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The Spatial Epidemiology of Pandemic Influenza in Seattle, 1918–1919

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In 1918 and 1919 a major influenza pandemic killed between 20 million and 50 million of the world's population. Its diffusion on an international basis has been discussed previously, but its intraurban characteristics are largely unknown. Within Seattle the outbreak struck young adults most seriously, and mortality was highest among immigrants from certain European and Asian countries. The diffusion of influenza mortality within the city is identified, using geostatistical and centrographic measures.

The impact of disease on civilization and on human history has been largely ignored by historians and other scholars of the past, as noted by William H. McNeill in his landmark synthesis *Plagues and Peoples* (1976). Ackernecht (1965) suggested that "medical geography will not be complete . . . if it is not supplemented by the history of diseases. Only when history and geography are integrated will they reveal a genuinely true picture."

As history progresses, civilizations seem to undergo an epidemiologic transition whereby chronic and degenerative diseases replace infectious diseases as the major causes of morbidity and mortality. Infectious diseases are clearly due to pathogens such as viruses, bacteria, and protozoa; the last are more ambiguous in their origins. They have been linked to habits of living such as diet, and to hereditary factors, although much research is being conducted on the pathogenic bases of chronic diseases, leading one medical geographer (Learmonth, 1978) to label them as "apparently non-infectious diseases."

More historical studies have been conducted to understand the cultural effects of infectious disease pandemics than the effects of chronic diseases. Moreover, more research has been completed on spectacular outbreaks of periodic epidemics that frequently have high attack rates and case fatality rates. Some examples include the bubonic plague in the fourteenth

century (Tuchman, 1978) and the influenza pandemic of 1918–19 (Crosby, 1976).

The worldwide outbreaks of bubonic plague in the fourteenth century and influenza in 1918 are probably the worst in recorded history. Bubonic plague killed a greater percentage of the world's population, but the influenza pandemic was responsible for a greater number of deaths. Precise estimates of the number of deaths due to influenza in 1918–19 are unavailable because of variability among countries in accuracy of records and diagnosis, and unavailability of medical care. In countries like the United States and England, approximately 0.5 percent of the population died in 1918–19 from influenza; in some countries, and in isolated communities such as islands, mortality was much higher (Beveridge, 1977). Somewhere between 20 million and 50 million deaths worldwide during this period are attributable directly to influenza. In the United States alone, about 500,000 deaths were due to influenza or its complications (Katz, 1974).

Like many infectious diseases, the influenza pandemic of 1918–19 exhibited regular spatial characteristics at a variety of scales. Its diffusion at the international scale, and at national scales within the United States and in other countries has been studied (Crosby, 1976), yet little is known about patterns of spread of this serious pandemic at more local scales.

In addition to the diffusion patterns manifested by the pandemic, another characteristic of geographical interest is that influenza had disparate attack rates among different migrant groups and social groups. Although many hypotheses have been developed, the explanation of this epidemiologic characteristic remains enigmatic.

A final theme of geographical interest relevant to this outbreak is more interpretive and conceptual. To try to understand how people perceived and reacted to the pandemic while it was an immediate threat, it is useful to consider the pandemic as a natural hazard because natural-hazards research has encapsulated so well the relationships between sudden, unexpected natural events and individual and institutional responses. Although epidemic disease has not been considered explicitly as a natural hazard, it is, in fact, a natural event that exhibits many of the characteristics of natural hazards.

All of these themes are applicable to the patterns of influenza in particular places. Based on death records maintained by King County, Washington, a medical geographic analysis and interpretation may be developed for Seattle. Such an analysis may suggest how influenza diffused at the intraurban scale, and provides an interesting example of how some immigrant groups in the United States were at higher risk of succumbing

to the disease. Some prefatory general comments on the nature of influenza are useful, as is an understanding of the historical context of this pandemic.

Characteristics of Influenza

The literature on the epidemiologic and virologic characteristics of influenza is extensive and need not be reviewed here except in the most general terms. For a grasp of the spatial aspects of influenza, several factors are relevant.

Influenza is an acute respiratory viral disease, characterized by fever, malaise, cough, sore throat, and other respiratory symptoms. It may range from asymptomatic to prostrating in severity. Influenza is usually of brief duration in an individual patient, with symptoms rarely lasting more than ten days. Diagnosis is mostly on the basis of symptoms, although serologic methods developed at midcentury are used to identify new strains. Treatment is symptomatic and nonspecific, although new drug protocols are under experimentation to lessen the course and severity of one type of influenza. Influenza vaccines may provide immunity for high-risk groups such as the infirm and elderly (Kilbourne, 1979; Davenport, 1976). Several characteristics of the virus and its epidemiology are particularly germane, and have been reviewed in greater detail elsewhere (Davenport, 1976; Mulder and Hers, 1972; Kilbourne, 1975; Pyle, 1980).

There are several types of influenza virus that belong to the myxovirus group. Influenza epidemics, which are localized outbreaks of the disease, and pandemics, which are worldwide outbreaks, occur periodically and have occurred historically. Outbreaks associated with high mortality and disability are caused by an Influenza A.

Mortality in connection with influenza is due to complications of influenza rather than to influenza itself. The complications are typically respiratory, and include viral and bacterial pneumonia, principally in the elderly or in patients with chronic respiratory or cardiac disease. Infrequent complications such as encephalitis are also found.

The periodicity of influenza epidemics and pandemics is striking, and is responsible for the equally periodic pattern of influenza diffusion. The periodicity of the disease is explained by the fact that people are immune only to those strains of the virus to which they have been exposed previously. The influenza viruses undergo periodic *antigenic drift,* whereby the surface antigens of the virus to which the human immune system reacts undergo minor changes, and *antigenic shift,* whereby the surface antigens undergo major changes. When the virus changes radically, as

in genetic shift, people lack immunity to it and are susceptible to disease. Because genetic shifts occur every ten to fifteen years—for whatever reasons—the result is generally a pandemic, with associated high attack rates and rapid diffusion in populations that lack antibodies to the new viral strain. The influenza virus also experiences recycling: earlier strains of the virus may reappear. Indeed, this was resonsible for the swine flu scare in the United States in 1976. The virus that was isolated from an epidemic confined to Fort Dix, New Jersey, was thought to have been the same virus that had been resonsible for the 1918–19 pandemic. Because the pandemic had been associated with such high mortality, there was tremendous concern in the public health community that a similar pandemic could occur in 1976 or 1977.

The incubation period of influenza is very short—usually a matter of days—and the virus reproduces in the respiratory tract and is readily spread by droplet. These factors account for the great rapidity with which influenza may spread through schools, communities, and families once a new strain appears. And once a new strain appears, "seeding" may occur before an actual epidemic, and a herald wave of cases may appear months before an epidemic is recognized as such. Herald waves have been identified in contemporary outbreaks (Glezen, Couch, and Six, 1982), and are also apparent retrospectively in the 1918–19 pandemic.

It is difficult to ascertain when influenza first emerged as a recognizable disease because of the absence of definitive methods of diagnosis and the absence of accurate historical records. Hirsch (1883) lists 299 influenza epidemics between 1173 and 1875, based on historical descriptions of outbreaks of disease with symptoms consistent with those of influenza. Discussions of similar outbreaks date as early as Hippocrates. There have been approximately 30 pandemics since 1580 (Davenport, 1976), and records suggest that epidemics and pandemics became more frequent in the nineteenth century, although this may be an artifact of better documentation. The term *influenza* dates from the early fifteenth century, where it was used to describe an epidemic in Italy thought to be due to the influence of the stars. According to Kaplan and Webster (1977), the term was introduced into the English language in the eighteenth century, when the French also coined the term *la grippe* for the same disease.

Pandemics prior to 1918 are described elsewhere (Beveridge, 1977), and much of the available documentation alludes to probable paths of diffusion. The 1918 pandemic, however, is probably the most noted influenza outbreak because of its high mortality.

The 1918–1919 Pandemic

The global history of the 1918–19 pandemic is described by Crosby (1976), and was also analyzed epidemiologically in the United States (Jordan, 1927). Herald waves in the spring of 1918 were recorded in many locations but were not recognized as such at the time. Only retrospectively did they seem related to the major crippling outbreaks of the fall of 1918 and winter of 1919. Until the subsequent outbreaks, deaths during the herald waves were thought to be an excess of pneumonia deaths that were exceptional and unrelated to influenza.

The pandemic occurred under conditions that were very conducive to rapid spread. It was contemporaneous with the height of World War I, and there were major troop concentrations living under crowded conditions at many places in the United States and Europe. Troops were very mobile: males were being conscripted and moved from their homes to military training stations, temporary bases, and the European battlefields, and some were returning. This was particularly significant because of the age distribution of soldiers. The 1918–19 pandemic was somewhat atypical in that young adults roughly between the ages of twenty and forty, the cohort predominant in the armed forces, were at greatest risk.

There is a great deal of controversy over the geographic origin of the pandemic. Crosby (1976) notes the existence of vague retrospective accounts of outbreaks in the spring of 1918 in China, India, and France, and in early March in the United States (Jordan, 1927; Le Count, 1919). He suggests that the only accurate accounts of a herald wave are those in regard to the United States. He also suggests that the disease spread to Europe along with the American Expeditionary Force, because of documented cases on U.S. troop ships en route to Europe in March and in a U.S. military camp near Bordeaux in mid-April.

Some scholars suggest that a herald wave may have occurred among British troops in France during the winter of 1916–17. Jordan (1927), noting the presence of "purulent bronchitis" during this period, observed nonetheless that "the 1918 pandemic of influenza resembles all previous pandemics of this disease in that its origin is largely shrouded in obscurity." There was a major wave of influenza in Spain in the spring of 1918, which may account for this pandemic's being named the "Spanish Flu" (Jordan 1927). Crosby (1976, 26) argues that it was called the Spanish Flu not because it originated in Spain but probably because Spain, a nonbelligerent, could not hide its health problems behind wartime censorship. The *Journal of the American Medical Association* noted, "The

whole of Spain was invaded by a disease sudden in its appearance, brief in its course and subsiding without leaving a trace." Jordan asserts that the first clear evidence of a herald wave was among U.S., British, and French troops and civilians in France in the first week of April 1918 (Pehu and Ledoux, 1918). The first wave reached Portsmouth, England, in June, and outbreaks in Japan and China were also noted (Crosby, 1976; Shope, 1958). Crosby (1976) believes that the United States was relatively unaffected by the spring herald wave, although ships with infected sailors and passengers were reaching U.S. ports.

The exact origins of the herald wave are, therefore, ambiguous. An interesting methodological question that has yet to be discussed formally is techniques for identifying the origin of a diffusing epidemic. In this case, the only method available is to identify existing accounts and records that are consistent with an outbreak of influenza; a method limited inherently by the nature and quality of documentation. Some of the best documentation leads to the notion that the herald wave first appeared in the United States. Other accounts deal with an outbreak of respiratory disease in February 1918 (Phipson, 1923); China as the origin of the diffusing epidemic (Lee, 1919); and localized outbreaks in Japan (Inada, 1919). However,

> it is thus plain that in widely separated parts of the world, in India, China, and Japan, in France . . . , in Germany . . . and in the military camps of Great Britain and the United States, more or less extensive outbreaks of acute respiratory disease often definitely identified by skilled clinical observers as influenza, had been occurring during the months that immediately preceded the onset of the definite first wave in April, 1918. The primary origin of the 1918 pandemic cannot be traced with any degree of plausibility to any one of these localized outbreaks [Jordan, 1927, 75–76].

Following the appearance herald wave in the spring of 1918, it diffused throughout much of the world, as discussed in more detail by Jordan (1927) and Crosby (1976). Some areas seemed spared from the herald wave, including South America, the West Indies, Canada, Iceland, and many island communities. Once the wave diffused from France, it followed established lines of communication and transportation. The epidemic waned in most places by July and August, although in some places the herald wave never waned completely.

The herald wave was of minor magnitude compared to the wave that appeared in late August and seemed to strike simultaneously in many locations (Shope, 1958). Major outbreaks occurred in the same week in Brest, France; Freetown, Sierra Leone; and Boston, Massachusetts (Crosby, 1976); thousands of people became ill and many died within a few weeks.

Many of the deaths and much of the illness were confined initially to the military but spread to the civilian population.

Although a detailed analysis of the spread of the second wave throughout the United States is beyond the scope of this study, there are some general identifiable characteristics. The wave almost certainly originated in Boston at a Navy pier. Crosby (1976, 45) cites 31 August as the first day the epidemic was noted in the city; Jordan (1927, 117), 28 August. Again, there are reports of isolated outbreaks elsewhere that predated August 28 and their role in the spread of the second wave remains uncertain.

From Boston, the epidemic diffused along lines of military travel and communication, remaining within the military, leading Crosby (1976, 57) to state that "in the pandemic's first weeks it was largely a Naval affair." A group of infected sailors arrived from Boston at the Philadelphia Naval Yard on 7 September, and an outbreak was noted there on 11 September. The Great Lakes Naval Training Station near Chicago had an outbreak that week: 8 September, according to Jordan (1927, 118), or 11 September according to Crosby (1976, 57). A week later 2,600 sailors were hospitalized there with influenza. From the training station, the epidemic apparently infected many civilians in Chicago, and then it spread southward and westward along major rail routes (Jordan, 1927, 118; Crosby 1976, 57).

The wave crested among army personnel about two weeks after it crested among naval personnel. It crested in civilian populations about two weeks thereafter. This conclusion is based upon reports in the news media, and on local health department reports. Influenza did not become a "reportable" disease until after there was widespread recognition of a major epidemic in progress.

There were some apparent anomalies in the spread of this second wave (Crosby, 1976, 64). Although the general pattern of spread was to the South and West, it peaked in Philadelphia and Pittsburgh at different times even though it reached them at the same time. It reached Seattle before it reached San Francisco. And it reached both Seattle and San Francisco at least two weeks before it reached Pittsburgh (Shope, 1958).

To generalize, the second wave and third wave, which developed in December 1918, seemed to follow a combined hierarchical and contagious pattern. Because of the central role of the military in the spread of influenza, the hierarchical pattern reflected military rail movements more than civilian. The disease spread along lines of military interaction in the United States, and in many cases civilian outbreaks came in cities near military bases. There then followed a distance-based pattern of contagious diffusion.

The epidemic reached Seattle roughly a month after the initial outbreak in Boston. Seattle was therefore somewhat of a laggard area in terms of the diffusion patterns. Although one might expect that the city's public health authorities might have taken some steps to control the spread of influenza, this was not the case. Indeed, there was substantial denial of the possibility of influenza's becoming a serious public health problem in Seattle.

The Seattle Context

Seattle was a relatively late-developing city in the history of U.S. urbanism. It remained a "frontier outpost" between 1850, when white men staked a claim on land that is now part of Seattle, and 1880, when its population reached 3,533 (Schmid, 1944; Sale, 1976). The city was given a charter by the Washington Territorial Legislature in 1869, when it had a population 1,107. The city's economic base was almost exclusively lumber.

Seattle experienced significant growth beginning around 1880. It served as the western terminus of the Northern Pacific Railway, which reached it in 1884, and of the Great Northern Railway, which reached it in 1896, operations that opened more markets for local lumber. The development of Alaska was also very significant for Seattle. Its population was 42,837 in 1890; 80,671 in 1900; 237,174 in 1910; and 315,312 in 1920. Growth in the first decade of the twentieth century was remarkable (Sale, 1976).

Seattle had a substantial number of foreign-born residents in the late nineteenth century and early twentieth century; the percentage peaked in 1890 at 30.7. The peak in absolute numbers occurred in 1920 (Schmid, 1944): 80,976 foreign-born were enumerated by the census (Bureau of census, 1922); in 1910 the number was approximately 68,000.

A detailed historic and geographic analysis of Seattle's foreign-born population is outside the scope of this research, but it is important to note major countries of origin at the time of the 1918–19 pandemic because earlier researchers have commented upon differential mortality rates in that regard (see table 1). Canadians were the largest group, followed by Swedes, Norwegians, English, Japanese, Russians, and Lithuanians, in that order.

Influenza in Seattle

Almost no mention is made of the influenza pandemic in Seattle in standard histories of the city. Thus, there is little information available about the outbreak in any secondary sources. One of the finest histories

TABLE 5.1
Foreign-Born Population of Seattle, 1920

Country of Birth	Number	Percent of Foreign-Born	Percent of Total
Canada	13,887	17.1	4.4
Sweden	10,253	12.7	3.3
Norway	9,119	11.3	2.9
England	7,807	9.6	2.5
Japan	6,016	7.4	1.9
Germany	4,827	6.0	1.5
Russia & Lithuania	3,504	4.3	1.1
Ireland	3,455	4.3	1.1
Scotland	3,195	3.9	1.0
Italy	3,095	3.8	1.0
Finland	2,256	2.8	0.7
Denmark	2,228	2.8	0.7
Austria	1,412	1.7	0.5
Greece	1,400	1.7	0.4
China	921	1.1	0.2
Poland	881	1.1	0.3
Other	6,721	8.3	2.1

Source: **U.S. Bureau of the Census, *Fourteenth Census of the U.S.*, vol. 2, *Population 1920: General Report and Analytical Tables* (Washington, D.C.: Government Printing Office, 1922).**

of Seattle says only, "In the fall of 1918 Seattle and the country were hit by an influenza epidemic" (Sale, 1976, 126).

The first mention of the pandemic in the *Seattle Post-Intelligencer,* one of the city's major newspapers, was on 2 August 1918, when it carried an item about an isolated outbreak at a military camp on the East Coast. Two days later it printed: "The epidemic has been thoroughly overcome, and the new cases are among men who are not accustomed to the cool nights" *Seattle Post-Intelligencer,* 1918a, 9). This was well before the major outbreak of late August in Boston. Throughout September the paper reported on the disease and related deaths on the East Coast, but there was no explicit acknowledgment until the end of the month of the possibility of its eventually reaching Seattle.

The first mention of influenza in the Northwest came on 25 September, when a hundred cases were reported at Fort Lewis, an army base south of Seattle (*Seattle Post-Intelligencer,* 1918b, 2). Still, a public health official

denied the presence of Spanish influenza at Fort Lewis. Two days later a headline in the *Post-Intelligencer* (1918c, 9) read: "Pandemic Delays Next Draft Call and Levies Death." On 4 October a *Post-Intelligencer* (1918d, 1) story began, "One death has occurred from Spanish influenza at the Naval Training Station, University of Washington, and 700 cases, 400 of them in hospitals, are reported." Influenza had arrived in Seattle just a month or so after its appearance in Boston.

The only statistical information by which to analyze the patterns of the pandemic in Seattle are the death records of King County. At the time, there was nothing like contemporary disease surveillance systems such as the Centers for Disease Control. It is impossible to study morbidity from the pandemic, not only because there was no formal surveillance system but also because such a system would have been of limited accuracy. Visits to physicians were less common than today, and hospitals were not the usual places of death (Shryock, 1979). Accordingly, the only available indicators of disease frequency are mortality statistics.

All death certificates for Seattle from 1 January 1917 until 31 December 1919 were examined. The certificates included information on the deceased's residence, date of birth, place of death, diagnosis, and other relevant data. When residence was missing, it could usually be ascertained from city directories for 1918 and 1919. Nearly all certificates were complete, however, and missing data were not a major consideration. Data from 1917 were included to serve as a pre-epidemic control against which data from 1918 and 1919 could be compared.

A total of 1,313 deaths listing influenza as the primary or secondary cause were recorded. To derive the influenza-specific mortality rate, it was first necessary to estimate the population of Seattle. This was done by interpolation, given the census-derived population in 1910 (Bureau of the Census, 1914) and 1920. It was necessary to consider influenza mortality in the population figures because influenza accounted for excess mortality during the period of the epidemic. A simple model was developed to be used in the estimation of total population and age-specific population. The same method was used to estimate the number of migrants from specific countries. The first day of January 1919, which was approximately halfway through the epidemic, was taken as the prevalence day.

The number of recorded deaths due to influenza was added to the 1920 population for a total representing an estimate of what the 1920 population would have been had the epidemic not occurred. Assuming a linear pattern of growth, the beginning population had the epidemic not occurred could be estimated by interpolation. Because the epidemic was approximately half over on 1 January 1919, half of the number of deaths in each age or migrant group could be subtracted from that

estimate to yield an estimate of the actual population on that date. The applicable equation for a sample group appears below:

$$P_{i,\,1919} = \left[\frac{\left(\left(P_{i,\,1920} + d_i \right) - P_{i,\,1910} \right)}{120} \cdot 108 \right] \frac{-d_i}{2} \qquad (1)$$

where

P^i1919 = estimated population of group i, 1 January 1919;
$P_{ij}1920$ = census estimate of population of group i; and
d_i = deaths in group i due to influenza.

In (1) the denominator 120 denotes the number of months in the decade 1910–19 to estimate population increases per month. The resulting rate is multiplied by 108 to estimate growth over the nine years (108 months) until 1 January 1919. From that figure, half of the number of influenza deaths are subtracted to estimate population in the midst of the outbreak.

According to the computations, the estimated population was 308,025. With 1,313 deaths due to influenza, the influenza mortality was 426 per 100,000. This is technically not a mortality rate, for rates are expressed in numbers per year; because the epidemic did not span a calendar year, 426 per 100,000 is more appropriately termed a *mortality ratio.* The derived ratio is consistent with mortality for the United States as a whole (Crosby, 1976). Specific locations such as Framingham, Massachusetts (Armstrong, 1919), Newark, New Jersey (Galishoff, 1969), and Nashville, Tennessee (Thomison, 1978) had comparable mortality ratios.

Temporal Patterns of Influenza Mortality

The patterns of influenza deaths in Seattle, by week, demonstrate a period of low mortality in 1917, followed by a gradual increase during the first forty weeks of 1918. During the week of 30 September to 6 October a notable increase in deaths due to influenza took place. Whereas fifteen influenza deaths had been registered during the first thirty-nine weeks, seventeen were registered during the next week alone. The increase was minor, however, compared with the rapid increases during the following three weeks. The peak of the epidemic was during the week of 21–27 October: 119 deaths. The next four weeks the numbers diminished gradually to 30, but during the week of 9–15 December a secondary peak of 114 occurred, probably because of another wave of influenza. The epidemic waned slowly through 1919; its curve is presented in table 2.

TABLE 5.2
Influenza Deaths in Seattle by Week, 1918–1919

Week	Dates	Number of Deaths	Percentage	Cumulative Percentage
1–39	1/1/18–9/29	15	1.1	1.8
40	9/30–10/6	17	1.3	3.1
41	10/7–10/13	63	4.7	7.8
42	10/14–10/20	117	8.8	16.6
43	10/21–10/27	119	8.9	25.5
44	10/28–11/3	88	6.6	32.1
45	11/4–11/10	78	5.9	38.0
46	11/11–11/17	48	3.6	41.6
47	11/18–11/24	30	2.3	43.8
48	11/15–12/1	51	3.8	47.6
49	12/2–12/8	82	6.2	53.8
50	12/9–12/15	114	8.6	62.3
51	12/16–12/22	75	5.6	68.0
52	12/23–12/29	63	4.7	72.7
53	12/30–1/5/19	46	3.5	76.1
54	1/5–1/12	58	4.4	80.5
55	1/13–1/19	53	4.0	84.5
56	1/20–1/26	37	2.8	87.2
57	1/27–2/2	27	2.0	89.3
58	2/3–2/9	23	1.7	91.0
59	2/10–12/31	120	9.0	199.0

By aggregating weeks corresponding to stages in the epidemic curve, one clarifies further the temporal patterns of the epidemic (table 3). Deaths were clearly most numerous between 14–27 October and 28 October–24 November.

Age-Specific Patterns

One of the features of the 1918–19 epidemic that has been noted in various countries and cities was the tendency for the epidemic to have the greatest mortality among persons aged twenty to forty rather than in younger and older age groups. Seattle was no exception to this pattern, as indicated in table 4; populations in each age group were estimated by the procedure described previously.

There are several notable patterns in the age distribution of influenza deaths. The age-specific death rates are much lower in the forty- and-over age groups than in younger age groups. This is different from most

TABLE 5.3
Influenza Deaths in Seattle by Aggregated Weeks, 1918–1919

Weeks	Number of Deaths	Percentage	Cumulative Percentage
1/1/18–9/29	15	1.1	1.8
9/30–10/6	17	1.3	3.1
10/7–10/13	63	4.7	7.8
10/14–10/27	236	17.7	25.5
10/28–11/24	244	18.3	43.8
11/25–12/8	133	10.0	53.8
12/9–12/22	189	14.2	68.0
12/23–2/9/19	307	23.0	91.0
2/10–12/31	120	9.0	100.0

TABLE 5.4
Age-Specific Influenza Death Rates in Seattle, 1918–1919

Age	Number of Deaths	Estimated population[a]	Rate/100,000
1	24	4,793	501
1–9	84	18,739	448
10–19	103	40,981	251
20–29	438	61,086	717
30–39	431	65,209	661
40–49	126	45,146	279
50–59	69	19,508	354
60–	38	19,326	197
TOTAL	1,313	274,788	478

[a]1 January 1919.

other influenza pandemics and epidemics but, as noted, consistent with the pattern of influenza deaths elsewhere during the 1918–19 pandemic (Crosby, 1976). If the population is dichotomized—under forty years old and over forty—the differences are extremely pronounced. The under-forty age group accounted for 69 percent of the city's population, and 82 percent of the influenza deaths. Peak mortality rates were among those aged twenty to twenty-nine, followed closely by peak rates among these aged thirty to thirty-nine. Over 66 percent of all deaths were in

the former age group, indicating even greater concentration in this group than has been noted elsewhere (Katz, 1974). Mortality was comparatively uncommon among the elderly; the differences between the groups are statistically significant: ($x^2 = 149.5$; $df = 7$; $p < .001$).

Several hypotheses have been advanced to explain the age distribution of deaths, notably high among age groups that usually demonstrate relatively low mortality rates from influenza. Winslow and Rogers (1920) suggest that those over forty had developed immunity to the strain of influenza prevalent in 1918–19, for the pandemic of 1889–92 may have been caused by the same strain as the 1918–19 pandemic. The problem with this hypothesis is that immunity, then, should also have extended to people thirty to forty years old, all of whom were alive during the earlier pandemic. Yet it was this group that had the second highest mortality rates in 1918–19. The hypothesis must be rejected.

Another hypothesis is that the pandemic struck mostly military personnel before spreading to the civilian population. Members of the military, therefore, had longer exposures to the influenza virus, and because the military comprised principally the younger age groups, such groups had higher mortality rates. The problem with this hypothesis is that the military consisted mostly of males and the attack rate was at least as high among females (Katz, 1974). The military may have been responsible for the initially rapid diffusion and the specific diffusion patterns during the early stage of the pandemic; it did not account for the particular age distribution of mortality.

If these two hypotheses are incorrect, how might the age distribution of mortality in Seattle and elsewhere be explained? Another hypothesis has been advanced: the pandemic in the United States seems to have affected certain immigrant groups to a greater extent than it affected the U.S.-born. A disproportionate number of migrants were between fourteen and forty-four years old, and a disproportionate number were originally from rural areas (Handlin, 1959; Katz, 1974). The consequences of this are clear:

> Most of the immigrants came from backgrounds where the chances of exposure to influenza would be small. Living in isolated, rural communities where contact with outsiders was rare, they would not have been exposed to the man-to-man droplet contact. The crowding, and the constant influx of new infective vectors vital to the spread of influenza—consequently, they would not have had the chance to develop immunity [Katz, 1974, 420].

Explanation of the age-distribution of mortality, then, hinges on the argument that a significant number of people in the most affected age

groups were migrants who had not been exposed to influenza previously. Because many worked in crowded environments and at physically demanding jobs, Katz (1974) suggests that the age-distribution is no longer a mystery; such circumstances are most conducive to the rapid spread of infectious disease.

There are two major problems with this hypothesis. First, it is difficult to prove or disprove. Second, the age-distribution of mortality in countries other than the United States was very similar to that in the United States. The age-distribution of deaths in Seattle and elsewhere remains a mystery.

Mortality Patterns and Place of Birth

One of the clearest characteristics of the influenza pandemic was that mortality was unevenly distributed among immigrant groups. In Connecticut, there was lower than expected mortality among American, Irish, English, and German groups, and greater than expected mortality in Russian, Austrian, Polish, and Italian stock (Winslow and Rogers, 1920; Katz, 1977). In Framingham, Massachusetts, the influenza attack rate was four times greater among Italians than among Irish (Armstrong, 1919), and observations of unequal mortality rates have been commonplace.

The distribution of mortality rates within Seattle by place of birth was as striking as it was elsewhere. It is possible to reconstruct the mortality rates by place of origin because the deceased's place of birth was noted routinely on the death certificates. The resulting patterns are clarified when countries of birth are grouped together into broadly defined regions. As table 5 indicates, the mortality rates are distributed differently among regions of origin ($x^2=91.4$; df=8, p. <0.01), based upon estimates of the population on 1 January 1919. Populations are estimated using the method discussed previously.

The overall influenza mortality rate in Seattle was 426 per 100,000 people: the mortality rate among its Austrian immigrants was nearly triple the overall rate; among natives of China and Japan, more than double the overall rate. Other marked deviations from the overall rate were the less than expected rates among German immigrants and the greater than expected rates among Russian, Greek, and Italian immigrants. Mortality rates among the American, Canadian, and Scandinavian residents were approximately equal to the overall mortality rate.

It is difficult to develop an adequate explanation for the distribution of mortality rates among the immigrant groups. This is because the crude rates (table 5) may be influenced strongly by the different age distributions

TABLE 5.5
Influenza Deaths in Seattle by Region of Origin, 1918–1919

Region Group	Estimated Population of Group[a]	Number of Deaths	Number Alive	Rate/100,000
U.S., Canada	228,994	979	228,015	428
Scandinavia Norway, Sweden Denmark, Finland	23,414	99	23,315	423
British Isles England, Scotland, Wales, Ireland	14,159	54	14,105	381
Austria	1,463	17	1,446	1,162
Germany	4,958	10	4,948	202
Greece, Italy	4,500	31	4,469	689
Russia	3,376	19	3,357	563
Poland	863	2	861	232
China, Japan	6,918	67	6,851	968

[a]1 January 1919.

among immigrant groups. Because influenza mortality rates were greatest among people twenty to forty years old, it is reasonable to expect that immigrant groups with a disproportionate number of people in that age cohort would have elevated mortality rates. The usual procedure to correct for age-related influences is to age-adjust the mortality rates to a standard population. However, such correction requires age-distribution facts for each immigrant group, and that information for 1920 cannot be ascertained. Age-adjusted mortality rates are therefore impossible to calculate.

Because age-adjusted mortality rates cannot be calculated, it is also impossible to determine whether the disparities in mortality rates among immigrant groups was an artifact of the population distribution, or whether the rates reflected epidemiologically important factors in the distribution of influenza. Katz's (1977) argument that differences among immigrant groups reflected differences in earlier exposure to the influenza virus may have held in Seattle, but it cannot be ruled out that the differential distribution among immigrant groups in fact reflected age distribution.

Spatial Diffusion of Influenza in Seattle

Much research has been carried out on the spatial diffusion of communicable disease, but most of it has considered patterns at the

international, national, and regional levels (e.g. Haggett, Ord, and Versey, 1981; Haggett, 1976; Hunter and Young, 1971; Pyle, 1969). At the local and intraurban levels, less is known about communicable-disease diffusion patterns. This reflects the inherent difficulties of discerning meaningful patterns when dealing with a rapidly diffusing disease in an area small enough to allow periodic movements of local residents over much of it. With a disease such as influenza, which is spread largely by droplet, close contact is necessary between infectives and susceptibles, and the contact may be short and fleeting. Once a disease appears within a city, though, are there meaningful and discernible patterns of diffusion as the disease spreads?

To explore this question, mortality data from the 1918–19 influenza outbreak in Seattle are useful. Mortality data are not ideal for this analysis because many people who were affected by the disease did not die, and also because some variation in the time lag between infection and death is normal. However, even at the intraurban level a regular progression in the distribution of deaths by residence might be expected. Deaths due to the disease might occur primarily in one area of the city early in the epidemic, and in another area later in the epidemic.

To test this expectation, the Seattle mortality data were stratified by the week during which the death occurred, and were digitized to a grid of approximately twenty meters. In the context of the overall population ecology of the urban area, the exact location of the death, as noted in the death certificate, is less important than the residence of the person who died. With out-of-town visitors excluded from the analyses, the relevant geostatistical and centographic measures of the distribution of deaths may be compared by time period to ascertain the diffusion patterns.

For an epidemic that progresses in a wavelike pattern of contagious diffusion, one might expect there to be little movement with time of the measure of central tendency, and a steady increase in the dispersion. For an epidemic that progresses in a "frontal" pattern or in a wavelike pattern with directional bias, analogous to relocation diffusion of innovations, the dispersion should remain relatively constant, while the point of central tendency would progress steadily in one direction. Thus, centrographic measures may assist in developing a profile of epidemic diffusion, as established in previous work (Morrill and Angulo, 1979). Centrographic measures may assist in ascertaining the directionality of spread, the temporal regularity of spread, and the dispersion of the epidemic.

The specific centrographic and geostatistical measures that have been selected for this analysis include the bivariate median, and the standard distance, and the skewness of the distribution. In addition, the raw and corrected nearest-neighbor statistics, and the relative dispersion are in-

cluded to provide further indication of the regularity and dispersion of the distribution of mortality, as indicated by Morrill (1971).

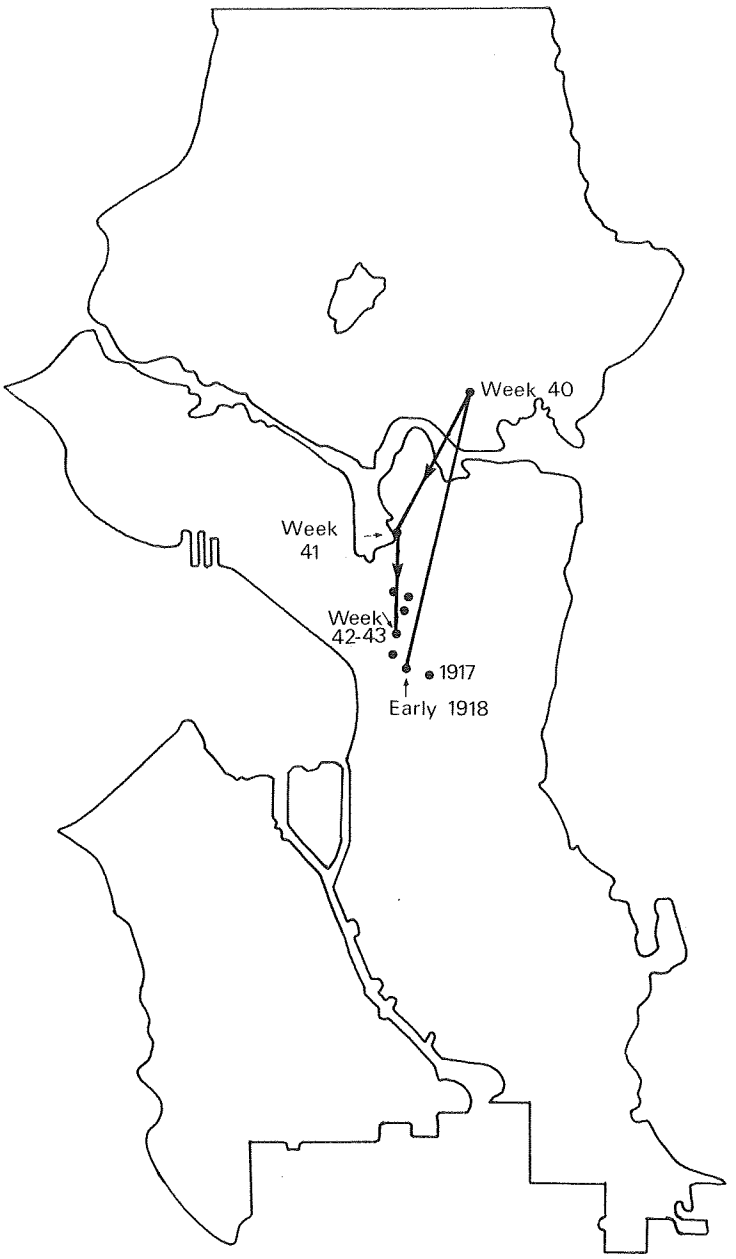
The bivariate median or point of minimum aggregate travel was selected because it provides a reasonable measure of central tendency and is not overly influenced by outlying points, as is the bivariate mean. The standard distance, rather than standard deviational ellipses, was selected to present a simple measure of dispersion that may be compared between time periods. The raw nearest-neighbor statistic reflects the standard nearest-neighbor formula, and the corrected nearest-neighbor statistic overcomes some of the bias described by Morrill (1971) that results when a distribution of points is of nonuniform density. The nearest-neighbor statistic is unbiased with respect to indicating the degree of uniformity, clustering, or randomness in a punctiform pattern only when the density of points is close to uniform. "Otherwise, local dispersion and even randomness in the arrangement of points to each other will be masked by the marked apparent clustering due to density variation (of concentration of points within the bounded area)" (Morrill, 1971, 39). Thus, an uncorrected nearest-neighbor analysis may indicate clustering, but this clustering may be an artifact of density variation. Morrill (1971) presents a method of calculating a corrected nearest-neighbor statistic that is unbiased by density variations; the statistic is interpreted in the same manner as is the raw nearest-neighbor statistic. The reader is referred to Morill (1971) for a more complete discussion.

The final measure used is the relative dispersion, and is defined as the ratio of the standard deviation of the points in a distribution $S_d = \sqrt{S_x^2 + S_y^2}$ to that of the bounded area within which the points are distributed were the points evenly distributed. The relative dispersion thus measures the degree to which the points are centrally or peripherally distributed (Morrill and Angulo, 1979).

The temporal progression of the bivariate median is indicated in figure 1. The distribution of influenza deaths in 1917 is used as a control against which the distribution during the epidemic waves may be compared. "Early 1918" refers to influenza deaths prior to the beginning of the epidemic in week 40 (30 September–6 October), during which there was tremendously elevated influenza mortality, which, by definition, constitutes the beginning of the epidemic.

The pattern that emerges in figure 1 is that the 1917 and early-1918 deaths were distributed around the central area of the city. During week 40, the central point of the distribution shifted to the north. Over the next three weeks of the epidemic, there was a steady movement southward toward Seattle's CBD. This was followed finally by seemingly random movements of the bivariate median, as indicated by the unlabeled points

FIGURE 5.1
Temporal Progression of the Bivariate Median



in figure 1. Thus, as the epidemic progressed, location of the bivariate median approached the locations of that point during the 1917 and early 1918 control periods.

What accounts for the northward "jump" of the bivariate median during week 40? This certainly indicates that the spatial distribution of mortality during this week differed from the previous and succeeding periods, and that it took several weeks for the distribution to shift back to the south. As noted previously, much of the disease's spread both nationally and internationally was through the military, particularly during the early period of the pandemic, along the major lines of military transport and communication. Influenza gained its entrance to Seattle at the U.S. Naval Training Station in Lewis Hall at the University of Washington, a building densely populated by recent recruits. The bivariate median in week 40 is located at almost the same coordinates at Lewis Hall; this reflects the fact that nearly all of the influenza deaths during week 40 were among naval trainees who were staioned there. As the *Seattle Post-Intelligencer* (1918d, 1) noted on 4 October, at the end of week 40, "Despite the rapid spread of the epidemic at the naval training station . . . there have been no city cases reported . . . by local physicians."

The probable cause of the temporal diffusion pattern, then, is explained largely by the concentration of deaths early in the epidemic at the Naval Training Station, and then by the gradual diffusion of the disease into Seattle's civilian population. By 10 October, during week 41, numerous civilian deaths were reported. The southward movement of the bivariate median to the central area of the city came to approximate the center of the Seattle population, and the movement would be entirely consistent with the disease's spread to the civilian population from the initial concentration in the military.

Were this explanation true, one would expect the standard distance to be relatively low initially, during week 40, when the mortality was concentrated. This would be followed by an increase in standard distance as the disease spread into the civilian population. As table 6 indicates, the standard distance increased from 3,021 m in week 40 to 4,740 m in week 41. Thereafter, the standard distance fluctuated around an approximate figure of 4,500 m. A gradual decrease in standard distance is evident between weeks 41 and 48–49; the statistic decreased from 4,611 m to 4,011 m. Weeks 48–49 represented the trough between the two major epidemic waves, and during this period there was greater spatial concentration of mortality than during the peaks—weeks 44–47 and 50–51. There was greater concentration throughout the epidemic than during the control year of 1917.

TABLE 5.6
Descriptive Geographical Measures

Week	Standard Distance (m)	Raw Nearest Neighbor	Corrected Nearest Neighbor	Relative Dispersion
1917	7338	1.06	.920	
15–21	4134	1.52	2.01	.960
40	3021	.950	.710	1.10
41	4740	.580	.640	.910
42–43	4611	.760	1.10	.790
44–47	4605	.580	.860	.740
48–49	4011	.690	1.11	.680
50–51	4500	.750	1.01	.790
52–58	4581	.570	.860	.730
59–End	4638	.710	1.03	.750

The patterns suggested by the nearest-neighbor statistics are somewhat puzzling. The raw nearest-neighbor statistic, during week 40, was near unity, suggesting a random distribution; it then decreased, suggesting that clustering was occurring. The corrected nearest-neighbor statistic, though, fluctuated in a seemingly random pattern around one, thereby suggesting that the pattern was random.

Finally, the relative dispersion was approximately 1 in week 40, and thereafter decreased. The value of this statistic for most of the epidemic is in the general range of that depicted in an outbreak of variola minor as described by Morrill and Angulo (1979), and indicates that mortality was relatively concentrated throughout the epidemic, compared with the territory in which the epidemic occurred.

Conclusion

By the time the epidemic waned in Seattle in 1919, nearly 1,400 people, or .5 percent of the city's population, had died because of influenza or its complications. The major epidemiologic features of the pandemic within Seattle did not differ from those for this outbreak elsewhere in the United States, and, indeed, throughout the world. It was more deadly among young adults than among children or the elderly. The precise explanation for this remains elusive. It affected immigrants from certain countries to a greater extent than immigrants from other countries or American-born citizens. This may have been an artifact of the population's

age structure, or may have been due to other unknown factors, such as the disproportionately affected immigrant population's having migrated from predominantly rural areas where exposure to previous outbreaks of this strain of influenza might have been lower than in urban areas.

Elementary spatial analysis is useful in depicting the diffusion of an epidemic. This particular outbreak followed some regular patterns in its diffusion at the intraurban scale. This scale of disease diffusion, as such, exhibits some similarities to diffusion patterns at the regional, national, and international levels.

The major areas for future investigation arise out of this particular research. The first concerns the behavior of geostatistical measures for certain types of disease diffusion. In contagious, hierarchical, and relocation patterns, do measures such as standard distance, nearest neighbor, and relative dispersion, actually exhibit regular patterns over time as the epidemic progresses? The question may be approached both by simulating epidemic spread, and by studying inductively actual infectious disease epidemics at a variety of levels.

The second major area for research is more contextual and interpretive. An analysis of primary and secondary historical sources confirms that the population and the bureaucracy were concerned about the epidemic, and attempted to prevent its spread within Seattle. Fear of the epidemic was somewhat eclipsed by attention to the progress of World War I. However, as Yi-Fu Tuan has observed (1979), fear of disease has been a universal aspect of human experience. Along with fear or perhaps because of it, there have been attempts at explaining and controlling disease.

Infectious diseases, when they reach epidemic or pandemic proportions, are responsible for a major increase in human morbidity, mortality, and anguish. The notion of an epidemic as a disequilibrating natural event that is relatively sudden in its onset, along with the ensuing perceptions and actions, suggests that epidemic or pandemic disease may be viewed as a natural hazard. The rich conceptual framework of natural-hazards research may add a great deal to understanding the dynamics of disease in its social, cultural, and historical contexts. Disease is usually excluded from discussions of hazards, yet the exclusion is both unfortunate and inappropriate. The human significance of disease may be measured and categorized using the scheme of Burton, Kates, and White (1978). Epidemic disease is a natural hazard: it is an "extreme" event, although not a geophysical event, that may have profound and devastating effects on human populations. Populations respond to the threat and actuality of epidemics through individual, collective, and bureaucratic coping mechanisms. A major goal of public health organizations is the mitigation of

the impact of disease. Further historical analysis is being conducted on the 1918–19 pandemic in Seattle within this conceptual framework.

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