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Source: *Ethnohistory*, Vol. 41, No. 1 (Winter, 1993), pp. 73-105

Published by: [Duke University Press](#)

Stable URL: <http://www.jstor.org/stable/3536979>

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“There Were Young People and Old People and Babies Dying Every Week”: The 1918–1919 Influenza Pandemic at Norway House

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Abstract. Analysis of Norway House Anglican parish registers during the 1918 influenza pandemic suggests that eighteen percent of the population perished in six weeks. Its strategic position in the fur trade and lack of substantial provisions in the subarctic winter contributed to the death rate. Population recovery occurred within five to ten years, owing to a modest post-epidemic marriage boom and the maintenance of birth rates. Analysis of parish records and twentieth-century virgin soil epidemics may help to develop models for early contact epidemics.

A lot of our people died in 1918 with the flu. There were young people and old people and babies dying every week.

—Felix Mahzenekezhik, 24 April 1979, cited in Higgins 1982: 95, *Whitefish Lake Ojibway Memories*

Introduction

The story of cataclysmic diseases introduced to the Americas has become the predominant leit motif of historical and anthropological analyses of the post-contact period. Indeed, disease stress has emerged as a central principle in the interpretation of culture change in the Americas since European contact (Cook and Borah 1971; Dobyns 1966, 1976) and epidemiological reconstructions as “the key to explaining and describing native American depopulation” (Dobyns 1984: 17). A large depopulation literature has developed, and the idea that large numbers of indigenous Americans died in “a wave of pestilence and death that rivals the Black Death in fourteenth-century Europe” (Roberts 1989: 1246) has become almost axiomatic.

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By the same token, most researchers are quick to cite a litany of problems associated with appraising the impact of specific diseases and in tracing the disease history of particular aboriginal American groups (see Henige 1990 for a particularly scathing review). The usual caveats are directed toward the quality and scantiness of the available data. Pre-contact health status and population sizes are acknowledged to be largely conjectural, based on the limited information encoded in bony remains, artifacts, and inferred from ethnographic analogies (Johansson 1982: 134–35). Historical demographic and epidemiological reconstructions stand or fall on the observations of a relatively small number of explorers, missionaries, and physicians—and on the extent to which the accuracy of their counts and comments is tainted by intentional or accidental bias (Ross 1977: 1–3; Ubelaker 1988: 289). In fact, *reliable* statistics on aboriginal population size and structure, morbidity, and mortality begin in the 1960s in Canada (Piché and George 1973: 369; Romaniuk and Piché 1972: 1). The lack of quantitative historical studies of the effects of disease on North American Indian populations (Cook 1973; Ray 1974: 104–9), therefore, comes as no surprise.

Such challenges to ingenuity, however, are not confined to research on disease and population history in the Americas. As recently as 1979, Hollingsworth noted that “the systematic study of population crises has scarcely been attempted previously” (1979: 17) and Arnold (1988: 1) stressed that disease and medicine in general have been much neglected outside of the European and North American contexts. The absence of a general theoretical approach for understanding disasters and their aftermaths (Bouckaert and Lechat 1987: 19), moreover, attests to the relative infancy of this area of inquiry. Nevertheless, new research has shown that post-crisis marriage and birth patterns can significantly buffer a population against losses and enhance the speed of its recovery (cf. Clark 1985; Mielke and Pitkanen 1989), undermining the traditional view that mortality crises conclusively checked population growth in the past.

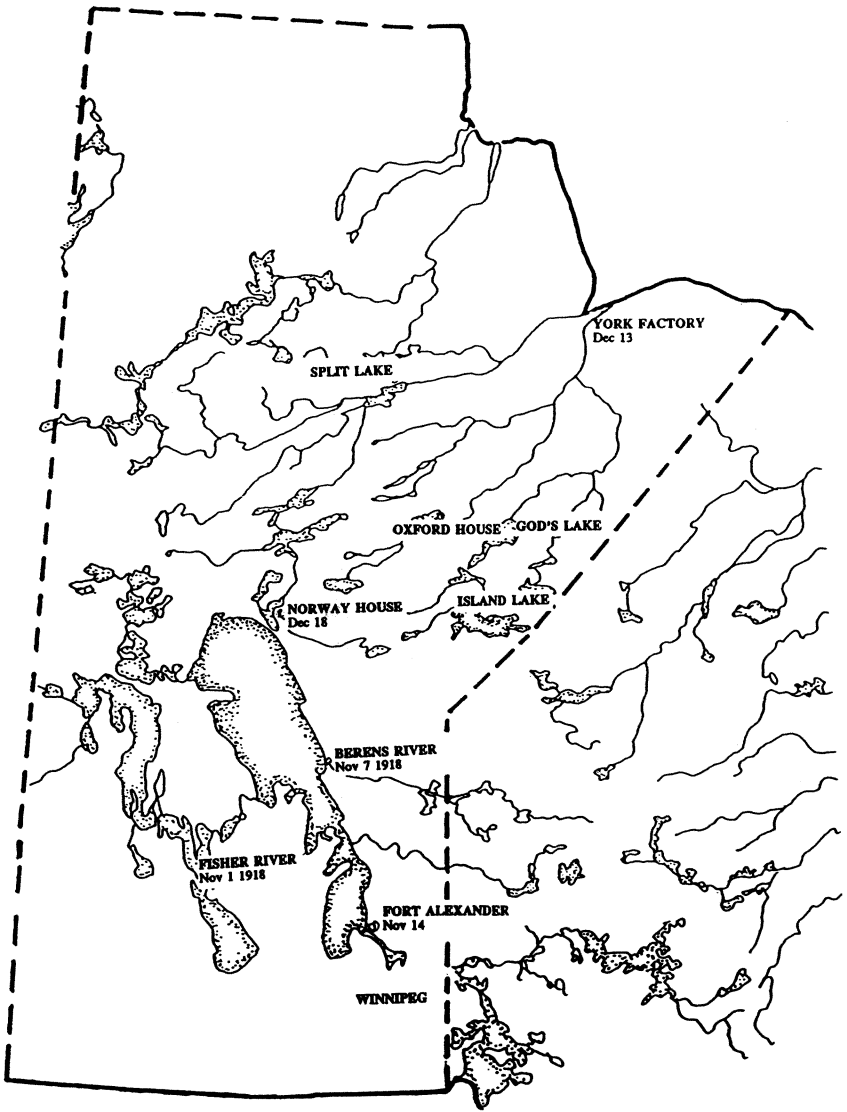
When situated in this wider historical demographic context, the idea of a virginal New World, helplessly ravaged by pandemics of Old World pathogens (cf. Crosby 1976, 1986; Dobyns 1976, 1983), warrants further scrutiny. New initiatives probing the evidence in support of the depopulation hypothesis include osteological analyses that demonstrate unequivocally that the New World was anything but free from infectious diseases prior to contact (Verano and Ubelaker 1992); development of revised population estimates for the time of initial European contact (Ubelaker 1988; 1992); synthesis of ethnohistoric, archaeological, medical, and demographic data to develop more complex models of post-contact dis-

ease diffusion (Ramenofsky 1987; Snow and Lanphear 1988); application of epidemiologic transition theory and principles of infectivity to historical studies of disease (Young 1988a, 1988b; Decker 1989); and computer simulations of infectious disease epidemics (McGrath 1988; Thornton et al. 1991).

The approach taken in this paper is to examine a relatively recent and better-documented virgin soil epidemic, with a view to contributing to the development of a range of scenarios that might have operated in the past. The effects of the 1918–1919 influenza pandemic are examined in the Swampy Cree/Métis community of Norway House in subarctic Canada (See Map 1). The paper is *not* intended to address directly the question of post-contact depopulation in the Americas, but rather to explore the impact of the epidemic at Norway House as a possible simulation of conditions that might have existed from time to time in some historic and prehistoric North American Aboriginal communities. Inferences about the effects of the epidemic on the community are drawn from a variety of historical sources, with a primary focus on Anglican church parish registers for the Jack River Mission at Norway House (ACCA 1902–1937). Beyond fleshing out for an indigenous community the poorly known details of a mortality crisis described as “the most appalling epidemic since the Middle Ages” (Collier 1974: 303) and as “the greatest disease holocaust of modern history” (American Journal of Public Health 1968: 2192), the study also affords a rare opportunity to examine marriage and fertility behavior in the post-epidemic period (cf. Ray 1974: 106–9).

The Community of Norway House

Norway House is a predominantly Swampy Cree/Métis settlement located at the northeast end of Lake Winnipeg in the central Canadian subarctic, about 280 miles due north of Winnipeg, the provincial capital of Manitoba (See Map 1). The Hudson’s Bay Company (HBC) established a supply base there in 1801 because of its strategic position at the southern end of the Nelson River trade axis, the main fur brigade route between York Factory and the northern interior. Norway House gradually rose to prominence in the nineteenth century along with the expansion of the fur trade into the western interior, so that by 1820 it had become the nexus of HBC provisioning activities in the region (Ray 1974: 128). The route to Hudson Bay from the Red River settlement in the south also passed through Norway House, making Lake Winnipeg “the crossroads of a continent” (Hallowell 1955: 114), to which the Athabasca fur brigades brought furs to trade for European goods shipped inland from York Factory (Ray 1974: 120).



Map 1. Norway House, Manitoba (Canada).

Because of its central location and point of connection for far-flung Indian groups, it was eventually selected as the headquarters for the British Wesleyan Methodist Missionary Society's effort to Christianize the Aboriginal peoples of the northwest. This brought the renowned and colorful John Evans—charismatic preacher, musician, and inventor of the syllabic writing system still in use today—to Norway House in 1840, to establish a Methodist mission some two miles beyond the Hudson's Bay Company Fort at Rossville (Donaldson and Abel 1985; Maclean 1925: 18). Through the work of subsequent clergymen, mission circuits covering HBC posts as distant as Nelson House, Oxford House, and Berens River were eventually established (Donaldson and Abel 1985; Maclean 1925: 18). The approximately 2,300 baptisms recorded for the Wesleyan Methodist Mission at Norway House between 1840 and 1889 attest to the zeal with which the gospel was promulgated in the region (PAM 1840–1889).

The last half of the nineteenth century, however, brought major changes to the economy of the northwest and to Norway House in particular. By the 1860s, York Factory had been eclipsed by Red River and St. Paul as the orientation of the fur trade shifted in concert with the construction of railways in the south. Indeed, as York Factory lost its prominence in the HBC fur trade, many people migrated south to Norway House (Stone 1926: 1–2).

The Norway House economy was also affected by the restructuring of trade routes, and its transshipment business faded as York Factory's depot was increasingly bypassed for southern locations. The local economy was further depressed by the introduction of steamboats on Lake Winnipeg in the 1870s (Warkentin and Ruggles 1970: 369). This effectively reduced tripping and freighting work, important alternatives to trapping which had diminished because of the depletion of game in the area. Consequently, when Treaty 5 was negotiated in 1876 with the Lake Winnipeg Cree and Ojibwa, some two hundred Norway House families moved south to Fisher River where better fishing and farming resources were available (Donaldson and Abel 1985: 13–14).

Although the halcyon days of the fur trade at Norway House were long past by the time of the Spanish Flu, subsistence activities were still primarily centered around fishing and fur-gathering and other fur-trade-related wage labor, such as boat building, freighting by canoe, or dog-training (HBCA 1918–1923, 1926: 76–77). Seasonal variation in activity patterns was still very much in evidence and similar to that described by Ray (1976: 140–42) for the early-nineteenth century; namely, winters tended to be passed in small family groups on traplines, some of which were three hundred miles away (Stone 1926: 9), while summers

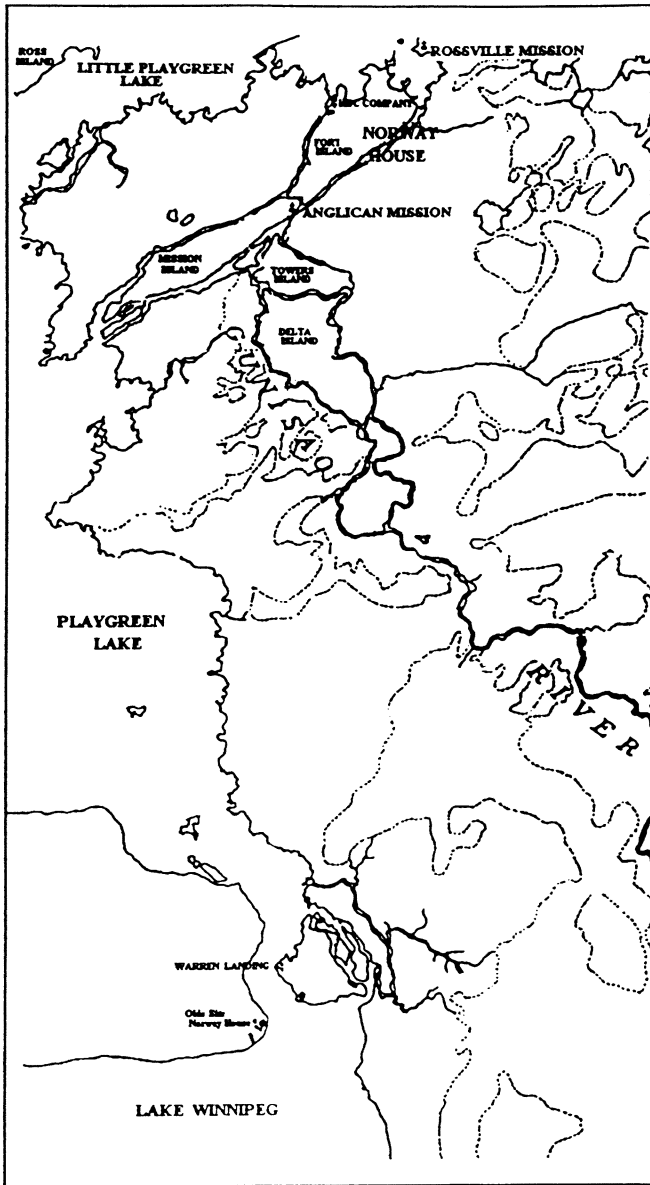
were characterized by larger agglomerations of people around the posts. The majority of traffic between posts occurred in summer when mobility was facilitated by open water; harsh winter weather and dispersed family groups tended to reduce contact with the post to a small number of trappers and mail couriers. Norway House nevertheless remained a bustling place, boasting three churches, a boarding school, a resident doctor and hospital, and the headquarters of the Norway House District.

Materials

Primary source information on the 1918–1919 influenza epidemic at Norway House was drawn from three sources: Hudson’s Bay Post Journal entries for 1918 and 1919 held at the Hudson’s Bay Company Archives in Winnipeg (HBCA 1918–1923); Rossville Methodist Day School attendance records located at the Provincial Archives of Manitoba in Winnipeg (PAM 1952–1957), and parish registers of baptisms, marriages, and burials from the General Synod Archives of the Anglican Church of Canada at Church House, Toronto (ACCA 1902–37). Unfortunately, the Methodist Mission registers for the study period did not survive to the present. Consequently, the ensuing statistical analysis is based on the Anglican Church parish records for the Jack River Mission.¹ The Post Journals and school records allowed cross-checking, corroboration, and amplification of inferences drawn from them.

The Jack River Anglican Mission was inaugurated in 1901, ostensibly to serve the children who lived too far away to attend the Methodist Mission School at Rossville (See Map 2). The congregation initially consisted of some “100 Indians and Halfbreeds,” most of whom had migrated from York Factory (ACCA 1904–1964). As Table 1 shows, the Anglican population at Norway House remained relatively small, never making up more than one-third of the total population. By 1916 it had grown to about two hundred, partly through the adhesion to Treaty 5 of a large number of York Factory migrants in 1909 (Government of Canada 1910).

The Jack River Mission registers of baptisms, marriages, and burials are available for the relatively short period of 1902 to 1941. They were maintained by a series of clergymen who evidently ran the mission with varying degrees of success until the appointment of Rev. J. F. J. Marshall in 1910. His arrival ushered in a sixteen-year period of relative stability at the mission until his transfer in 1926 to the newly opened Indian Residential School near Sioux Lookout, Ontario (ACCA 1907–1960). Rev. Marshall was the resident clergyman during the 1918–19 influenza epidemic, and his eight years of prior experience there confers a measure of confidence in



Map 2. Jack River Anglican Mission at Norway House.

Table 1. Anglican Population at Norway House 1909, 1914, 1916, 1924.

Year	Total	Anglican		Methodist		Roman Catholic		Source
		n	%	n	%	n	%	
1909	536	90	16	402	75	44	8	Government of Canada, 1910: 44–45.
1914	746	216	28	500	67	30	4	Government of Canada, 1915: 16–17.
1916	734	208	28	494	67	32	4	Government of Canada, 1917: 18–19.
1924	714	189	26	483	67	42	6	Government of Canada, 1925: 37.

the extent of his knowledge of his parishioners and in the accuracy of his records of their vital events.

Parish records are often biased. There are always individuals in a community whose life events are omitted because participation in the rites of the church is not compulsory and may, in fact, be avoided. Underenumeration of the population is exacerbated when there are competing religious groups, a propensity to migrate or leave the site of observation for even relatively short periods of time, or when famines, epidemics, or other disastrous occurrences make it difficult to maintain church rosters (Levine 1976; Pitkanen 1977; Wrigley 1978). Clearly, these sources of distortion operated at Norway House during the period under study. A previous study of tuberculosis mortality, moreover, showed some evidence of underreporting of infant deaths from 1901 to 1929 (Herring 1984). Careful cross-checking of the data, which will be discussed later, minimized distortion in the analysis which could have stemmed from this.

The 1918–1919 Influenza Pandemic

The 1918 influenza pandemic was unlike any known flu epidemic before or since (cf. Crosby 1989 for a review). Attacking in three waves in the spring and fall of 1918 and in the winter of 1919, it is conservatively estimated to have killed over twenty million people worldwide—more than were killed by all the armies in World War I over the span of four years (Graves 1969: 13). Indeed, it ranks with the Black Death and the Plague of Justinian as one of the most destructive human afflictions known (Walters 1978: 856).

Illness came on without warning, heralded by a sudden shivering,

severe ache in the head and eyeballs, and pain in the legs and kidneys, followed by collapse. High fever set in rapidly, coupled with a hacking cough that not uncommonly produced thin, rusty sputum. This particular flu manifested itself so swiftly and with such consistent symptoms that it was possible for many to pinpoint exactly when they became sick. All age groups were struck, rather than the usual case of the very young and very old, and it attacked children between the ages of five and fourteen and young adults in the twenty-to-forty age group with unprecedented lethality (American Journal of Public Health 1968: 2, 193; Walters 1978: 855; Patterson 1986: 89). Most recovered uneventfully within a week; nevertheless, about twenty percent of the ill developed secondary streptococcal or staphylococcal infections leading to pneumonia, sometimes within twenty-four hours (Burnet and Clark 1942: 88). It was the deadly complication of influenza pneumonia, untreatable in the pre-antibiotic era and still a persistent problem today (Stuart-Harris et al. 1985: 106), that eventually killed forty-to-fifty percent of them (Burnet and Clark 1942: 88). Those with severe pneumonia lapsed into a “‘typhoidal state’ characterized by cracked lips; a dry, shrunken, glazed, brown tongue; lint-picking fingers; incontinence; and a semi-conscious stupor” (Walters 1978: 860). When faces turned an ashy purple, indicative of heliotrope cyanosis and dangerously depleted hemoglobin, death was usually imminent.² Curiously, the afflicted at this point often chirped that they felt marvelous,³ despite frightfully purple countenances and a horrible “influenza stench, reminiscent of rotting corpses on a battlefield” (Walters 1978: 860). Not surprisingly, some were convinced the flu really *was* the Black Death (MacDougall 1985: 2090–91).

Its origin remains murky. Worldwide epidemiological data suggest, nevertheless, that the outbreak was closely tied to conditions in Europe at the twilight of World War I and, more specifically, to the arrival of American troops in France. The misnamed “Spanish Flu” may, in fact, have emerged at Fort Riley, Kansas,⁴ traveling explosively around the world from this single site in the wake of American and other troop movements (Burnet and Clark 1942: 69; Graves 1969: 21; Beveridge 1977: 42–43; Kaplan and Webster 1977: 89).

The causative agent eluded identification until 1933 when English researchers isolated the virus now known as Influenza A (Smith et al. 1933). Three major forms are recognized—Influenza A, B, and C—based on distinctive antigenic features and a lack of cross-reacting antigens (Berlin 1980: 353). All three are usually acquired via the airborne route when the virus is expelled from an infected individual through sneezes and coughs and inhaled into the respiratory epithelium of an uninfected person. Infec-

tion rapidly leads to acute febrile illness after a one-to-two-day incubation period because the respiratory epithelium is both the portal of entry and the site of infection (Turk et al. 1983: 177). While attacks of Influenza B and C are mild and rarely fatal, the A form is extremely dangerous and can provoke widespread, lethal epidemics and pandemics (Kaplan and Webster 1977). Its characteristic plasticity results in major phenotype changes and the emergence of new strains to which few are resistant and to which all age groups are potentially susceptible (Bannister 1983: 58).

Although the full story is still unclear, current evidence suggests that the 1918–1919 strain of Influenza A was new (Kaplan and Webster 1977: 102). Since immunity from Influenza A is highly strain-specific (Turk et al. 1983: 178), enormous numbers of people succumbed to the new variant that began to circulate in March 1918 because they lacked protective antibodies. Indeed, the 1918–1919 flu pandemic is a classic example of a virgin soil epidemic.

Influenza-like epidemics appear to have afflicted human populations for at least four hundred years (Stuart-Harris et al. 1985: 1), and possibly longer (Graves 1969: 14; Thacker 1986: 129), but the 1918 pandemic was extraordinarily deadly. The reasons for this remain obscure, but an association between encephalitis lethargica, Parkinsonism, and the 1918 influenza pandemic has long been postulated (Walters 1978; Ravenholt and Foege 1982; Maurizi 1985: 3). This may help explain the “utter uniqueness of the 1918 outbreak,” its explosive spread, and high mortality rate (Patterson 1986: 91).

There was no cure, though a huge array of prophylactics abounded in Canada, as elsewhere (see Graves 1969: 27). Some tried to ward off the flu with camphor or salted herring bags worn about the neck, while others assiduously applied sulphur to their shoes (MacDougall 1985: 2093). Salves, elixirs, herbal poultices, hot bran, lard mixed with camphor and chloroform, patent medicines spiked with opiates and alcohol, aspirin, and epsom salts were other common remedies (Pettigrew 1983: 110–14). Aboriginal people from the Little Pine Reserve in northern Saskatchewan credited wild ginger root and muskrat food with sparing many from illness. At Cumberland House Reserve, needles were dipped in the musk gland of a skunk, stirred into a glass of water, and drunk down; in the Peace River District, wild ginseng, yarrow, and wild cranberry bark were favored (Ibid.: 114). Government intervention at the local, provincial, and federal levels led to a number of public health measures. Public meetings were banned in the Province of Manitoba and elsewhere in Canada, based on the observation that the disease was airborne and contagious and that crowds facilitated its spread (Manitoba Provincial Board of Health

1919: 4). Citizens were ordered to wear masks in Alberta; some communities even attempted to impose total quarantines (MacDougall 1985: 2092–93). And a public outcry forced the federal government to permit the sale of whisky for medicinal purposes (Graves 1969: 27). In the end, the only effective remedy was good nursing care (Kaplan and Webster 1977: 88).

Influenza at Norway House

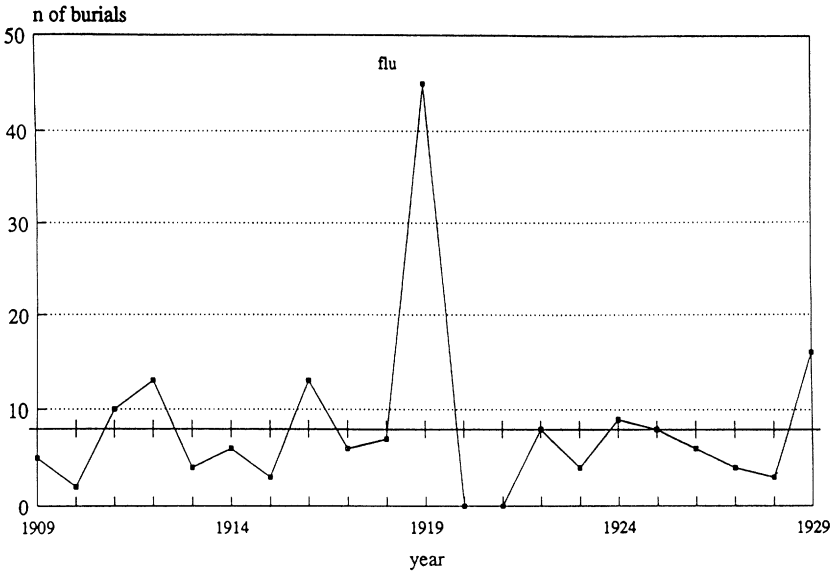
Although there is some dispute about precisely when Spanish Flu reached Canada, Pettigrew (1983: 8) suggests that the first civilian outbreak occurred in Victoriaville, Quebec on 8 September 1918.⁵ The major source of infection was returning troops exposed to the virus in Europe while fighting for Britain during World War I. As soldiers decamped and headed home, influenza steamed across the country with the speed of a trans-continental train. The disease was carried to Winnipeg, the capital of the Province of Manitoba, on 30 September 1918 by a group of sick soldiers traveling westward on a troop train. Just four days later, the first civilian influenza death occurred there (Ibid.: 56–57). From this urban focus, Spanish flu fanned out to rural and isolated parts of the province via an elaborate network of train lines, roads, and water routes.

Steamships, for instance, had become swift conveyers of disease since their introduction onto Lake Winnipeg in the mid 1870s (Warkentin and Ruggles 1970: 369). Lake Winnipeg was busy as usual with steamer traffic until freeze-up in the fall of 1918. At the end of October the passenger steamer *Wolverine*⁶ made its last stop of the year at Berens River. Within three days almost all the people there were overcome by the flu, brought from the south by the *Wolverine*'s ailing crew (Pettigrew 1983: 78–79).

Duration of the Epidemic

Scrutiny of the annual burial totals for Norway House from 1902 to 1941 indicates the unusual nature of mortality in 1919 and provides a dramatic visual impression of the impact of Spanish Flu (See Graph 1). Although a saw-toothed pattern of mortality is evident over the four decades, typical of periodic infectious disease epidemics and a small population, the mortality peak for 1919 is striking and unique. That year there were forty-five burials, thirty-five of which were ascribed to influenza. This compares to a mean of 6.4 ± 4.3 burials per annum for the ten years on either side of the epidemic, suggesting an astonishing sevenfold increase in mortality that year.

The chronology of the epidemic, derived from the Anglican Church

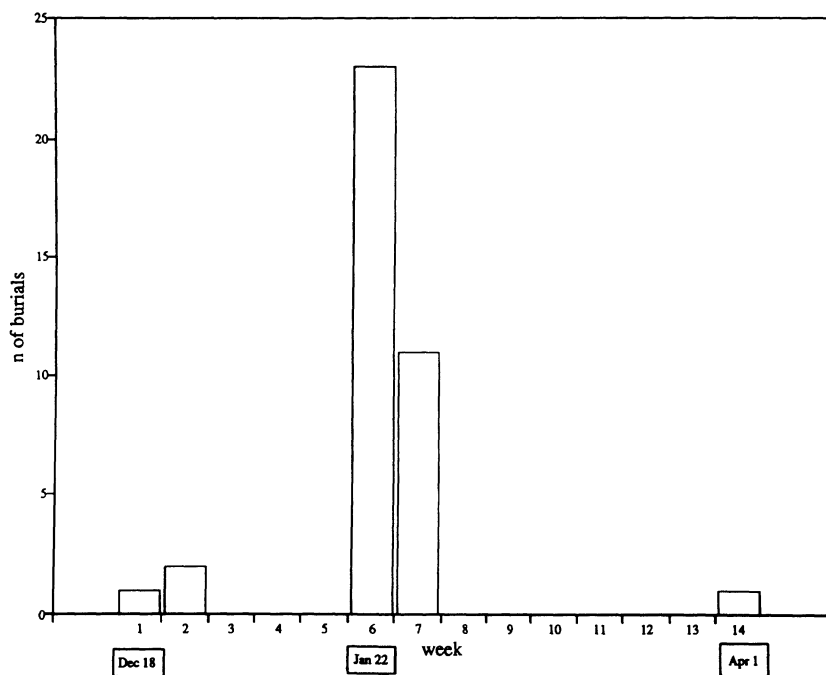


Graph 1. Anglican burials at Norway House, 1909–1929.

burial records (See Graph 2) shows that: (1) the epidemic began in December, 1918 during the second wave of the pandemic; (2) thirty-eight people who died from influenza in the winter of 1918–1919 were buried by the Anglican Church minister; and (3) the epidemic was effectively over by February 1919. Other evidence from the Hudson’s Bay Company Post Journal, the Rossville Methodist Day School records, and the Oxford House Post Journal confirms that the burial register accurately reflects the duration of the epidemic. This will be discussed in greater detail below.

It is difficult to determine the route by which Spanish Flu arrived at Norway House. Even though its heyday as a North American crossroads had long since passed, the post still served as a regional depot and transshipment center for furs, cargo, and people. Steamers, including the infamous *Wolverine*, stopped regularly at Warrens’ Landing from spring to fall. However, it is unlikely that influenza was transmitted directly by steamer since the last load of freight arrived in mid-October, far in advance of the December outbreak (HBCA 1918–1923: fo. 3). In fact, the Hudson’s Bay post journal implicates a more circuitous overland route from the northwest, rather than directly by water from the south.

The first allusion to the epidemic occurs in the Post Journal entry for Tuesday, 3 December 1918:



Graph 2. Anglican burial chronology during influenza epidemic, 18 December 1918 to 1 April 1919.

I walked over to Rossville to ascertain if there is any truth in the rumour that the war is over. Word was apparently sent from Cross Lake to this effect, but I was unable to assure that the source of the rumour is anything more than an Indian Story. "Spanish Flu" was also reported at an Indian camp at Clearwater Lake some 2 or 3 days north of Cross Lake. It is to sincerely hoped [sic] that the latter is not correct, but if the former is true, and we can only hope that it is, the Lord be praised! (HBCA 1918-1923: fo. 9)

The outbreak at Clearwater Lake is confirmed in the next entry which reports seventeen deaths there (*Ibid.*), conclusively demonstrating that the epidemic was already raging to the north well before it struck Norway House.

There is also evidence that influenza traveled south from Clearwater Lake to Norway House via Cross Lake, a nearby post to the north. The Norway House Post journal duly notes the arrival of the Cross Lake packet on 4 December (*Ibid.*). This constitutes the last communication

with another post known to have been struck by the epidemic.⁷ Five days after the arrival of the Cross Lake packet, the HBC choreboy was sick with an unspecified ailment, possibly influenza. Certainly the timing of the arrival of the packet and the boy's illness is consistent with influenza's two- to three-day incubation period (Bannister 1983: 58). Even though packets also arrived from Island Lake and Oxford House during this time (HBCA 1918–1923: fos. 9–10), both places escaped the epidemic and therefore could not have infected Norway House (HBCA 1918: fo. 51).

Spanish Flu also appears to have struck Cross Lake before Norway House. On 17 December Norway House's resident physician⁸ noted that sickness had taken hold at Cross Lake and killed eleven people (HBCA 1918–1923: fo. 11). No one at Norway House had yet succumbed to the disease, suggesting that Cross Lake was in a later phase of the epidemic wave.

Since the Cross Lake Post Journal for this period has been lost, this reasonable inference cannot be verified. However, there are two probable primary sources of virus: Winnipeg to the south, or York Factory to the north (See Map 1). From Winnipeg, influenza could have diffused along the northern railway line to The Pas, spread east and northward to Clearwater Lake, south to Cross Lake, and south again to Norway House. From York Factory, where Spanish Flu is mentioned on 4 November (HBCA 1917–1922), the virus could have spread south to Norway House through the old Nelson River trade axis. In light of the absence of influenza deaths at Split Lake during the epidemic (Herring 1989), it would seem that Winnipeg is the more likely primary source. On the other hand, it is possible that influenza was brought back directly by decamped soldiers, one of whom was buried at Norway House on 16 February 1919, having died from the effects of mustard gas (ACCA 1902–1937).

Regardless of its origin, the Spanish Flu evidently engulfed the community swiftly. "Sickness at Norway House" is mentioned in the Post Journal on Friday, 13 December (HBCA 1918–1923: fo. 10), the last day that year that the Rossville Methodist Day School was open (PAM 1952–1957: fo. 9). The school remained closed until 28 January and "EPIDEMIC" was written in bold letters where student attendance for 16 to 31 December would normally have been recorded. Attempts to muster a dog team to carry mail on 19 December failed because "every man here that has dogs is unwell," and the usual Christmas rush at the HBC store did not materialize because "most of the people sick" (HBCA 1918–1923: fos. 11–12). And the losses from flu had climbed to fifty on 6 January 1919, nineteen days after the first death on 18 December 1918 (Ibid.: fo. 13).

The Anglican Church burial chronology shows that the epidemic lasted about six weeks. Almost all of the flu burials took place in January, and the epidemic was effectively over by February (See Graph 2). Indirect corroboration of this appears in the Hudson's Bay Post Journal entries. Although the entries for January, 1919 regularly include observations on the progress of the flu, they cease by mid-February (*Ibid.*, fo. 12–17). More convincing evidence is found in the Oxford House Post Journal, which notes on 30 January 1919 that Spanish Flu at Norway House “is practically over now—no fresh cases having been reported” and that the quarantine had been lifted (HBCA 1917–1924: fo. 10). The resumption of attendance at the Rossville Methodist Day School on 28 January is another indicator that the crisis had passed and that daily life had begun to regain its normal rhythm (PAM 1952–1957).

Proportionate Influenza Mortality

Nevertheless, the roster of deaths from the flu was terrible. Graph 2 shows that thirty-eight individuals died from the virus between 18 December 1918 and 7 April 1919. Given a total Anglican population of 208 in the 1916 Norway House census (See Table 1), the epidemic appears to have carried off almost one-fifth of the population in six weeks. This is substantially higher than the three percent mortality estimated for all Canadian Indians (Graham-Cummings 1967: 149). On the other hand, the proportionate mortality estimate of 18.8 percent derived from the Anglican register is in line with the 21.8 percent estimate yielded by dividing the 160 flu deaths that occurred at Norway House (Krotz 1990: 31) by 734, the total population at risk in 1916.⁹ The results suggest that the Jack River flu burials provide a reasonable gauge of the epidemic at Norway House as a whole and that the Spanish Flu epidemic was more intense at Norway House than was generally the case for aboriginal communities in Canada.

It is important to bear in mind, however, that the maintenance of parish registers during epidemics is usually irregular, owing to the sheer volume of deaths and the pressing need to get food, keep fires going, and care for the ill. The disorderly listing of deaths in the Anglican Church burial register during the epidemic, a significant deviation from an otherwise chronological progression, is one small indicator of the upheaval caused by widespread illness, raising the specter of underreported deaths. On the other hand, the circumstances of daily life at Norway House lead to a counterbalancing suspicion of overenumeration of deaths arising from the frequent comings and goings of dog-trains from other posts, the documented deaths of men from God's Lake and Oxford House at Norway

House during the epidemic,¹⁰ the inclusion of five influenza burials by William Saunders (the resident clergyman at Fisher River),¹¹ and the April 1919 burial of a boy who actually died from the flu on 18 October 1918.

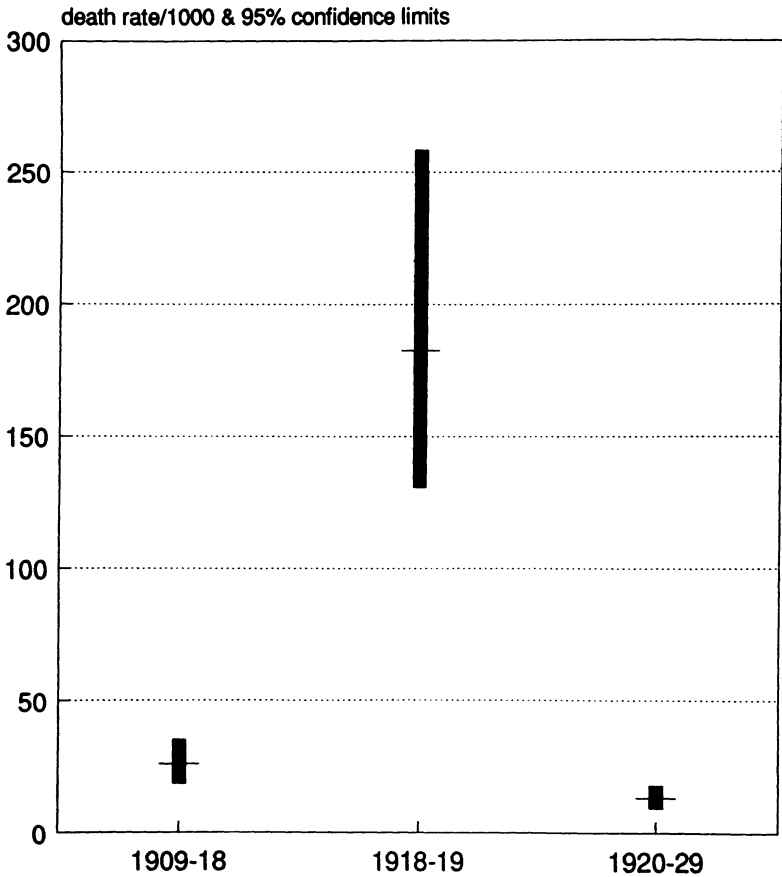
To determine whether individuals from other communities were inflating Norway House's death rate, the genealogical records for each flu victim were examined to establish whether there was evidence of their presence in the community prior to the epidemic. Sixty-four percent ($n=25$) were listed in either the baptism or marriage registers prior to 1919. This is strong presumptive evidence that they were part of the Norway House community before the epidemic. Another twenty-eight percent ($n=11$) had surnames present either in the Jack River records, the Rossville Methodist records, or in the band lists prior to 1919. Only two individuals could not be linked to the community before the epidemic.

Total Influenza Mortality Rate

An influenza mortality rate was derived by using the thirty-eight flu burials as the numerator and the 1916 census of Anglicans as the population at risk (Government of Canada 1917). Crude mortality rates were estimated for the preceding (1909 to 1918, minus the 1918 influenza deaths) and succeeding decades (1920 to 1929) using the Anglican population at risk in 1914 and 1924, respectively (Government of Canada 1915, 1925). Ninety-five percent confidence limits were then drawn around the mortality estimates, based on the Poisson distribution (Lilienfeld and Lilienfeld 1980: 336–38).

As Graph 3 shows, the Jack River Mission experienced an extraordinary influenza death toll of approximately 183 deaths per 1,000. This compares to a death rate of 26 per 1,000 for the preceding decade (1909–1918) and 13 per 1,000 for the subsequent ten-year interval (1920–1929). It is gratifying to note that the ninety-five percent confidence limits of 130 to 248 flu deaths per 1,000 encompass Stone's (1925: 80) mortality estimate of 146 per 1,000 for Norway House. Once again, the values produced from the Anglican Church burial register are not significantly different from those for the total Norway House population in 1919. Not only did the parish records chart the duration of the epidemic accurately, but they also appear to have provided a reasonable approximation of mortality for the community as a whole.

The high death toll at Norway House was undoubtedly influenced, however, by factors other than simply the presence of a new virus. Because of the community's hunting and gathering subsistence economy, there were few provisions on hand beyond what was available at the Hudson's Bay Company store.¹² As an apparently astonished Dr. Stone observed in 1926:



95% confidence limits based on Poisson distribution (Lilienfeld & Lilienfeld 1980:336-38)

Graph 3. Mortality rates per 1,000 for Anglicans at Norway House: 1909-18, 1918-19 influenza epidemic, 1920-29.

“Their meat supply is often in the net in the river, or in the rabbit snare in the bush. Going about from house to house, one is at a loss to see where the food produced at meal times is kept between meals. . . . When adversity comes they quickly come to want. . . . The hardship of the sick and aged would be worse than it is if there did not obtain among them a very admirable community spirit” (HBCA 1926: fo. 4).

No written accounts of the epidemic at Norway House are available, but the distress faced by the people can be imagined by analogy to the experience of other aboriginal settlements in the Canadian Prairies and North. Village life undoubtedly broke down when the flu struck and entire families would have been without an able-bodied individual to feed them or keep the fire going in the cold, subarctic winter. Many of the ill contracted pneumonia after going out to replenish dwindling food and fuel supplies, dying shortly thereafter (PAM 1902–1930: 8). The horror of the Spanish Flu in northern communities is perhaps best expressed in Reverend Henry Gordon's description of the influenza epidemic at the Grenfell Mission at Cartwright, on the Labrador Coast:

It has struck the place like a cyclone, two days after the Mail boat had left. After dinner I went on a tour of inspection among the houses, and was simply appalled at what I found. Whole houses lay inanimate all over their kitchen floors, unable to even feed themselves or look after the fire. . . . I think there were just four persons in the place who were sound. . . . A feeling of intense resentment at the callousness of the authorities, who sent us the disease by the Mail-boat, and then left us to sink or swim, filled one's heart almost to the exclusion of all else. The helplessness of the poor people was what struck to the heart. . . . It was very upsetting, people crying, children dying everywhere. (cited in Pettigrew 1983: 25–27)

The dead were often wrapped in sheets and placed on rooftops to keep the dogs from eating them. These ghostly white bundles remained potent reminders of the ravages of the epidemic until spring, when they could be buried in the thawing ground (PAM 1902–1930: 8).

Progress of the Epidemic

Epidemic curves, such as Graph 2, often yield important information about the etiology and intensity of an epidemic (MacMahon and Pugh 1970: 157–59). The Anglican Church burial records, however, proved to be a poor barometer of this aspect of the epidemic. There were often large gaps between the dates of interment and the dates of death. Most of the burials, in fact, were clumped in a four-day period between 26 and 30 January, creating the superficial impression that the epidemic was slow to take hold, but accelerated suddenly at the end of January. The mortality peak proved to be illusory, an artifact of the Anglican clergyman's three-week incapacitation with flu from 1 to 22 January (HBCA 1918–1923: fos. 12, 14). The removal of eight bodies on 30 January to the "English Church Graveyard" for burial also served to inflate the death toll at this time (*Ibid.*, fo.

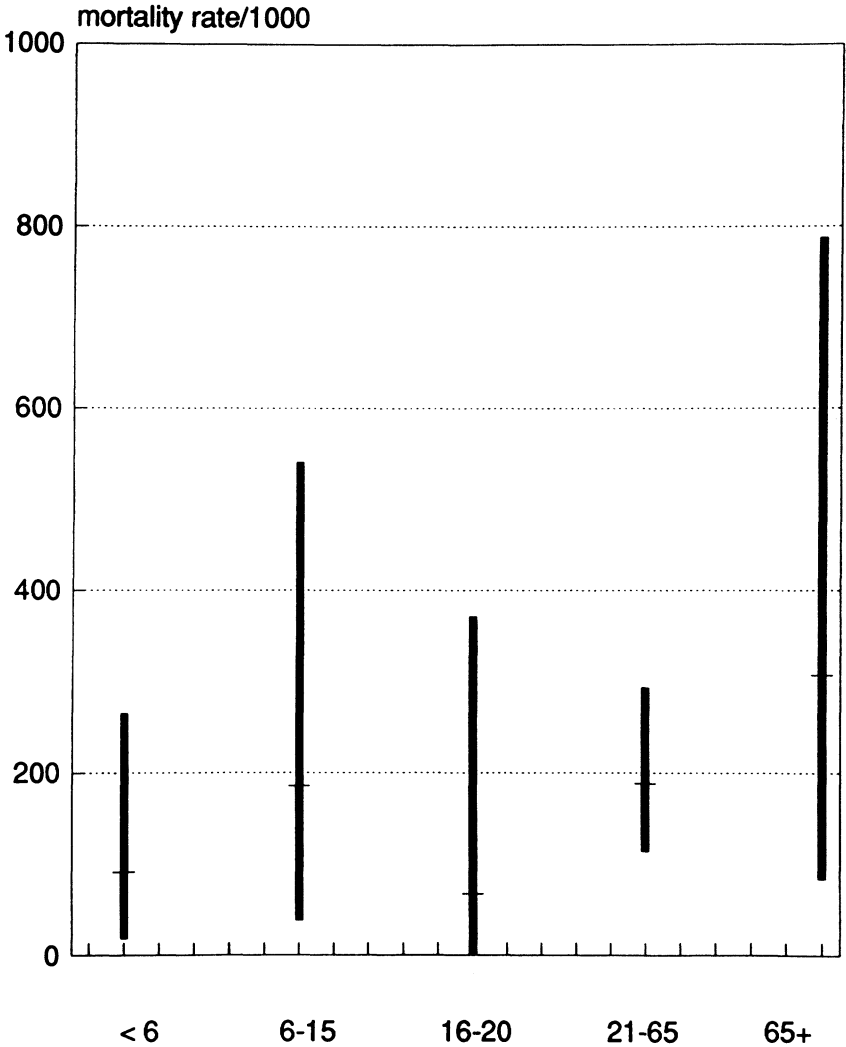
15). Thus, the apparently high death toll at the end of January is more properly viewed as the conjunction of a number of consequences of the epidemic: widespread sickness, including that of the Anglican Church missionary, community disarray, the problem of digging graves in frozen ground, and the need to retrieve bodies from outlying camps.

Age-Specific Mortality

The extensive effects of the flu epidemic on the whole community are mirrored in the age-specific mortality rates per 1,000 (See Graph 4). No age group was spared. Table 2 shows, moreover, that the age-distribution of mortality during the epidemic was substantially different than in either of the two decades that surround it ($LRX^2 = 13.73$, $p = .033$). During the epidemic there was a significant deficiency in deaths under the age of one (standardized residual = -2.04) and a significant increase in deaths in the 1–14 age group (standardized residual = $+1.75$).¹³ The high death toll in the 1–14 age group fits the observation that the disease occurred most frequently among children 5 to 14 years of age (Walters 1978: 855), but may still be a low estimate if children's deaths were underreported.

Age-specific mortality rates alone, however, cannot possibly represent other ramifications of an epidemic, such as the shock to families shattered by the deaths of parents, children, and other kin, or the despondency and fatalism that widespread sickness produces (see Krech 1983: 137–38). The death of children is often the hardest to bear for they are the hope of the future. The people at Little Pine Reserve in Saskatchewan, for example, were completely demoralized by the epidemic until word came from Alberta's Hobbema Reserve that someone had a dream telling them to dance and hold a pow-wow—which they did, feasting, praying, dancing, and raising their spirits (Pettigrew 1983: 80). At Wabauskang Reserve in Ontario, over a thousand people are believed to have died during the epidemic, leading the people to relocate to sacred ground at the old Grassy Narrows reserve (Shkilnyk 1985: 57–58).

Mortality rates also provide little insight into other social repercussions of epidemics. The Norway House fur returns for 1918 were down because “Many good hunters died as a result of the disease” which struck “at a time when Fur is usually at its best” (HBCA 1918: fos. 51–52). It is also difficult to gauge the impact of the loss of knowledge and leadership that ensues when elders die or sociocultural transformations that may be set in motion by high epidemic mortality (cf. Taylor 1977; Hader 1986: 24–30). Mortality rates, moreover, provide no indication of the time required for a community to regain its numbers following the crisis. However, the potential for post-crisis nuptiality and natality booms to increase the



Graph 4. Age-specific influenza mortality rates per 1,000 for Anglicans at Norway House, 1918-19 influenza epidemic.

reproduction rate and thereby spur quick demographic recovery cannot be ignored (Del Panta and Livi Bacci 1979: 76). Post-epidemic birth rates, for instance, are critical for assessing the impact of disasters, but are insufficiently studied (Bouckaert and Lechat 1987: 26-28). Examination of the Anglican Church Marriage and Baptism Registers suggests that post-

Table 2. Age Distribution of Anglican Burials at Norway House: 1909–18, 1919, 1920–29.

Age Category	Death Cohort			Likelihood	
	1909–18	1919	1920–29	Ratio X ²	*p-value
<1	10	0	4	13.73	.033
1–14	11	16	4		
15–44	23	14	10		
45+	12	10	7		
STANDARDIZED RESIDUALS ¹					
<1	+1.29	-2.04	+0.66		
1–14	-0.87	+1.75	-0.92		
15–44	+0.29	-0.38	+0.06		
45+	-0.38	+0.13	+0.40		

df=6

* Yates's correction for continuity

¹ (observed-fitted)/√(fitted)

epidemic marriages and births influenced the speed of population recovery at Norway House.

Marriages

To determine whether the epidemic had affected nuptiality in 1919, the number of marriages occurring that year were compared to those in the two decades on either side of it (See Table 3). Although the numbers in Table 3 are admittedly small (but commensurate with a population of about two hundred individuals), a modest marriage boom appears to have followed on the heels of the epidemic. Seven marriages were sanctified in the Anglican Church in 1919, more than twice the annual average of 2.8 marriages for the twenty years surrounding it. Interestingly, one or both of the spouses had been widowed in each of the 1919 marriages, producing a surfeit of marriages containing widowed individuals (+3.13) and a deficit in the never-married category (-2.13), compared to the surrounding decades. The disparity proved to be highly significant (LRX² = 24.94, p < .001, 1df).

It seemed reasonable to interpret the post-epidemic rise in marriages as the remarriage of individuals widowed during the influenza crisis (cf. Mielke and Pitkanen 1989: 388). Examination of the reconstituted fami-

Table 3. Marriages and Marital Status of Spouses at Norway House: 1909, 1919, 1920–29.

Type of Marriage	Marriage Cohort			Likelihood	
	1909–18	1919	1920–29	Ratio X_2	*p-value
both never married	19 (-0.37)	0 (-2.13)	24 (+1.59)	24.94	<.001
one or both previously married	11 (+0.55)	7 (+3.13)	1 (-2.34)		

df=2

* Yates' correction for continuity

lies, however, failed to support this hypothesis. Surprisingly, only one of the ten widowed individuals who remarried in 1919 actually lost a spouse during the epidemic. Perhaps the sudden loss of nineteen people, one-fifth of the productive and procreative age group, impelled the formation of new marriages simply to maintain community functioning. This may have been especially important in a fur-trade community like Norway House where the division of labor along sex lines and cooperation of a network of kin is vital to its success. Alternatively, it is conceivable that individuals who had not submitted to the Christian marriage ritual might have been persuaded to do so if the epidemic were represented as the wrath of God. More pragmatically, the marriages may also have involved returning soldiers from World War I.¹⁴ Regardless of the motivations behind them, the increase in marriages immediately following the epidemic may have served to buffer the community somewhat from further declines through reduced fertility and hastened the resumption and restructuring of social and economic life.

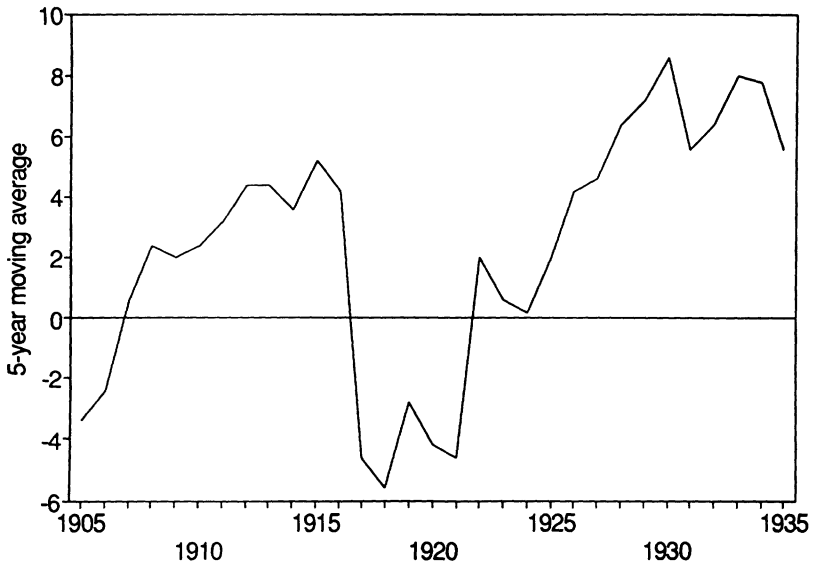
Baptisms

In light of the widespread sickness and trauma associated with the epidemic and the loss of a large proportion of adults, it seemed reasonable to expect a decrease in baptisms following the epidemic (see Sogner 1979: 317–19). Examination of the register of baptisms for the ten years on either side of the epidemic failed to support this hypothesis (See Table 4). No significant change in the mean number of baptisms was detected (Mann-Whitney $U=60$, $p=.447$, $df=1$), an observation independently confirmed by Medical Officer Stone who commented that birthrates in

Table 4. Baptisms at Norway House: 1909–18 vs. 1920–29.

Period	Mean	S.D.	Mann Whitney U Value	p Value
1909–1918	10.6	2.3	60	.447
1920–1929	9.1	4.3		

df= 1



Graph 5. Five-year moving averages: Anglican baptisms minus burials at Norway House, 1905–35.

the Norway House District had remained stable from about 1915 to 1923 (Stone 1925: 80).

Population Recovery

Graph 5 depicts the results of a simple procedure whereby the excess baptisms over burials are counted in succeeding years until they surpass the number of individuals lost during the epidemic (Drake 1974: 102).¹⁵ The graph shows that the Anglican population expanded from 1914 to 1918, only to plummet following the flu epidemic. It regained its pre-epidemic position between 1928 and 1930 and in 1931 surpassed the number lost during the epidemic. It appears, therefore, that some ten years elapsed before the Anglican parish at Norway House recouped its pre-epidemic numbers,

despite the marriage boom in 1919 and the maintenance of birth rates in the post-epidemic period. Referring back to Table 1, however, and comparing the *total* Norway House population in the 1916 and 1924 censuses, produces a different conclusion. The total Norway House population rebounded to ninety-seven percent of its pre-epidemic numbers within five years; in contrast, the Anglican segment had only regained ninety-one percent of its pre-epidemic size by 1924. Perhaps other factors, such as emigration, disaffection from the church, or the small size of the reproductive segment of the group, slowed the growth of the Anglican component.

Conclusions and Speculations

It is evident that parish records and family reconstitution techniques can be used effectively to help ascertain historical patterns of disease and death in historic aboriginal communities. When used judiciously and carefully, they offer an important means of amplifying and complementing other historical documents, such as Hudson's Bay Company post journals and reports. In particular, they contain crucial information on fertility and marriage patterns essential for evaluating the demographic consequences of infectious disease epidemics. These parameters are usually unavailable or neglected in mortality analyses that attempt to assess the impact of disease on aboriginal North Americans. In the case of Spanish Flu at Norway House, the Anglican Church marriage and baptism records suggest that a modest post-epidemic marriage boom and the maintenance of birth rates helped cushion the community against the loss of about one-fifth of the adults between the ages of twenty and sixty-four. This, in turn, facilitated the recouping of pre-epidemic population numbers within five (total population) to ten years (Anglicans only).

The excessive toll of adult deaths in the Spanish Flu pandemic remains one of its enigmatic features. If we consider that high mortality in the reproductive segment of a population is one of the features of a potentially depopulating epidemic, then the relatively rapid demographic recovery of the Norway House community becomes particularly instructive. It shows that even when conditions conducive to depopulation are present, they nevertheless may be insufficient to provoke an irreversible loss of population.

The results of this study also serve as a reminder that virgin soil epidemics exact variable tolls of mortality in different communities. While about eighteen percent of the people at Norway House died during the 1918–19 influenza epidemic, this was far in excess of the estimated three percent of indigenous Canadians who perished across the country

(Graham-Cummings 1967: 149). Preliminary comparative analysis of this epidemic in several subarctic aboriginal communities¹⁶ has confirmed that substantial mortality differentials occurred, even within the Norway House Fur District alone (Herring 1990). Indeed, both God's Lake and Oxford House escaped the epidemic altogether (HBCA 1918: fos. 51–52). Similar diversity undoubtedly occurred in less well-documented early contact virgin soil epidemics and it is clearly dangerous to assume that such epidemics were uniformly devastating, or derive regional epidemic mortality rates from a small number of locations. Overestimates of the death toll can ensue if they are based on worst-case scenarios; specifically, communities in close contact with European recorders and hence, European diseases. As Zubrow (1990: 760) notes: “There are multiple diseases which act distinctly on populations and they affect different types of societies dissimilarly.”

It is still unclear why Norway House was so severely struck by the 1918–19 influenza epidemic. Its key position in the fur trade network and frequent contact with locations to the west, northwest, northeast, and south left it particularly vulnerable to imported microorganisms, as Ray (1976: 156) suggests was the case in the mid-nineteenth century. Certainly, the number of routes of disease spread to Norway House had increased substantially by 1918, with steamer and railway routes offering swift passage to infectious agents. Transportation and exchange networks figured prominently in the diffusion of the Spanish Flu, channeling the movements of microorganisms and patterning their dispersion across some regions while bypassing others (see also Dobyns 1983: 12–13; Reff 1991: 119–24; Sattenspiel and Powell 1993). It follows that the establishment of Hudson Bay and Northwest Company fur trade posts in the central subarctic not only created new dependencies, stimulated significant relocations of peoples, restructured inter-ethnic mobility and exchange networks, and led in some instances to inter-ethnic hostility, resource depletions, or starvation (Ray 1974; Krech 1983: 131–38), but in so doing created new disease pools and patterns of diffusion.¹⁷ In fact, social organizational changes associated with the fur trade era may have been more momentous for the subsequent epidemiologic history of the central subarctic than the introduction of novel European pathogens per se.

Once it was established at Norway House, attempts to contain the influenza epidemic through quarantine may ironically and tragically have served to increase mortality by concentrating virus and infected individuals there. The lack of substantial stores of food and wood, in the midst of a subarctic winter, likely led to higher mortality rates among the ill from secondary pneumonia. As McGrath (1991: 418–19) emphasizes, social re-

sponses are instrumental in determining an epidemic's course and hence, its biological impact. Obviously, no single factor accounts for the Norway House experience of the Spanish flu epidemic. Should we assume that early contact virgin soil epidemics were less complex?

Notes

I am grateful to the Norway House Band Council for giving me permission to study their records. The staff of the Provincial Archives of Manitoba, Hudson's Bay Company Archives, Anglican Church of Canada Archives (Toronto), and the United Church of Canada Archives (Winnipeg) provided unstinting and cheerful assistance throughout the research process. Chris Egan, Andrew Mathiesson, Henry Weilenmann, and Scott Mackellar helped with the collection of primary source data. Larry Sawchuk made valuable comments on an earlier version of this paper which was presented at the Canadian Association for Physical Anthropology Meetings in 1989. The research was supported by Social Science & Humanities Research Council award #410-89-0638 and Arts Research Board of McMaster University award # 5-58631.

- 1 The baptism, marriage, and burial registers were hand-transcribed and then converted into separate databases using Paradox386 software (Ansa/Borland 1987). The individual entries in each database were subsequently concatenated into nuclear families using a combination of manual and computer-assisted linkage procedures, according to traditional historical demographic techniques of family reconstitution (Fleury and Henry 1965; Henry 1967; Wrigley 1966, 1973; Tilly 1981). Statistical analysis was carried out using SYSTAT (Wilkinson 1986).
- 2 The purplish hue of heliotrope cyanosis is also produced by strangulation or hanging (Graves 1969: 31).
- 3 Symptoms of neural disease, such as delusions, inability to concentrate, mental aberrations, insomnia, irritability, and depression during convalescence have suggested a link between the 1918 flu epidemic and encephalitis lethargica (Walters 1978: 561; Ravenholt and Foege 1982; Maurizi 1985).
- 4 Beveridge (1977: 39–44) points out that influenza broke out in China and Japan at about the same time as the first wave of spring outbreaks in Europe, leading him to suggest that either the United States or China could have been the primary focus of the first wave. Graves (1969: 22) offers the possibility of an independent, coeval Asian epidemic.
- 5 Graves (1969: 19–20) dates the arrival of the epidemic in Canada to 21 September 1918, during the second wave of the pandemic, noting that the first American victim died on 5 March 1918 during the first wave at Camp Funston, Fort Riley, Kansas.
- 6 The *Wolverine* was a screw-driven passenger, fisheries, and freight steamer built in 1903. It had no scheduled run. Indeed, "She popped into what ever harbours had cargo to offer, popped in often at the most inconvenient hours of the night. Her passengers were the workers of the land, fishermen, lumbermen, Indians. She was part of the country's life." (PAM 1859–1946).

- 7 The Annual Report for the Keewatin District, Outfit 1918, of the Hudson's Bay Company states unequivocally that only Norway House, Berens River, and Cross Lake Posts were severely hit while the rest of the posts "fortunately escaped" (HBCA 1918: fo. 51).
- 8 Although many outlying parts of the Province of Manitoba had no physicians, a permanent hospital had been constructed at Norway House in October 1914, staffed by Dr. H. C. Norquay and a trained nurse since 1904 (Government of Canada 1916: 55). Dr. Norquay and Dr. Larose of The Pas monitored the progress of the 1918 flu epidemic in the Norway House District. Both traveled large distances to care for the sick, in the dead of the subarctic winter (HBCA 1918-1923: fos. 11, 15-17, 19).
- 9 Calculation of ninety-nine percent confidence limits around the two proportions according to the method described by Lilienfeld and Lilienfeld (1980: 333-36) indicated there was no significant difference between them. The burial records intervals are: $.1826 \pm .0693$ and the intervals based on Krotz's (1990: 31) values are $.2180 \pm .0393$.
- 10 Examination of the Methodist burial registers for Oxford House (1918-1951) and God's Lake Narrows (1894-1926) (UCCA 1918-1951b, 1894-1926), cross-referenced to their respective post journals (HBCA 1918-1923, 1917-1924), shows that the deaths of these men were recorded in their home parishes, not at Norway House. Evidently, burial registers were not simply used to record interment ceremonies, but were also used to keep track of the fate of parishioners away from home.
- 11 A cross-check of Methodist Mission records for Fisher River from 1918-1951 (UCCA 1918-1951a) revealed that none were duplicates of the burials conducted by William Saunders in the Jack River Anglican burial register.
- 12 Although the Hudson's Bay Company apparently sent wood to some families and medicines to Gull Lake during the epidemic (HBCA 1918-1923: fos. 12-13), the Post Journal contains no references to deliveries of food.
- 13 Standardized residuals compare each observed cell frequency with its expected value (observed-expected/ $\sqrt{\text{expected}}$). Standardized residuals in excess of 1.64 (95th percentile of the standard normal distribution) indicate a significant deviation in the observed cell values from those expected (Reynolds 1977: 11-13).
- 14 I am grateful to Raymond Beaumont of the Frontier School Division for having drawn my attention to this possibility.
- 15 The technique assumes a closed population.
- 16 Berens River, Fisher River, Oxford House, and God's Lake.
- 17 For a discussion of the idea of the "confluence of disease pools," see McNeill 1977: 69-131.

References

- ACCA (Anglican Church of Canada Archives, General Synod Office, Toronto, Canada).
 1904-1964 Mf. 77-75, Reel #1, Diocese of Keewatin Records, History by Mission, Vol. 1.
 1907-1960 Mf. 77-75, Reel #1, Diocese of Keewatin Records, Biographical Sketches of Keewatin Clergy.

- 1902–1937 Mf. 77-75, Reel #3, Diocese of Keewatin Records, Norway House Burials.
- American Journal of Public Health
1968 Editorial. 58(2): 2, 192–94.
- ANSA/Borland
1987 Paradox. Scotts Valley, CA: ANSA Software.
- Arnold, D.
1988 Introduction: Disease, Medicine, and Empire. *In* *Imperial Medicine and Indigenous Societies*. D. Arnold, ed. Pp. 1–8. Manchester, U.K.: Manchester University Press.
- Bannister, B. A.
1983 *Infectious Diseases*. London: Ballière Tindall.
- Berlin, B. S.
1980 Influenza. *In* *The Biologic and Clinical Basis of Infectious Diseases*, 2nd ed. G. P. Youmans, P. Y. Paterson, and H. M. Sommers, eds. Pp. 353–66. Toronto: W. B. Saunders.
- Beveridge, W. I. B.
1977 *Influenza: The Last Great Plague*. New York: Prodist.
- Bouckaert, A., and M. Lechat
1987 Consequences of Catastrophic Mortality Arising from Epidemics and Natural Disasters for Social Structures and Economic Systems. *Genus* XLIII (1–2): 19–40.
- Burnet, F. M., and E. Clark
1942 *Influenza*. London: Macmillan.
- Clark, P., ed.
1985 *The European Crisis of the 1590s: Essays in Comparative History*. London: Allen and Unwin.
- Collier, R.
1974 *The Plague of the Spanish Lady*. London: Macmillan.
- Cook, S. F.
1973 The Significance of Disease in the Extinction of the New England Indians. *Human Biology* 45(3): 485–508.
- Cook, S. F., and W. W. Borah
1971 *Essays in Population History: Mexico and the Caribbean*, Vol. 1. Berkeley and Los Angeles: University of California Press.
- Crosby, A. W.
1976 Virgin Soil Epidemics as a Factor in the Aboriginal Depopulation of America. *William and Mary Quarterly*, 3rd series, 33: 289–99.
1986 *Ecological Imperialism: The Biological Expansion of Europe, 900–1,900*. New York: Cambridge University Press.
1989 *America's Forgotten Pandemic: The Influenza of 1918*. Cambridge: Cambridge University Press.
- Decker, J. F.
1989 'We Should Never Be Again the Same People': The Diffusion and Cumulative Impact of Acute Infectious Diseases Affecting the Natives on the Northern Plains of the Western Interior of Canada, 1774–1839. Ph.D. diss., York University.
- Del Panta, L. and M. Livi-Bacci
1979 Chronology, Intensity, and Diffusion of Mortality in Italy, 1600–1850. *In* *The Great Mortalities: Methodological Studies of Demographic*

- Crises in the Past. H. Charbonneau and A. Larose, eds. Pp. 69–81. Liège, France: Ordina Editions.
- Dobyns, H. F.
 1966 An Appraisal of Techniques for Estimating Aboriginal American Population with a New Hemispheric Estimate. *Current Anthropology* 7: 395–416.
 1976 Scholarly Transformation: Widowing the “Virgin Land.” *Ethnohistory* 23: 161–72.
 1983 Their Number Become Thinned. Knoxville: University of Tennessee Press.
 1984 Native American Population Collapse and Recovery. *In* *Scholars and the Indian Experience*. W. R. Swagerty, ed. Pp. 17–35. Bloomington: Indiana University Press.
- Donaldson, B. F., and K. M. Abel
 1985 The Rossville Mission, 1840–1900. Winnipeg: Historic Resources Branch, Manitoba Ministry of Culture, Heritage and Recreation.
- Drake, M.
 1974 *Historical Demography: Problems and Projects*. Rustington, U.K.: The Open University.
- Fleury, M., and L. Henry
 1965 *Nouveau manuel de dépouillement et d'exploitation de l'état civil ancien*. Paris: L'Institut National d'Études Démographiques.
- Government of Canada.
 1910 Sessional Paper No. 27, Edward VIIA: 14, 44–45, 114.
 1915 Sessional Paper No. 27, George V (A): 16–17.
 1916 Sessional Paper No. 27, George V (A): 55.
 1917 Sessional Paper No. 27, George V (9–10): 18–19.
 1925 Sessional Paper No. 27, George V (A): 37.
- Graham-Cummings, C.
 1967 Health of the Original Canadians, 1867–1967. *Medical Services Journal* (Feb): 115–66.
- Graves, C.
 1969 *Invasion By Virus*. London: Icon Books.
- Hader, J. M.
 1986 The Effects of Epidemic Illnesses on the American Indians. *Western Canadian Anthropologist* 3: 19–34.
- Hallowell, A. F.
 1955 *Culture and Experience*. Philadelphia: University of Pennsylvania Press.
- HBCA (Hudson's Bay Company Archives, Winnipeg, Canada)
 1917–1922 B.239/a/190, York Factory Post Journal.
 1917–1924 B.283/a/8, God's Lake Post Journal.
 1918 A.74/48 “Annual Reports from District Officers—Outfit 1918 (formerly HBCA D.FTR.9)
 1918–1922 B.156/a/44, Oxford House Post Journal.
 1918–1923 B.154/a/87, Norway House Post Journal.
 1926 A.95/53, fo. 6–33. Health and Disease at the Norway House—Indian Agency. Stone, E. L.
- Henige, D.
 1990 Their Numbers Become Thick: Native American Historical Demography as Expiation. *In* *The Invented Indian: Cultural Fictions and Gov-*

- ernment Policies. J. A. Clifton, ed. Pp. 169–91. London: Transaction Publications.
- Henry, L., ed.
1967 *Manuel de demographie historique*. Genève-Paris: Librairie Droz.
- Herring, D. A.
1984 Tuberculosis Mortality Patterns in Three Manitoba Indian Communities: 1900–1929. Paper presented at the First Annual Meeting of the Canadian Association of Medical Anthropologists, Montreal.
1989 Diffusion of the 1918–19 Flu Epidemic in Manitoba Native Communities. Paper presented at the 17th Annual Meeting of the Canadian Association for Physical Anthropology, Vancouver, BC.
1990 The 1918 Flu Epidemic in Manitoba Aboriginal Communities: Implications for Depopulation Theory in the Americas. Paper presented at the American Society for Ethnohistory Meetings, November, Toronto.
- Higgins, E.
1982 *Whitefish Lake Ojibway Memories*. Cobalt, Highway Book Shop.
- Hollingsworth, T. H.
1979 Background Paper to First Theme: An Introduction to Population Crises. *In* *The Great Mortalities: Methodological Studies of Demographic Crises in the Past*. H. Charbonneau and A. Larose, eds. Pp. 17–20. Liege, France: Ordina Editions.
- Johansson, S. R.
1982 *The Demographic History of the Native Peoples of North America: A Selective Bibliography*. *Yearbook of Physical Anthropology* 25: 133–52.
- Kaplan, M. M., and R. G. Webster
1977 The Epidemiology of Influenza. *Scientific American* 237(6): 88–106.
- Krech, S.
1983 Disease, Starvation, and Northern Athapaskan Social Organization. *American Ethnologist* 5: 710–32.
- Krotz, L.
1990 *Indian Country*. Toronto: McClelland & Stewart Ltd.
- Levine, D.
1976 The Reliability of Parochial Registration and the Representativeness of Family Reconstitution. *Population Studies* 30(1): 107–22.
- Lilienfeld, A. M., and D. E. Lilienfeld
1980 *Foundations of Epidemiology*, 2nd ed. New York: Oxford University Press.
- MacDougall, H.
1985 The Fatal Flu. *Horizon Canada* 8(8): 2089–95.
- Maclean, J. S.
1925 James Evans—1801–1846: Inventor of the Cree Syllabic System. *The Beaver* 6(1): 18–19.
- MacMahon, B., and T. H. Pugh
1970 *Epidemiology*. Boston: Little, Brown.
- Manitoba Provincial Board of Health.
1919 *Annual Report for 1918*. Winnipeg, MB.
- Maurizi, C. P.
1985 Why Was the 1918 Influenza Pandemic so Lethal? The Possible Role of a Neurovirulent Neuraminidase. *Medical Hypotheses* 16: 1–5.

- McGrath, J.
 1988 Social Networks of Disease Spread in the Lower Illinois Valley: A Simulation Approach. *American Journal of Physical Anthropology* 77:483–96.
 1991 Biological Impact of Social Disruption Resulting from Epidemic Disease. *American Journal of Physical Anthropology* 84: 407–20.
- McNeill, W. H.
 1977 *Plagues and People*. New York: Doubleday.
- Mielke, J. H., and K. J. Pitkanen
 1989 War Demography: The Impact of the 1908–09 War on the Civilian Population of Åland, Finland. *European Journal of Population* 5: 373–98.
- PAM (Provincial Archives of Manitoba, Winnipeg, Canada)
 1840–1889 GR 1212, Item 10 Wesleyan Methodist, Norway House Mission, Register of Baptisms.
 1859–1946 MGII A11 Parker, John E. Correspondence, Data, and Pictures Re: Steamboats of Manitoba.
 1902–1930 MG 8 B47 O'Reilly, Anna M. Notes, Re. History of The Past-Le Pas, Northwest Territories—later Manitoba (Reminiscences re: The LaRose Family and The Pas).
 1952–1957 MGII Miscellaneous Copies of Steamboat Bill of Facts, P4675, fo. 9. Norway House Indian Band. Rossville Methodist Day School attendance records.
- Patterson, K. D.
 1986 *Pandemic Influenza 1700–1900*. Totowa, NJ: Rowman & Littlefield.
- Pettigrew, E.
 1983 *The Silent Enemy. Canada and the Deadly Flu of 1918*. Saskatoon, SK: Western Producer Prairie Books.
- Piché, V., and M. V. George
 1973 Estimates of Vital Rates for the Canadian Indians, 1960–1970. *Demography* 10(3): 367–82.
- Pitkanen, K.
 1977 The Reliability of the Registration of Births and Deaths in Finland in the Eighteenth Century: Some Examples. *Scandinavian Review of Economic History* 25(2): 138–59.
- Ramenofsky, A. F.
 1987 *Vectors of Death: The Archaeology of European Contact*. Albuquerque: University of New Mexico Press.
- Ravenholt, R. T., and W. H. Foegen
 1982 1918 Influenza, Encephalitis Lethargica, Parkinsonism. *The Lancet*, Oct. 16: 860–64.
- Ray, A. J.
 1974 *Indians in the Fur Trade*. Toronto: University of Toronto Press.
 1976 Diffusion of Disease in the Western Interior of Canada, 1830–1950. *Geographical Review* 66(2): 139–57.
- Reff, D. T.
 1991 *Disease, Depopulation, and Culture Change in Northwestern New Spain, 1518–1764*. Salt Lake City: University of Utah Press.
- Reynolds, H. T.
 1977 *The Analysis of Cross-Classifications*. London: Collier, Macmillan.

- Roberts, L.
1989 Disease and Death in the New World. *Science* 246: 1245–47.
- Romaniuk, A., and V. Piché
1972 Natality Estimates for the Canadian Indians by Stable Population Models, 1900–1969. *Canadian Journal of Sociology and Anthropology* 9: 1–20.
- Ross, W. S.
1977 Whaling and the Decline of Native Populations. *Arctic Anthropology* 14(2): 138–59.
- Sattenspiel, L., and C. Powell
1993 Geographic Spread of Measles on the Island of Dominica, West Indies. *Human Biology* 65: 107–29.
- Shkilnyk, A. M.
1985 A Poison Stronger Than Love: The Destruction of an Ojibwa Community. New Haven, CT: Yale University Press.
- Smith, W., C. H. Andrewes, and P. P. Laidlaw
1933 A Virus Obtained from Influenza Patients. *Lancet* 2: 66–68.
- Snow, D. R., and K. M. N. Lanphear
1988 European Contact and Indian Depopulation in the Northeast: The Timing of the First Epidemics. *Ethnohistory* 35(1): 15–33.
- Sogner, S.
1979 Nature and Dynamics of Crises (Including Recent Crises in Developing Countries). In *The Great Mortalities: Methodological Studies of Demographic Crises in the Past*. H. Charbonneau and A. Larose, eds. Pp. 311–32. Liège, France: Ordina Editions.
- Stone, E. L.
1925 Tuberculosis among the Indians of the Norway House Agency. *The Public Health Journal* 16(2): 76–81.
1926 Health and Disease at the Norway House Indian Agency. *PAM HBCA*. A/95/53, pp. 6–33.
- Stuart-Harris, C. H., G. S. Schild, and J. S. Oxford
1985 *Influenza: The Viruses and the Disease*. 2nd ed. Baltimore, MD: Edward Arnold.
- Taylor, S.
1977 Sociocultural Effects of Epidemics on the Northern Plains, 1734–1850. *Western Canadian Journal of Anthropology* 7(4): 55–81.
- Thacker, S. B.
1986 The Persistence of Influenza A in Human Populations. *Epidemiologic Reviews* 8: 129–42.
- Thornton, R., T. Miller, and J. Warren
1991 American Indian Population Recovery Following Smallpox Epidemics. *American Anthropologist* 93(1): 28–45.
- Tilly, C.
1981 *As Sociology Meets History*. Toronto: Academic Press.
- Turk, D. C., I. A. Porter, B. I. Duerden, and T. M. S. Reid
1983 *Medical Microbiology*. Toronto: Hodder and Stoughton.
- Ubelaker, D. H.
1988 North American Indian Population Size, AD 1500 to 1985. *American Journal of Physical Anthropology* 77: 289–94.

- 1992 North American Indian Population Size: Changing Perspectives. *In* Disease and Demography in the Americas. J. W. Verano and D. H. Ubelaker, eds. Pp. 169–76. Washington, DC: Smithsonian Institution Press.
- UCCA (United Church of Canada Archives, Winnipeg, Canada).
 1894–1926 81-32 God's Lake Narrows Burial Register.
 1918–1951a 78-27 Fisher River Burial Register.
 1918–1951b 78-28 Oxford House Burial Register.
- Verano, J. W., and D. H. Ubelaker, eds.
 1992 Disease and Demography in the Americas. Washington, DC: Smithsonian Institution Press.
- Walters, J. H.
 1978 Influenza 1918: The Contemporary Perspective. *New York Academy of Medicine* 54(9): 855–64.
- Warkentin, J., and R. I. Ruggles
 1970 *Historical Atlas of Manitoba*. Winnipeg: Historical and Scientific Society of Manitoba.
- Wilkinson, L.
 1986 SYSTAT. Evanston, IL: SYSTAT, Inc.
- Wrigley, E. A., ed.
 1966 *An Introduction to English Historical Demography from the Sixteenth to the Nineteenth Century*. London: Wiedenfeld & Nicolson.
 1973 *Identifying People in the Past*. London: Edward Arnold.
- Wrigley, E. A.
 1978 Births and Baptisms: The Use of Anglican Baptism Registers as a Source of Information about the Numbers of Births in England before the Beginning of Civil Registration. *Population Studies* 31(2): 281–312.
- Young, T. K.
 1988a *Health Care and Culture Change*. Toronto: University of Toronto Press.
 1988b Are Subarctic Indians Undergoing the Epidemiologic Transition? *Social*
- Zubrow, E.
 1990 The Depopulation of Native America. *Antiquity* 64: 754–65.