The Social Geography of Diphtheria Mortality in Hamilton

Heather T. Battles et Bobbie-Leigh Jones

Volume 110, numéro 1, spring 2018
URI : https://id.erudit.org/iderudit/1044327ar
DOI : https://doi.org/10.7202/1044327ar

Résumé de l’article
La ville d’Hamilton en Ontario présente aujourd’hui des fortes disparités socioéconomiques en termes de santé qui se moulent à la division nord/sud de la ville. La diphtérie nous permet d’examiner la nature historique de ces disparités. Des données recueillies de l’enregistrement des décès depuis 1900 jusqu’à 1921 ont permis l’analyse de 321 décès dans ArcMap. Malgré le fait que le taux de mortalité de la diphtérie était plus élevé au nord en général, aucune différence n’a été repérée dans le groupe d’âge de 5 à 16 ans. Dans le nord, le taux de mortalité plus élevé chez les enfants de moins de 5 ans serait alors lié aux facteurs associés avec une faible position socioéconomique. Ces résultats démontrent la dimension géographique de l’inégalité sociale en matière de santé qui existait à Hamilton au début du 20e siècle.

Citer cet article
1. Introduction

Hamilton, Ontario, Canada is a city of strong social geographical contrasts, which can have substantial impacts on health. The discrepancies in health and mortality between the north and south of Hamilton emerged during the initial industrialisation of the city in the nineteenth and early twentieth centuries, as factories and heavy industry became increasingly concentrated in the northern end of the city on the edge of Lake Ontario. The surrounding neighbourhoods became more crowded and housed a population of lower socioeconomic status (SES) than the southern part of the city towards the Niagara Escarpment. The current social geography of health is a topic of research and public discussion in Hamilton today.1 In contrast, according to Rosemary Gagan, in the early twentieth century “no one” in Hamilton commented on the mortality differences between wards.2 Gagan examined mortality patterns in Hamilton for the 1900-14 period, and found that generally mortality was highest in the wards with the lowest standards of living.3 However, data regarding mortality from individual infectious diseases by ward prior to 1910 was largely unavailable at the time of her research.4 Using data from the Ontario Sessional Papers and the Hamilton Spectator newspaper, Gagan was only able to partially reconstruct mortality from contagious diseases by ward for 1901, for example. By using individual death records and identifying the residential locations of these individ-

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3 Ibid., 211.
4 Ibid., 87.
uals, we can now gain a better understanding of the mortality disparities in Hamilton at this time.

Specifically, mortality due to the infectious disease of diphtheria can be used as a lens through which to glimpse the impact of these socioeconomic and geographic disparities. Diphtheria today is known to be linked to poor social conditions such as crowding. However, it has also been viewed in different times and places as more of a socially neutral disease or even a disease that targeted the better-off. Anne Hardy describes how the new diphtheria pandemic manifested in London, England, first in the 1850s-60s as a disease which seemed to target wealthy areas, before becoming more common among those of lower SES. Seen as a ‘socially ubiquitous’ disease in the 1880s, in the 1890s it was observed that while wealthy districts often had high diphtheria mortality rates, the highest were in the poorest districts.

Below, we provide some background information on Hamilton and diphtheria. We then describe the data sources used for this analysis as well as the spatial and statistical methods employed. We present the results of analysis of diphtheria mortality rates for the northern versus southern city wards and of the age


7 Ibid.
patterns in each area, showing that diphtheria mortality rates were higher in the northern wards and that this is likely due to higher mortality in the 0-4 age group in the north. Finally, we discuss possible explanations for these findings, specifically the roles of crowding, economic pressures impacting treatment and recovery, and insult accumulation.

2. Background

2.1 Hamilton, Ontario

The City of Hamilton is located on the south-western shore of Lake Ontario in southern Ontario. It was growing rapidly in the early twentieth century in both population and geographic area. The City’s tax assessment books record a total population of 51,277 in 1900, reaching 118,243 in 1921. This increase was the result of a combination of in-migration, natural increase, and annexations that incorporated surrounding areas into the city. Hamilton’s urban population was largely blue-collar working class, with an increasing number of men employed in the steel and construction industries. It styled itself as “The Ambitious City” and “the Birmingham of Canada.” It was then, and is still today, characterized by strong socioeconomic geographic divisions (residential segregation), primarily between the northern and southern parts of the city. The northern end (wards Four through Eight) bordered the lakefront and was dominated by industry and housing for the industrial workers and their families; the northeast in particular was criss-crossed by rail lines and affected by pollution from industrial activities and sewage. Wards Six and Seven had the highest population densities in the city. The southern part of the city (wards One through Three) bordered the Niagara Escarpment and had larger homes and much lower densities, lowest in wards One and Two.

A number of studies have examined various aspects of Hamilton’s health and disease history in the early twentieth century. As mentioned above, Gagan’s 1981 Master’s thesis provides a microanalysis

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8 For details see John C. Weaver, Hamilton: An Illustrated History (Toronto: James Lorimer & Company, 1982), 199.
10 See for example the promotional booklet Hamilton: The Electric and Natural Gas City of Canada (Hamilton: Times Printing Company, 1906).
12 Gagan, “Disease, mortality, and public health.”
13 Ibid.
of cause-specific mortality for the 1900-14 period in the context of public health efforts. Gagan concluded that mortality worsened in the first decade of the century, before beginning to improve again, and found that working class infants and children were especially impacted by worsening health conditions. In 1900, the highest crude death rates were in the northern wards, at 14.0-15.7 deaths per 1,000 population, and lowest in the southern wards at 11.1-12.2 per 1,000.

2.2 Diphtheria

The pathogen responsible for diphtheria is toxigenic *Corynebacterium diphtheriae*. The bacterium is infected with a virus (*corynebacteriophage*) which carries a *tox* gene, resulting in the production of a toxin. Classical diphtheria, the type investigated here, is spread primarily from person to person through respiratory droplets. Symptoms include sore throat and low fever, though some people can be asymptomatic carriers. In serious cases, the formation of a pseudomembrane can block the respiratory tract and cause suffocation. The toxin can also enter the circulatory system and damage the nervous system, heart, and other organs, with death resulting from myocarditis or renal failure. Death may occur during the initial acute infection or weeks later in the convalescent period. Diphtheria case fatality rates are generally higher for children under age five than other age groups. Dr. John G. Fitzgerald and colleagues at Toronto’s Connaught Laboratories noted that during the pre-vaccine diphtheria era in Canada, children under age five accounted for one quarter of all diphtheria cases but half of diphtheria deaths.

The understanding of diphtheria upon which Ontario’s Board of Health based their decisions in this period is described by Dr. W. Logie in an article in the board’s report for the year 1904. Logie, a physician from Sarnia, Ontario, defined the disease as “an acute infectious and contagious disease with or without pseudo membrane, produced by the presence and development of Klebs...”

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15 Gagan, “Disease, mortality, and public health.”
16 Ibid.; Gagan, “Mortality patterns and public health in Hamilton, Canada.”
17 Gagan, “Disease, mortality, and public health,” Table 14, 87.
Loffler bacillus.” Logie cautioned that since other bacteria can also produce similar inflammations in the throat, microscopic examination was the only real means of positive diagnosis of diphtheria. However, he also cautioned that the presence of the “Diphtheritic Bacillus” did not define diphtheria as a disease in the absence of any clinical illness. Logie described three types of diphtheria, nasal, pharyngeal/tonsillar, and laryngeal, the latter having been otherwise known as ‘membranous croup’ as it frequently resulted in a hoarse, high-pitched cough or stridor as in viral croup. He noted that diphtheria could be complicated by a secondary infection such as pneumonia, or occur as a ‘mixed’ infection along with another disease such as one of those caused by group A streptococcus (e.g. scarlet fever).

The early twentieth century saw the end of a diphtheria pandemic which began in the 1850s. An antitoxin serum was developed in 1894 and was quickly adopted for use in cities around the world, including Hamilton. Public health officials in Hamilton, and in Ontario generally, seem to have been convinced of the curative powers of antitoxin and attributed declines in mortality to its increased use. Logie was especially rapt in his description, stating that it “acts like magic” and that “no remedy has done so much for the human race... except perhaps vaccine.” It was considered a great improvement over previous interventions such as tracheotomy, which Logie called “a last

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22 Ibid.; previous Board of Health annual reports mention specific diseases which diphtheria was often mistaken for, such as strep throat and oral thrush, in Ontario Board of Health Annual Report, 1902 (Toronto, 1903), 165, and laryngitis, ulcerated sore throat, and tonsillitis, in Ontario Board of Health Annual Report, 1901 (Toronto, 1902), 93. These reports similarly used such examples as arguments for the necessity of laboratory testing.
24 Ibid.
25 Ibid.
27 See for example D.A. McClenahan’s comments in Ontario Board of Health Annual Report, 1918 (Toronto, 1919), 14; McClenahan was a District Officer of Health from Hamilton, in charge of the District Three, which included Wentworth County and thus the City of Hamilton. However, physicians at the time, and historians since, have debated whether the advent of antitoxin had any substantial effect on diphtheria mortality rates. Condran argues diphtheria mortality rates were already declining when antitoxin was put into use, but that the antitoxin did help to accelerate that decline; see Gretchen A. Condran, “The elusive role of scientific medicine in mortality decline: diphtheria in nineteenth- and early twentieth-century Philadelphia,” Journal of the History of Medicine and Allied Sciences 63:4 (2008), 484-522. Nelson, “Diphtheria in late-nineteenth-century Sweden,” also argues that antitoxin is the most likely explanation for the decline in case fatality rates observed in Sweden. Hardy, The Epidemic Streets, argues from her study of diphtheria in London, England, that a shift to a milder type of diphtheria was already underway when the antitoxin came into use there.
29 Ibid.
resource.”29 Some risk with the antitoxin was acknowledged, though it was not to the patient; rather, if antitoxin made a child well sooner, there was a risk that they might be prematurely returned to school while still infectious, and thus spread the disease to others.30 A continuing theme in the annual Provincial Board of Health reports is the emphasis on encouraging earlier administration of antitoxin serum, as well as the use of larger doses.31 Physicians were advised not to wait for a laboratory diagnosis, but to use antitoxin immediately in all suspected cases.32 This belief in the critical value of antitoxin contributed to the decision to make it available free of charge to all Ontarians in 1916.33

2.3 The End of Diphtheria in Hamilton
Immunisation against diphtheria began early in Hamilton compared to elsewhere. Toxin-antitoxin (TAT) immunisation campaigns were carried out in the city between January 1922 and 1926.34 Hamilton was also among the sites of the toxoid immunisation trials in 1925, followed by more extensive campaigns beginning in 1926 as the new toxoid vaccine proved effective.35 It is because of these early immunisation campaigns that this study ends in 1921.

Table 1 provides a brief timeline of relevant dates.

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30 Ontario Board of Health Annual Report, 1900 (Toronto, 1901), 8.
31 See for example Chairman E.E. Kitchen’s comments in Ontario Board of Health Annual Report, 1905 (Toronto, 1906), and John W.S. McCullough’s comments in Ontario Board of Health Annual Report, 1918 (Toronto, 1919), 3 that it was still not being used early enough or in large enough doses despite now being free of cost. Roberts gives his own slogan as “Antitoxin early and in large doses” in Ontario Board of Health Annual Report, 1915 (Toronto, 1916), 129.
32 For example, Logic’s comments in Ontario Board of Health Annual Report, 1904 (Toronto, 1905), 99; see also comments in Ontario Board of Health Annual Report, 1915, 72. H.M. Lancaster’s comments regarding the laboratory’s activity in the Ontario Board of Health Annual Report, 1916 (Toronto, 1917), 80 indicate that this advice was being heeded, at least by some.
33 While public health officials credited the free availability of antitoxin with reducing the diphtheria death rate in 1916, they also blamed the war for limiting their ability to reduce it further; the Provincial Board of Health’s Chief Officer noted that they would not be able to launch a full campaign aimed at increasing the use of antitoxin “early enough and in sufficient quantity” until after the war, Ontario Board of Health Annual Report, 1916, 1-2. D.A. McLenahan described the financial limitations imposed by the war but expressed the hope that “with the incoming year peace may once more be restored to the Empire and then we shall be able to pick up the loose threads of public health work…”, Ontario Board of Health Annual Report, 1916, 18.
34 Toxin-antitoxin immunisation involved injecting a mixture of diphtheria toxin and antitoxin to provide protection against diphtheria that lasted longer than the protection offered by injection of antitoxin alone. However, the protection offered by toxin-antitoxin immunisation was still relatively short-term, at up to two years; see Evelynn Maxine Hammonds, Childhood’s Deadly Scourge: The Campaign to Control Diphtheria in New York City, 1880-1930 (Baltimore: The Johns Hopkins University Press, 1999), 176-77.
3. Sources and Methods

3.1 Data sources

Data were collected from death registrations for the City of Hamilton for the period 1900 to 1921. Death registrations for Ontario are considered generally complete from the beginning of the twentieth century. All registrations for city residents whose Cause of Death (COD) information included “diphtheria,” “diphtheritic croup,” “membranous croup,” or “membranous laryngitis” were recorded (n=399). These were transcribed into a Microsoft Excel spreadsheet both directly from Archives of Ontario microfilm (for 1900-04 and 1920-21, in previous research in 2008) and from Archives of Ontario records via Ancestry.ca (for 1905–19) in 2014. We excluded those with insufficient residential address information for mapping in ArcGIS and those whose residences fell outside the city boundaries in the year of their deaths (and thus not covered by the assessment record population counts). We also excluded two individuals who lacked age information. The final total for analysis consisted of 312 individuals.

Assessment records for the City of Hamilton held at the Hamilton Public Library’s Local History and Archives provided annual population data by ward. This included not only total ward population by year but also specifically the population aged 5-16. Analysis was done on total population (all ages) and well as on diphtheria mortality rates for ages 5-16 specifically. In the northern wards, the mean percentage of the population in that age category over the 1900-21 period was 22.2 per cent compared to 19.6 per cent in the south.

The assessment data should be considered with some caution. The city’s population was growing at this time and its physical boundaries were expanding rapidly. Furthermore, there was substan-

<table>
<thead>
<tr>
<th>Year</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>1895</td>
<td>Diphtheria antitoxin becomes available in Ontario for general use</td>
</tr>
<tr>
<td>1907</td>
<td>Ontario Board of Health resolves that antitoxin should be available free of charge for indigent and poor patients</td>
</tr>
<tr>
<td>1914</td>
<td>Antitoxin becomes available from the University of Toronto Antitoxin Laboratory at much lower prices than commercial labs</td>
</tr>
<tr>
<td>1916</td>
<td>Antitoxin becomes available free of charge from Ontario government</td>
</tr>
<tr>
<td>1922</td>
<td>Toxin-antitoxin (TAT) immunisation campaign begins in January</td>
</tr>
<tr>
<td>1925</td>
<td>Toxoid vaccine trials in Hamilton</td>
</tr>
<tr>
<td>1926</td>
<td>Toxoid immunisation campaigns begin</td>
</tr>
</tbody>
</table>

36 Archives of Ontario, Registrations of Deaths, 1900–21, MS 935, reels 95-284 (Toronto, Ontario, Canada).

37 Hamilton Assessment Department, City of Hamilton Assessment Rolls, 1901-22 (Hamilton, Ontario, Canada).
tial residential mobility into, out of, and within the city. Thus, the ward population counts in the assessment records taken at the mid-point of the year might differ from the ward population at another point in the year, especially in years when annexations occurred and ward boundaries shifted. Also, discrepancies between population estimates for Hamilton from different sources have previously been noted, particularly in regards to the effect on calculated mortality rates. The fact that the assessment records provide population counts by ward on an annual basis makes them far preferable to decennial census data for the purposes of this study. Nevertheless, the diphtheria mortality rates calculated here are best considered relative to each other, rather than directly compared to rates from other populations.

3.2 Mapping in ArcMap

Mapping was done using ESRI’s ArcMap geographic information system (GIS) software version 10.3.1. Modern map shapefiles were obtained from the City of Hamilton containing public sector data made available under the City of Hamilton’s Open Data license. Residential addresses of diphtheria deaths were checked for changes in street names, georeferenced, and subsequently plotted in ArcMap. Ward boundaries as of 1910 and 1920 were drawn and saved as separate layers, and used to assign each death to specific ward according to the boundaries for their year of death (see Figures 1 and 2). Plotted deaths were also checked against boundary changes over the period as areas were annexed to the city. For further analysis, wards One through Three were grouped as ‘south’ and wards Four through Eight as ‘north’. Ward Eight deaths were included as of 1910, when assessment records first gave population counts for that area.

Comparison of the mapped versus unmapped diphtheria deaths showed minor differences between the two groups. Addresses which could not be mapped were slightly more likely to be in northern wards, thus their absence potentially biases outcomes in favour of the null hypothesis that there was no difference in diphtheria mortality between the wards.

3.3 Statistical methods

Statistical analyses were conducted in Microsoft Excel and IBM’s SPSS Statistics 22 software. The distributions of the annual diphtheria mortality rates were assessed for normality using visual

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38 See Gagan, “Disease, mortality, and public health,” 113-15. Gagan notes that in calculating mortality rates for Hamilton, Ontario’s Provincial Board of Health used population estimates which were likely too low, thus producing rates which overestimated mortality in the city.


40 Ward boundaries for 1910 were drawn according to the details in Hamilton Public Library, “Wards in 1910” (2011), available on <http://www.myhamilton.ca/myhamilton/LibraryServices/Genealogy/wards+in+1910.hm> (accessed 8 February 2011); ward boundaries for 1920 were drawn according to information in Weaver, *Hamilton*.

41 Changes detailed in Weaver, *Hamilton*. 
Figure 1. Map of Hamilton showing ward boundaries as of 1910. Ward boundaries drawn according to information from Hamilton Public Library, “Wards in 1910” (2011).

Figure 2. Map of Hamilton showing ward boundaries as of 1920. Ward boundaries drawn according to information from John C. Weaver, Hamilton: An Illustrated History (Toronto: James Lorimer & Company, 1982).
assessment of histograms, z-scores of skewness and kurtosis, and Kolmogorov-Smirnov tests. Both the overall distribution of rates and the distribution for the 5-16 years age group were assessed, including north and south separately and collectively. Due to various indications of non-normality, particularly skewness, the non-parametric Mann-Whitney $U$ test was selected for analysis of mortality rates. Fisher’s exact test was used for comparison of frequencies between north and south. Statistical significance was set at $p < .05$. Specific $p$ values are reported to $p = .001$.

### 4. Results

A Mann-Whitney $U$ test indicated that annual diphtheria mortality rates (deaths per 10,000 population) for the 1900-21 period were higher in the northern wards (median = 1.8) versus the southern (median = 1.2), $U = 106$, $Z = -3.19$, $p = .001$, $r = -.48$ (see Table 2 and Figure 3). Epidemic waves track generally across both areas of the city over
the study period, with the exception of 1919-21 (see Figure 4).

The age span of the 312 mapped diphtheria deaths ranged from an infant aged 1 month and 10 days to an older adult aged 65 years. There are very few deaths over the age of 17 years ($n = 13$; see Figure 5). Looking then at the 5-16 years age group (diphtheria deaths $n = 157$), there is no evidence of a difference in the annual diphtheria mortality rates between north and south ($U = 170.5, Z = -1.68, p = .093$) (see Table 3 and Figure 6).

This leaves the 0-4 age group as the probable source of the difference in mortality rates between north and south. It is not possible to test this directly without annual population data on 0-4 year-olds. However, an examination of the proportions of deaths in each age group in north and south provides some evidence (see Figure 7). In the north, 47.3 per cent of diphtheria deaths were under age five, compared to 39.4 per cent in the south, though this difference is not statistically significant according to a Fisher’s exact test ($p = .279$). A Mann-Whitney $U$ test also found no evidence of a difference in overall age at death in the north (median = 5.0 yrs, $n = 241$) versus the south (median = 6.0 yrs, $n = 71$), $U = 7437.5, Z = -1.680, p = .093$. However, one-year-olds in particular appear to stand out with

![Figure 3. Box plot displaying distribution of annual diphtheria mortality rates (deaths per 10,000 population) for Hamilton’s southern and northern wards for the 1900–21 period.](image)
10.8 per cent of deaths ($n = 25$) in the north compared to 2.8 per cent ($n = 2$) in the south, though the difference does not reach statistical significance according to a Fisher’s exact test ($p = .054$).

Finally, there were no significant dif-
ferences in time (year of death) between north ($n = 241$; mean = 1912.1; SD = 7.1; median = 1913) and south ($n = 71$; mean = 1912.5; SD = 6.2; median = 1915), $U = 8513.5$, $Z = -.063$, $p = .95$.

5. Discussion

Higher diphtheria mortality rates in the north of Hamilton can possibly be explained by a combination of crowding, economic pressures impacting treatment and recovery, and insult accumulation.

In terms of the most proximate or direct influences, diphtheria mortality might be increased by crowding both in terms of overall population density by ward and crowding within homes. One study of boarding schools in Russia found that both physical proximity and duration of contact influenced spread of diphtheria infection, with most transmission occurring in dormitories at less than 1 metre distance.42 Crowding might also have contributed to increased severity of diphtheria infections as well as incidence, as has been demonstrated in other childhood infectious diseases, both viral and bacterial.43

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42 L.A. Favorova, “The influence of overcrowding and prolonged contact on transmission of the diphtheria pathogen,” *Journal of Hygiene, Epidemiology, Microbiology, and Immunology* 13 (1969), 73-82.

Table 3. Annual diphtheria mortality rates for ages 5–16 years per 10,000 population aged 5–16 years in Hamilton, Ontario, 1900–21

<table>
<thead>
<tr>
<th>Year</th>
<th>South (Wards 1–3)</th>
<th>North (Wards 4–8)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mortality rate</td>
<td>Mortality rate</td>
</tr>
<tr>
<td></td>
<td>n</td>
<td>n</td>
</tr>
<tr>
<td>1900</td>
<td>0.00</td>
<td>9.91</td>
</tr>
<tr>
<td>1901</td>
<td>4.25</td>
<td>8.05</td>
</tr>
<tr>
<td>1902</td>
<td>6.59</td>
<td>5.25</td>
</tr>
<tr>
<td>1903</td>
<td>2.21</td>
<td>2.04</td>
</tr>
<tr>
<td>1904</td>
<td>4.39</td>
<td>2.04</td>
</tr>
<tr>
<td>1905</td>
<td>6.09</td>
<td>7.18</td>
</tr>
<tr>
<td>1906</td>
<td>4.52</td>
<td>3.22</td>
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<td>1907</td>
<td>0.00</td>
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<tr>
<td>1908</td>
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<td>0.89</td>
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<tr>
<td>1909</td>
<td>3.86</td>
<td>6.81</td>
</tr>
<tr>
<td>1910</td>
<td>5.24</td>
<td>5.56</td>
</tr>
<tr>
<td>1911</td>
<td>4.59</td>
<td>2.69</td>
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<td>1913</td>
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<td>1914</td>
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<td>1917</td>
<td>3.98</td>
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<td>1918</td>
<td>2.58</td>
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<tr>
<td>1919</td>
<td>7.04</td>
<td>1.99</td>
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<tr>
<td>1920</td>
<td>3.02</td>
<td>11.57</td>
</tr>
<tr>
<td>1921</td>
<td>0.00</td>
<td>11.16</td>
</tr>
</tbody>
</table>

Sources: Ontario Death Registrations, Archives of Ontario; City of Hamilton Tax Assessment Records, Hamilton Public Library Local History and Archives.

Health officials were well aware of overcrowding problems in Hamilton. Gagan suggests that between 1905 and 1909, the city was dependent on immigration to maintain its industrial workforce and would not have been keen on the health department making noise about housing and health in the city.\textsuperscript{44} But this changed in 1910, she says, when a new by-law was passed relating to sanitary conditions, which expanded the powers of the city’s Board of Health.\textsuperscript{45} And in fact, in his report for the year 1912, Medical Officer of Health (MOH).

\textsuperscript{44} Gagan, “Disease, mortality, and public health,” 175-86.
\textsuperscript{45} Ibid.

James Roberts stated that in his perception housing conditions had worsened in Hamilton in the past few several years.  

He described an investigation that had found severe overcrowding in the central city, with many families living in rented basements and attics, remarking, “we have evidence that the germs of the slum are here, alive, making vigorous efforts at growth....” As Gagan noted, the highest population densities in the city were found in the north, especially wards Six and Seven.  

The *Hamilton Spectator* newspaper highlighted poor living conditions in the city centre within its pages to elicit donations for its “fresh air outings” fund for local children. Alongside photographs of houses and yards in Hamilton’s central city were captions such as this:  

“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.”  

These problems continued throughout the period. Visiting nurse Mrs. C.H.  

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46 *Ontario Board of Health Annual Report*, 1912 (Toronto, 1913), 449-52.  
47 Ibid. According to Roberts, the Chief Inspector described such basement dwellings as “more suggestive...of a morgue than a dwelling house,” in *Ontario Board of Health Annual Report*, 1912, 450.  
48 Gagan, “Disease, mortality, and public health.”  
49 *Hamilton Spectator*, 6 August 1910.
Jarvis noted in her report to Hamilton’s MOH in 1920, “Houses in Hamilton, as everyone knows, have been scarce and there has been a tendency to overcrowding.”50 Regarding how much Hamilton’s public officials were aware of and acknowledged the socioeconomic and geographic disparities in health in their city, it is useful to look at the data obtained from the 1922 immunization campaign. Toxin-antitoxin immunization work began in January of that year, and focused on a clinic and six schools, mostly in the city’s northern wards.51 Schick tests were conducted to see who lacked immunity.52 The decision to concentrate on schools in the northern part of the city suggests an awareness of where diphtheria rates were highest. This is supported by the following passage by James Roberts in his 1923 report:

It is interesting to note that the children from the poorer districts gave a lower percentage of positive reactions and especially those from the districts where diphtheria is most prevalent. In one school, out of 203 children, only 36 gave a positive reaction. This is no doubt due to the widespread immunity acquired by frequent contact with the disease.53

Economic pressures might also have been at play in increased diphtheria mortality in the north. One way in which this could affect mortality is in how much time the sick individual is able to take to recover. This can potentially be illustrated in the case of one death in 1921, a ten-year-old in the north, the son of Polish immigrants, whose death registration specifically states that he was “let up too soon.” In contrast, Figure 8 is a photo of Archie Dixon taken in March 1909 entitled “Archie convalescent.” It is unclear whether or not the illness he was recovering from was diphtheria; however his sister Marion had died suddenly of diphtheria two months previously. The Dixons lived in the south of Hamilton.

Economic pressures could also impact medical treatment. While for much of this period antitoxin was available free of charge or at low cost (see Table 1) from the provincial government, physicians’ fees and hospital fees could still be significant costs. Cortiula’s examination of social class at Hamilton’s City Hospital (renamed Hamilton General Hospital in 1919) notes that the poor were placed in crowded rooms while those with funds could get private rooms.54 The industrial boom of 1900-13 brought more people to Hamilton and further increased the pressure on the hospital.55 Among the diphtheria...
no indication that hospital deaths were more frequent in the north; 92 of 241 diphtheria deaths, or 38.2 per cent, died in hospital in the north versus 33 of 71 deaths, or 46.4 per cent in the south. Care at home was sometimes possible, with the potential benefit of not being exposed to additional infections in hospital. However, this usually meant that the entire household was placed under quarantine, a severe burden for the wage earner, and increased the risk of contagion within the household. New provincial regulations in 1903 required that diphtheria (and scarlet fever) patients be removed to hospital, yet Hamilton’s MHO at the time, Walter Langrill, noted that this...

56 In 1907, a dispute arose regarding the death of 21-year-old Hamilton resident Clayton Young. There was an accusation that he contracted diphtheria in hospital (after developing erysipelas from being struck with a puck), and thus that hospital staff were in some way at fault. His death registration recorded his cause of death as “erysipelas complicated with diphtheria”; see Archives of Ontario, Registrations of Deaths. An inquest was held, and the jury concluded his death was due to erysipelas alone; see “Clayton Young’s death due to erysipelas,” Globe, 28 March 1907, 8. The case highlights the concerns about contamination between patients with contagious diseases in hospital, and perhaps helps to explain the reluctance and resistance of many parents and guardians to allow their children to be removed to hospital, which frustrated public health officers; see Hamilton Medical Health Officer (MHO) Walter F. Langrill’s comments in Ontario Board of Health Annual Report, 1903 (Toronto, 1904), 36.

57 Gagan, “Mortality patterns and public health in Hamilton,” 170. Gagan mentions that in Hamilton’s 1905-06 scarlet fever outbreak, only 55 of 147 cases could be accommodated in the isolation wing of the hospital, which was an economic burden on families of those who could not be accommodated since they then had to be quarantined for six weeks. Thus James Roberts, who became MHO in 1905, pushed (unsuccessfully) for a separate isolation hospital.
could not always be accomplished. Those of lesser means might also delay calling a physician, as early symptoms might be attributed to a less serious sore throat.

In terms of more indirect or complex contributors to diphtheria mortality risk, insult accumulation could explain higher diphtheria mortality in the northern wards. As Gagan has shown, the northern wards had higher mortality from many causes, indicating a heavy disease burden. Janjua found that for the 1925-35 period, slightly later than that considered here, infant mortality rates for airborne and respiratory diseases as well as food- and water-borne diseases were higher in Hamilton's north, though the rates were then declining throughout the city. Higher diphtheria mortality might thus be a reflection of overall poorer health in the north, rather than indicating that conditions were particularly conducive to diphtheria. Co-infections and super-infections give some direct insight into this, as multiple infections were listed on death registrations. We know that such additional infections, particularly with streptococcus bacteria, contribute substantially to diphtheria morbidity and mortality.

In Hamilton, 7 out of 71 diphtheria deaths in the south (9.9 per cent) had co-infections listed (measles, strep, bronchopneumonia, bronchitis, scarlet fever), compared to 14 of 241 in the north (5.8 per cent) (measles, scarlet fever, mumps, pneumonia, polio, erysipelas, bronchopneumonia, influenza). Interestingly, the individual with the highest number of co-infections (diphtheria, scarlet fever, measles, and bronchopneumonia) was in the south. A Fisher's exact test showed no evidence of a difference in the frequencies of co-infections between north and south ($p = .279$). It is possible that diphtheria patients in the south were more likely to be cared for by private physicians.

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58 According to Langrill, “In a good many instances,... parents have refused to allow me to remove their children to hospital, and I was obliged to leave them in their homes, believing that forcible means and harsh measures would ultimately defeat the attempt to win public opinion....” Ontario Board of Health Annual Report, 1903, 36.


physicians, who might have been more likely to return multiple causes of death.\textsuperscript{64} Therefore, the recorded co-infections on the death registrations might not reflect actual numbers of co-infections in diphtheria deaths, especially for the north. However, as discussed above, there is no direct evidence from this sample that diphtheria deaths in the south were more likely to have been cared for by private physicians rather than in hospital.

One caveat to this study is that it is based on residential geography, not personal SES. Occupation information was only provided in some of the death registrations; since most were children, often this line is left blank, or else occupation was given as “student,” “infant,” etc. However, there are some examples of low SES deaths in the south. The individual mentioned above as having the highest number of co-infections was a three-year-old whose death registration gives “Occupation” as “Father is a labourer.” This child lived in Ward Three, the generally well-off southwest area of the city. Previous work on the cities of Hamilton and nearby Toronto has shown that even where early twentieth-century neighbourhoods could be broadly classified along socioeconomic lines, there was often substantial heterogeneity in the social geography that was revealed in examination of mortality patterns.\textsuperscript{65}

6. Conclusion

This study has found that from 1900 through 1921, the City of Hamilton’s annual diphtheria mortality rates were higher in its northern wards. The evidence presented here suggests that this difference was concentrated among children under five years old, with no evidence of a difference between north and south in mortality rates for the 5-16 year age group. This is supported by the results of the 1922 Schick testing, which found widespread immunity in children of school-age in Hamilton’s “poorer districts.”\textsuperscript{66} These socio-geographic differences in diphtheria mortality persisted despite the decreasing cost of antitoxin. Crowding, a known problem in Hamilton’s northern wards in this time period, provides a likely explanation for some of the higher mortality in the north. Other factors associated with poverty and low SES, such as economic pressures reducing recovery time, delaying treatment, or increasing exposure to additional pathogens, are also likely to have played a role in producing the observed mortality differentials, though the available information makes it difficult to implicate any of them directly.

These results demonstrate the social inequalities in health that existed in early twentieth-century Hamilton and their geographic dimensions. They show that

\begin{itemize}
  \item Condran, “The elusive role of scientific medicine,” 507.
  \item Ontario Board of Health Annual Report, 1923, 364.
\end{itemize}
residents of the city’s northern, lower SES wards were at higher risk of dying from diphtheria, and that this risk was likely concentrated in Hamilton’s youngest residents: children under the age of five. This pattern suggests that those most vulnerable to diphtheria in early-twentieth century Hamilton were found among the large populations of industrial working class immigrants living in crowded areas close to the factories. These families lived disproportionately under economic pressures that led to sick children spending less time resting and recovering, and in conditions that fostered repeated infections and further increased their susceptibility.

Thus, these findings support Gagan’s speculations that “chances for survival” were in fact lower in Hamilton’s northern wards, especially for its infants and children, than in the comparatively well-off south, at least for this particular serious infectious disease. They add to a recent growing literature on this aspect of Hamilton’s health history, which has revealed similar mortality disparities for other infectious diseases. For example, Natalie Ludlow’s investigation of Hamilton’s mortality patterns in the late nineteenth and early twentieth centuries found that industrialization had major consequences for human health, increasing inequalities within the city. Ludlow attributes these disparities in large part to uneven development and infrastructure that did not keep pace with rapid population growth. More broadly, this research expands our understanding of social and economic inequalities in one of Ontario’s major urban, industrial centres by elucidating the consequences of such inequalities for human health and mortality. It also highlights how local and provincial public health officials perceived the problem of the diphtheria mortality in Hamilton and its relationship to social inequalities, and the ways in which they tried to address it—particularly, their focus on the “miracle” of antitoxin treatment.

Future research could utilize SES information on individuals and households, such as that from occupations listed on the death registrations or other linked records, to dig deeper into these patterns of SES and residential geography. This could illuminate some of the more complex patterns of urban social geography and their relationship to health and mortality.

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67 See Janjua, “Infant mortality during the Great Depression in Hamilton”; Herring & Korol, “The north-south divide”; and Battles, “Differences in polio mortality by socioeconomic status.”

68 Ludlow, “Historical population health.”

69 Ibid.