1918-1919 INFLUENZA PANDEMIC AT NORWAY HOUSE AND FISHER RIVER, MANITOBA

DIFFERENCES IN THE EXPERIENCE OF THE 1918 – 1919 INFLUENZA PANDEMIC AT NORWAY HOUSE AND FISHER RIVER, MANITOBA

By

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Abstract

This thesis discusses the impact of the 1918 influenza pandemic at Norway House and Fisher River, Manitoba. Despite sharing similar overall mortality rates during the pandemic, the two communities showed substantial differences when the distribution of deaths are examined at the family level. Reconstituted family data show that deaths were more tightly clustered within a small number of families at Norway House, while at Fisher River they were distributed amongst more families. Adults perished more often at Norway House than Fisher River. Historical documentation suggests, moreover, that the day-to-day functioning of Norway House was more severely disrupted than was the case for Fisher River. I argue that the differences in the family distribution of mortality at the two communities is linked to differences in social organization and, specifically, to the presence or absence of the Hudson's Bay Company. To test this hypotheses the data are examined using aggregate techniques, reconstituted family data and a technique outlined in Scott and Duncan's 2001 work.

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Chapter 1 Introduction

This thesis was written at a very interesting time in the exploration of the influenza pandemic of 1918 –1919. Up until just over a year ago this pandemic was viewed by the majority of scholars as simply forgotten. According to Alfred Crosby (1989), "The average college graduate born since 1918 literally knows more about the Black Death of the fourteenth century than (of) the World War 1 pandemic" (1989: 314-315). According to Tomes (1998), North America has been in an era of viral panic since the emergence of the HIV/AIDS virus in 1985. Although the overall perception of the new microbial menaces is focused primarily on viral pathogens (Tomes 2000: 194), influenza still remained on the periphery of public interest for the populace seemed to be obsessed with other newly emerging pathogens. The public, however, would soon come to fear influenza after a new microorganism, likened by many investigators to influenza, appeared in North America.

Severe Acute Respiratory Syndrome (SARS) arrived in North America on the 13th of February 2003 bringing with it new perceptions of vulnerability to person-toperson transmitted viruses. This new virus had an incubation period which ranged anywhere from 2 to 11 days (Tsang et al. 2003: 1), with transmission patterns that indicated droplet or contact related infection (Poutanen et al. 2003: 1). Symptoms for SARS include fever (temperatures in excess of 38°C for over 24 hours), rigor, dry cough, dyspnea, malaise, headache, hypoxemia, and shortness of breath (Tsang et al. 2003: 1), a description many medical practitioners and media personalities equated with influenza.

As a result a new fascination with emerging viruses developed and a population already engaged in an era of viral panic latched on to the idea of an imminent influenza pandemic. This fear sparked new interest in understanding past pandemics of influenza and the publication of a number of studies that detail the social conditions that gave rise to the pandemic (see for example Barry 2004, Phillips and Killingray 2003) as well as works that strove to decode the pandemic at the molecular level (see for example Kolata 2001; Davies 2000).

As a result of this new fascination, information regarding the 1918 – 1919 influenza pandemic has been transforming quickly as estimates of a potential international death toll shifted from 21.5 million in 1927 (Johnson and Mueller 2002: 108) to current estimates of 100 million (Phillips and Killingray 2003: 4). This thesis began as an exploration of a little remembered pandemic and has evolved into a study of the family context of mortality that contributes to this growing body of literature on Spanish Influenza¹.

¹ Spanish Influenza is the popular term used for the 1918 – 1919 influenza pandemic. This name was adopted because the first reported cases of the epidemic came from Spain (Afkhami 2003: 368).

This thesis explores the dissemination of influenza within Aboriginal communities in Canada during the pandemic of 1918 – 1919. The project focuses on two

Aboriginal communities: Fisher River and Norway House. Both are located in the vicinity of Lake Winnipeg, Manitoba (see figure 1.1). Norway House's population was comprised predominantly of Swampy Cree/ Métis. Geographically, the settlement is located at the northeast end of Lake Winnipeg in the central Canadian subartic, about 450 km north of Winnipeg (Herring 1994: 75). Its prime location led it to become a

prominent Hudson's Bay Company trading post within the region. Fisher River First Nation is located on the

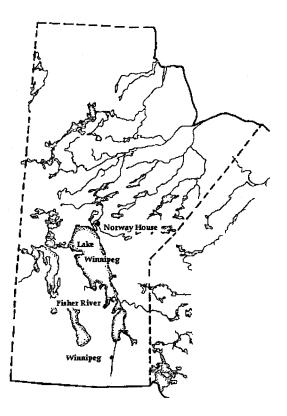


Figure 1.1 – Map of the Province of Manitoba Outlining Study Communities in Relation to Winnipeg. Adapted from Moffat 1992: 7

western side of Lake Winnipeg, 200 km north of Winnipeg (Moffat and Herring 1999: 1823). This site once housed a HBC post, which was closed in 1893 (Moffat and Herring 1999: 1825). By concentrating on these two communities I aim to study how influenza spread within and between families in these two communities. To achieve this end I utilize primary source data compiled and collected by Dr. Ann Herring and her students.

I also employ epidemiological concepts outlined by Scott and Duncan (2001: 21 - 44; 228 - 248).

The following quote helps to explain the theoretical underpinnings that shape the scope of this thesis. According to Dubos (1977: 34),

Exposure to one of several viruses is a *necessary* condition for the development of the common cold but not a *sufficient* condition. Exposure results in disease only when the exposed person is in a receptive state. This receptivity is in turn affected by the season, the weather, and almost certainly by a host of other ill-defined factors...

One of the central tenets of this work is not to forsake the richness of people's lives but rather to help elucidate the "ill defined" conditions that help to shape the disease experience. In part, this is achieved by exploring the conditions that were present prior to the disease experience as well as delving into "the way people *really* approach illness and cope with death" (Nations 1986: 97). In this way, it is possible to gain a better understanding of the interactions between microscopic agents and human beings and how this relationship in turn affects "the process by which behavioural choices are sorted out in a developing sociocultural system."(Alland 1970: 52).

In order to understand the conditions that were present prior to the influenza epidemic this thesis examines the political and economic conditions that influenced everyday life at Norway House and Fisher River. One of the central features of the political economic approach used in this thesis is to understand "power, not as socially diffuse or "capillary" but as structured by and in terms of the control of social labor" (Goodman & Leatherman 1998: 79).

This thesis also strives to study how existing local systems of community organization at Norway House and Fisher River influenced the effects of the epidemic at each location. As Sattenspiel and Herring state "the key to understanding the effects of the Spanish flu ... lies within the domain of social structure and community organization" (Sattenspiel and Herring1998: 112). To achieve this end biological, ecological and sociocultural systems must be explored and understood within the context of the individuals being studied (see Inhorn and Brown 1990: 31 – 55 for a detailed discussion). Lastly, it is important to understand how communities construct and respond to the disease experience at the individual and community level. Herzlich and Pierret (1985: 147) state, "The conceptions that a society forms about sick people – orient, organize and legitimate social relationships and, to a certain degree, 'produce' the 'reality' of sick people".

This research is based primarily upon reconstituted family data from parish registries for these two communities, along with Treaty Annuity Pay Lists for Norway House. It attempts to take current thinking within the field of infectious disease research a step further by using the influenza pandemic of 1918 – 1919 to focus on individual and family health, as opposed to gauging the general well being of a community. This is an important endeavour as the vast majority of studies of the transmission of influenza attempt to understand the macro environment in which the virus flourished (see Crosby 1989 and Kolata 2000 for examples). They aim to reconstruct how societies transform at a city, province/state or country level, rarely taking into account the experiences of the individual, family or household (See for example Barry 2004 and Kolata 2000).

Using the individual and household as the primary units of analysis provides a researcher with numerous advantages. One of the most valuable tools to the historical demographer is parish registry data. This is information based on individual accounts of birth, death and marriage. Thus, while inferences can be made about community-level experiences, the individual experience can be reasonably examined. A focus on the individual also helps to illuminate aspects of a disease, such as poverty, social structure and social class, that contribute to individual differences in the experience of disease.

This thesis is also intended to be a companion for research that has been carried out for the region (see Herring 1994 a and b, Moffat 1992, Moffat and Herring 1999, Sattenspiel and Herring 1998, Herring and Sattenspiel 2003, Sattenspiel et al. 2000). It aims to extend their work by focusing in on the local level/family experiences of the 1918 – 1919 influenza pandemic.

The aims of this research are therefore:

- to document and interpret the distribution of deaths due to influenza during the 1918 – 1919 pandemic at the family level;
- 2. to analyze the dissemination patterns of influenza from family to family; and
- to develop a profile that details how mortality was structured within the two study communities with special reference to how local political, economic and social structures influenced deaths within the Norway House and Fisher River populations.

This thesis is organized into eight chapters. Chapter 2 provides background information on the study area as well as a brief synopsis of details of the virus, pandemic

and the epidemic within each of the two communities. Chapter 3 describes and evaluates the documents utilized for this thesis, while Chapter 4 outlines the three main analytical methods utilized. Chapters 5 through 7 each present a different method of analysis, which allow this thesis to compare how influenza was experienced within each of these communities. Chapter 5 is concerned with aggregate data and findings, providing the reader with a sense of the overall patterns of mortality experienced within each community. Chapter 6 employs techniques outlined by Scott and Duncan (2001), which permit an in-depth analysis of the spread of the microorganism within and between families. The pattern of spread of the pathogen is estimated by examining the latent and infectious periods for the virus and deciphering a pattern of person-to-person transmission from them. Owing to a lack of available information for Norway House, only Fisher River is included in this analysis. Chapter 7 utilizes family reconstitution methodology to create a detailed picture of mortality within the family context in each community. Chapter 8 is a synthesis of information derived from the preceding three chapters, a discussion of the pertinent findings of this thesis, and offers suggestions for future research.

The purpose of this thesis is to demonstrate that mortality from the 1918 – 19influenza pandemic was structured differently at Norway House than at Fisher River. This, I hypothesize, results from the presence of a Hudson's Bay Company trading post at Norway House or, more aptly, from how this institution restructured daily life and family interactions in this community. Although this speculation is advanced with caution, this

thesis demonstrates clear differences in the distribution of mortality at these two locations.

Chapter 2 Historical Context

The History of the HBC and its Consequences for Indigenous Social Structure

The aim of this chapter is to introduce some of the key elements that are examined within this thesis. This section begins by looking at the history of the Indigenous people that occupied the two study areas. This overview mainly focuses on the formation of the relationship between the Aboriginal inhabitants of the communities and European settlers. Special attention is paid to the foundation and development of the Hudson's Bay Company within the region to the introduction of pandemic influenza within each of the communities. There is also a section devoted to exploring the subsequent relationship that developed between these communities and the Government of Canada. A brief review of the basic subsistence strategies employed by the inhabitants of Norway House and Fisher River at the time of the epidemic is also provided.

The next section outlines the characteristics of the influenza virus and gives a brief synopsis of the 1918 – 1919 pandemic. Lastly, a brief summary is provided of the microorganism's entry into the Province of Manitoba as well as into Norway House and Fisher River.

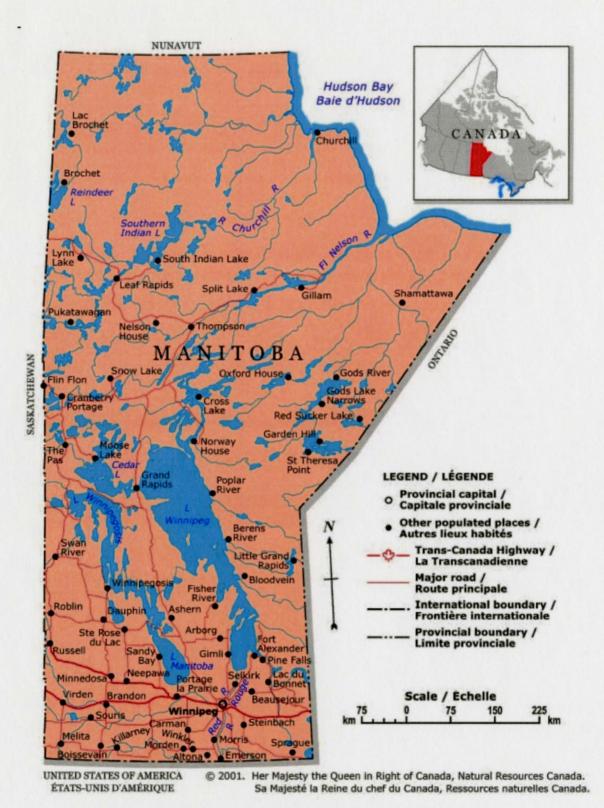


Figure 2-1 Map of Manitoba. Source: The National Atlas of Canada

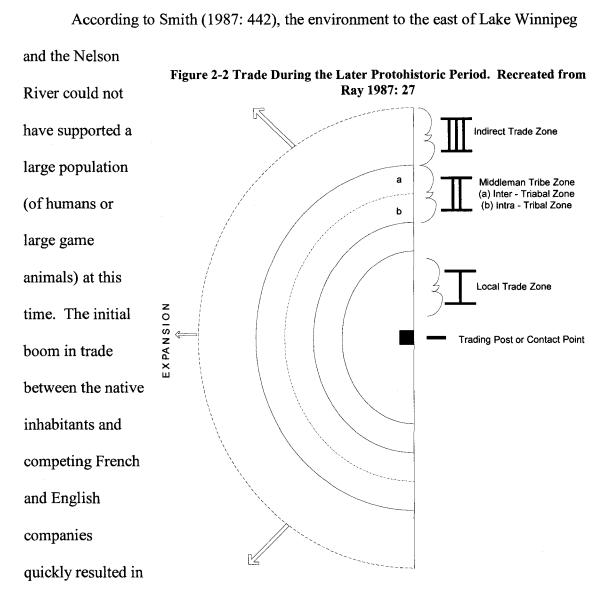
Contact and Competition

The communities explored in this study are primarily Swampy Cree, also referred to as Wood Cree or Muskegon (Skinner 1912: 9). Some argue that the Western Woods Cree inhabited the areas to the east of Lake Winnipeg and the Nelson River prior to European contact, although others maintain that this estimated land use prior to contact is far too restrictive (see Smith 1987).

In order to understand the complexities of the relationship between the Cree and the Hudson's Bay Company (HBC), the historical context within which it was forged must be fully understood. The Hudson's Bay Company was founded in 1670 (Heidenreich and Ray 1976: 34). After an initial period of adjustment, Indians are said to have willingly taken on the specialized roles of provisioners, traders and trappers (Ray 1978: 26). Tough (1990) states, "the observations of the HBC employees indicate that Indians understood price concepts and made special efforts to improve their incomes" (1990: 391). Not long after the English established the HBC the French began to expand their St. Lawrence-based fur-trading operation and by the late 1680's they had erected a number of small posts expanding into the north and west as far as Lake Nipigon and Rainy Lake (Ray 1978: 28).

This was also the period in which York Factory was founded and the history of Manitoba as an HBC territory was sparked. York Factory (or York Fort as it is also referred to) was instituted in 1682 (Smith 1981b: 137) when Pierre-Esprit Radisson and Médard Chouart Sieur Des Groseilliers, two French-Canadian adventurers, came upon the site (Alcock 1920: 68). They are credited with the formation of the Hudson's Bay

Company as they led the first expedition to the Bay and built Fort Bourbon, the HBC's first post, on the northwestern bank of the Hayes River (Alcock 1920: 68). The Cree who resided at York Factory occupied the full boreal forest where the major game animals were moose and woodland caribou. Beaver and bear were also hunted for fur and food and the latter for use in ritual ceremonies (Smith 1981b: 135).



the overexploitation of local resources and a shift in hunting practices away from the

Bay. Figure 2.2 is recreated from Ray (1978: 27) and serves to show how trade was structured during the later protohistoric period. According to Ray (1978: 26) the protohistoric period refers to the transitional period within which Aboriginal inhabitants had access to European goods but limited contact with European settlers. This shift inland created a niche for Aboriginal middlemen. These individuals traded with trappers from outlying areas and brought goods back to the trading post or contact point.

The Treaty of Utrecht in 1713 ended the rivalry between the French and English over control of the Hudson Bay leaving the region under the authority of the Hudson's Bay Company (Ray 1978: 28). However this did not cause competition to end, rather it provoked the French to pursue more aggressively the creation of inland posts. In the later 1720s and early 1730s the French, led by La Vérendrye, opened a number of posts in the area occupied by the middlemen portion of the York Factory settlement (Heidenreich and Ray 1976: 41) with the hopes of cutting off the HBC from its hinterland (Ray 1978: 28). By 1751 the French had established inland posts as far west as the foothills of the Rocky Mountains and the English still only had one inland post at Henley House (Smith 1981: 258). French trade peaked in 1763 with the conquest of New France and the signing of the Treaty of Paris, which was accompanied by the arrival of "Montreal peddlers" into the west (Smith 1981: 258).

According to Bishop (1972: 66) "dependence upon the trading post came early and was associated with a relatively poor ecological adjustment after contact". By 1793 this reality was truly taking form. Hanks (1982: 109) notes that between the 1793 and 1830 big game steadily declined, causing many Cree to become fishermen and small

game hunters. Beardy and Coutts (1997: xxv) add that the effects of diseases were also significantly altering population dynamics during this time. This period (beginning in 1801) also marks the establishment of an HBC post at Norway House (Herring 1994: 75). Its strategic position on the northern end of Lake Winnipeg and the southern end of the Nelson River trade axis linking the west, northwest, northeast and south (Ray 1976: 156, Herring 1994: 75) would make it an important district headquarters in subsequent decades (Tough 1990: 395).

The Creation of Dependency: The Rise of the HBC and the Marginalization of the Swampy Cree

At the same time as the ecological effects were beginning to depress Indigenous populations, political and economic consequences also were taking hold. The rivalry between the French's North West Company and the English's Hudson's Bay Company, which in many ways had contributed to Indigenous autonomy, ceased in 1821 when the two companies became one (Smith 1981: 258) and a monopoly was created. Competing posts were shut down as were posts that did not demonstrate profitable yields causing Indigenous groups engaged in the fur trade to become localized and oriented around a specific post (Smith 1981: 258). The company also began to marginalize Indian trappers "by reducing the buying price of furs and increasing the selling price of trading goods" (Tough 1990: 389). European trade goods by this point were no longer viewed as luxuries but rather necessities. Items like kettles, knives, guns, blankets and twine were things on which Indigenous peoples had come to depend (Bishop 1972: 60). More and

more natives had become specialized in trapping and trading, so much so that they had allowed their own subsistence needs to fall by the way side, again causing them to rely on the supplies sold by the Hudson's Bay Company (Hanks 1982: 107). The implementation of a debt system that consisted of the HBC issuing equipment to trappers in the fall with the onus of the return of fur in the spring (Hanks 1982: 107) resulted in a state of "company welfarism" (Tough 1990: 390).

The once autonomous Indian was now an unwitting servant of the HBC system. It was also around this time, the mid-nineteenth century, that missionary activity began in earnest (Smith 1981: 259). Because of its prominent position, Norway House became the headquarters for the British Wesleyan Methodist Missionary Society, whose aim was to Christianize the northwest Aboriginals (Herring 1994: 75). These efforts were so intense that, nominally at least, by the twentieth century the once diverse Cree population was largely Christian (Smith 1981: 259).

By the 1870s the Cree way of life had become a shell of what it had once been. Fur yields were no longer abundant after decades of protracted decline (Tough 1990:385) causing natives to return to the post during winter to beg for provisions (Hanks 1982:107), further entrenching them in a cycle of debt with the HBC. It was during this time that Fisher River was founded. In 1876 ninety Swampy Cree families moved south from Norway House to escape the hardships that were plaguing them. By 1893 the Hudson's Bay Company closed its post at Fisher River and these former traders and trappers shifted to wage labour, working at lumber camps, fisheries, saw mills and on steamboats (Moffat and Herring 1999: 1825).

Factoring in the Canadian Government

The Hudson's Bay Company served as the *de facto* government as well as the country's chief commercial enterprise. According to Peake (1972: 72) "as the government... the company (HBC) had some responsibility for public order and wellbeing and for the administration of justice". Government of Canada involvement came in the decades following the cession of Hudson's Bay Company territories to the Dominion of Canada in 1870 (Smith 1981: 259). This period was marked by the establishment of a number of treaties (Figure 2.3). Of primary concern for this thesis is Treaty 5.

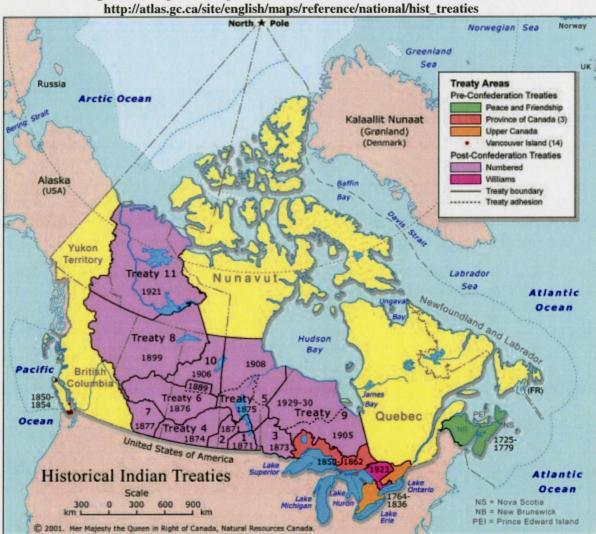


Figure 2-3 Map Outlining Historical Indian Treaties Boundries. Source: http://atlas.gc.ca/site/english/maps/reference/national/hist treaties

During the first few years of steam navigation the Department of the Interior discovered that valuable minerals and timber were located in the vicinity of Lake Winnipeg and "For these reasons it was essential that the Indian title to all territory in the vicinity of the Lake should be extinguished so that settlers and traders might have undisturbed access to its waters, shores, islands, inlets and tributary streams" (Indian Affairs Annual Report 1875: viii). Treaty number 5 was signed on a number of dates beginning on September 20, 1875 at Berens River and ending on September 7, 1876. Norway House signed the document on 24th of September 1875.

For the purposes of this thesis the two most important features of this document are the stipulations for the creation of the Fisher River Band as well as the allocation of treaty payments. Tough points out that these treaty payments further threatened Aboriginals' way of life as the payments from the Department of Indian Affairs "challenged the old system of exchange and distribution" (Tough 1990: 397).

Subsistence Strategy and Status of Norway House Prior to the Pandemic

In its heyday Norway House occupied a position of importance within the trade network second only to York Factory. Goods that had been shipped from England in the previous summer to York Factory were repackaged in the winter and shipped to Norway House where they were stored over winter, ensuring supplies over the difficult season; in the spring they were then shipped to Methye Portage for further distribution (Alcock 1920: 71). The post rose to prominence throughout the nineteenth century so that by 1820 it had "become the nexus of HBC provisioning activities in the region" (Herring 1994: 75). However, Norway House was not immune to social upheaval and environmental depletion and, much like the communities surrounding it, by 1918 its halcyon days had long past.

The annual cycle at Norway House remained similar to post-contact patterns elsewhere in the Subarctic, with summer concentration and winter dispersal into traplines

in the form of small hunting groups or local bands serving as the primary social unit (Smith 1981b: 147). However Waldram et al. (1995: 60) add that Norway House was basically 'trapped out' by the mid-nineteenth century causing some trappers to travel as far as three hundred miles to hunt fur.

Subsistence Strategy and Status of Fisher River Prior to the Pandemic

As previously stated, Fisher River's role as an HBC post was short lived but although its position as a post was not lucrative, reports to the Department of Indian Affairs make it appear as though the community was faring well as an agricultural centre. A report for the year ended June 30, 1906 reads "at Fisher River, the most desirable, reserve in the agency, about 300 head of cattle are kept, as well as a number of horses" (Indian Affairs Annual Report 1906: 87). This contrasts with the few cattle housed on most other reserves. In 1913 the Indian Agent states that "Many of the homes in the Peguis and Fisher River reserves are a credit to the occupants" (Indian Affairs Annual Report 1913: 88). Moffat and Herring (1999: 1825) state, however, that although agriculture was practiced to some extent, work in lumberyards was far more lucrative.

The annual cycle at Fisher River differed from that of communities engaged in the fur trade. As opposed to heavy reliance on trapping and trade networks with the HBC, the Fisher River subsistence strategy relied on a mixed economy with a strong emphasis on wage labour (Moffat 1992: 10). Moffat (1992) also stresses the importance of lumbering and fishing to the Fisher River community. The primary social consequence

that is created within these different modes of subsistence is that Norway House was a much more mobile community. As traplines were overexploited trappers had to travel further away from the post. Herring (1994b: 77) states that at the time of the pandemic some families had to travel as far as 300 miles to reach their family trapline. A report from 1910 records that at Fisher River there was "some very fine timber" (Indian Affairs Annual Report 1910: 97) located in the vicinity of Fisher River and it can therefore be assumed that mobility was somewhat more confined here.

The Great Influenza

The year of 1918 was not the first time influenza rocked the Keewatin District². According to Ray (1976), who explored the diffusion of disease throughout the region between 1830 and 1850, epidemics of influenza were recorded in 1835, 1843, 1845, and 1850 (Ray 1976: 142). He goes on further to state that Norway House, owing to its key position within the transport system, was one of the unhealthiest communities in the region (Ray 1976: 156). Although the 1918 strain of influenza was by no means the first epidemic to hit this area there was something peculiar about this particular virus.

Influenza Epidemiology

The Influenza virus genome consists of eight separate pieces of ribonucleic acid (RNA). Due to the segmented nature of these RNA strands, differing flu viruses can very

 $^{^{2}}$ The Keewatin District refers to the majority of Manitoba North the name given to the region from the 1870s to 1912 (Friesen 1992: 44)

easily exchange genes to produce "hybrid progeny viruses with bits of RNA from each parent virus" (Laver et al., 2000: 2). There are two glycoprotein molecules that define the virus: *hemagglutinin* and *neuraminidase*.

Hemagglutinin is a triangular, rod-shaped molecule whose function is to attach the virus onto its host's cell. It fuses to cells that contain sialic acid, which allows the RNA of the virus to produce thousands of new virus particles. The second component is *neuraminidase*, which is a mushroom-shaped enzyme whose job it is to remove all the sialic acid from the newly infected cell. This enables the virus to escape the primarily infected cell and spread



Figure 2-4 Microscopic Image of the Influenza Virus. Source: DeSall 1998

throughout the rest of the body in order to contaminate subsequent cells with which it comes into contact (Laver et al., 2000: 2).

There are three types of Influenza: A, B and C. Type C is relatively uncommon. Type B has given rise to epidemics but it only infects humans, and outbreaks have been predominantly limited to children (Beveridge 1977: 9). Type A is perhaps the most interesting, common and adaptable, and was responsible for the 1918 – 1919 pandemic. Its lethality rests in the fact that it not only affects humans, but pigs, horses, seals, whales and many kinds of birds as well (Laver et al., 2000: 2).

Influenza A may always be endemic to our planet because the virus has a natural host in wild water birds. Populations of ducks found in northern Canada appear to carry

most kinds of Influenza A, acting as a reservoir for the disease. Although infected with the virus, no harm is done to the ducks, possibly because they carry the pathogen in their intestinal tracts. It is here that strains of Influenza A survive and are able to recombine (Laver et al., 2000: 5). The virus may be transmitted to other mammals through intestinal excretions. Another complication adding to the complexity of the Influenza A virus is the role of the pig, which can act as a mixing vessel for the virus. Pigs can become infected with avian influenza virus after the consumption of duck droppings; simultaneously they can catch the human virus from a farmer's cough or sneeze. If the pig becomes infected with both viruses at the same time, the genetic material can mix, which has the potential to create a new strain that may find its way back to the respiratory tract of a farmer and thus spread a novel influenza to the human population (Scholtissek 1992: 4).

Influenza is contracted when a 'healthy' person inhales the aerosolized droplets containing the virus that have been expelled via a cough, sneeze or any other method that could launch the virus out of the infected person's respiratory tract (Herring 1994: 368). The incubation period of this ailment is incredibly short, ranging from twenty-four to forty-eight hours (Ohadike 1991: 1397). People who are only mildly hindered by the virus infect the susceptible members of their community most effectively because the disease does not debilitate them. Symptoms of influenza include headaches, fever, hacking cough, feelings of chilliness, malaise, pain in the legs and kidneys, muscular pain, vomiting, dizziness, profuse sweating, sneezing, and labored breathing (Herring 1994: 367; Herring 2000: 7; Ohadike 1991: 1397; Laver et al., 2000: 1).

1918 – 1919

The influenza pandemic of 1918 – 1919 was possibly the greatest infectious disease-related catastrophe that the world has ever seen. The global death toll lies somewhere between twenty and possibly 100 million people. It is estimated that over a billion people, which would have been equivalent to half the entire human population, were infected with the virus during this twelve month period (Collier 1974: 305). During the height of the pandemic morbidity rates hovered around 50% (Reid et al 2001: 82) with a 2.5% mortality rate reaching as high as 70% in isolated regions (Taubenberger 2003:40).

There are many different hypotheses regarding the origins of the virus that caused this massive outbreak. The most widely accepted view is that the Influenza A strain associated with the 1918 – 1919 pandemic originated somewhere in the United States. Many scholars see Fort Riley³, Kansas, as the start point for this outbreak (see Crosby 1989). There are reports from the fourth of March that the hospital at Camp Funston (Kansas) was swamped with soldiers complaining of flu-like symptoms (Herring 2000: 6). This predates the first wave of illness that swept across the world. Jeffery Taubenberger's team (the US Armed Forces Institute of Pathology) was able to sequence the tissue samples of three exhumed bodies, consisting of two soldiers and an Inuit woman. They sequenced the entire gene for both *hemagglutinin* (HA) and *neuraminidase* (NA) components of the 1918 strain and their analysis showed some very interesting results. They report that the 1918 HA shares characteristics with both the

³ Also referred to as Camp Funston

human clade and swine clade, suggesting that it derived from a combination of the two. On the other hand, the NA gene is most closely related to avian isolates. They go on to say however that the 1918 "sequences share enough characteristics with mammalian isolates to distinguish them from the avian clade" (Reid et al 2001: 85)⁴. It is believed the new influenza strain traveled across the Atlantic with the American troops on the *Leviathan* and arrived in France on October 7, 1918 to start the first of three epidemic waves that would rock the world (Crosby 1989: 125).

There is also a theory put forth by Oxford et al. (2001, 2002) who argue that the pandemic should be renamed French influenza and that the date marking the entry of the elusive influenza strain be re-ascribed to 1916 rather than 1918 (Phillips and Killingray 2003: xix). He and his team point to outbreaks of respiratory disease in France and the UK during the years 1915 - 1917. They claim that in 1916 - 1917 there were outbreaks at army camps at Etaples (France) and Aldershot (United Kingdom) that caused very high mortality among 25 - 35 year olds (Oxford et al. 2002: 112). They suggest that this new interpretation of the dispersal of Spanish Flu is indicative of the characteristics displayed by pandemic influenza. They arrived at this conclusion by looking at the behaviour displayed by the 1957 and 1969 pandemics. The H₂N₂ pandemic of 1957 was isolated in China in February yet did not spread to the rest of the world until July through to September, indicating that a period of 10 months was required for global spread. The influenza A (H₃N₂) pandemic of 1968 spread throughout China in July yet did not reach the UK and Europe for 12 - 14 months (Oxford et al. 2001: 159, Oxford et al. 2002: 113).

⁴ This is a oversimplified summary of their findings, for a more detailed discussion please see Reid et al. 2001, Taubenberger et all. 2000, Reid et al 2000, Worobey 2002, and Phillips and Killingray 2003.

They go on to claim "The protracted period that we postulate for the emergence of the Great Pandemic of 1918, almost 2 years, could be explained by the absence of air travel and the distortion and restriction of travel during the Great War itself" (Oxford et al. 2001: 160).

Through a critical examination of their work a number obstacles toward accepting their hypothesis arise. In their analysis Oxford et al. (2001: 155 - 161) cite Spain as the 'accepted' country of origin for modern day understandings of influenza dissemination. However, there is little credence given to the fact that Spain was the country of origin for the pandemic. Again, the widely accepted hypothesis is that the virus originated in North America or China, with the former being the most likely candidate. It is believed that the misconception of a Spanish flu was a by-product of Spain's involvement, or rather neutrality, throughout the Great War. It has been noted that countries involved in warfare censor information about epidemics occurring on the front as well as back at home. Spain's lack of participation in WWI provided a climate for accurate reporting and the subsequent placement of proactive measures such as guarantine and dissuasion against public gatherings (Echeverri 2003). This point is noteworthy because flu was not reported in Spain until the 29th of June 1918 (Echeverri 2003: 173), some four months after it was recorded in Kansas. As well, Oxford et al.'s (2001; 2002) claim that travel was restricted as a result of the war is a little one sided. Many authors attribute the conditions created by military travel as aiding in the dissemination of the virus rather than acting as a hindrance (see Ewald 1994 and Crosby 1989 for examples).

Regardless of its origins, there is agreement that the influenza pandemic of 1918 – 1919 was characterized by three successive waves. The first wave hit in the spring of 1918. Although it boasted the strain's high morbidity rates, which ranged from 20 – 50% (Reid et al 2001: 82), it produced nothing terribly out of the ordinary in terms of death toll (less than 0.1%) (Taubenberger 2000: 241). The only odd characteristic that it possessed was that rather than 'attacking' the very young and the very old, as influenza is known to do, it instead was striking down individuals in their prime (between the ages of twenty to forty) (Osborn 1977: 5-6). Still, due to the all-consuming nature of war, it was able to sweep by barely noticed. This wave waned by July and the bulk of the deaths were "blamed largely on malnutrition and the general weakness of nerve power known as war-weariness" (Crosby 1989: 27).

The second wave occurred in the autumn of 1918 when the virus is believed to have mutated. There is some debate over the succession of these waves with some scholars believing that this second strain involved novel recombination of the influenza virus and not a mutated continuation of the virus associated with the first wave (Reid et al. 2001, Crosby 1989). Students of the pandemic are quick to note however that in places where both the spring and fall wave hit "victims of the first wave either escaped or experienced only mild illness in the fall" (Reid et al 2001: 82), indicating the reemergence of a remarkably similar virus. This second wave was characterized by unprecedented mortality. It was definitely the most lethal of the three waves, killing up to seventy-four people in one U.S. town in one day (Crosby 1989: 38). Another incredible aspect of the second wave was that it arrived at the ports of Freetown, Sierra

Leone; Brest, France; and Boston, Massachusetts within the span of seven days (Crosby 1989: 37) causing chaos worldwide. This proved that this strain was not only extremely lethal but highly communicable as well.

The third wave came in the winter of 1919. It was much less severe due to the fact that much of the world's population had already been stricken by the sickness. It was, however, evident that this epidemic was tied to the two that came before it because the age at death distribution was similar to the previous two outbreaks (Johnson and Mueller 2002).

It is also interesting to note that while all thee waves showed uncharacteristic mortality within the 20 - 40 age group⁵ a study by Luk et al (2001: 1376 - 1377) shows that there was a negative excess mortality⁶ displayed among elderly individuals. They go on to conclude that this negative excess mortality was likely the result of previous exposure to an influenza virus that was similar to the 1918 strain. There was a reported pandemic of influenza that began in late spring of 1889 and subsided in early 1892 (Reid et al 2001: 82).

Accounting for Abnormality

The plague of influenza that struck in 1918 was much more devastating than the flu epidemics that had preceded it and much more deadly than any ever seen. The

⁵ Although the most commonly expected definition of this age group is 20 - 40 (i.e. Phillips and Killingray 2003, Luk et al 2001, Crosby 1989) some scholars define the group displaying the highest mortality as individuals 15 - 45 (Reid et al 2001, Taunenberger 2003), 25 - 39 (McCracken and Curson 2003) or those 20 to under 65.

⁶ This means that there were fewer deaths in this age category than is usually experienced during a typical experience with this virus.

Spanish flu targeted people in their prime. Healthy young men and women were stricken down all around the world leaving influenza's common targets, the infirm and the young, without the support system they required. Influenza took an unbelievable death toll. In the 1918 – 1919 epidemic, "about twenty percent of the ailing developed pneumonia, sometimes within the short span of one day, and this deadly and uncontrollable complication eventually killed approximately half of those that contracted it "(Herring 1994: 367). This is most likely due to the fact that this virus had originated, or mutated, in swine as has already been discussed. The third factor was, of course, World War I.

Paul Ewald utilizes an approach that integrates "the fundamental principals of evolutionary biology with our knowledge of epidemiology" (Ewald 1994: 13) in order to understand infectious disease within human populations. An oversimplified summary of Ewald's fundamental findings would read: evolutionary relationships between humans and microbes have molded both the parasite and its prey.

He views the virus that sparked the pandemic of 1918 – 1919 as being an evolutionary abnormality, which must have been the product of extraordinary circumstances rather than biological fitness. He attributed the conditions created by trench warfare and the overcrowding of hospitals and transport vehicles as being the determining factors in Influenza A's success. He identifies a general populace that was imuno-comprised both psychologically and physically by the ravages of war, and attributes this to the spread of the pathogen. Ewald terms this mode of thinking as a 'cultural vector hypothesis' which suggests "that most of these influenza deaths were

caused evolutionarily by the war rather than being just coincidental with the war" (Ewald 1994: 115).

In order to illustrate this point Ewald looks at the account of an ambulance driver who recounted his travels during the height of the pandemic. From the front line, to overcrowded evacuation hospitals to troop ships that crated the sick back to their country of origin, this vehicle brought together infected individuals and susceptibles. In this manner, the influenza virus was carried into every crevasse of the war and eventually to the world. Individuals normally debilitated by the microorganism were moved about via these cultural vectors. This enabled them to infect others who too, while the virus incubated within their systems, returned to the trenches and aboard transport vehicles.

The Epidemic In Canada

According to Eileen Pettigrew the first major civilian outbreak occurred in Canada on September 8, 1918 when two students attending Victoriaville College in Quebec came down with a new virulent strain of influenza (Pettigrew 1983: 8). This incident was precipitated by outbreaks of influenza on steamships among servicemen returning home from battle. By the 30^{th} of September the disease had reached the province of Manitoba via a troop train arriving in Winnipeg. By October 3^{rd} two soldiers had fallen from the disease, as well as the province's first civilian death (Pettigrew 1983: 56 - 57). The virus was brought from Winnipeg to Berens River at the end of October by the passenger steamship, Wolverine (Pettigrew 1983: 79).

The first mention of the virus in the Norway House region comes from the Hudson's Bay Company Norway House post journal where the December 03, 1918 entry reads "'Spanish Flu' was also reported at an Indian camp at Clearwater Lake, some two or three days north of Cross Lake" (HBCA 1918 – 1923). The next entry confirms the outbreak at Clearwater Lake and mentions seventeen deaths. Between the 9th of December and Monday December 15^{th} illness is reported at Norway House. The first person mentioned with influenza is the chore boy, Alex Roberts (on the 9th), then Mrs. Kirkness (on the 13^{th}) and Mary Roberts (on the 15^{th}). There is no evidence in the post journal or the Anglican Mission parish records of these individuals succumbing to the virus. On Wednesday, December 18^{th} the Hudson's Bay Company post journal reads, "Roderick Roberts's wife died today, this being the first death from the "Flu" here" (HBCA 1918 – 1923).

The wealth of information for Norway House is not available for Fisher River. This is in large part due to the fact that Fisher River was not part of the Hudson's Bay Company fur trade network and therefore did not have a post, and hence a Factor, who would report daily activities in a post journal. As well, Fisher River was a relatively new community comprised of predominantly agriculturalists and loggers, and would not have prompted much attention from researchers of the fur trade period. Much of the information available for the community comes from the Fisher River Oral History Project undertaken in 1991, and the well maintained records and memoirs of Reverend Frederick G. Stevens (UCCA Frederick Stevens n.d.) and his wife Frances G. Stevens (UCCA Frances Stevens n.d.).

The first influenza death at Fisher River is recorded in the Methodist Church parish records (UCCA 1918 - 1951) as having occurred on the 30th of October 1918, some two months before it appeared at Norway House, or for that matter before it is even recorded in the post journals as plaguing the neighbouring communities of Berens River and Clearwater Lake. The epidemic at Fisher River had actually tapered off before it hit the community of Norway House on the 18th of December 1918. It is possible, however, that this virulent strain of influenza struck from Berens River to the north, and on the opposite shore of Lake Winnipeg, where it was first reported around the end of October when the steamship the *Wolverine* brought with it more than just supplies to the unsuspecting community. There is also a possible connection between the outbreak at Fisher River and The Pas, both locations having experienced their first deaths within a day of one another. We know from the Anglican burial record that the first individual at Fisher River died on October 30th so the flu must have arrived by October 28th at the latest, taking into account the 2-day incubation period, and probably even earlier.

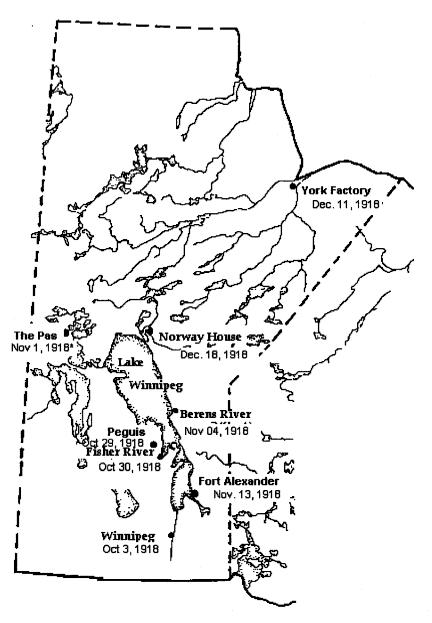


Figure 2-5 Map of Winnipeg Indicating Date of First Death Within Each Community⁷

Figure 2.5 is a map of Manitoba that indicates the first influenza death within several Aboriginal communities. All dates listed were gathered from parish registry data with the exception of The Pas⁵, which was gleaned from newspaper accounts. It appears

 $^{^{7}}$ The date for The Pas identifies the first case; first death is suspected to have occurred on the 6th of November.

that the virus moved from South to North through the province of Manitoba, beginning in Winnipeg with the arrival of a troop ship. Herring (1994: 83) suggests that the epidemic was spread throughout the province along train lines, roads, and water routes.

Exploring Differential Mortality Through Historical Documentation

Earlier it was alluded that the impact of mortality was greater at Norway House than Fisher River. Both the Anglican and Methodist community at Norway House and Fisher River (respectively) experienced the same number of deaths, 38. However at Norway House the overall mortality rate was greater with 183⁸ deaths per thousand compared to Fisher River's 102 deaths per thousand. Although Fisher River's 102⁹ deaths per thousand may appear to be notably less than what was experienced at Norway House this difference is not statistically significant and therefore cannot entirely account for the overall greater impact of the pandemic at Norway House (Herring 1994b: 374).

Newspaper accounts from The Pas discuss in detail the disarray rampant at Cross Lake, Norway House, Pelican Narrows, Beaver Lake, Herb Lake, Lac la Ronge and Stanley yet make no mention of the virus plaguing Fisher River. An article from Friday February 07, 1919 with the header *Heavy Toll of Deaths Among Northern Indians* reads

Influenza is taking away large numbers of Indians in the outlying tribes. The figures show that there have been 316 carried away in the past month, viz." Cross Lake 135; Norway House 107; Red Earth 23; Pelican Narrows 30; Cumberland 13; Beaver Lake 8. The disease is raging at Pelican Narrows. In one house there were 20 lying on the floor helplessly sick, with four dead bodies lying in amongst them. The reports coming in say that the conditions at the Narrows are horrible, and every family is down with the flu and helpless. It is thought that, unless relief is given

 ⁸ Calculated using the population from the 1917 census for the Anglican population at Norway House.
 ⁹ Calculated using the population from the 1917 census for the Methodist population at Fisher River.

immediately, the entire Indian tribe at Pelican will be wiped out (The Pas Herald and Mining News, Friday, 7 February 1919, p.3)

Of the 107 deaths at Norway House "A letter from Mr. R. Talbot, manager H.B. co. post, Norway House, dated Jan 22nd, states that the Spanish Flu hit them three weeks previously, and at the date of writing there were 107 deaths, representing 12 percent of the population" (The Pas Herald and Mining News, Friday, 7 February 1919. p.1)

It is possible that the omission of Fisher River from the newspaper accounts could result from its situation within the greater landscape of the Keewatin District. Its designation as an agricultural/forestry base (as opposed to a Hudson's Bay Company trading post) may have isolated it from the attention of neighbouring communities or the press. Consecutive newspapers articles from 1918 to 1921 were scrutinized in vain for any mention of Fisher River. As a point of reference, it is noteworthy that prior to the introduction of the influenza pandemic there were only two mentions made of Norway House in the same newspapers. Much of what is reported in The Pas Herald and Mining News is in reference to the HBC or the functioning and maintenance of the railway.

The impact of the epidemic however is not only gauged by mention made within newspaper accounts. Firsthand historical accounts as well as reports made to the Indian Affairs department also help establish the dichotomy between the experiences of people living at Fisher River and Norway House. One of the most useful tools utilized to gain deeper insight in the situation at Norway House is the post journal. This document details the effect of the epidemic and entries like the ones for December 19, 1918 and January 6, 1919 help establish that the outbreak had devastating consequences for the community. They read as follows:

Thurs Dec 19 1918

Adam Dickson, who was to go off with the second mail today, unable to go, having contracted the "Flu" during the night. Tried to get another man in his place, but this was impossible, as every man here that has dogs is unwell.

Mon Jan 6 1919

Sent team off again today with wood to Indians on Jack River. Garson around today for first time since taken ill, but not able to do anything. Mrs. Arthurson died during the night. The total deaths at Norway House now amount to 50. Mr. Mercer, who arrived from Cross Lake reports 58 deaths amonst the Indians there. (HBCA 1918 – 1923: fos # 11, 13)

Because Fisher River was not a HBC post, it did not have a daily log of events,

however there are two excellent manuscripts from which information can be drawn.

They were written by the community's Methodist minister and his wife and are therefore

an excellent gauge of how the community was impacted. Reverend Stevens' account

takes on the form of the Sandy Lake Story, an autobiographical tale that outlines his

'heroic' mission to convert Indians from neighbouring communities on the outskirts of

established parishes which he felt were "heathen but longing to hear more of religion."

(UCCA, Frederick G. Stevens, n.d: 2). The only mention made of flu is as follows:

Soon after our departure from Deer Lake a man was taken sick and could not be cured. All the time some of the older men were just over the border between paganism and Christianity. Other means failing them, they bought out their drums. The man died. Then they were very much afraid of what they had done. That was the year of the 'flu' and, although they were isolated and none of them took it, they were excited all winter. More or less conjuring went on all the next spring. (UCCA, Frederick G. Stevens, n.d: 12)

We know from Reverend Stevens' wife, Frances Stevens, that Frederick was at Fisher River at the time of the epidemic yet he makes no mention anywhere here about the experience within his own community. Frances Stevens' account pertains to her life

as the wife of Reverend Stevens. It details her everyday life and demonstrates her interest in documenting, as well as understanding, disease patterns. She speaks of her fears of the community contracting diphtheria from her daughter, Frances, in 1913. She states that she determined that her daughter was ill with the bacteria when she noticed, "her throat was covered with a grey film" (UCCA, Frances G. Stevens, n.d.: 89). During the smallpox epidemic of 1915 she comprehensively documents every detail of the community's confinement and subsequent illness. She states

What was called Smallpox spread like wild fire through out Reserve and out on the Lake. There were no fatalities nor no marks left and we wondered what it really was. For six weeks, mounties and a couple of visiting doctors and their drivers had to be housed at the Mission. For two weeks the entire family slept in one room in order to accommodate the party. The Indians objected loudly to the Quarantine though they had rationed sufficient food for the duration of the epidemic (UCCA Frances Stevens n.d: 99).

She goes on to speak of individuals' experiences with the restraints of quarantine and the perils of illness, yet her accounts of the influenza pandemic seem to lack the vivid detail of the community experience and conscientious concern that was displayed during the smallpox epidemic. She states

the smallpox epidemic. She states

The summer had quickly passed and we found ourselves facing the "Spanish Flu'. As elsewhere, it was a terrible experience. The work in the dispensary never ended while my husband was the first to get it, he kept going. When a doctor arrived in due time, he ordered him to confine his work to dispensary and burying the dead. It was a miracle that he could accomplish that. One day he buried seven, two in one grave. At Berens River thirteen bodies lay in the mission store house because there was not a sufficient number of men able to dig resting places (UCCA Frances Stevens n.d: 103)

This account implies that the community was disrupted by the virus, but instead

of detailing the events that were unfolding within her own community, Frances takes this

time to detail the disorder that was ravaging neighbouring communities. It can be inferred from her statement that although Fisher River had a terrible time during the pandemic, they were still able to cope with the strain placed upon the community, and therefore the impact could be construed as moderate when compared to some of the other communities in the region.

Lastly, there are the reports of the Indian agents. The two communities fall within the same district and therefore share a common general report which reads:

The Indians of Manitoba in common with other sections of the population suffered very severely from the epidemic of influenza, and the mortality among them as a result of this cause was high. The department's medical officers and the agency staff spared no effort in their efficient and energetic efforts to prevent the spread of the disease. Unfortunately it was impossible to secure adequate medical attention for the Indians living in the more outlying parts, a circumstance which is not remarkable in view of the fact that a similar situation existed in the majority of the white communities throughout the Dominion (Indian Affairs Annual Report 1919: 47 - 48).

The true difference here lies within the reported government financial support. Figure 1 is comprised of information collected from the Dominion of Canada Annual Report of the Department of Indian Affairs for the year ended March 31, 1919 under the heading General Expenditures 'Epidemic of Influenza' page I-52.

Norway House Agency	Cost	Fisher River Agency	Cost
Dr. A. Larose Services 60d @ \$15	\$900.00	Medical Attendance	\$132.00
assistants 60 d. at \$15	\$900.00	Medical Supplies	\$283.75
assistant 60 d. at \$15	\$840.00	Treatment in Hospital	\$47.24
travel of Dr. Larose & Assistant	\$656.28	Relief	\$427.30
Sundry Services	\$95.00	Burials	\$334.10
Medicines	\$37.15	Freight	\$62.80
Rations to Men Visiting Camps	\$114.08		
Blankets 8 pr	\$58.00		
Relief Supplies	\$1,030.33		
burials	\$93.77		
Total	\$3,223.61		\$1,287.20

Table 2-1 - Expenditures Incurred By the Canadian Government (1919) to Manage Influenza atFisher River and Norway House. Source: Indian Affairs Annual Report 1919: I-55

This table clearly illustrates differences in how the pandemic was treated in the two locations. The expenditures at Norway House speak much more to a community that is geographically vast, and in need of aid. This is represented in the cost of travel (\$656.28) as well as the high burden of relief supplies (\$1, 030.33 at Norway House compared to \$427.30 at Fisher River). This table also indicates differential access or perhaps demand for care. According to this table Norway House relied heavily on the capability of the local visiting doctor from The Pas and his assistants. Historical documents show that a temporary hospital was erected at Norway House in October of 1904 after the community suffered a number of epidemics. There is evidence of a permanent hospital being erected 1913 from the following report to the Department of Indian Affairs

Dr. Norquay was appointed medical officer of this agency during the year, and he has been in residence at Norway House since August, 1913; he has been provided with a liberal supply of drugs. He has associated with him a trained nurse; so that the people are well looked after. The hospital to be erected at Norway House will be completed during the coming season of 1914, and when equipped, should furnish a splendid institution to meet the

demands of that district, and with a resident physician and a capable nurse, in charge, should prove to be an asylum for the sick and stricken unfortunates that may seek relief and attention there (Department of Indian Affairs. Reports of Indian Agents. Sessional Paper No. 27 5 George V., A. 1915: 53)

A report published in the following year confirms the erection of the hospital; it can therefore be assumed that it was fully operational in 1918 – 1919. Unfortunately there are no records available as the hospital burned down in 1922 and again in 1952 (Krotz 1990: 31). There are also reports of aid being received by the local resident physician who fell sick for a spell during the height of the epidemic. Fisher River's residents appear to have relied on paid medical attendants as well as the local hospital for treatment. There also appears to be a stark contrast in the availability or demand of medical supplies/medicines between the two communities as indicated by the disparity in the amount of money that is allotted to Fisher River (\$283.75) and what is afforded to the Norway House residents (\$37.15). The contrast in the cost of burials between Norway House (\$93.77) and Fisher River (\$334.10) may relate to the timing of the epidemic within each community. The report consists of information recorded until March 31, 1919; however, it is possible that burial information only included deaths up to December 1918. The majority of burials had already taken place at Fisher River by this time but the epidemic has not yet truly commenced at Norway House.

These accounts though informative, help explain how the pandemic was experienced, but they do very little to explain why two communities merely 715.8 kilometres apart had such different experiences of this new strain of influenza. By reviewing the family context of influenza mortality, one is offered another avenue for

exploring these disparities. Before turning to this problem, the materials and methods used to investigate it are addressed in the next two chapters.

Chapter 3 Materials

Parish records provide historical demographers with an irreplaceable tool for uncovering aspects of the past. Current advances in methodology using information extracted from parish registries have also allowed ideas about past populations to be more adequately explored. The aim of this chapter is not only to highlight the importance of parish registry information but also to identify possible pitfalls associated with relying on this source. This is accomplished through the identification of problems associated with Anglican and Methodist parish registers that form the basis of this thesis research. Where applicable, I offer explanations of why these obstructions arise as well as information pertaining to how these obstacles may be overcome in future studies.

This chapter also provides a detailed discussion of the records available for each of the study communities. The benefits and difficulties of using parish registry data within an Aboriginal context are also considered.

Parish Records

Parish records have the ability to speak volumes about the past if they are methodically and comprehensively analyzed. They were originally sought out by "historical demographers as sources of nominative data for the analysis of family-level fertility behavior" (Willigan and Lynch 1982: 58). They represent the compilation of events within a parish through the recording of baptisms (which track a community's

births), marriages, and burials within consecrated ground (which track a community's deaths). This thesis draws upon parish registers for Fisher River, Manitoba that were maintained by the Methodist clergyman Frederick G. Stevens, who served this community from June 1907 to December 1939 (Moffat 1992). It also draws upon the Anglican registers maintained by Reverend J.F.J. Marshall who presided over the Jack River Mission at Norway House from 1910 to 1926; prior to his appointment the records were kept with varying degrees of competency by a secession of clergymen (Herring 1994: 78). The problems associated with inconsistency within and between ministers will be explored later in this chapter, as will other issues that add to the problems of reconstituting families in the past.

As it is "not possible to reconstitute fully all of the families in a parish by making use of the register of that parish alone" (Wrigley 1966: 96), my thesis draws upon records collected by Dr. Ann Herring for the neighbouring communities of Berens River, Oxford House, York Factory, Split Lake and Peguis. Other studies have shown that information sometimes absent from the registers can be supplemented from other primary source material (see for example Herring and Sattenspiel 2003; Hoppa 1998 and Desjardins 1995, 1996). To this end Treaty Annuity Pay Lists (TPAL) are used to supplement information from parish records for Norway House (ACCA 1902-1937). TAPL documents (DIAND 1896 – 1959) are censuses that were compiled annually for the Government of Canada by Treaty Party officials who visited each reserve during the summer. Other primary source materials that were consulted include Hudson's Bay Company post journals (Hudson's Bay Company Archives 1917-1922, 1918-1922, 1918-

1923) the unpublished memoirs of Frederic Stevens and his wife, Frances (UCCA, Frances G. Stevens, n.d.; UCCA, Frederick G. Stevens, n.d.) as well as published census data (Government of Canada 1917).

The Parish Registry As A Source of Information

Although parish records provide vital information on the past, they are in no way invulnerable to criticism with reference to the results they expose.

The clergyman is an integral aspect of the parish record, for it is through his pen that these documents are created. Therefore the conscientiousness of the clergyman in maintaining his records (Nair 1988: 184: see also Razzell 1972) is paramount for any study that strives to utilize the parish registry. The relationship that a minister forms with his parish contributes to the construction and quality of the registries. This is shown by the appearance of "notable lapses in the quality of the recording... when a registrar who had lived for a long time in the community was replaced with a newcomer" (Willigan and Lynch 1982: 62). Population fluctuation, an increase in duties, geographic limitations and other catastrophic events all play a role in how these relationships are expressed and experienced.

These problems can also potentially lead to breaks in registration resulting in a complete absence of data, which can result in the loss of a community for family reconstitution (Wrigley and Schofield 1981: 4). Even when all of the data are present and accounted for there are periods when registrations are seriously defective (Wrigley and

Schofield 1981: 4) as "[t]he skills of individual registrars varied widely" (Willigan and Lynch 1982: 62).

Registry creation is not a sufficient condition for reconstruction. There are many elements that hinder a demographer's ability to interpret the past. Immaculate records can go missing while others are entirely illegible. Human errors, problems with interpretation, and the natural degenerative processes that eat away at parish registries are all factors that contribute to this phenomenon.

As well, it is important to note that error is not always the result of poor vicar compliance with the ecclesiastical rules for record keeping. Many problems arise when secondary transcription of these vital records takes place. Family reconstructions can be hindered by "typographical errors in computerized records from the originals" (Weiss et al. 1980: 59). Difficulty deciphering original handwriting (Srivastava 1987) can also skew what information is presented for analysis. This difficulty often rests in the fact that individuals transcribing original sources may not be familiar with the handwriting of the recorder. When secondary sources are unavailable to support the material bound in parish registries, these errors can have a significant impact on interpretations.

Again even when registers are intact and the material contained therein is amenable to analysis their utility may be questionable at best. "Much of the criticism of parish registers over the centuries was directed at the insufficient detail" (Krause 1965: 381) they held. In most instances there were no standardized forms that parish priests were obligated to fill in (Hollingsworth 1969: 156). As a result of this and prevailing pressures on the priest's time "[S]ome registers provided very little information,

sometimes being mere lists of persons baptized, married, and buried without any other information. At best, the highest level attained by most parishes was the naming of both parents in the case of baptisms and burials of children" (Krause 1965: 381).

Other potential stumbling blocks include under-registration of vital events (Ruggles 1999: 106). The delay between birth and baptism (Razzell 1972: 122) resulting in under representations in both births and deaths of infants and young children can affect demographic analysis. As well there are a plethora of problems associated with errors that arise from differential spelling of peoples' names (Weiss et al. 1980: 59).

After it has been established, by following a systematical protocol to ensure that the registries have been meticulously entered and that their quality has not deteriorated (e.g. Drake 1974), one must still be aware that the material presents an inherent sampling bias. The registers were not compiled to act as demographic tools to aid in the reconstruction of entire villages or towns; rather, they were tools to assist the conscientious clergymen in watching over his flock. It is also important to note that the church was not solely a place of worship; it also served as a business and a regulating body entrenched in government policy.

Since "registers normally only record the Anglican ceremonies" (Wrigley and Schofield 1981: 15), there is a clear omission of members from other religious groups (Cox 1974: 96). Currently there is a debate over the extent to which this factor influences statistical findings. According to Ruggles (1999: 114), many analysts who practice family reconstitution state that this bias is minor. Others "point out that the non-migrant

population over-represents farm-occupiers, artisans, and fishermen, while underrepresenting both the rich and the poor" (Ruggles 1999:114).

As this thesis is devoted to understanding the impact of the 1918 pandemic some discussion must be directed toward the impact of infectious disease on family reconstitution. Munkhoff (1999) makes a brief reference to plague as having had a deleterious effect on both the community and on the governing parish priest. Lovell (1991) also notes this as a possible hindrance to reconstructing the population of Jacaltenango, Guatemala. It is important to note here, however, that much of the material pertaining to instances of disease outbreak within a particular community is clearly documented in parish records. Eversley (1965: 396) tells us that " [t]here is much additional information in many registers. In years of exceptional sickness, the prevailing epidemic is often indicated". Pestilence, however, definitely poses a problem in research aimed at reconstructing families. Often the sudden onset of illness and death prevents individuals from returning home. In addition, the short duration of many air-borne person-to-person transmitted diseases can mean that if a priest or minister falls ill records for the epidemic may be lost.

Wrigley et al. (1997) provide the best summation of the place of parish registries within historical demography. Expanded upon by Scott and Duncan (1998: 16) the idea is as follows, "although parish registers should be used with discretion and with an appreciation of their shortcomings, 'the comparative precision with which population characteristics can be defined from the information contained in Anglican parish registers is striking". When conducting a study that utilizes parish registry data the saying

'hindsight is 20/20' comes to mind. Since we have the ability to look back at what conditions were present when the material we seek to understand was recorded, we have the ability to overcome some of the limitations that may skew our studies by taking into consideration these conditions when conducting our analysis.

The Parish Record and Family Reconstitution in an Aboriginal Community Context

As has been noted, family reconstitution was originally developed in large part to understand family-level fertility within the context of French parish records. Many authors who explore the applicability of the standards established to extract information from these records aim to apply them to English registers. The same guidelines that were developed to explore French registries, however, cannot be equally applied to English records (See Wrigley and Schofield 1981, Willigan and Lynch 1982 and Laslett 1972). Willigan and Lynch (1982: 60) note that English registers have a tendency to contain less information than the French ones, most notably "a lack of information on mothers' maiden names on the baptismal records of children". They go on to state that this is the result of differential laws imposed upon this form of record keeping. Although much has been written regarding the applicability of applying French standards to English records, there is virtually no information regarding the application of European standards to reconstituting Aboriginal populations, which are the focus of this thesis.

There are three main areas that must be understood and explored within an Aboriginal context if one is to apply European standards to record-keeping among

colonized populations. The first is that much of the discussion of problems encountered when studying records of the New World focuses on European settlers and not on the original inhabitants of the area. For example, the problem of population expansion within parish boundaries located within colonies of The British Empire is often addressed. Willigan and Lynch (1982: 62) and Krause (1965: 385-389) highlight changes in population size as a result of migration as a major hindrance to studying frontier populations. Their discussion seems to center on population changes due to overseas migration and has little to say about internal population changes that result from conversion and mobility within and between communities by Indigenous populations. This ties in to the second and third obstacles to reconstituting Aboriginal communities: understanding and accounting for mobility within populations as opposed to migration, and the impact of proselytizing or active conversion on community composition. Hoppa (1998) suggests the use of a cross comparison between multiple sets of recorded vital events to overcome this obstacle. As well he cautions that material extrapolated from communities that consider historic Aboriginal demography take into account the nature of seasonal mobility "associated with subsistence and economic pursuits" (Hoppa 1998: 183).

There is some debate regarding the degree of distortion that active conversion has had on the sampling bias presented in the registries of a given parish priest. Piché and George (1973: 367) comment that even though active conversion of First Nations people was the norm there is still a lack of information about these groups. This point is further expanded by Willigan and Lynch (1982: 63) where they state that "The Roman

Catholic parish registers for the unfree populations of the New World generally contain much less complete information than those for free populations". Ubelaker (1988: 289) distills these problems down to three contributing factors: incomplete records are either the result of intentional bias, inadequate exposure, or improper counting procedures. On the other hand, Walker and Johnston (2003) point out that Franciscan priests kept careful records of important events in the lives of Chumash neophytes under their control (2003: 55). Pilgrimages taken by a minister with the intent of converting 'lost souls' also had the potential to skew community-based data. In areas of dispersed or sparse settlement, which was most often the case in places of European colonization, the clergy's strategy was to make periodic visits to outlying populations. These pilgrimages were carried out in order to baptize, marry, or solemnize burials that had occurred in prior months. Although this practice ensured that members of the clergy registered vital events in these remote areas, "the long lag between visits tended to result in the underregistration of baptisms because of child deaths shortly after birth, or the artificial "bunching" of vital events in the registers for those periods when the visits took place"(Willigan and Lynch 1982: 63).

Usually it is claimed that populations and families characterized by great mobility are less suitable for reconstitution techniques (Eversley 1967: 44). As a result "fully reconstituted families are made up only of nonmigrant couples (at least, individuals who have not migrated since the time of their marriage) and their children" (Willigan and Lynch 1982: 178). This presents a major problem to any researcher attempting to

understand the individual level experiences of Aboriginal populations, especially those involved within the fur trade industry. For this reason this thesis distinguishes between migrant individuals and mobile populations. This distinction is divided into three classifications: the first is seasonal mobility, referring to individuals, families or communities that move in relation to a seasonal round to meet the demands of their subsistence cycle. The second grouping includes individuals who move due to economic demand (economic mobility), for example movement around posts or logging trips. The third category simply encompasses migration (Herring Pers Comm).

Willigan and Lynch (1982: 178) suggest that one way of coping with the problem of mobility "by tracing the movements of local migrants, is to attempt to reconstitute a series of villages or parishes that comprise an ecological region". Wrigley (1966: 105) reaffirms this technique by stating: "It is always possible that by combining the information from the registers of a number of small contiguous parishes the same results can be achieved as may be had from a single large parish".

The Norway House Parish Record

The Jack River Mission registries utilized for this study are available from 1902 – 1941 (Herring 1994: 78). They were maintained by a succession of Anglican clergymen until the appointment of Reverend J.F.J. Marshall in 1910. This marks the beginning of comprehensive record keeping until the end of his tenure in 1926. As has already been noted, changes in clergy can have a deleterious effect on the quality of the register. In the case of Norway House, there are quite a few missing or incomplete entries, which

presents an obstacle in reconstituting the earlier generations represented in this study. This is discussed in greater detail below.

The standard baptismal record for Norway House after the appointment of Rev. J.F.J. Marshall includes the name and sex of the individual as well as parents' names (although in most instances the mother's maiden name is omitted). Father's occupation is listed as well as the residence at birth and dates of birth and baptism. A complete marriage record gives both the surname and given name of the bride and bridegroom, as well as their ages, residence when married, place of birth, previous marital status, parents' given and surname (again the mothers' maiden name is often omitted), and the date of the event. The standard burial record contains the surname and given name of the deceased as well as the age at death and a listing of quality/trade/etc. The latter proved to be very useful in reconstitution, as Reverend Marshall would often include information on familial relationships in this space. The date of death and date of burial were also included for the majority of the records. It has been noted that the Anglican clergyman was incapacitated by flu from January 1 1919 through to the 22 (Herring 1994: 90), which accounts for the lack of recorded dates of death for burials during much of the 1918 - 19 influenza outbreak, as well as the clumping of burials between January 26 - 301919 (Herring 1994: 90).

Sadly in the case of Norway House lost registers do play a significant role in what information is present to analyze, as the Methodist Mission records for the period were lost in a fire prior to being subject to any secondary transcription. There are records for the Roman Catholic Church but at the time of writing this thesis they were unavailable.

The following methods for collection and transcription of the Anglican records for the Norway House population were carried out. Initial collection was conducted at the General Synod Archives of the Anglican Church of Canada at Church House, Toronto (ACCA 1902 – 37) by Henry Weilenmann, a student assistant of Dr. Ann Herring. She also carried out family reconstitution and oversaw correction and transcription into Paradox. Later the records were imported into Quattro Pro by Joseph Parish, a student assistant.

If the small size (< 250) at of the Anglican population at Norway House is taken into account, these records pass Drake's protocol (See Appendix 1). However, it is important to note that during the time of the epidemic Rev. Marshall's illness did compromise the quality of the registry data. To further check the accuracy of the records they were compared to the Norway House Cree First Nation Treaty Annuity Pay Lists (TAPL) (DIAND 1896 – 1959). Seven families were chosen at random and the two sets of records were compared for a fifteen-year period (1905 – 1920 inclusive). Of the 46 entries¹⁰ of births, deaths and marriages 41 appeared within both the TAPL and Anglican parish registry. The missing records consisted of three births, one marriage and one death. One child died within a few months of birth; as well, one of the chosen families did not have a record within the parish, which accounts for two missing births and a missing marriage.

There are questions about whether inferences can be made about the entire population at Norway House from the data collected from the Anglican registry.

¹⁰ There were 17 births listed in the TAPL, 3 marriages and 22 deaths. From parish registry data 20 births were recorded, 2 marriages and 23 deaths (14 of which were flu deaths).

According to a Census taken by the government of Canada in 1917 (Department of Indian Affairs. Sessional Papers No. 27 George V, A.1917) the entire population of the Norway House band was 734; of this there are a reported 208 Anglicans, 494 Methodists and 32 Roman Catholics. This means that only 28% of the population at Norway House was Anglican. Krotz (1990: 31) reports there were 160 deaths due to influenza in 1918, which would represent 22% of the entire population. According to Herring (1994: 87) the difference in the influenza mortality rate among Anglicans and the total populaiton is not statistically significant.

Both seasonal and economic mobility seem to have been present at Norway House. Due to the limited amount of provisions on hand, trappers and traders were forced to come to the post to acquire necessities at the Hudson's Bay Company store (Herring 1994: 88). Seasonal mobility was also a feature of life at the time. Herring (1994) suggests that there may have been an artificial inflation of influenza deaths during the pandemic as a result of temporary population fluctuations. She points out that the

circumstances of daily life at Norway House led to a counterbalancing suspicion of overnumeration 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, the inclusion of five influenza burials by William Saunders (the resident clergyman at Fisher River), and the April 1919 burial of a boy who had actually died from the flu on 18 October 1918 (Herring 1994: 87 – 88).

To address this problem I decided only to include deaths within the Anglican burial record for Norway House. Although I am aware that this study includes individuals who came in from God's Lake and Oxford House, and misses individuals from Norway House who perished in neighbouring

communities¹¹, to begin to pick and choose who was included and excluded would only increase sampling bias.

The Fisher River Parish Record

When Frederick G. Stevens first arrived at Fisher River on July 2, 1894 it was to act as the resident schoolteacher; his travelling companion, Egerton R. Steinhauer, took on the duties of parish priest. In 1896 Stevens left Fisher River to travel northeast to Oxford House where he served as an educator for 11 years, returning to Fisher River in 1907, to serve as their minister. He spent 33 years presiding over the Methodist Church, which became part of the United Church of Canada after its formation in the late 1920s (Moffat 1992: 31 - 33). This continuity in clergy helped insure the quality of the parish registry. Stevens' devotion and apparent concern for his parishioners also contributed to the precision with which the registers were kept. This concern is evident in Stevens' ability to speak with his flock in their native tongue, Cree. He was also conscientious to write and present his sermons in Cree (Moffat 1992: 33). His devotion to caring "was corroborated by Flora Kirkness, who described how Stevens used to make visits around the community, and always visited those who were sick [Fisher River Oral History Project, 1991]" (Moffat 1992: 33).

The Methodist/United Church parish records were maintained for Fisher River from 1888 to 1988. Only marriage records exist for this entire period; the baptismal records terminate in 1961 and the burials are recorded until 1979. The best period of

¹¹ There were four men reported from Norway House in the York Factory burial registry.

registration coincides with Stevens' tenure as there are no gaps in his registration (Moffat 1992: 43). The dedication of this minister transferred itself into high quality records that meet the standards established by Drake's protocol for systematically evaluating instances of underreporting within the parish data (See Moffat 1992: 44 - 52 for a detailed analysis).

The standard baptismal entry includes the individual's name, sex, residence, parent's names, date of birth, place of birth, date of baptism and place of baptism. Complete marriage records indicated both the surname and given name of both parties as well as their previous marital status, their residence when married, respective places of birth, religion, occupation or calling (predominantly for males), both of their parents' names and the date of the event. Finally a completed burial record indicated the deceased's surname and given name, age at death, date of birth and birthplace as well as a date of death, date of burial and the place of burial.

According to census reports for 1917 (Department of Indian Affairs. Sessional Papers No. 27 George V, A.1917) the number of individuals in the Fisher River band was 493. Of this 284 are reported as Methodist and 209 are recorded as having other Christian Beliefs. Thus 58% of the population at Fisher River were Methodists.

The role of active conversion could potentially have an impact on Fisher River as Reverend Stevens was very much engaged in the practice of soul saving. His personal accounts depict him as a devout man of God who views Indigenous peoples who have not yet converted to Christianity as being "heathen but longing to hear more of religion" (UCCA, Frederick G. Stevens, n.d.: 2). He did occupy much of his time travelling to

neighbouring communities where no missions were established in an attempt to spread the word of God. We know that Rev. Stevens was at Fisher River at the time of the influenza outbreak from his unpublished autobiography (UCCA, Frederick G. Stevens n.d.), as well as his wife's account of his illness (UCCA Frances Stevens n.d.: 107) and the accurate record of deaths during the epidemic. It can also be surmised that Revered Stevens did not include individuals buried, baptized or married during his travels to outlying areas within the records he maintained for Fisher River. In Rev. Stevens' '*The Sandy Lake Story*' he recounts travelling to Deer Lake in June of 1918 where he states he "baptized many children, performed nine marriages" (UCCA, Frederick G. Stevens, n.d.: 12). During this period, there are no marriage and baptisms recorded at Fisher River for individuals who resided at Deer Lake, evidence that he kept the records for other locations separate from those of his home parish.

The issue of mobility is likely to be a much more significant feature of the Norway House parish record data than it is for Fisher River. I hypothesize that this is in large part the result of the different subsistence activities employed at each of these sites. Fisher River was founded as a fishing and agricultural centre (Moffat 1992: 6). It was also a site of wage labour (Moffat 1992: 10), which would have increased the sedentisim of its residents¹².

¹² It is however noteworthy that the majority of individuals who had to be removed from the family reconstitution portion of this study did not have a birthplace listed or had one that was listed other than Fisher River, with the exception of three individuals who were recorded has having been born at Fisher River, but had not yet been baptized. This may indicate some sampling bias. The lack of available data on previous generations is most likely the result of the newness of the community.

Chapter 4 Methods

Introduction

In order to tackle the problem of understanding how mortality was structured at Norway House and Fisher River this project took a multi-layered analytic approach. The use of multiple lines of evidence was employed not only to understand the impact of influenza in these two communities but also to check the consistency of each of the findings for each methodology. The three methods used to understand the impact of influenza were an aggregate approach, a technique developed by Scott and Duncan (2001) that charts the spread of infection, and family reconstitution methodology. The ultimate goal is to compare the findings for each community to determine if the influenza epidemic's impact differed between them. The following chapter is designed to explain each of these techniques.

Aggregate Approach

Traditionally there are two ways to analyze information extracted from the parish register. The first, and most common method is aggregate analysis. Aggregate methods serve most commonly as counting techniques that provide information on the flow of events (Knodel 1988: 4), such as trends in birth rates, marriages and so forth. Although this thesis is not constructed primarily around information collected through aggregate techniques, it does rely on this methodology indirectly in two instances. First, much of

the work already carried out on issues pertaining to health and disease for this region is based on aggregate techniques (see Herring and Sattenspiel 2003; Herring 1994 and 1994b; Beckett 1998; Moffat and Herring 1999). Eversley (1966) suggests that aggregation can act as a prelude to reconstitution as aggregate methods act as a comparatively rapid system of analysis. With it researchers are able to pinpoint areas and periods within a population's history which show the most striking demographic changes. Family reconstitution can then be carried out on these recognized periods resulting in a more detailed study, which may give final answers to certain questions (Eversley 1967: 44 - 45). Had aggregate analysis not already pinpointed that these communities had been hard hit by the pandemic, the level of analysis embodied within this thesis would not have been possible (see Herring 1994, Herring 1994b, Moffat 1992, Moffat and Herring 1999, Sattenspiel and Herring 1998, Sattenspiel and Herring 2003, Sattenspiel et al. 2000). The second way in which methods of aggregation are utilized within this thesis is to explore deaths for the region that cannot be linked to a particular family. The cardinal rule for working with aggregate methods is to "throw nothing away" (Eversley 1966: 61) as well as to create a foundation of information upon which subsequent analysis can be based.

In order to carry out this methodology all of the burial, baptismal, and marriage information for Norway House, Fisher River, Berens River, and Oxford House were scrutinized. As well Treaty Annuity Pay Lists for Norway House were consulted. The first step undertaken was to identify each individual who died from influenza in the two study communities. To this end, I consulted burial records transcribed into Microsoft

Excel by Carol DeVito for Fisher River and QuattroPro for Norway House by Joseph Parish to compile a list of deaths where the cause of death in 1918 or 1919 was listed as flu or a derivative of the word. For Norway House the vast majority of burials were recorded between December 18th 1918 and January 30th, 1919. The terms Spanish Flu, influenza, flu, and "flu and age" were used to identify the malady. Fisher River was affected by the pandemic predominantly between the dates of October 30th, 1918 and February 15th, 1919 as indicated by dates of death from influenza recorded by Rev. Stevens. In these registries flu was referred to as Spanish Flu, flu, la grippe, "after the effects of flue", and tuberculosis following Spanish Flu.

A separate database was created in Microsoft Excel for Norway House and Fisher River. The data were transferred from the original file provided by Dr. Herring, and then verified using the original records for Fisher River (UCCA 1895 – 1908, 1918 - 1951) and the original transcriptions for Norway House (ACCA 1902 – 1941). The deaths were then organized according to date of burial, or date of death where applicable for Norway House, and by date of death for Fisher River. Some dates of death for Norway House were gathered from Hudson's Bay Post Journal reports (HBCA B.154/a/87), as Reverend Marshall did not record dates of death into the Anglican burial registry.

After all of the individuals were listed and organized within the spread sheet I then turned to the reconstituted families to gather further information about each individual who died from influenza. I created separate forms that synthesized the information from all sources for each individual (See Appendix II for a copy of these forms).

After the forms were completed I used digitized data, converted from Paradox to Quattro Pro by Joe Parish, as well as other primary source data, collected and compiled by Dr. Ann Herring to fill in all gaps. The following files were searched: Baptisms and Births at Berens River (UCCA 1885 – 1889, 1909 – 1952); Births and Baptisms at Fisher River (UCCA 1908 – 1929); Baptisms and Births at Norway House (ACCA 1902 – 1937); Baptisms and Births at Oxford House (UCCA 1894 – 1917, 1918 – 1951). Burials and Deaths at Fisher River (UCCA 1907 – 1941); Burials and Deaths at Norway House (ACCA 1902 – 1941); Marriages at Berens River (UCCA 1884 – 1909 and 1909 – 1952); Marriages at Fisher River (UCCA 1888 – 1903, 1911 – 1920); Marriages at Norway House (ACCA 1903 – 1937); Marriages at Oxford House (UCCA 1894 – 1917, 1919 – 1951). Quattro Pro files containing information gathered from Treaty Annuity Pay Lists for Norway House for the year ending 1918 and 1919 were also utilized. Drs. Ann Herring and Tina Moffat collected the annuity lists and Joe Parish converted the files into Quattro Pro.

To combat the problem of errors in transcription I manually searched the photocopies of the original records for Oxford House, Fisher River and Berens River. The original records were not available for Norway House so I relied on the original transcriptions reproduced by Dr. Ann Herring. This method proved to be quite fruitful for identifying individuals recorded within the documents, however the lack of documents prior to 1888 for Fisher River and 1903 for Norway House posed a significant problem for identifying older individuals within each study community.

These forms were referred to in order to determine for each community the index influenza mortality case, how many people died, the age and sex distribution of mortality, the impact of mortality, and whether influenza mortality cluster in families. This standardization of information insured that the same data were consistently drawn upon and that no information was omitted.

Scott and Duncan (2001) Approach

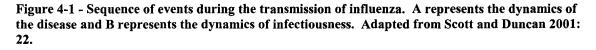
The Scott and Duncan (2001) approach was used to investigate how the influenza virus spread within and between families at Fisher River. This technique could not be employed at Norway House because it relies on actual dates of death and these were not recorded there during the pandemic. The technique is designed to illustrate how the infectious and latent periods that help to define the transmission possibilities of a particular virus can be used to estimate a pattern of dissemination throughout a given community.

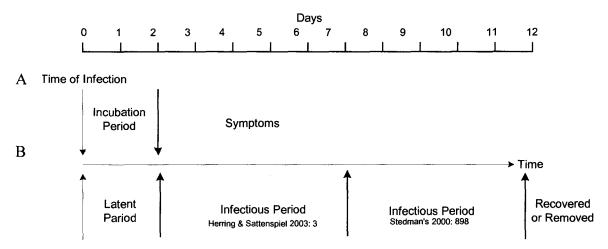
To understand the complexities of extended kin networks, this phase of the research relied heavily on the above-mentioned forms created for the aggregate analysis methodology, although larger family reconstitutions were also consulted. This process began with a review of Scott and Duncan's 2001 work to gain a working knowledge of the methodologies they employed.¹³ It was important to determine which of their strategies I could utilize in my own work as their book set out to disprove the idea that

 $^{^{13}}$ Special attention was paid to chapters 2 (Epidemiological Concepts pp.21 – 46) and 5 (Case Study: the plague at Penrith 1597 – 98 pp.115 - 248)

the Black Death was caused by *Yersinia pestis*. This thesis project, however, is centered on undisputed pathogen and therefore did not have to employ all of the methodologies they used. The key to employing their technique within my own research was to recreate the spread of the virus throughout the community by tracing infection patterns using established infectious and latent periods for the influenza virus.

To this end I began by establishing the latent and infectious periods of the influenza virus to construct a time period that captured an individual's period of infectivity. The incubation period for influenza ranges between 24 to 48 hours (Ohadike 1991: 1397), and the infectious period can last up to ten days (Stedmans 2000:898) until the individual has recovered or is removed from the cycle. Alternatively, it is suggested by Herring and Sattenspiel (2003: 3) that adults on average shed the disease for only about 3 - 5 days after which they are no longer infectious. Figure 4.1 demonstrates the sequence of events during the transmission of influenza.





To address the question of how influenza spread from family to family at Fisher River, the deaths within and between families were studied. Using the period of infectivity as the primary unit of analysis I attempt to estimate which individuals in different families could have had contact with one another during the infectious period of their illness. Using primary source material I then attempt to unravel relationships through kin and occupation.

As much of this analysis is based on information gleaned from research carried out by Ann Herring and Lisa Sattenspiel (1998, 2000 and 2003), I chose to work within the guidelines of infectivity that they established in their most recent publication (2003), namely a 1 to 2 day incubation period and a 3 to 5 day infectious period. This resulted in the creation of seven possible infectious patterns, each of which plots the spread of the virus with varying length of incubation and infectious periods. The seven combinations used are:

- 1 day latent and 3 days infectious
- 1 day latent and 4 days infectious
- 1day latent and 5 days infectious
- 2 days latent and 1 day infectious
- 2 days latent and 3 days infectious
- 2 days latent and 4 days infectious
- 2 days latent and 5 days infectious

Relationships were assessed with the aid of parish documents, Reverend Stevens' *Sandy Lake Story* (UAC Frederick G. Stevens n.d.), Frances Stevens' unpublished

memoirs (UCCA Frances Stevens n.d.), Government of Canada census documents (Department of Indian Affairs. Sessional Papers No 27 George V, A. 1917) as well as the Indian Affairs Annual Reports available from 1864 – 1990 (Government of Canada).

Reconstituted Families Approach

"Family reconstitution is the bringing together of scattered information about the members of a family to enable its chief demographic characteristics to be described as fully as possible" (Wrigley 1966: 96). First introduced by Louis Henry with *Anciennes Families Genevoises* in 1956, family reconstitution methods expose demographic intricacies at the individual and group level (Willigan and Lynch 1982: 177). Henry expanded the applicability of this technique with his study on the French Parish of Crulai, co-authored by Étienne Gautier (1958). Fleury and Henry (1976) took this method to the next level by developing a set of standards in their work *Neuveau manuel de dépouillement et d'exploitation de l'état civil ancient*. This methodology allows a researcher to view the family and/or household as the primary level of analysis.

This thesis is based primarily on analysis derived from family reconstitution methods. Although family reconstitution is often praised for its ability to penetrate the parish registers and reconstruct a history with unparalleled detail, there must be some discussion of the limitations of this approach. These problems can be reduced to two main limitations: first, the quality of the register used for reconstruction, and second, the need to disregard much of the parish record data.

In order to carry out this methodology for this thesis all of the burial, baptismal, and marriage information for Norway House (ACCA 1902 – 1937) and Fisher River (UCCA 1918 - 1951) were scrutinized. As well Treaty Annuity Pay Lists for Norway House (DIAND 1862 - 1959) were consulted. This section also relies heavily on primary source data written at the time of the epidemic, such as reports to the department of Indian Affairs (Government of Canada 1919), newspaper accounts (The Pas Herald and Mining News 1919) personal memoirs (UCCA Frances Stevens n.d., UCCA Frederick Stevens n.d.) and Norway House Post Journals (HBCA B.154/a/87).

The first step was to consult reconstituted families that already existed for the two communities, which were created by Dr. Herring and her student assistants. After these reconstitutions were checked, wherever possible, information was linked from other sources (above mentioned TAPLs, newspapers, post journals, memoirs and government documents) and communities (Oxford House, The Pas, Berens River, Gods Lake and York Factory). The search for kin connections between each nuclear family was then undertaken, to construct a more generalized 'genealogy' of influenza mortality. The deaths were divided by surname and placed onto separate spreadsheets to look for patterns of infection within families. It quickly became apparent that surname was not a sufficient feature by which to establish relationships between individuals because many surnames were common in the study communities. The forms created for the aggregate analysis were also consulted, to gauge the impact of mortality at the family level. However, it quickly became apparent that this data set would not allow for full disclosure of the complexities of extended kin groupings.

In order to reconstruct accurately the spread of the virus through families in each community it was necessary to construct family trees or genealogies. I accomplished this by returning to the reconstituted family data and re-checking the marriage and birth records. In chronological order of death, I looked up the individuals who died from flu within the reconstituted families. Once the individual was located, a genealogy marking the influenza victim as 'ego' was constructed. The genealogical links were then traced through the other reconstituted families for each community.

Vital records for births and marriages were checked for every individual over the age of 15. This age was chosen because I saw no indication of marriages taking place prior to this age; in fact, the earliest age at marriage in the sample was 17. No information regarding marriages or baptisms subsequent to 1919 was included. Often times events were recorded out of sequence so in order to insure that no vital pre-1919 events were excluded, the records were checked until 1925.

Where a date of birth was absent and no age was provided it was virtually impossible to positively identify an individual. Linkages between families at Norway House were rather difficult to establish, for often the parents of children who died were not listed. On the other hand when Rev. Marshall created the burial records he included the deceased's date of birth, or in the absence of a date of birth, the age of the individual was most often indicated.

After individual influenza families were completed, all of the genealogies, and the links between them, were drafted onto a larger canvas. The large genealogy of influenza mortality was divided into three sections, consisting of two marriage cohorts (pre 1889

and 1889 – 1919) and an adolescent, unmarried group. These time periods were chosen because they take into account migration patterns for both study communities. Norway House people founded Fisher River in 1885 with adhesions in 1908 (Moffat 1992: 6). Norway House itself was established in 1801 as a HBC supply base but Herring (1994) notes that the Anglican mission opened in 1902, as indicated by the commencement of parish records for the community. The marriage cohorts were also chosen to help highlight key periods in the life cycle. The first category represents a post menopausal, or non-reproductive category. The second, is a reproductive, or early adult cohort, and the third is an adolescent cohort. The genealogy also attempted to highlight age at death patterns, placing the youngest individuals at the bottom of the diagram and older individuals at the top.

I chose to leave the first marriage cohort (prior to 1888) open to include all of the parish registry data. Marriage records for Fisher River are available from 1888 onwards and the marriage records for Norway House exist for 1903 - 1937. Information about unions prior to 1888 usually consists of the names of the parents of brides and bridegrooms. Where this was the case, and a marriage record could not be found, the date of birth and birthplace for a couple's first-born child was entered. This made it possible to search birth and marriage material from other communities. Even with this measure there was a lot of information that could not be retrieved.

At Fisher River, the vast majority of individuals who perished from influenza were connected through birth or marriage. Consequently, it was possible to distinguish the wider kin network of the families in which an influenza death occurred. For Norway

House, where fewer of the families were interlinked, it was possible to distinguish families that were employed by the Hudson's Bay Company (HBC) from those that appeared not to be connected to it. The process of creating kinship diagrams proved to be an invaluable aid for familiarizing myself with the data. Patterns that were vague in the initial stages of my research became wonderfully visualized within the genealogies.

After reviewing the parish registry data I turned to the Treaty Annuity Pay Lists (DIAND 1896 – 1959) for Norway House to help confirm deaths and family information. Again using the search tool within the Quattro Pro database modified by Joe Parish in 1999, I ran queries using the male head of household's surname. If that failed to render results I then manually searched for entries to account for alternate spelling of surnames and given names. If I was still unable to find a link, I referred back to the reconstituted family data to retrieve other possible surnames connected to the family. In one instance, a wife's maiden name unearthed a genealogical connection. I also searched the deaths in the 1919 TAPL records (DIAND 1896 – 1959) in an attempt to connect them to influenza deaths in the parish records and reconstituted family files. It was hard to make definitive connections. In another instance I assigned a Treaty Annuity Pay List number to a reconstituted family because the death registry included information that linked individuals into a single family¹⁴. This particular family lost 3 of 4 family members (according to the reconstituted family), which corresponded with what was recorded within the TAPL for 1919. Also, the surname listed on the TAPL was a common derivative of the name listed on the death registry.

¹⁴ Under the quality/trade section a note appeared that read "wife of said individual", and so on.

Once the influenza death data were collected and confirmed for both Fisher River and Norway House, it was necessary to assess which families would be included in the reconstituted family portion of the study. Since the central aim of my project is to understand how influenza spread within families, and ultimately from family to family, it was important to establish guidelines for inclusion. The question that I was confronted with is what happens to the sample of families from Fisher River and Norway House if individuals are removed from the analysis? On the other hand, it is also necessary to assess how the data become skewed if people are included who do not have appropriate information. Since all individuals were included in the aggregate chapter of this thesis and the aim of this section was to comprehend the family context of mortality, only individuals who could be definitively linked to a nuclear family were included.

Chapter 5 The Aggregate Analysis

Introduction

This chapter is designed to address some fundamental questions about the dynamics of the epidemic by examining some basic questions:

- Who died first?
- How many people died?
- What was the age and sex distribution of mortality?
- What was the impact of mortality?
- Did influenza mortality cluster in families?

These questions help expose general patterns in transmission and dissemination of the pathogen. By delineating who died first it may be possible to decipher how the virus entered the community. Numbers generated for total mortality and age and sex distribution allow for easy comparison between a number of communities. As well, looking at the impact of the epidemic also allows these raw numbers to be contextualized. Finally since the focus of this thesis is to understand the family context of mortality an exploration of how families were affected by the epidemic is essential. Although this section focuses on aggregate analysis, it is important to note that family reconstitutions were relied upon heavily to help flesh out a more detailed mortality profile.

How Many People Died?

A total of 76 individuals (38 from each community) are listed as having died from influenza during the epidemic within the data collected from the Methodist and Anglican burial records for Fisher River and Norway House respectively. According to census reports (Department of Indian Affairs, 1917) available for 1917 there were 208 Anglicans out of a total population of 734 at Norway House and 284 Methodists registered at Fisher River out of a total population of 493. Thus, about 18.27% (183 deaths per thousand) of the Anglican population perished from influenza during the epidemic at Norway House while 10.21% (102 deaths per thousand) of the Methodist population died from influenza at Fisher River. The Norway House estimate is corroborated by published reports for the region (Herring 1994; Herring and Sattenspiel 2003). Krotz (1990: 31), reports 160 deaths occurring out of the total population of 734 (proportionate mortality = 21%). The estimates for Fisher River corroborate Herring's 1994b estimate of about 100 deaths per thousand (Herring 1994b: 375). This is a high mortality rate relative to Lux's (1992: 25) estimated average of 37.7 deaths per thousand or just under 4% for the Canadian Aboriginal population during the pandemic.

According to Herring (1994: 374), the difference in the number of deaths

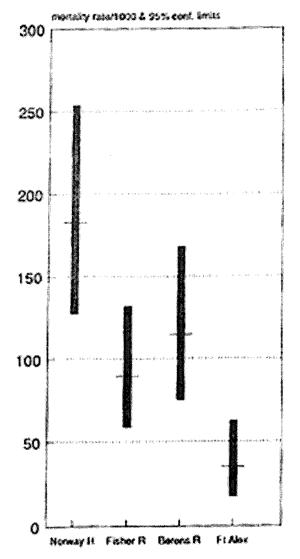
Figure 5-1 Estimated Spanish Flu Mortality Rates per 1000. Reproduced from Herring 1994b: 374.

experienced within these two communities is not statistically significant. In order to arrive at this conclusion she estimated influenza mortality rates utilizing methods described by Lilienfeld and Lilienfeld (1980). To account for the small sample size she calculated the 95% confidence levels using

a Poisson distribution and a crude mortality rate of deaths per 1000 for each community (Figure 5.1). Even though there appears to be a marked difference in the point estimates for mortality at Fisher River (102 deaths per 1000) and Norway House (183 deaths per 1000), when considered within the confines of the small size of these populations this difference is negligible.

Tracing the Spread and Impact of the Epidemic

The epidemic seems to have struck Fisher River prior to arriving at Norway



House. According to the Methodist burial register, the first individual at Fisher River to

succumb to the flu virus was Clara Clairson¹⁵ on October 30, 1918. By the time she was buried (November 01, 1918) Spanish Flu had already claimed its second and third lives in the tiny community of Fisher River. From the family reconstitution data we know that Miss Clairson was the unmarried daughter of Elijah Clairson and Mary Budd. She had nine siblings, four brothers and five sisters, of which two were younger than her. Three of her first cousins also died of influenza during the epidemic. The son of her sister, Mary Ann and Mary's husband, Benjamin Crane (married August 2, 1909), died on the 5th of November 1918 at two years of age. The son of her sister, Georgina, and her husband, John Mase (married November 20, 1912) died on the 19th of November 1918 at five months of age, and the daughter of her brother, John R., and his wife Addie Clairson, (married September 12, 1907) on the 8th of June, 1919 at approximately two and one half years of age.

Mrs. Effie Roberts was the first individual at Norway House to perish from influenza (HBCA B.154/a/87; ACCA 1902 - 1941). She died on December 18th, 1918. Not only is Mrs. Roberts the first death identified in the post journals, but the Anglican parish registry corroborates her date of death. From the reconstituted family data and the post journal entry, we know that Effie Roberts, whose maiden name was Keamawini, was married to Roderick Roberts. His occupation is listed as 'Indian'. Effie was the mother of six children; at the time of her death two had already died, one of an unknown cause at 2 months and the other at 12 years of age of tuberculosis. Three of her four remaining children later succumbed to tuberculosis.

¹⁵ All surnames with the exception of the ministers for the two communities and Mrs. Stevens are pseudonyms

Although these two individuals seem to have been the first to perish from the epidemic in their churches, it is unlikely that they were the first to contract the disease within their respective communities. The first recorded case at Fisher River appears to have been, Reverend Frederick Stevens, as indicated by the account kept by Frances Stevens. She states "The "Spanish Flu", as elsewhere, it was a terrible experience. The work in the dispensary never ended and while my husband was the first to get it, he kept going. When a doctor arrived, in due time, he ordered him to confine his work to dispensary and burying the dead. It was a miracle that he could accomplish that..." (UCCA Frances Stevens n.d.: 107). There is evidence from her memoirs that their mission was the only lodgings available for travellers to the area. This may account for the entry of the virus into the community although there is no mention made in her accounts of anticipating or entertaining a visitor.

The onset of the epidemic at Norway House is clearer. It appears to have entered the community via the mail system. According to Herring (1994: 85 - 86) the epidemic was spread to Norway House from Clearwater via Cross Lake. The post journal from December 03, 1918 reads that ""Spanish Flu" was also reported at an Indian camp at Clearwater Lake, some two or three days north of Cross Lake" (HBCA 1918 – 1919: fo. 9). On the fourth it is reported that a packet arrived from Cross Lake (HBCA 1918 – 1919: fo. 9) at Norway House and by the ninth of December 1918 the local chore boy is recorded in the post journal as being indisposed with an unnamed ailment, which arguably could have been the influenza virus.

An interesting dichotomy arises when we explore the entry point of the virus within these two communities. At Fisher River the epidemic may have begun with the arrival of an unknown individual to the home of the Methodist minister. Subsequent spread seems to have been a product of community interactions with either this individual or the newly infected minister. Clara Clairson is the index *death* for the Methodists, but likely not the index *case* for the greater community. As a result, establishing the conditions under which the disease entered the community becomes more difficult. A detailed discussion pertaining to her possible role in the entry and subsequent spread of the virus in the Fisher River community is provided in the next chapter. For the purposes of this analysis it is likely that her infection was the result of interaction within her community.

At Norway House it can be stated with a high degree of confidence that the HBC served as the entry point for the virus within the community. Both individuals first identified as being afflicted with the virus have a strong relationship to the post. The first was in its direct employ, acting as the local chore boy while the other was the wife of an employee of the company. This relationship is mentioned in an entry in the post journal which indicated that the death of Roderick Roberts wife as the first death from flu (HBCA 1918 – 1923; fo. 9). The spread of the virus, therefore, seems to be linked to economic factors that influence these individuals' daily lives, or more aptly, the peoples' reliance on the post for basic necessities (Hanks 1982: 107).

I would be remiss however if I did not address the simple fact that these conclusions may merely be the products of the research material available for this project.

Much of the information from Fisher River is linked to the local clergyman through diaries and parish records and therefore one would expect that he, his family, and close associates would be mentioned in the documents. The same is true for the data available for Norway House. The illness of the resident clergyman at Norway House and the availability of post journal entries may incorrectly identify the HBC as a portal of entry for the virus.

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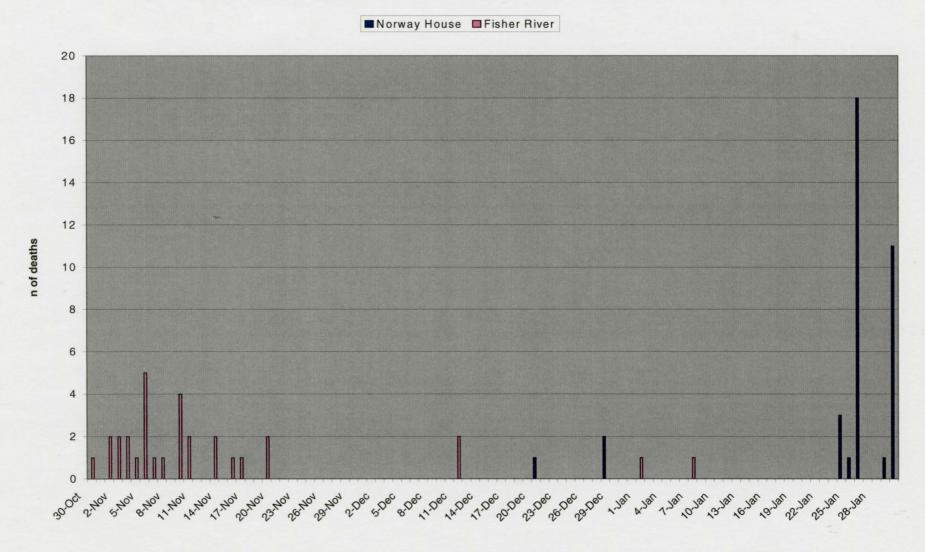


Figure 5.2 Burial Chronology Norway House & Fisher River From October 1918 to January 1919

Figure 5.2 charts the chronology of the epidemic at Fisher River (pink) and Norway House (blue),¹⁶ as seen throughout the parish registers. With the omission of one individual at Norway House and the five at Fisher River, the pandemic seems to have been somewhat more protracted at Fisher River lasting seven weeks and two days compared to Norway House's five weeks and six days¹⁷. Fisher River experienced a fall epidemic beginning on the 30th of October and tapering off by the 19th of November with sporadic deaths after the height of the pandemic. On most days between the 4th to the 10th of November the community seems to have experienced anywhere from 3 to 5 burials daily. The number of deaths peaks on the 5th of November with five deaths and then again on the 9th with four. It is hard to consider the impact of mortality as being anything less than overwhelming at Fisher River. The deaths are distributed across many families, which indicates that there were probably very few families untouched by this epidemic. Reverend Stevens acknowledges the disruptive nature of influenza and the quick succession of burials is incontrovertible evidence of a society in turmoil.

Norway House's epidemic took hold in winter with the first death occurring on the 20th of December. There are two more deaths recorded on the 28th with none recorded until the 24th of January, nearly one month later. Thereafter, burials seem to

¹⁶ A number of Spanish Flu deaths were omitted from this figure. They include the following 5 deaths from Fisher River: one that occurred on the second of February 1919; one from the twenty second of April 1919; one from the twenty seventh of May 1919; one on the eighth of June 1919, one from the fifth of July 1919; and one on the fifth of November 1919. One death on April 8, 1919 for Norway House was omitted. They were removed due to the large amount of time that elapsed from the onset of the pandemic and subsequent period of heightened mortality.

¹⁷ Time line based on time elapsed from the first burial to the last within each community.

peak over the next seven days with three burials on the 24^{th} , one on the 25^{th} , eighteen on the 26^{th} , one on the 29^{th} and eleven on the 30^{th} .

Herring (1994) suggests that the gap in burials may be the result of the local clergyman falling ill himself. This explanation seems very plausible and is supported by a post journal account from Wednesday January 1, 1919, which reads "Visited Mr. Marshall, who is down with the 'Flu'" (HBCA 1918 – 1923: fo.12). It can be stated with almost absolute certainty that this entry pertains to Reverend Marshall as the date of his illness coincides with the break in burial registration. The date of his recovery, January 22, 1919, also is noted in the post journal, reads "Mr. Marshall visited the Fort, this being the first time he has been out since taken down with the "Flu"" (HBCA 1918 – 1923: fo.12). This matches up with the period in which burials resumed (January 24, 1919).

There is further evidence of a delay between death and burial in the HBC post journal accounts are scrutinized. Of the individuals mentioned from the post journal, three were also found within the Anglican Church's burial register. The first death, that of Effie Roberts, helps to establish the accuracy of the post journal account. According to William Campbell on Wednesday December 18, 1918 "Roderick Roberts's wife died" (HBCA B.154/a/87: fo.8). Reverend Marshall supports this date by listing her date of burial as December 18, 1918. The next death listed in the post journal that is found in the burial records is that of Jennie Oman Arthurson [Artherson]. According to the factor "Mrs. Arthurson died" (HBCA B.154/a/87: fo.8) on Monday January 6, 1919. Reverend Marshall recorded her burial on January 25, 1919 some nineteen days later. Finally on Wednesday January 15, 1919 the post journal reads "John Gunn died" (HBCA

B.154/a/87: fo.8), yet the burial record indicates that he was not interred until the 24 of January 1919.

The impact of mortality at Norway House was staggering. Its immediate effects can be witnessed from post journal entries and other reports from the time. We know from the post journal that by December 19th communication with neighbouring communities had broken down as dog teams could no longer be assembled to carry on even the most basic services, such as mail delivery. The first mention in the post journals of attempts to get provisions out to 'Indians' on trap lines occurs on the 4th of January, 1919 when the factor reports sending a "team off to deliver wood" (HBCA B.154/a/87: fo.8). This is seventeen days after the first death is reported at Norway House. As well the usual Christmas rush did not occur as "[m]ost of the people [were] sick" (HBCA 1918 – 1923 fo.9) and unable to make the trip to the post. On the 29th of December the factor, William Campbell, visited Dr. Norquay (the local physician) and reports that there had been eleven deaths from flu thus far (HBCA 1918 – 1923 fo. 9). Three more deaths were reported the next day and by the 6th of January the number had risen to fifty.

Herring (1994b) conducted interviews with residents of Norway House that help articulate some of the disarray that was experienced first hand. An informant described to her the "difficulties of burying so many people who died so suddenly, noting that they were brought to a cabin and stacked like wood until it was possible to bury them later" (Herring 1994b: 376). The unpublished autobiography of Frances G. Stevens titled "My Experience Living On A Mission" also indicates the problem of finding able-bodied

individuals to help prepare and bury the deceased. She states that at "Berens river thirteen bodies lay in the mission storehouse because there was not a sufficient number of men able to dig resting places" (UCCA Frances Stevens n.d.: 107).

Accounts from neighbouring communities describe complete breakdowns in dayto-day functioning as well. At Berens River Harry Everett recalled that everyone but his father had succumbed to the illness and it was all that his father could do to simply ensure that there were adequate food and wood supplies in all of the individual residences.

Herring (1994) gives a very thorough account of the long-term impacts of the 1918 influenza epidemic at Norway House. She examined the Anglican registers to see if the epidemic had any impact on either the birth or marriage records. She found no significant change in the number of births in the 10 years following the epidemic, but noted a small but dramatic increase in the number of marriages. She attributes the increase to the fur-trade based economy, which lends itself to a division of labour along sex-lines. In order to maintain community functioning in such an economy it is essential to re-establish gender-based cooperative labour formalized through marriage (1994: 93 – 94). She also notes that it took approximately ten years for the population to bounce back to pre-epidemic size. This level of analysis could not be carried out for the Fisher River data due to breaks in the registration of vital events for the community.

	Norway H	louse	Fisher River		
	N	%	N	%	
0 - 6	3	1.44	15	5.28	
6 - 15	11	5.29	6	2.11	
16 – 20	1	0.48	4	1.41	
21 – 65	18	8.65	4	1.41	
65+	3	1.44	1	0.35	
Unknown	2	0.96	8	2.82	
Total	38	208	38	284	

Age and Sex Specific Proportionate Mortality at Norway House and Fisher River

 Table 5-1 - Age at Death Distribution for Influenza Mortality 1918 - 19 per 100 Population. Based on Categories Derived from the 1917 Census for Norway House and Fisher River

Table 5.1 displays the age at death distribution for influenza using categories derived from the 1917 census (Department of Indian Affairs. Sessional Papers No. 27 George V, A.1917) for Norway House and Fisher River. At Norway House there were very few deaths in the under 6 category (n = 3, or $7.89\%^{18}$), the 6 – 15 age category experienced the second highest mortality encompassing just under 29% (n = 11) of the total influenza mortality experienced at Norway House. The 16 - 21 category is represented by one death (2.63%). The majority of deaths occurred among individuals between the ages of 21 – 65, comprising just over 47% (n = 18) of the total mortality for the Anglican population. The last age category of over 65 also experienced only a few deaths (n = 3 or 7.89%).

At Fisher River there appears to be an overrepresentation of deaths in the 0-6 category (n = 15 or 39.47% of the total mortality). The 6 - 15 age category encompassed

¹⁸ Percent recorded in relation to number of deaths (38), not entire population at risk within the Norway House community.

the second highest mortality with 6 deaths (15.97%). Four deaths occurred in the 16 - 20 age grouping leaving only 4 deaths in the 21 - 65 age range and one in the over 65 category.

Although this table yields some interesting results they are not amenable to a chisquare analysis, as chi-square tests should not be used if any of the expected frequencies is less than 5 (Huck 2004: 475). To combat this shortcoming the data were organized into two broad age categories, created to reflect current ideas about the age distribution of mortality during the 1918 pandemic. Influenza mortality normally takes the highest toll among the very young and the aged. Beveridge (1977: 50) suggests that the risk of developing fatal pneumonia as a sequela from an influenza infection increases after the age of fifty. One of the defining characteristics of the strain that was responsible for the 1918 – 1919 pandemic was that it caused a peak in mortality among individuals between the ages of 20 and 40 (Osborne 1977, Crosby 1989, Herring 1994, Phillips and Killingray 2003 to name a few). Beveridge (1977) also indicates that those studying mortality from the pandemic witnessed an increase in infant and perinatal mortality. He states that "In 1918 – 19, one series of 1350 pregnant women who had influenza were observed: abortion, stillbirth or premature labour occurred in 26 per cent of those without pneumonia and 52 per cent of those with pneumonia, which was a feature not expressed either in the 1957 – 58 or 1968- 69 pandemics" (Beveridge 1977:15).

To work within the confines of the pre-established parameters of the 1917 census the two age categories that were chosen were 0- 20 years (encompassing the 0- 6, 6 - 15

and 16 - 20 age groups) and 21 - 65 + (which subsumes the 21 - 65 and 65 + age classification).

These groups were first

Table 5-2 Age and Sex Distribution for Influenza
Deaths at Norway House, 1918 - 1919

	Male		Female			
	n	%	n	%		
0 - 21	7	19.44	8	22.22		
21 - 65+	13	36.11	8	22.22		
Total	20	55.56	16	44.44		
				36		
$x^2 = 0.8229, df = 1, p = 0.3643461$						

checked at the individual community level to ascertain if there was any difference in the distribution of mortality by sex. The first community examined was Norway House (Table 5.2).

The null hypothesis for this test

is that there is no difference in the age distribution of influenza deaths, by sex. The chi square results in Table 5.2 show no differences and therefore the null hypothesis is

Table 5-3 Age a	nd Sex Distributi	ion for Influenza	a Deaths
at	t Fisher River, 19	918 - 1919	

	Male		Female		
	n	%	n	%	
0 - 21	15	51.72	9	31.03	
21 - 65+	2	6.90	3	10.34	
Total	17	58.62	12	41.38	
				29.00	
$\chi^2 = 0.8636$, df = 1, p=0.6670265 yates corrected					

accepted ($x^2 = 0.8229$, df = 1, p 0.3643461). Table 5.3 presents influenza mortality by sex for Fisher River. Again the null hypothesis is that there is no difference in the age distribution of influenza deaths by sex. The cell values for the over 21 age groups have values less than 5, and so the results of the test must be interpreted with caution. The Yates' corrected chi square test detected no differences in the two distributions ($x^2 =$ 0.8636, df = 1, p = 0.6670265, the Fisher exact 1-tailed p-value = 0.3924767 and 2-tailed p-value = 0.6220791).

	Norway I	louse	Fisher River			
	n	%	n	%		
0 - 21	15	39.47	24	82.76		
21 - 65+	23	60.53	5	17.24		
Total	38	100.00	29	100.00		
$x^2 = 12.6, df = 1, p < 0.0001$						

 Table 5.4 Age at Death Comparison of Influenza Deaths 1918 - 1919 for Norway House and Fisher

 River

Table 5.4 compares the age structure of mortality at both Norway House and Fisher River. In order to determine whether the mortality patterns differed, the chisquare test was employed. The null hypothesis for this test is that there is no difference in the age distribution of deaths at Fisher River and Norway House when two categories are employed: under 21 and over 21. The results for the chi-square reject the null hypothesis. This indicates that the distribution of deaths within the cells is not the result of chance. The chi-square was constructed to check two probabilities. The first was if the number of deaths differed within the two age groupings (under 20 and 21 and above) and the second was to compare the distribution of deaths at Norway House and Fisher River. Both of these questions resulted in statistically significant findings ($x^2 = 12.6$, df = 1, p = 0.0003720).

There are a few possible reasons to explain the differential distributions by age of flu mortality at Fisher River and Norway House. The first possibility is that the difference is simply an artifact of the data, that is, the record keeping practices of the two clergymen. Reverend Marshall (Norway House) included the age at death of the individual within the burial record, even for individuals who did not appear to be affiliated with his parish (n = 6 or 16%). This meant that even individuals without a connection with the parish (or a valid family reconstitution) were included at this level of analysis. Therefore at Norway House all 38 influenza deaths were included in the chisquare test for probability. This is in contrast to the 29 (76%) included at Fisher River.

Reverend Stevens (Fisher River) demonstrates clear methodical thinking and record keeping in relation to his parishioners but there is very little evidence that this same level of care was extended to those who weren't members of his parish. He does not even appear to have kept records on converts he encountered and either baptises, buries or marries during his visits to neighbouring communities. As a result of his record keeping practices there were nine individuals out of the original 38 who could not be included at this level of analysis. Of the nine omitted there were three males of unknown age, three females of unknown age and three individuals of unknown age and sex.

It is not clear to what extent population size comes into play in this analysis. According to census data the Norway House population in 1917 was 734 with 208 Anglican's contributing to the total population (Government of Canada, Sessional Papers No. 27 1917). Although the entire population at Fisher River was only 493 the Methodist population accounted for more than half of this number (n = 284). The age distributions within these two communities are quite similar, and would not contribute to the differences expressed in the findings of the chi-square analysis. For example at Fisher River the under 6 age category accounts for 17.85% (n = 88) of the entire population;

similarly at Norway House this same age grouping accounts for 20.57% (n = 151) of the entire population.

The differential distribution of ages at death between these communities is a harder problem to tackle; again it could be a result of the record keeping. Researchers caution scholars undertaking population studies based on parish records that there is a high incidence in underreporting of infant deaths. Razzell (1972: 122) suggests that the delay between birth and baptism can result in under representations in both births and deaths of infants and young children and therefore can sway demographic analysis. This observation could account for the limited number of infant and young children reported as having perished at Norway House. Reverend Stevens' investment in his 'flock' could suggest why this same anomaly did not occur at Fisher River. The relatively large number of infant deaths at Fisher River could also be a natural product of the 1918 influenza strain.

It is important to note however that although Drake's analysis was carried out on the Norway House records and indicates that the records were accurate for the short time period they are available for (see Appendix I), extra steps were taken to check for underreporting of infant deaths. The parish records were cross-referenced against Treaty Annuity Pay Lists (TAPL) (DIAND 1896 – 1959) for a fifteen-year period. This is a technique suggested by Hoppa (1998: 180). Six families were chosen at random¹⁹. Out of the 20 children that were included in the sample only two discrepancies arose in the reporting of the number of vital events. One was a child noted in the TAPL but not in the

¹⁹ All of the families were assigned a number then using a random number generator available at <u>www.random.org</u> the families that corresponded with the first six numbers generated were analysed.

birth registry of the Anglican mission and the other was a child listed in the Anglican records as having died one month after birth who was omitted from the TAPL documents.

Year	Men	Women	Boy	Girl	D.	Man	D.	Women	D.	Boy	Β.	Girl	Notes
1905													
1906	1	1		1									girl born
1907	1	1		1									
1908	1	1		1									
1909	1	1		1									
1910	1	1		2									girl born
1911	1	1		2									
1912	1	1	1	2									boy born
1913	1	1	1	2								-	
1914	1	1	1	2									
1915	1	1	2	2									boy born
1916	1	1	2	2									
1917	1	1	1	3									girl born
1918	1	1	2	3									
1919	1	1	3	2								1	boy born and girl died
1920	1	1	3	2									

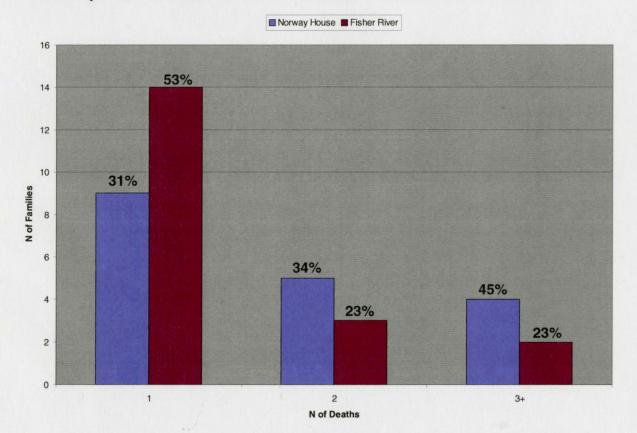
TREATY ANNUITY PAY LIST DATA

RECONSTITUTED FAMILY DATA

Husband: Appatag, Fredericl	k Margham	Occ. Indian	Born:
Married:	Died:	Cause:	
Husband's Father:		Mother:	
Other Wives:			· · · · · · · · · · · · · · · · · · ·
Wife: McLeod Mary Ann		OccWhite (?)_	Born:
Married:	_ Died:	Cause:	
Wife's Father:		Mother:	
Other Husbands:			
Children			
Given Name, Surname. Born	-		ed. Cause.
1Kanena Eva 4/21/1906_D			
2. Norah Jane 2/27/1908 I	DD~3/24/1908	@.08 Whopping Cou	ıgh
3. Sarah Ann 2/1/1910			
4. Walter Eli 3/14/1912]	DD~5/30/1922	@ 12 Influenza	
5. Donald Stanley 9/30/1914	1		
6. Maggie (Margaret Jane) 2			
7. Moses Lawrence 7/3/19	19		

Figure 5.3 - Comparison between Reconstituted Family Data and Treaty Annuity Pay List Information for a Family Chosen at Random

Figure 5.3 is a comparison of TAPL documents to a reconstituted family data derived from the Anglican mission record. This family was chosen to act as an illustration of this methodology because it is the first name to appear when the families were sequenced alphabetically. Under traditional family reconstitution methodology this family would not be included as no marriage information is available for this union. It is evident nevertheless that the two lines of evidence support one another with the exception of the omission of the birth and death of Norah Jane who perished just under one month after birth.



A Comparative Look at the Distribution of Death Within Families

Figure 5-4 Distribution of Influenza Deaths for Norway House and Fisher River Per Nuclear Family October 1918 - July 1919

Figure 5.4 compares the number of deaths experienced in each nuclear family at Norway House and Fisher River. Of the 38 individuals included in Reverend Stevens' burial records at Fisher River, 26 (68%) can be linked to reconstituted families²⁰. Of these twenty-six, the deaths per family break down as follows: one death each in fourteen families; two deaths each in two families; and three deaths each in two families. This equates to 53%, 23%, and 23% of all deaths reported in this community respectively. No families experienced four or more deaths.

Of the 38 individuals included in Reverend Marshall's burial records for Norway House, 29 (76%) can be linked to reconstituted families. Of these twenty-nine the breakdown of deaths per family is as follows: one death each in nine families, two deaths each in five families, three deaths each in two families and four deaths (or the complete removal) in one family. This equates to 31%, 34%, 31% and 14% of all deaths reported in this community respectively. From this figure we can ascertain that Fisher River had the highest proportion of deaths in the one member per family range (n = 14 or 53%). In contrast, multiple deaths within households appears to be the norm at Norway House with just under 50% of all deaths taking place within four families. The difference in the distribution of deaths per nuclear family, however, is not statistically significant ($x^2 = 2.23$, p = .3282, df = 2).

²⁰ Individuals were linked to families if their first and last names were recorded. Individuals with possible links were not omitted from the sample, i.e. no marriage record, as is usually the norm with family reconstitution techniques. Individuals were included if there was a strong likelihood that there was a family connection (i.e. probable family cluster without parents' ages).

Chapter 6 The Scott and Duncan Approach

Methods outlined by Scott and Duncan (2001) utilize mathematical modeling techniques to track the spread of disease within and between families. Their aim is to disprove the hypothesis that *Yersinia pestis* was solely responsible for the mortality experienced in Europe during the Black Death pandemic during the 1300s and the Great Plague of London in 1665.

In order to assess the impact and spread of the 1918 influenza pandemic within Norway House and Fisher River it is my aim to trace the dissemination of the disease throughout families, and ultimately throughout the two communities, by utilizing some of the methods outlined in Scott and Duncan's *Biology of Plagues* (2001). Specifically, I am interested in understanding how the infectious and latent periods that help to define the transmission possibilities of the influenza virus may allow researchers to estimate a pattern of dissemination throughout a given community. Scott and Duncan (2001) utilize the following statistical concepts in order to expose their 'mystery' ailment as well as rule out the presence of *Yersinia pestis:*.

- transmission probability (the r umber of contacts required to create subsequent infection)
- secondary attack rate (the number of persons exposed who develop the disease divided by the number of susceptibles)

- basic reproductive number R_o (number of contacts per unit time x the transmission probability per contact times the duration of infectiousness)
- virulence (measure of the speed with which a parasite kills an infected host)
- case fatality ratio (probability of dying from a disease before recovering or dying of something else)

For the purposes of this thesis, it is not necessary to establish these parameters for they are known for the influenza virus (Beveridge 1977, Krause 2000) and the 1918 outbreak (Taubenberger et al 2001, Reid et al 2000, Laver 2000). Consequently, these statistical concepts are used to help reconstruct the characteristics of the spread of influenza rather than to aid in the identification of an unknown pathogen. This subtle but important distinction affects how mortality is viewed and structured by this type of analysis.

Assessing the Practical Application of the Scott and Duncan (2001) Approach

In order to carry out the Scott and Duncan (2003) technique it is necessary to comprehend the requirements that are essential to this type of methodology. Scott and Duncan (2001) based their 'plague' research on reconstituted family data. They establish that the registers for the region of their study²¹ begin in 1557, then go on to state that they "have carried out a full family reconstitution from this date until 1812" (Scott and

²¹ Although their book deals with the epidemic in England (London, Stratford-upon-Avon, Richmond, Durham), France, Italy, Germany, and Switzerland, the area they explore in most detail is Penrith. This is also the region in which much of their previous work is based.

Duncan 2001: 115). It is important to note that the quality of the registers for this 255year period does not seem to be addressed in their 2001 publication. In a 1993 article in which they worked with the data from published records of the parish of Penrith, Cumbria they state that they "made no attempt to test the robustness of the records, to consider the effects of immigration and emigration, or to estimate the underregistration of baptisms" (Scott and Duncan 1993: 408). In a subsequent article published in 2000 in which they examine nutrition, infant mortality and fertility for the parishes of Penrith they state

The quality of the registers of births and deaths was tested by the techniques advocated by Wrigley et al. (1997) in which the birth intervals are divided into the following three categories: (1) previous child known to have died within the first year of life; (2) previous child known to have survived its first year; (3) fate of previous child unknown (Scott and Duncan 2002: 73).

This sentiment is echoed in their 1999 article which states that they used reconstitution methods outlined by Wrigley et al. (1997), and make no mention of the validity of the records.

Scott and Duncan also draw upon 'plague' sufferers' wills and last testaments. They mention that there was information available in the form of Hearth Tax documents as well as the Compton Census data, although they state that the "Hearth Tax is not an entirely reliable source, since there is no certainty that all households were included and possibly up to 40% were never recorded" (Scott and Duncan 2001: 118). They also have access to an account of the plague written in 1894 by William Furness entitled *The History of Penrith from the Earliest Record to Present Time* which the researchers dismiss as "anecdotal and conjectural" (Scott and Duncan 2001: 117).

By attempting to replicate their analysis for the purposes of studying the 1918 influenza pandemic, it became apparent that specific information must be at hand. First, it is not sufficient to work from aggregate data; family reconstitutions must be carried out. Although transmission patterns can be examined by simply plotting burial sequences in cases where family relationships are noted (as was the case for the Norway House data), even so no further analysis can be conducted as relationships within and between families cannot be fully detailed. Dates of death are also essential to this form of analysis, especially when working with a pathogen, like influenza, that exhibits short incubation and infectious periods. This poses a serious problem as epidemics have a tendency to disrupt daily life and record keeping. This occurred at Norway House. The post journal (HBCA B.154/a/87) notes that Reverend Marshall, the sole Anglican minister, contracted influenza during the height of the epidemic (at or around January 01, 1919) and was unable to continue his duties of burying the dead. As a result the community experienced a backlog of burials clustered near the end of the epidemic. Because the burial records do not include dates of death, it is impossible to carry out a Scott and Duncan type analysis on the Norway House Anglican records. Consequently, this analysis focuses on Fisher River, where the data requirements are met.

	Scott & Duncan (2001)	Slonim (2004)
	Case Study at Penrith	Fisher River Case Study
Pathogen	Unknown	Influenza
Latent period:	10 - 1 2 days	1 - 2 days
Infectious Period before symptoms:	20 - 22 days	hours if anything
Period of Symptoms:	5 days	2 - 5 days
Total infectious period:	25 - 27 days	2 - 5 days
Total time - infection to death:	37 days	3 - 7 days
Materials	1 - Full family Reconstitution from 1557 - 1812	1 - Family Reconstitution 1888 - 1920 - Few breaks, meets Drake's protocol (Moffat 1992: 43 - 53)
	2 - Wills	2 - The Stevens' Accounts UAC Fredrick G. Stevens n.d. UCCA Frances Stevens n.d.
	3 - Census	3 - Census
	4 - Historical Accounts	4 - Newspapers Accounts
Objective	Rule out pathogen -	Micro-level analysis of
	Yersinia pestis	dissemination
Results	Modeled Morbidity	Analyzed Mortality

Table 6-1 General	comparison between	Scott and Duncan	(2001) a	nd this analysis

Table 6.1 compares the characteristics of the microorganism as well as the materials used by Scott and Duncan (2001) and here. The primary difference between the two is that their research was carried out primarily to demonstrate that the transmission pattern of the disease they were tracking in Penrith mimicked a Reed-Frost distribution, indicative of person-to-person transmission, rather than the more widely held belief that the Great Plagues (1347 – 1670) were caused by a vector-borne pathogen. Their primary focus then, was to establish a parameter by which the data would lend itself to this type of dissemination. My focus on the other hand was simply to chart the spread of the H₁N₁ influenza virus throughout Methodist families at Fisher River.

There are pros and cons to both approaches. Working with an identified, welldocumented disease brings with it guidelines that are exceedingly rigid, which leaves the timelines of transmission less open to interpretation. Let me refer the reader back to Figure 6.1 in order to illustrate this point. There are five general time periods that must be established when employing the Scott and Duncan (2001) approach; they are the latent period, the infectious period before symptoms, the period of symptoms, the total infectious period and the total time from point of infection to time of death.

Scott and Duncan (2001) created their timelines for the Penrith pathogen by working backwards. They began first by establishing a transmission pattern. They then set about creating time periods that would adhere to their original hypothesis. In order to have their collected data represent an infectious disease transmitted from person-to-person they had to establish the presence of an infectious disease that is characterized by a long, asymptomatic infectious period (20 - 22 days), a sizeable latent period (10 - 12 days) and a comparatively short symptomatic period (5 days). The fact that this pattern is not necessarily indicative of any known pathogen does not seem to alarm them for they claim that the offending microorganism was a progenitor of today's hemorrhagic filoviruses (Scott and Duncan 2001: 384 - 389). To support this hypothesis they draw upon current epidemiological theory of the well-known, but poorly understood filovirus, Ebola. They state that with Ebola "symptoms appear after an incubation period of 2 - 21 days" (Scott and Duncan 2001: 386).

The advantage for them is that even though their findings are not supported by modern epidemiological theory their hypothesis cannot be disproved, as there is no documentation that can discredit their claim. As a result of this fact they are able to model a very smooth transmission of the pathogen through the communities and families within their study. Their 2001 publication (pages 129 to 137) illustrates a clear pattern of

infection moving slowly through one nuclear family at a time. However it is also important to note that it is not evident from their description that they have included all individuals afflicted with the pathogen or simply those belonging to their study families.

My findings are much less subject to interpretation, but what they gain in credibility and testability they lose in clarity and definition. Unlike Scott and Duncan I was able to plot the spread of infection by using pre-established parameters. My data as a result do not lead to the same clean transmission within families before continuing on to subsequent families. This is because of the disparity between mortality and morbidity. Through the use of modeling techniques Scott and Duncan (1993, 1999, 2000, 2001) often infer results based on morbidity data, where as this thesis is restricted to a discussion of actual mortality. To apply the full technique used by Scott and Duncan (2001) for the influenza pandemic of 1918, a 50% morbidity estimate would have to be inferred from a 2.5% mortality pattern.

Assumptions

Many difficulties arise when researchers attempt to reconstruct events of the past due to the intricacies of everyday life and human relationships. No two people will ever have the same experience of the same day for there will be variations in physical actions and emotional reactions. There is no way to code for fingernail biting, excessive hand washing, the random decision to decide not to show up for work or unknown family feuds. Since there is no way to encompass all of the variants of human behaviour, guidelines laden with inherent assumptions must be established. The best a researcher

can do is to introduce their assumptions and hope that their line of reasoning is consistent with the actual events that transpired. That said, the following are the main assumptions of this analysis.

Due to the short incubation and infectious periods of the influenza virus my decision to include the date of death as a possible date of transmission had quite a large impact on the postulated chains of infection. Inherent in this assumption is the idea that people died later in the day and therefore had an opportunity to infect others within the community. This is justified because this project is concerned with the spread of the virus within families. It is feasible to assume that family members would console and care for the sick until the time of their death.

Another major assumption made in this analysis was that mortality mimicked morbidity. One of the primary aims of this approach is to estimate how the microorganism spread throughout the community yet all that was available was parish records of burials, so a true analysis of spread was not possible. Those familiar with the work of Scott and Duncan (2001) will note that their analysis included individuals who did not perish from the disease. How this was possible was not explained, and therefore it was reasoned for this thesis that the assumption that mortality mimicked morbidity was more defensible than the assumption that all individuals in the community were afflicted by influenza. We know that the case fatality rate for influenza is generally very low, so within the confines of this study the assumption that mortality mimics morbidity cannot be accepted even if we take into account the higher case fatality ratio at Norway House and Fisher River.

The third assumption that was formulated was that there was a single point of infection for each community and that all subsequent cases are in some way linked to the index case. There are two problems with this conclusion: the first is the assumption that Clara Clairson was the first individual to be infected with the influenza virus. This is already a bone of contention as Frances Stevens states that her husband was the first individual to display signs and symptoms of the disease (UCCA Frances Stevens n.d.: 107). The second reason is that there is no clear evidence that the epidemic at Fisher River was the result of a single contact with a single infected individual. There is evidence of flu raging in neighbouring communities prior to and during the epidemic at Fisher River. Peguis experienced its first death from flu on the 29th of October, just one day before the first death at Fisher River.

Results

Since this project is based upon extensive research carried out by Herring and Sattenspiel (1998, 2000, 2003), I decided to use the influenza parameters they established. For consistency and clarity only the one-day incubation and five-day infectious period diagram, derived from these parameters is presented here. Appendix II contains the six other Scott and Duncan (2001) type diagrams that trace the spread of influenza throughout Fisher River.

Figure 6.1 charts the hypothesized spread of influenza throughout Fisher River from the first recorded Methodist death on October 30, 1918 to the end of the first wave on November 19, 1918. The Methodists experienced ten more deaths between December

11, 1918 to November 10, 1919 that are not incorporated into this diagram²². The individuals who are recorded within Reverend Stevens' burial registry are listed on the left-hand side of the figure in the order of their date of death. Individuals are identified by two components: the first is the name of the male head of the household²³ and the second is their relationship with the male head of the household. H indicates head/husband, W refers to wife, S to son and D to daughter. Ages appear in red where known. The line to the right of the individual represents their period of infectivity and the solid vertical line divides the period of infectivity into latent and infectious periods, represented here by a one-day latent period and subsequent five-day infectious period. The black square at the end of the line indicates the death of the individual. Dotted vertical lines demarcate the period in which individuals can infect one another, or rather the day from which they are infectious up to and including their date of death. Index denotes the first individual to contract and perish from the disease, S represents a secondary case, T indicates tertiary infection, Q, quaternary; and F is representative of a fifth generation of infection.

²² The purpose of this section is to understand how the virus traveled throughout and between households; under the predefined infectious and latent periods these individuals could not have infected one another.
²³ Pseudonyms are used

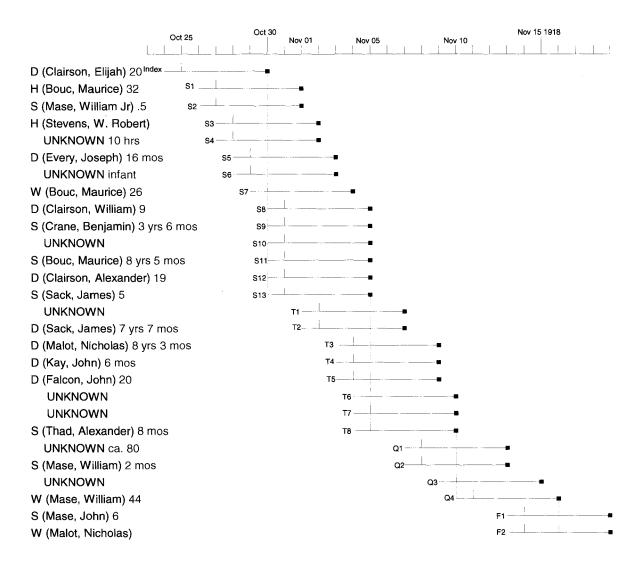


Figure 6.1 – Spread of influenza within and between families at Fisher River as represented by a one-day latent period and five-day infectious period. A standardized line that is divided into a latent and infectious period represents each individual. The first section of the line is representative of the latent period and the remainder of the line following the solid vertical line is infectious period. The square at the end of the line indicates the day the individual died. Dashed vertical lines indicate the period in which the primary case is infectious (from the beginning of the infectious period to death). Once the primary case has been removed a new dashed vertical line is established to represent tertiary cases and so on. Removed individuals are listed on the left hand side of the figure. They are represented by the name of the male head of household, which is preceded by their relationship to this individual. H represents head/husband, W, wife; S, son and D, daughter. The letter and number preceding the solid horizontal line that indicates the period of illness for each individual represent their order infection by group (S, secondary; T, tertiary; Q; quaternary; and F, fifth generation) and with in groupings (1 – 13). Age is indicated in red where known and the scale is in days beginning on October 23rd 1918. Although the first victim suspected to have contracted the influenza virus was said to be Reverend Stevens (UCCA Frances Stevens n.d: 107) there unfortunately is no date given from which to derive the commencement of his period of infection. For the purposes of this analysis only individuals listed in the Fisher River Methodist burial record are included. Clara Clairson is the first recorded Methodist death and is therefore considered to be the index case of 'Spanish Flu' for the Fisher River community. With a five-day infectious period and one-day latent period, Ms. Clairson would have had to come into contact with the virus on or around October 24, 1918. The co-secondaries are 36-year-old Maurice Bouc and the six-month-old son of William Mase, each seemingly infected on the 26th of October. They are two of a possible thirteen infected by contact with Clara Clairson. With the exception of Robert Stevenson (age unknown), 26 year old Sarah Bouc and 19 year-old Mabel Clairson, all of the secondary infections occurred among children between the ages of 10 hours to eight years.

Eight individuals fall within the tertiary group of infections. This grouping is comprised of older children as well as a few adults. Although Alice Amos (T1) and Janet Clairson (T7) are listed as unknown individuals, it is likely that they are adults. This is surmised through probable family reconstitution links. Unknown (S6) is listed in the burial registry as Amos, Baby born November 3rd. It is my assumption that a cause of death listed as influenza is only possible if the mother of the child or another member of the immediate family is quite ill with the virus at the time of the birth. There is no record of Alice Amos anywhere in the Methodist records, so it is therefore my assumption that this individual was pregnant, passing through Fisher River at the time of her own and her

baby's early demise. There are four quaternary infections (one 80-year-old woman, a 2month-old child, a forty-four year-old woman and perhaps a six-year-old child (Q2). There are two fifth generation infections consisting of a 6 month old infant and a mother of five children.

Interesting patterns emerge when individual families are examined. Four nuclear families and one extended family experienced multiple deaths. The four families that contained more than one death did not appear to be tied to larger kin groups so an analysis that highlighted transmission from nuclear family to extended family does not seem possible for this group of families. The number of deaths clustered in families that do not appear to be linked to a wider kin network is interesting as the majority of singular deaths within families tended to be linked to a larger kin network (for a detailed discussion refer to the aggregate and family reconstitution sections of this thesis). To examine the spread within families, I created diagrams that trace transmission. The first family to experience multiple deaths was the Bouc family.

Figure 6.2 Spread of Influenza Through the Bouc Family

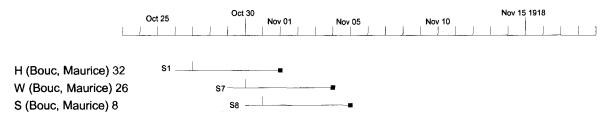


Figure 6.2 illustrates how the virus spread. The head of the household apparently came into contact with the virus on or around the 26th of October rendering him one of the first secondary cases. By the 29th his wife seems to have contracted the virus and by the fifth of November their son had contracted and lost his battle with the virulent

microorganism. This family was survived by their six-year-old son who died of pneumonia in 1921.

The Sack family was the second family to lose multiple members (Figure 6.3).

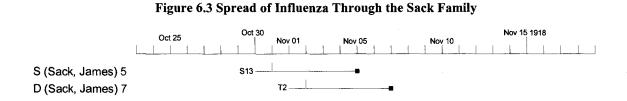


Figure 6.3 shows how the virus spread through this family. The first individual to be removed from the family was their youngest son, who was five years old at the time of infection. Some two days later, his sister contracted the disease and subsequently perished. She was just over seven and a half years old at the time of her death. These children were survived by their parents and two brothers, both of whom died in subsequent years of tuberculosis at ages 20 (in 1928) and 33 (in 1930).

The third family that experienced an excess of one death was the Malots (Figure 6.4).

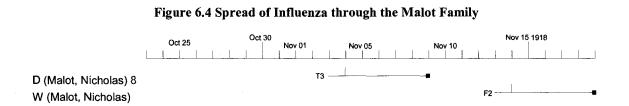
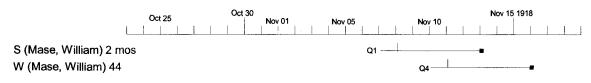


Figure 6.4 illustrates how influenza affected this family. Within the parameters of a one-day latent period and five day infectious period established for this study it would

appear that this family came in contact with the influenza virus twice. The first death that occurred was that of the youngest girl in a family of five. Her mother, who passed away on the 19th of November, was 44. It is also possible that the virus remained active within members of their immediate or extended household between their bouts with the virus.

The last nuclear family to experience multiple deaths was the Mases (Figure 6.5).

Figure 6.5 Spread of Influenza Through the Mase Family



The virus seems to have been brought into the family by the couple's two months old son, one of eight children (Figure 6.5). This is a highly unlikely scenario as two month old infants are unlikely to venture out on their own. This example helps demonstrate why the Scott and Duncan (2001) approach must be understood within the confines of its assumptions. The most likely situation is that this virus was brought in by a surviving sibling or perhaps even by his mother who passed away three days after the death of her youngest son. Four-year-old Russel Gordon died on the 11th of December 1918, less than a month after his younger brother. His death was one of the 10 deaths not included in this analysis because they fall outside of the inclusion parameters of the study in that his death occurred after the 20th of November.

Finally, using the methods outlined by Scott and Duncan, I was able to view a possible spread throughout one extended family (Figure 6.6).

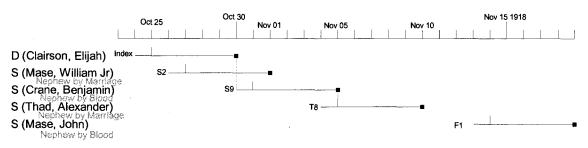
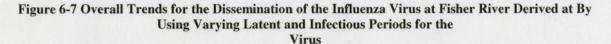
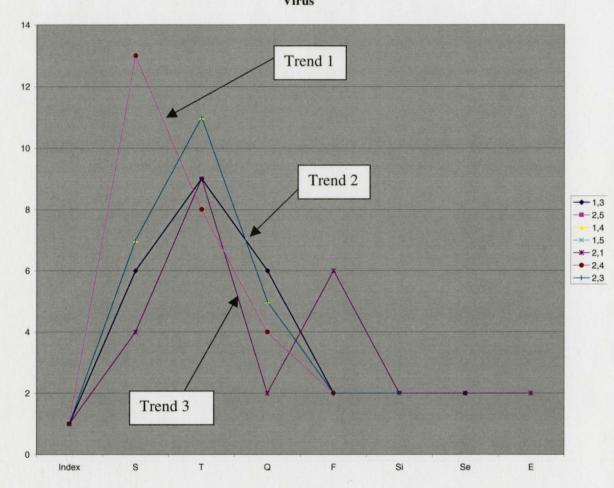


Figure 6.6 Spread of Influenza through the Extended family of Clara Clairson

Figure 6.6 depicts this spread. Clara Clairson, the daughter of Elijah Clairson, has been identified as the primary index death for this study of the Methodists. This figure suggests that she may have spread the disease to her nephews shortly after her own infection. It is also possible that they all came into contact with the virus at the same time. These children range in age from six months to three years although three of them are under one year of age. With the exception of Edgerton Ryerson Mase it appears that all of the subsequent infections can be related to contact with Clara Clairson.

Approximated Dissemination Throughout The Community





The Scott and Duncan (2001) approach also allows researchers to approximate the spread of infection within the afflicted community. Figure 6.7 charts the spread of the disease using the 7 possible combinations of infectious and latent periods described on page 66^{24} . The pattern of dissemination throughout the Fisher River community would have been same for 2 days latent, 5 infectious, 1 day latent and 5 days infectious, and 2

²⁴ These numbers come from the figures generated to examine varying infectious and latent periods as outlined by Scott and Duncan (2001) (See Figure 6.1 and Appendix II)

days latent and 4 infectious. As well the pattern that emerged for 1-day latent and 4 infectious and 2 days latent and 3 days infectious was identical. Although 1, 3 and 2, 1 were not completely undifferentiated, their general patterns were similar.

Trend 1 (2, 5; 1, 5 and 2, 4) commences with one index death, spikes at 13 secondary deaths, and begins a gradual decline in mortality with eight tertiary deaths, four quaternary and two fifth generation deaths. Trend 2 (1, 4 and 2, 3) is represented by a more gradual incline and decline with the largest number of deaths occurring in tertiary phase of the dissemination of the illness. Trend 3 (2, 1) is perhaps the most interesting for it exhibits two peaks: one during the tertiary phase of the illness and a second within the fifth generation of sufferers.

Discussion

Two main areas of interest must be examined to understand the spread of the influenza virus at Fisher River. The first pertains to the origins of the outbreak and the second is to uncover any trends or patterns of transmission among community members.

One of the most interesting questions about the deaths at Fisher River was finding an identity for the index death, Clara Clairson. Regrettably often the most interesting questions are the ones that prove to be unanswerable. Scouring every source failed to turn up any supplementary information on Clara Clairson. The only information in this case was the absence of information. Therefore, aside from her genealogical context and age, it can be ascertained that Clara was an unmarried woman who was not in the employ of any governmental agency.

The only real Clairson to turn up any leads is one John Clairson, conveniently the name of one of Clara's brothers. The reconstituted family data indicate that four John Clairsons were residing at Fisher River during the epidemic. There are two relevant entries linked to this name. The first is the listing of a John Clairson being paid as a schoolteacher for twelve months in 1909 (Indian Affairs Annual Report 1909: 46). There is no subsequent recording of a John Clairson teaching at the Fisher River Methodist School (Indian Affairs Annual Report 1910 - 1920). As well it is noteworthy that with the exception of Mr. W. Stevens and Mrs. F. G. Stevens, none of the teachers at the Fisher River Methodist School can be found within Frederick Stevens' parish records for the Methodist population. This could perhaps be indicative of the mobile nature of government employ. A search of the Indian Affairs Annual Reports for 1905 – 1920 (Indian Affairs Annual Report 1905 – 1920) shows a number of teachers re-locating to and from neighbouring areas.

The second, and perhaps more intriguing entry comes from an appointment taken up by a John Clairson in 1911 as Foreman for the Peguis reserve. The year end report reads "The department placed John Clairson in charge of the new Peguis reserve of this agency as foreman, whose duty it was to exercise supervision over the new settlers coming to this locality from the surrendered St. Peter's reserve" (Indian Affairs Annual Report 1911: 117). It is likely that the John Clairson in question was indeed the brother of Clara Clairson. Returning back to the information gathered from the reconstituted family data some tentative inferences can be made.

The first John Clairson was married to Ann (date unknown). In 1909 had a child, Margaret, at Jackhead. There is no other information on this couple and their marriage is assumed from information contained in Margaret's baptismal record. The second John Clairson was married to Nancy Kay. Their daughter, Eliza, was born in 1912 at Jackhead. Again this is not a 'true' reconstitution as there is no marriage record and like the previous John Clairson there is no indication of age or occupation. The third John Clairson was born in 1902, which would make him too young to be Clara Clairson's brother, and only 7 years old at the time of appointment.

The final possibility is of Clara Clairson's brother. He was born to Mary Budd and Elijah Clairson in 1887 at Fisher River. On September 12, 1907 he married Addie Clairson. The family seems to be quite prominent at Fisher River and although he was listed as a carpenter in 1907, at the time of his marriage, it is still a possibility that he could have taken on this appointment in 1911. The first death at Peguis is recorded on October 29, 1918 so it would appear that the two communities experienced simultaneous outbreaks, if indeed this connection was a possible point of entry.

The disease did not exhibit any clear patterning in association with its spread. There was no clear pattern of transmission within or between families, as was exhibited in the models developed by Scott and Duncan (2001). Some observations can be made, but it must be noted that they are mere speculations and should be viewed with caution.

It appears that the outbreak may have begun and ended with the deaths of individuals in their prime (between the ages of 20 - 40); after the death of Clara the next two deaths are the only 2 men over eight included in this study. Their dates of death are

November 1st and second of November 2nd, 1918. The end of the peak of the epidemic seems to be marked with the deaths of two adult women on the 16th and 19th of November. As well there appears to be a cluster of four infants deaths between November 1st and the 3rd and four children between the ages of seven and a half to nine dying between November 5th to the 10th. And finally, between November 9 through to the 13th three infants are removed from the Fisher River population. This could indicate that a school, or other gathering place for children, may have been responsible for some of the early spread among the 7 to 9 year olds, which subsequently led to the infection of their infant siblings. To tie everything back to Clara Clairson it is possible that due to her status as an unmarried woman she may have been involved with caring for infants within the community. This is especially feasible if we refer back to Figure 6.6, which denotes that one of the initial removed infants who died was one of her nieces.

Chapter 7 The Family Reconstitution Approach

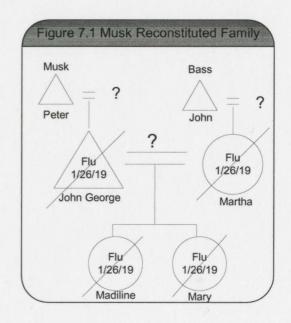
Introduction

This chapter examines the impact of the pandemic at the family level in the two communities. As has already been demonstrated in earlier sections of this thesis, influenza mortality differed at Fisher River and Norway House. This chapter is designed to take an in depth look at mortality from the point of view of reconstituted families to further explore the impact of the epidemic in these two locations.

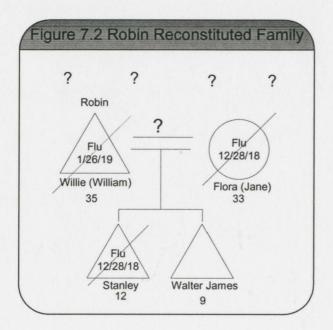
The Shift from Distress to Disorder

The aim of this section is to take an in depth look at families that experienced multiple deaths at Norway House and Fisher River in order to ascertain how these

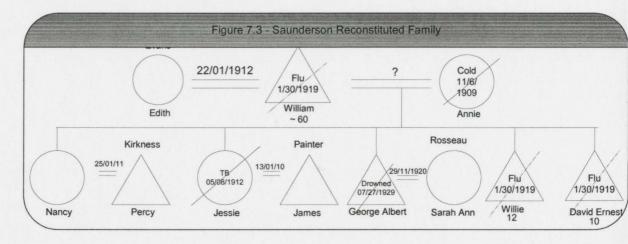
collective deaths affected individual families and the communities in which they resided. It is not the goal of this chapter to report findings of statistical significance; rather, this section aims to put a face on an at times faceless epidemic. To this end reconstituted families that lost three or more members to the virus were examined. Figure 7.1



displays all of the reconstituted family data for the Musk family. It is of note that Mary Musk's (Bass) place of birth is listed as Gods Lake and that John George hails from Oxford House. They were married at Oxford House on August 29, 1898. It is unclear where Madiline and Mary were born as there is no record for baptisms for them at Oxford House, Gods Lake or Norway House. As no birth records exist at Norway House it is probable that they were born elsewhere. There is no question about the relationships in this family as they are listed on the burial record. The children's ages are listed as fourteen and eight. John George is listed as a trapper in his burial record and as a hunter in the marriage record. It is therefore likely that this family was out on the trap line at the height of their illness with influenza. It is also of note that this family collected treaty payments at Norway House (TAPL 1918) (DIAND 1896 – 1959) . The removal of this entire family from the Treaty Annuity Pay Lists in 1919, coupled with the burial records in the Anglican registry, indicates that it was wiped out during the epidemic.



The second example explored in this section is the Robin family (Figure 7.2). There is no marriage record for this family nor are there any birth records. Their relationships are established through notes on the burial documentation as well as their Treaty Annuity Pay List payment (TAPL 1918). William was a carpenter for the Hudson's Bay Company, which would have placed him and his family at the post at the time of the pandemic. Stanley and Flora were among the first to die from the pandemic and their dates of burial most likely reflect their date of death as they both fell ill prior to Reverend Marshall's illness²⁵. The only information known about Walter James was that he was residing at Norway House at the time of his father's burial.



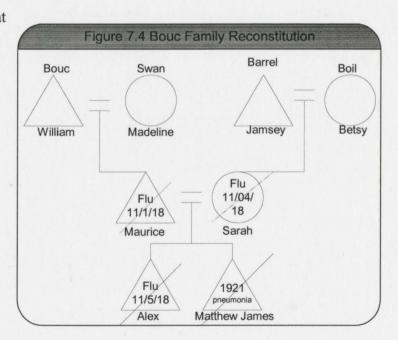
The third family to be explored in detail are the Saundersons (figure 7.3). William Saunderson married Edith Evans nearly three years after the passing of his wife, Annie. Together, he and his first wife had five children, two of whom had married prior to Mr. Saunderson's remarriage in 1912. There are two possibilities for the composition of this family at the time of the ep⁻demic. The first is that at the time of his death only three of his children were residing with him; the second is that through the practice of extended kin groupings he and his mature sons were co-operating on the trap line. His first two children are listed as being born at York Factory (1886) and Split Lake (1891),

²⁵ He is reported to have contracted the disease on January 01, 1919

and although there are no birth records that indicate that any of their children were born at Norway House there is evidence that they were at Norway House in 1909 (as indicated in Annie Saunderson's burial record) and 1912 (again indicated in the burial record for Jessie Painter). The records also indicate that the surviving family remained at Norway House until at least 1929, as indicated in the burial record of George Albert. Willie Saunderson is listed as a trapper on his burial record and it is therefore possible that he was out on the trap line with his two sons at the time of their illness and subsequent deaths.

There are two more families at Norway House that experienced more than two deaths: The Gunns and the Bass. But due to a lack of information on the earlier generations they cannot be definitively linked to reconstituted families, and as a result will not be included in this discussion.

Only two families at Fisher River experienced more than two deaths, and of these only one can be reconstituted with confidence. This is the Bouc family (Figure 7.4). It is important to note that this family is unusual at



Fisher River. In most instances families are much larger and this is the only recorded

instance in which both parents passed away during the epidemic. The virus swept through this family perhaps after it was introduced by the father, Maurice, sometime on or around the 25th of October. At the time of death it is unknown if there were any surviving grandparents to look after Matthew James until his passing in 1921.

It is also worthwhile to note that at Norway House ten partners were left widowed by the virus, as opposed to three at Fisher River. At both Fisher River and Norway House, there was one child that was orphaned as the result of the loss of both parents. Finally there was the impact of losing an entire family, which occurred at Norway House but was not experienced at Fisher River (Figure 7.1).

The aim of this discussion is to demonstrate how the family patterns of mortality played out differently in these two communities. The sheer impact of dealing with multiple families devastated by the pandemic would have greatly influenced the general morale of these communities; as well, multiple deaths within single families speak to a community's ability to deal with an epidemic.

Accounting for Age at Death

The other main determinant of how the pandemic played out in each of these communities is the age of death of the individuals. The age specific proportionate

mortality has been explored

previously in this thesis (for a detailed statistical analysis of these results please refer to pages 82 -89). Table 7.1 illustrates the age at death

 Table 7-1 - Age at Death Distribution for Norway House and

 Fisher River. Categories Created to Reflect Social Organization

 in Cree Life. Red Indicates Cells with the Highest Value.

	Norway House		Fisher River	
	N	%	N	%
< 1	1	2.63%	7	18.42%
1 - 12	11	28.95%	12	31.58%
13 - 69	21	55.26%	10	26.32%
70+	3	7.89%	1	2.63%
UNKNOWN	2	5.26%	8	21.05%
Total	38	100.00%	38	100.00%

distributions at Norway House and Fisher, according to categories developed to represent social organization in Cree life. As it is difficult to fully comprehend the complexity of Cree social organization, especially in the climate of change that was being experienced at the time of the pandemic, four broad age categories have been developed for this analysis. These are infants under 1, children 1 - 12, adults 13 - 69 and the elderly, 70 years of age and older. The highest mortality at Fisher River was experienced in children between the ages of one to twelve (31.58%). At Norway House the 13- 69 age range was hardest hit by the epidemic (55.26%).

According to Moffat (1992) the population at Fisher River experienced an excess infant mortality rate after the fifth month of age (1992: 84). She goes on to state that approximately 70% of all infant deaths at Fisher River between 1910 and 1939 were caused by infectious disease, and almost half of these deaths were caused by acute airborne respiratory infections (1992: 86). It is important to note that Moffat removed

deaths due to Spanish Flu so as to avoid skewing her results. These findings therefore are not the result of an overrepresentation of deaths during the 1918 pandemic. This is important for two reasons: first, infant mortality, although grieved, was not an atypical occurrence at Fisher River and therefore would not likely have had a significant impact on the Fisher River community. Second, the fragility of infant life at Fisher River may help to explain why mortality from influenza at Fisher River did not display the common characteristic of the 1918 strain of targeting individuals in their prime.

The intention of this section is to demonstrate the impact of *who* dies rather than concentrating on the number of victims. As the stages in one's life change, so too does the commitment one has to one's community. An oversimplified way of looking at this idea is to break up the life stages into three general categories: past, present and future. In this framework, the past would consist of the elderly within the community, for the purposes of this analysis; this would consist of individuals 70 years of age and over. These people may still contribute to the day-to-day functioning of the community but their main contribution occurs at a household level. It is important to note that they were not perceived by the community as weak but rather as strong, tough and healthy (Beardy and Coutts 1997: 90). They were important keepers of wisdom and provided their community with a wealth of information based upon life experience. Joseph Saunderson, a resident of York Factory states the elders "stayed home. They often looked after the children while the women were outside working or out hunting" (Beardy and Coutts 1997: 90). So although they are often still active, their immediate physical impact is usually restricted to the family level.

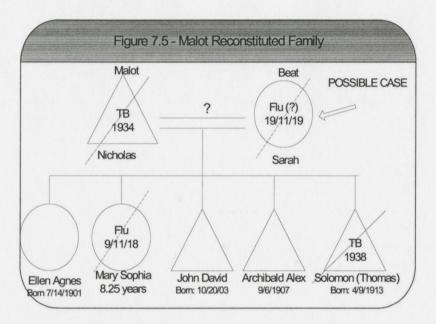
The future category in this framework references children and teenagers within a given community; for the purposes of this study, this includes the infants under 1 and children 2 - 12. They comprise what a community will be, or rather, its future. This group consists of individuals who are not yet an autonomous part of the functioning of the community; again their importance at this juncture is limited to the family and not the general populace. Puberty often marked the shift from student to hunter in Cree society for although a child may make his first kill prior to this rite of passage they were usually still in the process of acquiring proficient skills (Higgins 1982: 83). Younger children would spend their day playing while older children might help with household chores suck as cutting firewood (Beardy and Coutts 1997: 44). The removal of individuals from these age categories has an impact on a community but it lacks the immediacy and severity of what is felt when individuals in their prime are removed.

This brings us to the third group in this framework: those who were the backbone of the community and responsible for daily functioning. This is the age group upon which a community relies; for the purposes of this study, this group is comprised of 13 -69 year-olds. When individuals are removed from this category the impact is immediately felt especially in a smaller community where few individuals are trained in certain areas. Certain professions only require a few practitioners under normal circumstances yet the job that they serve at the time of an outbreak may be essential for the daily functioning of a community. Examples of these types of professions are clergymen, doctors, gravediggers, nurses, store clerks and so on. Individuals in this age range also are more likely to have dependents, whether they take the form of children or

the elderly. These dependents are reliant on these individuals as primary caregivers and when after they are removed from a community those who depended on them become more vulnerable to death and starvation.

To demonstrate how mortality was shaped within each of these communities two deaths are explored more fully within the context of family reconstitution. In order to eliminate sampling bias, individuals were chosen using the random number generator available at www.random.org. A random sequence was chosen (to eliminate the possibilities of duplicate numbers), the smallest value was set at one and the largest value was set at thirty-eight. The first two numbers that were generated were 17 and 3. For Fisher River this corresponds with the death of Mary Sophia Malot and Kenneth McLure Mase. For Norway House the corresponding individuals are Madeline Musk and Flora Jane Robin, both of whom have already been reconstituted in the previous part of this chapter. The next two numbers generated turned up an individual from the Saunderson family and an unknown individual. The next two numbers, 13 and 21, correspond to Moses Bass and Edward Falcon. This sampling approach was adopted for two main reasons. First, ordering individuals alphabetically lent itself to a bias toward families with multiple deaths. Second, the chronological sequence of deaths within each community also presented an imbedded bias.

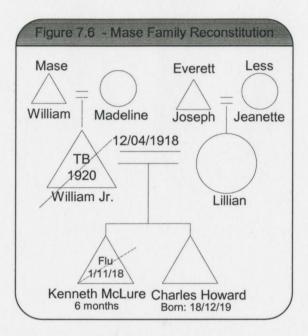
 $; ^{+}$



Sample Mortality at Fisher River

The Malot family, depicted in Figure 7.5, consisted of two parents and five children. At the time of the outbreak the children's ages ranged from 5 to 17 years of age. Mary Sophia was the second youngest in her family. It was not uncommon at Fisher River for the youngest or close to youngest child to perish during the epidemic. Of the 19 deaths of children under 13 at Fisher River 12 were the youngest and 4 were the second youngest. It is possible that Sarah, the postulated mother of the Malot family,

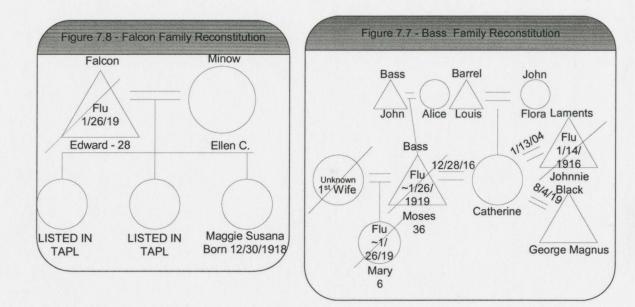
also perished during the flu pandemic but due to the manner in which Rev. Stevens kept his burial registry it cannot be determined definitively²⁶.



The Mase family, as represented in Figure 7.6, was headed by a couple that had been married for seven months at the time of their child's death (Kenneth). Lillian was 22 and William was 26 years old when they were wed. Charles Howard was born just over a year after the death of his brother. It is also important to note that this reconstitution only depicts William's nuclear family; a full reconstitution is shown in Appendix IV. William had a large extended family consisting of a brother and sister, as well as many individuals related through the marriages of his siblings. His brother, John, also lost a child (Edgerton Ryerson 0.5 years old) during the pandemic.

²⁶ Sarah Beat does not have a recorded age at death or birth date, as well no relationships are listed, therefore definitively linking her to this family is very problematic.

In both of these families children have died during the influenza outbreak. There also appears to have been some degree of family support available to individuals who lost a loved one, exemplified by the Mase's large extended family.



Sample Mortality at Norway House

The two families randomly chosen to represent mortality at Norway House are depicted in Figure 7.7 and Figure 7.8. Figure 7.7 tells a very interesting tale about life at Norway House. Catherine Laments (Barrel) was married three times in the span of fifteen years. Her first husband passed away of influenza ten years after they were married; within eleven months of his passing she was wed to her second husband Moses Bass, who too died from influenza along with her stepdaughter. Catherine married her third husband in just under seven months after the death of her second. It is also of interest to note that George Magnus, Catherine's third husband, had also been touched by the influenza pandemic. As it swept through Norway House it carried away his father and brother-in-law. The treaty payment for 1919 affirms the deaths of both Moses and his daughter, Mary during the previous year.

There is very little information available for the Falcon family depicted in Figure 7.8. The father, Edward, died from influenza during the outbreak, and there is also a birth record for their third (or possibly only) child available within the Anglican registry for Norway House. The TAPL for 1919 indicates the family had three children (there are only two listed for 1918). Although there is some question regarding how many children Ellen Tadpole and Edward Falcon had together, the Anglican clergyman's records do provide some indication of how this family may have been affected by the virus. According to the burial record, Edward Falcon was listed as a trapper in 1918. His daughter's birth date is listed as December 21, 1918, which is 36 days after the death of her father.

In both of these reconstitutions at Norway House parents were removed from the family unit during the pandemic. The records were scoured in order to locate a remarriage record for Ellen Falcon (Tadpole) and nothing was turned up. Whether this is the result of her removal from the immediate community or her inability or lack of desire to remarry is unknown. However, we do know from aggregate analysis carried out by Herring (1994) that there was an increase in the number of re-marriages after the pandemic. Both of these families would have struggled to regain a semblance of normalcy in its aftermath. Due to the subsistence strategy employed at Norway House,

multiple adults were likely required to support a family, and the loss of a partner would have had detrimental effects on the development and survival of young children.

Chapter 8 Discussion and Conclusion

Introduction

When I began working on this thesis I wanted to explore the possible role of the Hudson's Bay Company in the dissemination of the 1918 influenza pandemic. In order to accomplish this it was necessary to understand how Norway House and Fisher River grappled with the presence of the virus. To this end I conducted a three-stage analysis. The first stage involved a view of mortality from the aggregate perspective, the second adapted techniques outlined by Scott and Duncan (2001), and the third employed the qualitative approach of family reconstitution. The purpose of this chapter is to summarize how each of these approaches contributed to unravelling the story of the dissemination of influenza within the communities of Fisher River and Norway House. It is important to state that none of the approaches can actually stand-alone; each was analysed with the aid of the other approaches. The results come from the synergy between multiple methodologies. That said, these approaches did uncover one definitive finding: the two communities experienced the pandemic differently. There is the strong impression that at Norway House normal daily functioning ceased. Whether this was the result of a few 'key' individuals falling sick (i.e. Rev. Marshall), or an entire community sick at the same time is hard to determine. At the same time although there was a fair amount of disruption at Fisher River, there is very little evidence of a community in turmoil.

Embedded in the composition of each of these communities is a hierarchal political and economic structure that constructs and reshapes the local inhabitants' daily lives. This has been demonstrated through an in depth look at the conditions that were created when Europeans first developed relationships with local Indigenous populations. Although the degree to which this relationship influenced the spread and overall mortality in each community cannot be assessed with absolute certainty, it can be stated that colonial institutions did influence the spread of the epidemic. That influence ranged from the creation of the conditions of war that were necessary for a virus of this magnitude to proliferate throughout the world to the creation of "company welfarism" amongst fur trade families in the Central Subartic.

Aggregation Revisited

There were a number of interesting trends that came to light when the material was examined using an aggregate approach. The first was the question of how the influenza virus entered the community. At Norway House the portal of entry for the microorganism appears to have been the Hudson's Bay Company (HBC). This is supported by both the first death as well as the first individual suspected of carrying the influenza virus (with symptoms). At Fisher River the local clergyman seems to be the first individual to have fallen ill with the disease. The loci of infection for both of these communities therefore, were institutions that helped to structure social networks and subsistence strategies: The HBC (Norway House) and the church (Fisher River). This

contributors that helped to shape the disease experience within each community. It can be argued further that the conditions created by each of these institutions may provide clues as to why the impact of the epidemic seemed more severe within one community (Norway House) and somewhat more protracted and yet less tumultuous at another (Fisher River). The HBC was an establishment that encouraged competition whereas the mission fostered a community atmosphere. If we are to conclude that these places were the mainstays of social organization, we can gain a better understanding of how they helped to shape community interaction and therefore the influenza experience.

The aggregate approach also helped to demonstrate general patterns about the dissemination of the epidemic. First, it appears that there is very little difference in the number of individuals that died in each community. Secondly, from this level of analysis, the difference in the distribution of deaths by family is insignificant (p = .3282). Thirdly, there is no difference in the sex of the deceased individuals within and between the communities examined. The population is too small to get an accurate idea of age-specific mortality at a precise level but when very broad categories are developed (0 - 20, 21 - 65+) there appears to be a statistically significant difference of the age at death of individuals between the two communities (x2 = 12.6, df = 1, p < 0.0001). At Norway House 61% of individuals who died during the epidemic came from the 21 -65+ age category, where as mortality at Fisher River disproportionately resided within individuals between 0 - 20 (83%).

Finally an interesting pattern develops when the spread of the epidemic is examined. It appears that one community experienced a fall epidemic (Fisher River)

while the other endured a winter epidemic (Norway House). This raises the following question: was the impact of mortality greater at Norway House because of the timing of the epidemic or rather the conditions under which the virus existed? Herring and Sattenspiel (2003: 163) state that "the impact of Spanish flu could have been shaped by the fact that it struck the central subartic in the winter, when social contact occurred primarily *within* families, rather than in summer, when social contact was extensive *between* families". If this thesis could address morbidity I think that there would be a clear patterning at Norway House of high mortality and somewhat more limited morbidity, which would be contrasted by Fisher River's lower mortality rates yet higher morbidity rates. This hypothesis would be consistent with the modeling carried out by Herring and Sattenspiel (2003).

Scott and Duncan (2001) Reviewed

What the Scott and Duncan (2001) approach lacks in applicability it more than makes up for in creativity as it provides a new and interesting way to analyze data. One of the key features that arose out of the Scott and Duncan chapter of this thesis is that their technique is hard to replicate. To begin with, this approach could only be applied to the data for Fisher River because the Norway House burial records lack dates of death. As well, difficulty arose in grappling with all of the assumptions that must be accepted in order to assess the findings generated by this technique. The most significant of these are the assumption that morbidity mimics mortality and that the first individual to perish

from the virus is also the one who introduced it into the community. This was clearly shown not to be the case either at Norway House or Fisher River.

That said, however, this technique permitted an in depth look at the micro processes in play at Fisher River by providing a new way to home in on the family context of mortality.

The results of this analysis helped to suggest some of the possible ways that the virus may have spread among Fisher River families. For example its introduction into the Methodist community by a young adult and the subsequent infection of young children between the age of 7-9 was followed by the death of a number of infants. Other possible spread patterns point to children as introducing the virus into the nuclear family and adults introducing the pathogen into the community. This would be consistent with Beckett's 1998 findings that schools (and children) may have played a key role in the dissemination of the 1918 flu pandemic at Berens River. However, the main contribution of this approach is to flesh out the family context of mortality and dissemination of the virus thus demonstrating again that social organization played a key role in the epidemic's progress. This technique also prompted new kinds of questions, such as under what conditions is mortality structured? How do family and community interactions influence the life course of a pathogen? So although Clara Clairson's role in the dissemination of the influenza virus at Fisher River remains speculative, the issue of her significance within the context of her family and community was not a trivial one. The Scott and Duncan approach, although flawed, contributed more ideas for future research, perhaps more so than the other two approaches combined, for it highlighted not

only the constraints of research based on limited archival sources but it also helped demonstrate what must be understood in order to model epidemics within an Aboriginal context.

Family Reconstitution Methodology Readdressed

Family reconstitution is the qualitative companion approach to aggregate quantification. What it lacks in statistical robustness it more than compensates for in depth. This methodology added insight into the Scott and Duncan approach by providing a family context, and the role within the family of individuals who died. Through this analysis, birth order, subsistence strategies, social organization and age at death were explored. The results are subject to interpretation but many of the families can now, by virtue of this methodology, be situated within a broader political and economic context. It demonstrated yet again how mortality played out differently within these two communities and helped to synthesize multiple lines of evidence in order to create a detailed tale of the pandemic as opposed to an aggregate snapshot. Family reconstitution methodology proved to be the backbone of this thesis.

This analysis has shown that the important question that needs to be addressed within these two communities is not *how many* individuals per family died but rather *which* individuals in each family died. At Norway House many individuals in their prime were removed from the community whereas at Fisher River there seemed to be an abundance of children and infants who passed away during the epidemic. This must have affected how the pandemic was experienced and might help explain how one community

appeared to shut down (Norway House) while the other, though jarred by the experience, was still able to maintain day-to-day functioning (Fisher River).

Although this thesis can stand as an autonomous piece of research, it is not meant to; rather, it serves as extension of much of the work that has already been conducted for the region (again see Beckett 1998, Herring 1994, Herring 1994b, Moffat 1992, Moffat and Herring 1999, Sattenspiel and Herring 1998, Sattenspiel and Herring 2003, Sattenspiel et al. 2000). This work has continued where Herring and Sattenspiel (2003) began using aggregate techniques (via Treaty Annuity Pay Lists) to calculate how influenza was distributed at the family level. It also uses their idea that "the key to understanding the effects of the Spanish flu ... lies within the domain of social structure and community organization" (Sattenspiel and Herring1998: 112).

Conclusions

Within the context of a historical understanding of Aboriginal health "[m]uch has been written about the effects of certain European diseases, most notably smallpox, on Indian populations; yet few scholars have attempted to assess the impact that a series of epidemics had on Indian life in various areas of North America" (Ray 1976: 139). With specific reference to the 1918 – 1919 influenza pandemic "...little is known about the epidemic in Canada, let alone how it affected aboriginal communities in remote northern regions" (Sattenspiel and Herring: 1998: 96). In Northern Manitoba, which is the focus of this thesis, comprehensive work has been conducted by Dr. Ann Herring and colleagues predominantly at an aggregate level. At the local level partial, family-based

reconstitution has been carried out for Norway House by Herring and Sattenspiel (2003) using Treaty Annuity Pay Lists as a gauge of mortality. This research has added to what is known about the 1918 – 1919 influenza pandemic within the Fisher River and Norway House communities at both a local and family level.

The most significant finding of this thesis echoes that of research already conducted for the region by Herring (1994b) and Beckett (1998). This finding is that Norway House and Fisher River had a markedly different experience with the 1918 strain of the influenza virus. I argue that these differences derived from the contrasting subsistence strategies employed within each. The differing political, economic and social conditions formed by the differing subsistence strategies employed within each community restructured human interaction within each neighbourhood, influencing not only informal support networks but individuals' susceptibility to the influenza virus and access to subsequent care. An example of this would be individuals at Norway House isolated on trap lines in contrast to people embedded within communities at Fisher River at the time of the epidemic. The conclusion of this work then is that mortality was not simply influenced by the virulence of the pathogen but the community conditions in which the pathogen resided. These factors, combined with the timing of the epidemic at each community and environmental conditions, structured the shape of the influenza experience.

I propose that the following research must be carried out: 1) More communities within the region must be studied in order to ascertain if there was a noticeable pattern in the structure of mortality within HBC and Non-HBC communities. 2) Modeling

techniques should be employed on the data already collected in order to establish some information on the morbidity associated with the 1918 – 1919 strain. 3) More work needs to be done to fully comprehend the factors that determined health or rather, ill health, within these communities with a strong focus on the role that informal networks of care played on the effects of epidemics.

In conclusion it is my belief that this thesis adds to the growing literature that views interactions with infectious disease as a multi layered process that encompasses not only the role of the microorganism but the conditions present within the communities they afflict as well. After all, the key to understanding infectious disease experiences is in decoding how cultural contingencies transform physical realities and thus shape the disease experience.

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(1888 – 1903) 78 – 27 Fisher River Marriage Register.

(1911 – 1920) 78 – 27 Fisher River Marriage Register.

(1908 - 1929) 78 – 27 Fisher River Baptism Register.

(1918 – 1951) 78 – 27 Oxford House Burial Register.

(1894 – 1917) 78 – 27 Oxford House Baptism Register.

(1919 – 1951) 78 – 27 Oxford House Baptism Register.

(1894 – 1917) 78 – 27 Oxford House Marriage Register.

(1919 – 1951) 78 – 27 Oxford House Marriage Register.

(1885 – 1889) 87 – 4 Berens River Baptism Register.

(1909 – 1952) 87 – 4 Berens River Baptism Register.

(1884 – 1909) 87 – 4 Berens River Marriage Register.

(1909 - 1952) 87 - 4 Berens River Marriage Register

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Appendix I - Evaluation of Norway House Anglican Church Parish Record Data

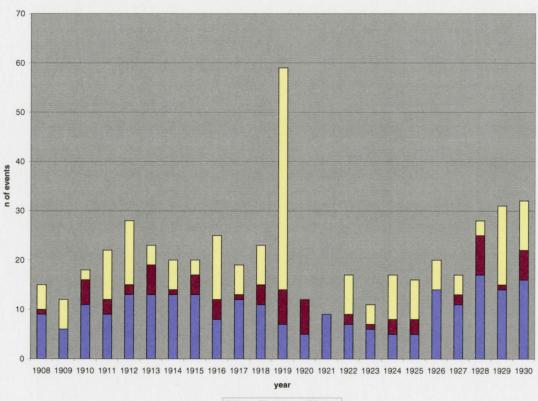
This test covers the years of 1908 - 1930 because this was the only time period where all three records of vital events were recorded for the Jack River Mission (ACCA 1902 - 37). This time frame is also appropriate because it sandwiches the time period of the epidemic explored within this thesis. For a detailed discussion of the information available within each of the parish registries please refer to the Materials and Methods chapters of this thesis (pages 41 - 69). This analysis uses Drakes' (1974) analysis and adopts modifications implemented by Moffat (1992) and Beckett (1998), who used this procedure to test the validity of records created for other Indigenous communities in Manitoba, Canada.

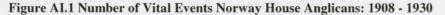
Establish the Mean Number of Entries Per Year

According to Drake's guidelines, registries must have at least 100 entries of vital events per year. These parameters were established to examine populations with much higher densities, specifically English parishes. Subsistence strategies of Indigenous populations, environmental conditions of the Canadian Central Subartic as well as the presence of many competing parishes during the period encompassed by this study would likely not have supported the large population that would have been necessary to generate this number of vital events. Moffat (1992) recorded a mean of 47.7 vital events for her study of Fisher River for the years 1908 - 1939 (1992: 45). Beckett (1998) records a

148

mean of 24 vital events annually at Berens River for the years 1909 – 1952 (1998: 84) and 11.9 entries annually for Poplar River between the years 1905 – 1946 (1998: 63 – 64). The mean number of vital events at Norway House for 1908 – 1930 was 21.5 vital events with a range of 9 in 1921 to 59 in 1919 (caused predominantly by influenza deaths). Figure AI.1 shows the number of events annually at Norway House, with blue representing baptisms, red marriages and white burials.





Births Marriages Burials

To explain the difference in the number of vital events sessional papers were collected to compare population sizes at Fisher River and Norway House (Table AI.1) for the years $1908 - 1930^{27}$.

Year	Total Population Norway House	Anglican	Total Population Fisher River	Methodist
1908	520		411	411
1909	563		420	420
1910	738			
1911	735		444	444
1912	765		455	455
1913	738	214	472	472
1914	746	216	472	472
1915	743	213	478	280
1916	734	208	510	290
1917	734	208	493	284
1929	732	(united) 417	529	290

Table AI.1 - Norway House and Fisher River Populations Collected from Sessional Papers: 1908 -1930

Although the overall population at Norway House appears to be larger than Fisher River if we compare the number of Anglicans at Norway House to Methodists at Fisher River, clear differences in population size come to light which accounts for the smaller number of recorded vital events at Norway House.

Determine Whether the Whole Community's Vital Events Are Being Registered

There were three competing religious groups at Norway House according to the 1917 census (Department of Indian Affairs. Sessional Papers No. 27 George V, A.1917). These were Anglicans, Methodists, and Roman Catholics. In 1917, the total population

²⁷ Only years where population sizes were available were listed

at Norway House was 734, of this 208 of which were Anglican (28%), 494 Methodist (67%) and 32 were Roman Catholic (4%), thus making the Methodists the predominant group at Norway House. Sadly these records can not be accessed because they were lost in a fire.

Scrutinize the Entries for Suspiciously Long Gaps

According to Drake (1974), any gaps in registration that exceed two years would bring into question the quality of the registry. At Norway House the only two-year break that exists in deaths occurs between the years 1920 – 1922. This could possibly be explained by the early or premature removal of susceptibles during the 1918 – 1919 influenza epidemic. It is of interest that there were eight deaths due to consumption following the cessation of the flu in 1919. As well, there are three years in which no marriages were recorded (1909, 1921 and 1926), which could have been the product of competing parishes, which contributed to the small Anglican population. Tables AI.2 through AI.4 provide a breakdown of vital events by month and year. The vital events, while low in number, appear to be fairly evenly distributed.

	Jan	Feb	March	April	May	June	July	Aug	Sept	Oct	Nov	Dec	Totals
1908	1		4			3						1	9
1909		1		1		3		1					6
1910			1			3	3		2	1		1	11
1911		2						3		2			9
1912		1	1	3	1	1	1	3			2		13
1913			3	1	1	1	3	2	2				13
1914		2				1	1	1	1	3		1	13
1915		2	1	1			1		1	3	2		12
1916			1			3			1	1		2	8
1917		1		3		1	3		2	1		1	12
1918		1	2		3	2		1				2	11
1919			1		1			5					7
1920	1	2	1						1				5
1921			2	1		1		2	2			1	9
1922			2	1			1	1		2			7
1923	1			1		3		1					6
1924			1	1	1			1	. 1				5
1925						1			3		1		5
1926								3	5	4		1	13
1927	1		1	2	1	1	1		2	2			11
1928	2	2		1	2			4	<u> </u>	2	1		15
1929	1	2	1	1			3	2	2	2			14
1930		4		3	3	2	1			1	1	1	16

Table AI.2 - Number of Baptisms Recorded For the Norway House Anglican Population 1908 - 1930

Table AI.3 - Number of Burials Recorded for the Anglican Population at Norway House: 1908 - 1930

							<u> </u>				-		
	Jan	Feb	March	April	May	June	July	Aug	Sept	Oct	Nov	Dec	Totals
1908			2	1			1			1			5
1909	1	1		1	1	1	1						6
1910			1							1			2
1911					1		1	1	1	3	2	1	10
1912			2	2	1	3		1		2	1	1	13
1913				3					2				5
1914					1		1		1			2	5
1915	1	1										1	3
1916	2		3	2	4		2						13
1917				1	1			2	1			1.	6
1918							1	2				5	8
1919	34	1		2	2		3		1	1		1	45
1920													0
1921													0
1922			1		2		5						8
1923			1					1			1	1	4
1924			1			1				4	2	1	9
1925	1		1	2							2	2	8
1926			2		1					3			6
1927							1		1	1		1	4
1928			1					1				1	3
1929	4	1	2			1		2	2	2		1	15
1930	1	1			2	3					2		9

	Jan	Feb	March	April	May	June	July	Aug	Sept	Oct	Nov	Dec	Totals
1908						1							1
1909					1	1							0
1910		1							1	1	1	1	5
1911	1							1			1		3
1912													2
1913	3						1	1			2		7
1914									1				1
1915		1	1					1	1				4
1916					1		1		1			1	4
1917							1						1
1918						2			2				4
1919			1	1				1	2	1		1	7
1920					1		1	2			1	1	6
1921													0
1922					<u> </u>								2
1923								1					1
1924						1							2
1925					1				2				3
1926									L				0
1927					1		1						2
1928			ļ		1	2			2	1		2	
1929			ļ			1							1
1930			1		l				2	1	1		4

Table AI.4 Number of Marriages Recorded for the Anglican Population at Norway House: 1908 -1930

Assess the Reliability of the Clergyman Recording Events

Reverend Marshall entered the Norway House community in 1910 after being ordained at Wycliffe in June. His appointment ended the preceding nine years of fluctuation in clergy²⁸. On August 11, 1911 he was ordained to the priesthood and formally appointed to the Jack River Mission. During his first few years at the Mission he was commended for his diligent work by the Diocese of the Keewatin District helped to erect a new mission house and his own lodgings (ACCA 77 – 75 #1). Marshall left Jack River in June of 1926 to take over the position of principal at the Indian Residential School near Sioux Lookout and was succeeded by Rev. W.H.J. Walter. There is no

 $^{^{28}}$ From 1901 – 1909 five different clergy presided over the mission, including Mr. L. Hart, Mr.C. Wilkins. Rev. A.A. Adams, Mr. W. Tomalin and Mr. J.H. Petter (ACCA 77 – 75 #1).

mention made of Rev. Walter's departure from the mission, but in 1933 there appears to be a new clergyman presiding over Jack River (ACCA 77 - 75 # 1).

The Sessional Papers indicate that Reverend Marshall and his wife, Mabel, were active participants within the Norway House community (Department of Indian Affairs. Sessional Papers No. 27 George V, A. 1911, 1912). In 1911 and 1912, Rev. Marshall is listed as the teacher at Jack River School, and Mabel Marshall is listed as a school teacher from 1913 - 1926²⁹. Rev. Marshall's eight years of experience at Norway House at the time of the influenza epidemic, combined with his wife's involvement in the community, suggests that he was familiar with his parishioners, which, in turn, lends credibility to the accuracy of his records especially in regard to proper identification of individuals. This assumption is supported by Rev. Marshall's maintenance of the Norway House burial records, in which he often listed the relationships or occupations of deceased individuals to members of the community (e.g. "son of...." or "wife of").

Assess the Quality of the Information on Individual Records

The type of information contained within each vital event record for Norway House can be found in the Materials section of this thesis. In her 1992 MA thesis, Moffat checked the following ancillary information to assess the quality of the individual records: birth date for baptisms, bride's age for marriages and age at death/date of birth for burial records. The same criteria were used for this study and the results are displayed in Table AI.5.

²⁹ There are no records available for 1923 and 1924.

Anaillan Information	n of records with	total n of	per cent of
Ancillary monthation	data	records	total
birth date	229	234	97.86
bride's age	68	71	95.77
age/birth date or both	173	188	92.02
	bride's age	Ancillary Information birth datedatabirth date229bride's age68	birth date229234bride's age6871

Table AI-5 - Quality of Ancillary Inflormaion for Parish Record Data. Norway House: 1908 - 1930

The quality of the records kept for the Norway House Anglican population between the years 1908 – 1930 is excellent. There appears to be a slight problem with age at death, which was the result of missing data from the some of 1919 influenza deaths. This likely occurred due to the backlog of deaths caused by Reverend Marshall's illness. This omission in the records did affect this study since it is predominantly based on the 1918 – 1919 time period.

The records that are available for the Norway House Anglican population for the years 1908 – 1930 are very well maintained. Reverend Marshall seemed to be a very conscientious clergyman devoted to precise record keeping; problems arise however in the disruption that preceded and succeeded his tenure. As a result, it is difficult to develop full family reconstitutions because older generations are harder to link to wider kin networks. However, the probability of accurate reporting of the number of deaths for the Anglican population during the 1918 epidemic derived is quite high as Rev. Marshall's records appear to adhere to the modified adaptation of a Drakes analysis.

Appendix II – Data Collection Forms

NORWAY HOUSE	
BURIAL RECORD	
Surname(1):	
Given Name(1):	-
Age (1):	
Quality/Trade/etc.(1):	
Date of Death:	
Burial Date:	
TAPL	
TAPL # :	
Surname, Given Name TAPL :	
TAPL 1918: M W B G	
Dead: $M W B_G$	
TAPL 1919: M W B G	
Dead: $M W B_G$	
MARRIAGE RECORD	
Surname, Given Name H or F ():	
Surname, Given Name W or M ():	
Age:	
Residence When Married:	
Place of Birth: Male: Female:	
Bachelor/Widow: Male: W B Female: W B	
Occupation:	
Parents Names (Paternal):Father: Mother:	
Parents Names (Maternal):Father: Mother:	
Date:	
BAPTISMAL RECORDS	
Name: M F	
Parents Names:	
Occupation:	
Residence:	
Date of Birth: Date of Baptism:	
Children:	
1. 5.	
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3. 7.	
4. 8.	
RECONSTITUTED FAMILY DATA Husband: Occ Born:	

Married:	Died:			Cause:			
Husband's F	ather:		Mot	her:			
Other Wives:							-
Wife:	Occ	•		Born:			
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Other Husba							
Children							
Given Name	, Surname.	Born.	Place.	Baptized.	Died.	Where	Died.
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FISHER RIVER

BURIAL RECORD								
Surname () :								
Given Name():								
Age (): Date of Birth:								
Birthplace:								
Date of Death: Date of Burial:								
Place of Burial:								
MARRIAGE RECORD								
Surname, Given Name H or F ():								
Surname, Given Name W or M ():								
Residence When Married:								
Residence When Married: Place of Birth: Male: Female:								
Bachelor/Widow: Male: W B Female: W B								
Religion: Male: Female:								
Occupation or Calling: Male: Female:								
Parents Names (Paternal):Father: Mother:								
Parents Names (Maternal):Father: Mother:								
Date:								
BAPTISMAL RECORDS								
	_ M F							
Name: Residence:	_ M F							
Name: Residence: Parents Names:								
Name:Residence:Parents Names: Date of Birth:Place of Birth:								
Name: Residence: Parents Names:								
Name:Residence:Parents Names: Date of Birth:Place of Birth:								
Name:								
Name:Residence: Parents Names: Date of Birth: Place of Birth: Date of Baptism: Place of Baptism: Children:								
Name: Residence: Parents Names: Date of Birth: Date of Baptism: Place of Baptism: Children: 1.								
Name:Residence:Parents Names:Date of Birth:Place of Birth:Date of Baptism:Place of Baptism:Children:1.6.2.7.3.4.								
Name:								
Name:Residence:Parents Names:Date of Birth:Place of Birth:Date of Baptism:Place of Baptism:Children:1.6.2.7.3.4.								
Name:Residence:Parents Names:Date of Birth:Place of Birth:Date of Baptism:Place of Baptism:Children:1.6.2.7.3.4.								
Name:								
Name:								

Married: Died: Cause: Husband's Father: Mother: Other Wives: Mother:

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Wife:	Occ	
Born:		
Married: Died:	Cause:	
Wife's Father:	Mother:	
Other Husbands:		
Children:		
Given Name, Surname.	Born. Place. Baptized. Died. Where	e Died.
Cause.		
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Marriage Information_		
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OTHER INFORMATION		
School Records:		_
Other:		-
Other:		

Appendix III – Supplemental Scott and Duncan (2001) Diagrams

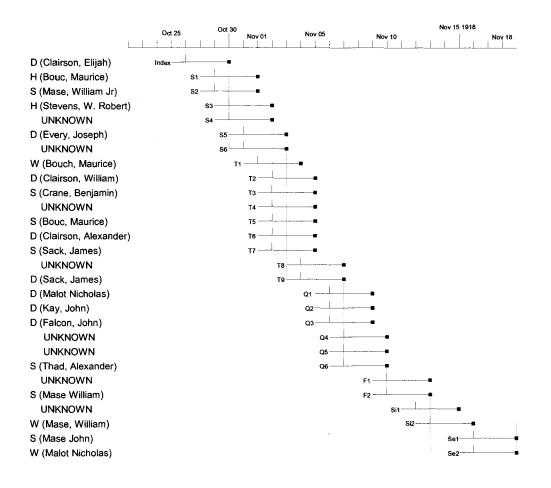


Figure 0.1 – Spread of influenza within and between families at Fisher River as represented by a one-day latent period and three-day infectious period. A standardized line that is divided into a latent and infectious period represents each individual. The first section of the line is representative of the latent period and the remainder of the line following the solid vertical line is infectious period. The square at the end of the line indicates the day the individual died. Dashed vertical lines indicate the period in which the primary case is infectious (from the beginning of the infectious period to death). Once the primary case has been removed a new dashed vertical line is established to represent tertiary cases and so on. Removed individuals are listed on the left hand side of the figure. They are represented by the name of the male head of household, which is preceded by their relationship to this individual. H represents head/husband, W, wife; S, son and D, daughter. The letter and number preceding the solid horizontal line that indicates the period of illness for each individual represent their order infection by group (S, secondary; T, tertiary; Q; quaternary; and F, fifth generation) and with in groupings (1 – 13). Age is indicated in red where known and the scale is in days beginning on October 23rd 1918.

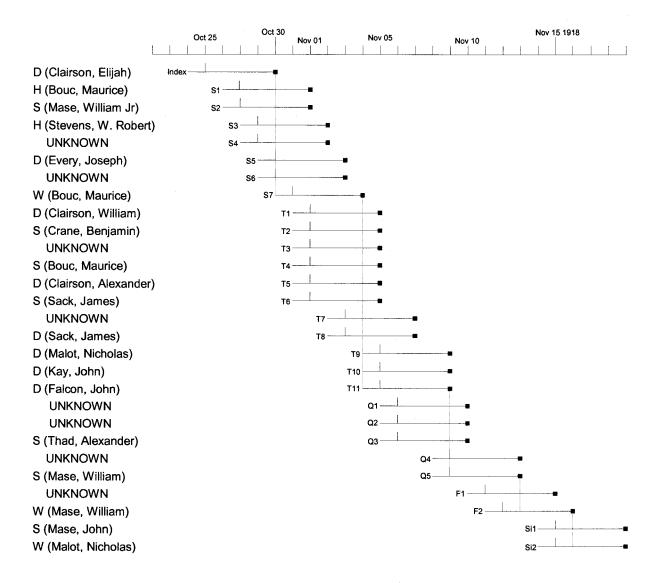


Figure 0.2 – Spread of influenza within and between families at Fisher River as represented by a one-day latent period and four-day infectious period. A standardized line that is divided into a latent and infectious period represents each individual. The first section of the line is representative of the latent period and the remainder of the line following the solid vertical line is infectious period. The square at the end of the line indicates the day the individual died. Dashed vertical lines indicate the period in which the primary case is infectious (from the beginning of the infectious period to death). Once the primary case has been removed a new dashed vertical line is established to represent tertiary cases and so on. Removed individuals are listed on the left hand side of the figure. They are represented by the name of the male head of household, which is preceded by their relationship to this individual. H represents head/husband, W, wife; S, son and D, daughter. The letter and number preceding the solid horizontal line that indicates the period of illness for each individual represent their order infection by group (S, secondary; T, tertiary; Q; quaternary; and F, fifth generation) and with in groupings (1 – 13). Age is indicated in red where known and the scale is in days heading in the scale is in days

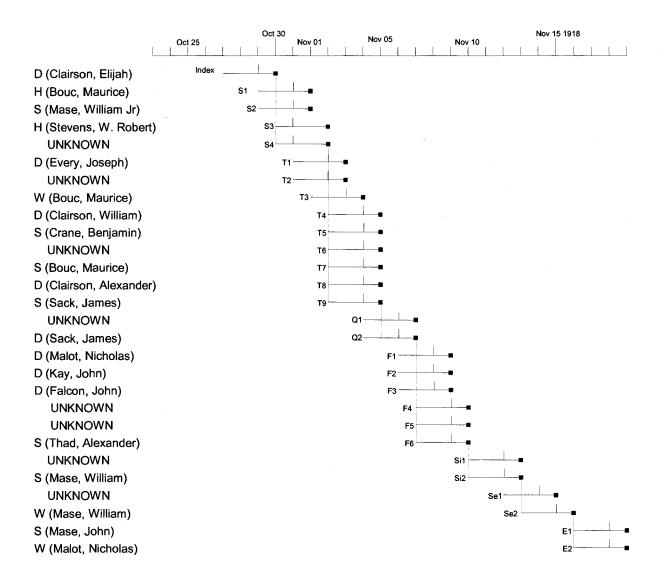


Figure 0.3 – Spread of influenza within and between families at Fisher River as represented by a two-day latent period and one-day infectious period. A standardized line that is divided into a latent and infectious period represents each individual. The first section of the line is representative of the latent period and the remainder of the line following the solid vertical line is infectious period. The square at the end of the line indicates the day the individual died. Dashed vertical lines indicate the period in which the primary case is infectious (from the beginning of the infectious period to death). Once the primary case has been removed a new dashed vertical line is established to represent tertiary cases and so on. Removed individuals are listed on the left hand side of the figure. They are represented by the name of the male head of household, which is preceded by their relationship to this individual. H represents head/husband, W, wife; S, son and D, daughter. The letter and number preceding the solid horizontal line that indicates the period of illness for each individual represent their order infection by group (S, secondary; T, tertiary; Q; quaternary; and F, fifth generation) and with in groupings (1 – 13). Age is indicated in red where known and the scale is in days beginning on October 23rd 1918.

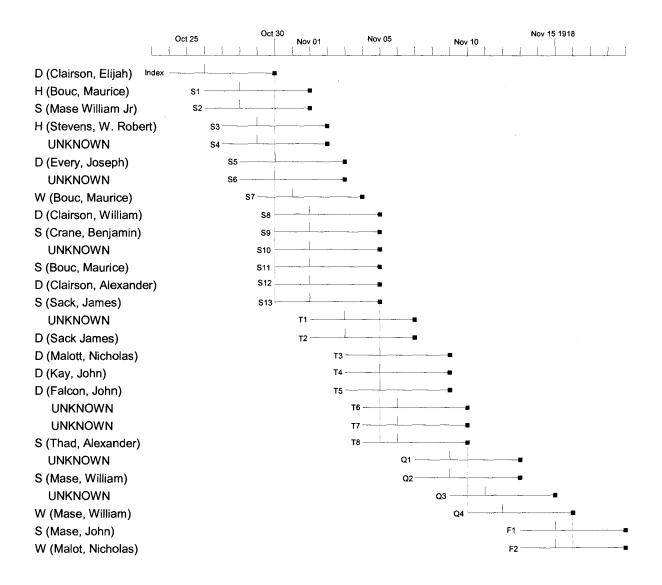


Figure 0.4 – Spread of influenza within and between families at Fisher River as represented by a two-day latent period and four-day infectious period. A standardized line that is divided into a latent and infectious period represents each individual. The first section of the line is representative of the latent period and the remainder of the line following the solid vertical line is infectious period. The square at the end of the line indicates the day the individual died. Dashed vertical lines indicate the period in which the primary case is infectious (from the beginning of the infectious period to death). Once the primary case has been removed a new dashed vertical line is established to represent tertiary cases and so on. Removed individuals are listed on the left hand side of the figure. They are represented by the name of the male head of household, which is preceded by their relationship to this individual. H represents head/husband, W, wife; S, son and D, daughter. The letter and number preceding the solid horizontal line that indicates the period of illness for each individual represent their order infection by group (S, secondary; T, tertiary; Q; quaternary; and F, fifth generation) and with in groupings (1 – 13). Age is indicated in red where known and the scale is in days beginning on October 23rd 1918.

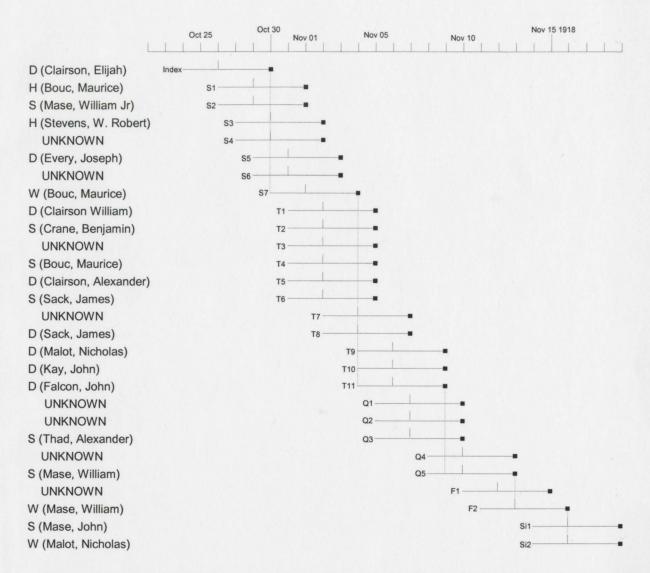


Figure 0.5 – Spread of influenza within and between families at Fisher River as represented by a two-day latent period and three-day infectious period. A standardized line that is divided into a latent and infectious period represents each individual. The first section of the line is representative of the latent period and the remainder of the line following the solid vertical line is infectious period. The square at the end of the line indicates the day the individual died. Dashed vertical lines indicate the period in which the primary case is infectious (from the beginning of the infectious period to death). Once the primary case has been removed a new dashed vertical line is established to represent tertiary cases and so on. Removed individuals are listed on the left hand side of the figure. They are represented by the name of the male head of household, which is preceded by their relationship to this individual. H represents head/husband, W, wife; S, son and D, daughter. The letter and number preceding the solid horizontal line that indicates the period of illness for each individual represent their order infection by group (S, secondary; T, tertiary; Q; quaternary; and F, fifth generation) and with in groupings (1 – 13). Age is indicated in red where known and the scale is in days beginning on October 23rd 1918.

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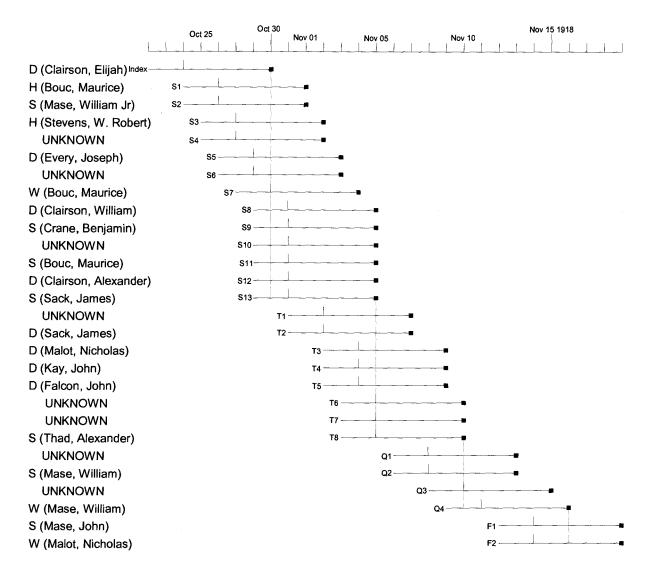


Figure 0.6 – Spread of influenza within and between families at Fisher River as represented by a two-day latent period and five-day infectious period. A standardized line that is divided into a latent and infectious period represents each individual. The first section of the line is representative of the latent period and the remainder of the line following the solid vertical line is infectious period. The square at the end of the line indicates the day the individual died. Dashed vertical lines indicate the period in which the primary case is infectious (from the beginning of the infectious period to death). Once the primary case has been removed a new dashed vertical line is established to represent tertiary cases and so on. Removed individuals are listed on the left hand side of the figure. They are represented by the name of the male head of household, which is preceded by their relationship to this individual. H represents head/husband, W, wife; S, son and D, daughter. The letter and number preceding the solid horizontal line that indicates the period of illness for each individual represent their order infection by group (S, secondary; T, tertiary; Q; quaternary; and F, fifth generation) and with in groupings (1 – 13). Age is indicated in red where known and the scale is in days beginning on October 23rd 1918.