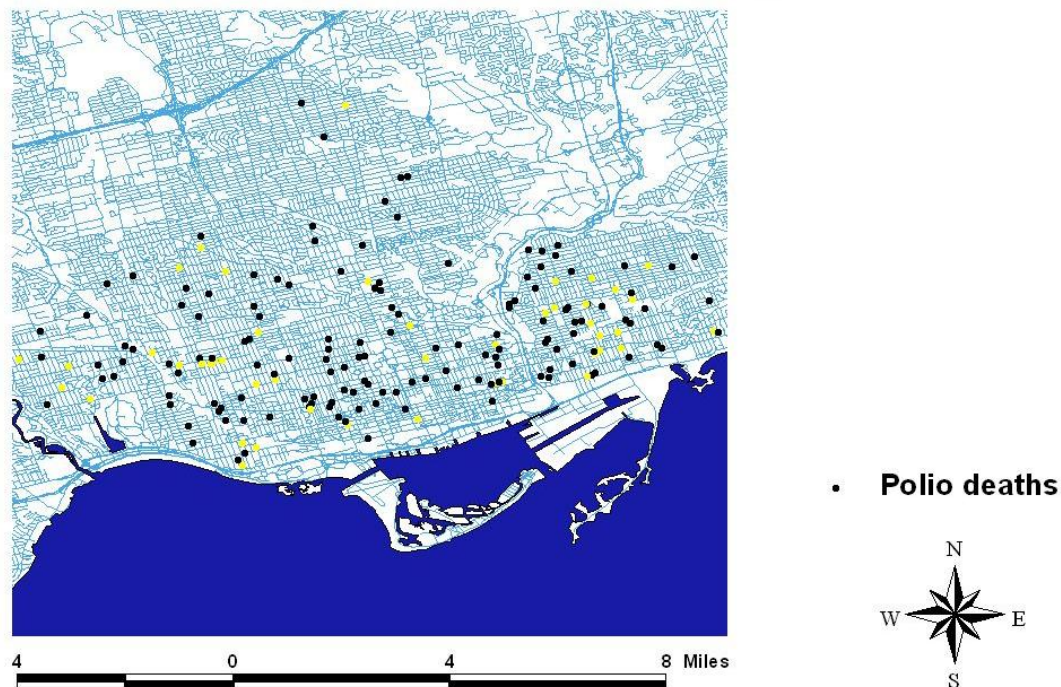


Figure H.1 Map of Toronto-area polio deaths for 1900-1937. Status score 3 deaths are highlighted in yellow. Not all deaths mapped have status scores.

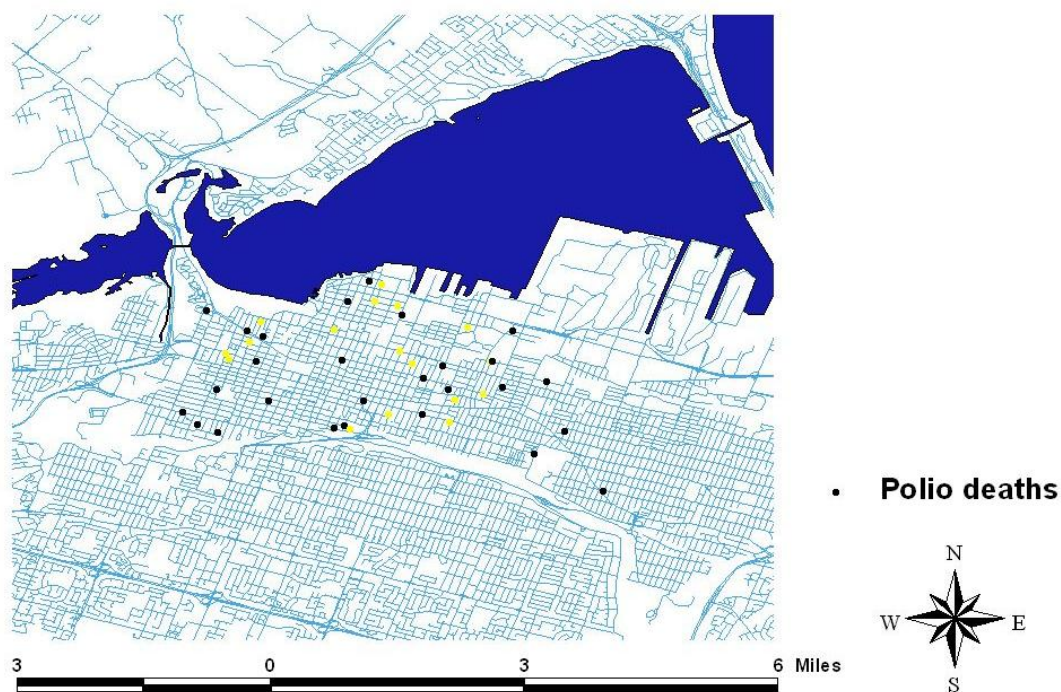


Figure H.2 Map of Hamilton-area polio deaths for 1900-1937. Status score 3 deaths are highlighted in yellow. Not all deaths mapped have status scores.