'A Very Remarkable Sickness': The Diffusion of Directly Transmitted, Acute Infectious Diseases in the Petit Nord, 1670-1846

by

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Submitted to the Faculty of Graduate Studies
in Partial Fulfillment of the Requirements
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ABSTRACT

This dissertation examines the epidemic history of the Petit Nord (eastern Manitoba and northwestern Ontario) in the period 1670 to 1846. It documents the incidence and diffusion of directly transmitted, acute infectious diseases (*ACIs*) within the region. In addition to identifying the internal patterns of diffusion, it also delineates the circumstances that carried these diseases into the region. It is thus concerned with both the events surrounding particular epidemics and with the changing conditions that saw *ACIs* introduced into the region from external sources.

There are four main findings emanating from this research. First, it documents the early arrival of *ACIs* in the Petit Nord, beginning in the seventeenth century. Among these were several epidemics that occurred prior to the 1779-83 smallpox epidemic, which has been characterised in the historical literature as the first epidemic in the Canadian Northwest. Secondly, this study identifies an epidemic transition that occurred in the region during the 1830s and 1840s. With this transition the Petit Nord was visited increasingly by *ACIs*, including diseases that had never before entered the region. Similar transformations occurred elsewhere in the New World, and this research provides data on the nature of the changing circumstances that brought about these transitions. Thirdly, this dissertation delineates an important characteristic that was common to all of the epidemics that spread into the Petit Nord: their inability to diffuse to the limits of the region. No matter how virulent, each epidemic left some people untouched while others suffered the full brunt of its effects. Evidence presented here suggests that these differential outcomes had a significant impact on the people of the region, particularly

with respect to changing territorial patterns. Finally, it documents a system of epidemic diffusion that regularly carried *ACIs* from eastern disease pools westward across the continent. This system appears to have been far more significant as a threat to the Petit Nord than that which carried diseases from Mesoamerica through the Central Plains. Although these findings reflect the epidemic history of the Petit Nord, they also provide insight into the history of neighbouring regions, and of the New World in general.

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Finally, this dissertation is dedicated to the memory of two women who had the often thankless task of raising me: my mother, Margaret Walker, and my sister, Mary Jane Caine.

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CHAPTER 1: INTRODUCTION

Study objectives

This dissertation is a geographical examination of the introduction and spread of directly transmitted, acute infectious diseases (*ACIs*[1]) among the peoples of the Petit Nord (northwestern Ontario and eastern Manitoba) in the period between 1670 and 1846. It has four main objectives. The first is to identify the major outbreaks and epidemics that appeared in the region during this study period. The second is to document their diffusion within the region. The third task is to reconstruct the broader, extra-regional, patterns by which *ACIs* entered the Petit Nord. Finally, the study seeks to understand the changes that occurred in the type, frequency and spatial extent of the *ACIs* that struck the Petit Nord during this period. These objectives are consistent with those called for by Ray (1976) in his pioneering study of epidemic disease in the Western Interior of Canada. Ray concluded that

> the geography of epidemics must be worked out in as much detail as possible to determine the frequency with which diseases occurred regionally, the origins and patterns of dispersals of the various epidemics, and the diffusion processes that produced the spatial patterns. (Ray 1976: 139)

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[1]The question of what, collectively, to call these diseases is not a simple one. Here, and throughout this dissertation, I will use the term *ACIs*. This is short for *acute crowd infections*, a term that Trimble (1985; 1989) employed in his studies of the village tribes of the Upper Missouri. See also Cockburn (1971: 50). *ACIs* such as smallpox, measles and influenza have a short duration in an individual and are spread, for the most part, directly from human to human. These characteristics will be more thoroughly explained in Chapter 2.

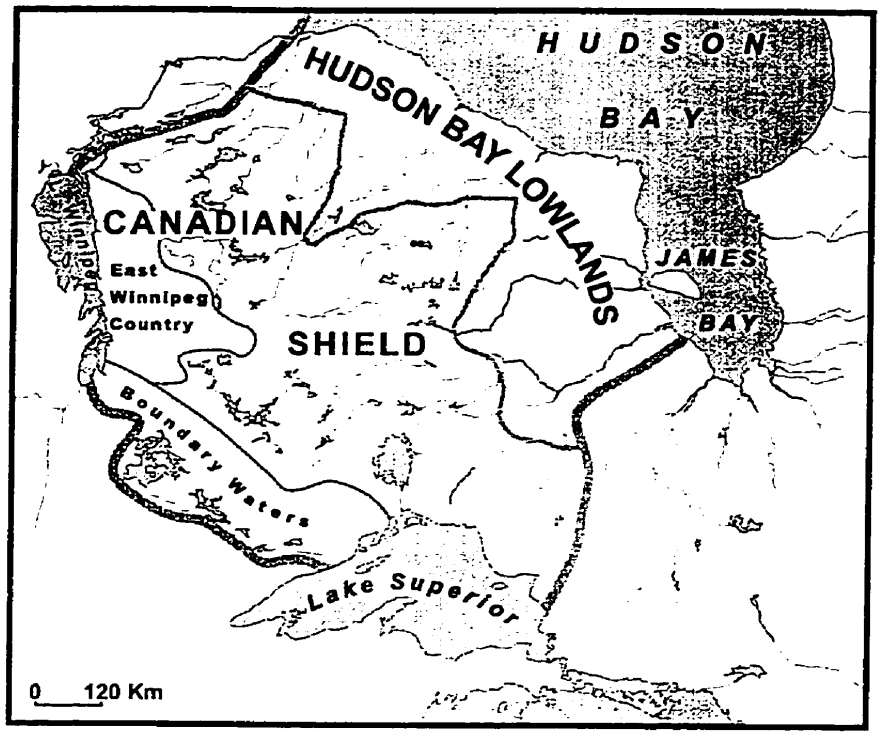

Figure 1: The Petit Nord

Study area

The term *Petit Nord* was an informal regional designation employed first by the French fur traders and later by the Montreal-based English and Scottish traders to describe the large and valuable fur trading country lying to the north of Lake Superior and east of Lake Winnipeg. While its boundaries were never precisely defined, the region can be identified as the area bordered on the north by Hudson and James Bays, on the east by the divide between the Moose and Albany River drainage systems and on the south by Lake Superior and the Boundary Waters that approximate the current U.S.-Canada boundary between Lake Superior and Lake Winnipeg. The region was bounded on the west by Lake Winnipeg and the Hayes River system. Together with the Grand Nord, whose lands extended to the north and west of Lake Winnipeg, it comprised part of the larger regional entity known as the Canadian Northwest (Figure 1).

The Petit Nord is comprised of two different physiographic regions: the Hudson Bay Lowland and the Canadian Shield. The northern third was part of the Hudson Bay Lowland, a large, flat, and swampy plain that is underlain by easily eroded sedimentary strata (Alwin 1978: 24, 27). It is crossed by broad rivers with gentle gradients that flow long distances from the interior of the region to Hudson and James Bays, dropping over fall lines as they emerge from the Canadian Shield (Ibid.: 24). Among the most significant are the Albany, Severn, and Hayes Rivers. The dominant vegetation of the Lowland is muskeg and bog, but there is also a narrow strip of tundra running along much of the length of the coast (Lytwyn 1993: 30-35). While researchers have long thought that the historical food resources of the Lowland were marginal and were insufficient to support native people prior to the fur trade period, recent archaeological evidence (Lytwyn 1993) has demonstrated that this was not the case. Instead, native people inhabited the region long before contact, subsisting on seasonably available food resources, notably the extensive caribou herds, marine mammals, fish and migratory birds. Nevertheless, during the historical period population densities in the Lowland were considerably lower than those found to the south.

The remainder, and by far the greater extent, of the Petit Nord was part of the Canadian Shield. This is an area of erosion-resistant, igneous and metamorphic Precambrian rock, heavily modified by glacial action, which created "a maze of lakes, swamps, rivers, and ice-scoured rock" (Lytwyn 1987). The Canadian Shield is characterised by an "intricate interconnected network of lakes and rivers" which provided access to the interior of the Petit Nord by water, while denying any long distance overland

travel (Alwin 1978: 27, 28). The dense tree cover that is characteristic of the Shield also hindered overland travel during the fur trade period (Bishop 1972: 59). The major routeways into the interior of the region were via the Nipigon and Albany Rivers, which connected with a dense network of interior waterways (Lytwyn 1986: ii; Lytwyn 1987). Travel was constrained during the long winters, when it was necessary to walk along the frozen rivers, and was largely impossible during the brief freeze-up and break-up periods each fall and spring (Figure 2) (Rogers and Smith 1981: 130, 137; Lytwyn 1986: 87). This limited contact between aboriginal groups from the late fall to the early spring, hindering epidemic diffusion during this period. The southern and western margins of the region were significant transport corridors that carried traffic beyond the Petit Nord, to the waterways of the Great Lakes in the east and to those of the Grand Nord in the west (Moodie 1987).

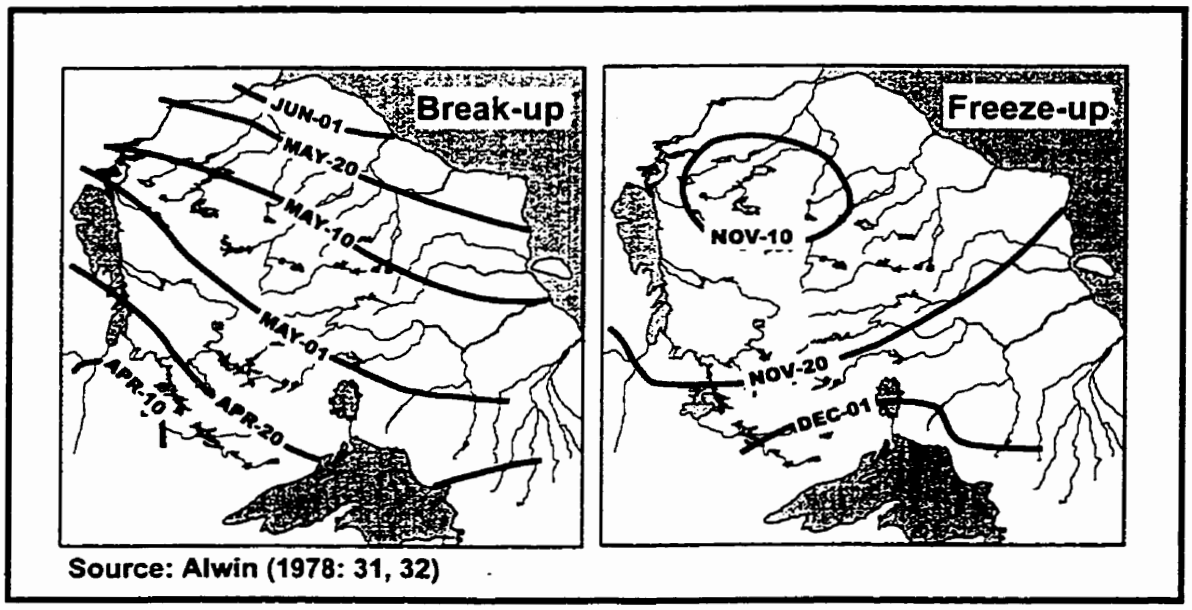

Figure 2: Average dates of freeze-up and break-up in the Petit Nord

In general, the food resources of the Canadian Shield were richer than those of the Hudson Bay Lowland, and this was reflected in the relative densities of their human populations within the region. However, these resources were not evenly distributed throughout the Shield, such that there was a general south to north gradient of decreasing density between the southern margins and Hudson Bay (Bishop 1978: 222). These differences in population density affected the speed with which an *ACI* could spread within the Petit Nord as well as the distance that it could penetrate into the region. For example, the Boundary Waters area provided an abundant selection of seasonably available foods, including big game, water fowl, maple sugar, wild rice and, especially, several varieties of fish[2] (Waisberg 1984: 127; Holzkamm *et al.* 1988; 1991; Vennum 1988; Moodie 1991b). The shores of Lake Superior afforded rich locations for fishing sites, particularly at Sault Ste. Marie, where large numbers of native people gathered from all around during the seasonal runs (Cleland 1982: 763; Goodier 1984; Thwaites 1959: LIV: 133-135; Hickerson 1988: 40-43; Kinietz 1965: 323). Gatherings at Sault Ste. Marie and along the Rainy River were the most conspicuous, where the fisheries supported groups in excess of a thousand people.

In the vast interior of the Petit Nord, however, group sizes were smaller and the people were more dispersed than in the south (Rogers and Black 1976: 21; Rogers 1983: 103). While big game and waterfowl were available, this was beyond the range of maple

[2]In the latter part of the study period several groups in the region also planted crops for subsistence and trade purposes. However, this does not seem to have been the case during the precontact period (Moodie and Kaye 1969).

sugar and wild rice, and the local conditions were such that the fish did not occur in the extraordinarily large runs found to the south (Rogers and Taylor 1981: 231; 232; Smith 1981: 257). As such, these people spent much of the year in smaller hunting and fishing groups of between 10 and 30 people (Rogers 1983: 103). Even during the seasons when the food resources were most abundant, as in the summer, the concentrations of people were much smaller than in the south, numbering at the most 200-300 people (Rogers and Smith 1981: 136; Ray 1976: 141).

While scholars have constructed epidemic inventories for many parts of the New World, none has yet been compiled for the Petit Nord. Indeed, there has been little research specifically intended to illuminate the epidemic history of this region prior to the midpoint of the nineteenth century. Regional inventories are considered necessary in light of the varied epidemic experiences of aboriginal peoples in the Americas[3]. In acknowledging this need, Taylor (1977: 56; 1989: 28) noted that the disease experience of each native group was different. He stated that "Each American Indian tribe... even though they resided in close proximity to each other will experience completely different contact histories. These contact histories will affect their populations". Thus, detailed studies of all aboriginal groups need to be undertaken in order to understand their particular disease experiences.

Such variability in the effects and diffusion of *ACIs* has been observed in the recent literature. For instance, Newson (1992: 108-109) concluded that the epidemics

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[3]For example, Trimble (1988: 4; 1985: 292); Ray (1976), Borah (1992: 12), Crosby (1992) and Villamarín and Villamarín (1992: 113).

that struck early colonial Ecuador were unlikely to have spread over extensive areas or to have been uniform in their impact for many reasons, including several of a geographical nature. Harris (1994: 615) found that there was no common epidemiological history among the aboriginal people of coastal British Columbia until long after the first epidemics had struck them. Aufderheide (1992: 166) warned of the danger of "extrapolating isolated observations of disease-related demographic changes to the population of an entire region or even a continent". By the same token, Milner (1992: 110-111) cautioned that applying mortality figures taken from one epidemic situation to another is problematic, as varying conditions affected the frequency and severity of the disease experience in North America. Moreover, he also suggested that it is unlikely that epidemics reaching the interior of North America would have had a uniform impact throughout. Instead, some groups would have been devastated while others escaped entirely (*Loc. Cit.*).

Although little in the way of historical epidemiological research has been done in the Petit Nord, the region was a key one in terms of the overall flow of diseases within the continent. It lay between the Interior Plains on the west, and northeastern North America on the east, and was epidemiologically connected to both. Thus, an understanding of the epidemic history of the Petit Nord, and also of the sources and patterns of spread of the diseases that penetrated to the region, can help to further explain the patterns of diffusion within the continent as a whole.

Study period

This study covers the period between 1670 and 1846. It begins with the founding of the HBC, which essentially launched the historical period in the Petit Nord and which soon led to significant changes in the movement of people between the region and the east (See Chapter 4). This was also the year of the first documented epidemic *ACI* to reach the margins of the region. The period ends with a series of devastating epidemics in 1846. Although not the last epidemics in the Northwest prior to the midpoint of the nineteenth century, they were among the most severe (Ray 1976: 157). *ACIs* were far rarer in the region during the previous century, suggesting that the situation had changed by this time, and that the Indians of the Northwest[4] were being exposed to these diseases on a far more frequent basis than had previously been the case. The lengthy period covered by this study allows it to examine both of these eras, and to identify the timing of the transition between them. The period begins long before the first direct observations by Europeans on the Northern Plains and over a century prior to the 1779-83 smallpox epidemic, which has been accepted as the first major epidemic in the literature of the Canadian Northwest. This study provides important insights into the nature of earlier epidemics among the peoples of the Petit Nord and the possibility that they spread to surrounding regions.

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[4]And of the Petit Nord. See Young (1991: 37).

Organisation

The organisation of this dissertation departs from that of most epidemic histories. Although chronological, its chapters alternately examine historical periods of varying length followed by single epidemics or epidemic years. This structure can be thought of as a series of vertical studies separated by cross-sections. Both of these are traditional modes of inquiry in historical geography but are not usually combined in a single investigation. The vertical studies document the *ACIs* appearing within a particular period but, more importantly, they also describe the ongoing changes (or processes) that affected the introduction and spread of acute infectious diseases within the Petit Nord. Taken together, these chapters explore the major influences on the epidemic activity within the region between 1670 and 1846. Conversely, the other chapters serve as historical cross-sections, considering only a single epidemic or set of epidemics within a given year, and in as much detail as the documents allow. While these epidemics are important in their own right, they also serve as examples, illustrating the historical forces of change outlined in the other chapters. To begin with, however, the following chapter examines the nature of the diseases being studied and the historical literature, while Chapter 3 deals with the introduction and protohistoric spread of *ACIs* in North America. These chapters provide the necessary background for an understanding of the epidemic history of the Petit Nord that is the subject of this study.

CHAPTER 2: EPIDEMIOLOGICAL AND HISTORICAL BACKGROUND

THE IDENTITY AND NATURE OF *ACIs*

As their name implies, *ACIs,* or directly transmitted acute infectious diseases, have three basic characteristics in common. First, they are *infectious*. Infectious, or communicable, diseases are those that are caused by a specific agent or organism, such as a virus, protozoan[1] or bacterium, which is passed directly or indirectly to a susceptible host from another source[2]. There are five basic modes of transmission: by air, by physical contact, by food and water, by insect and by being there already (Jones and Moon 1992: 147-148). This last includes infectious organisms that normally inhabit the body without producing disease. Many diseases that affect humans are not infectious, but instead are triggered by other factors such as environmental hazards or old age, or are the result of genetically inherited conditions. *ACIs*, by contrast, require the communication of an organism to the victim in order for the disease to develop. Table 1 lists major *ACIs* and their causative agents.

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[1]A single celled organism that is larger than a bacterium and which can cause diseases such as malaria and amoebic dysentery.

[2]When identifying particular *ACIs*, this study will refer to the disease's ICD (International Classification of Diseases) number, the tenth version of a system of unique classification of disease established by the World Health Organisation. For example, measles is classified as ICD-10 B05 (Benenson 1995).

DISEASE	AGENT
Varicella (Chickenpox)	Varicella-zoster virus
Influenza	Influenza virus
Rubeola (Measles)	Measles virus
Mumps	Mumps virus
Rubella (German Measles)	Rubella virus
Variola (Smallpox)	Variola virus
Scarlet Fever	Group A streptococci (Bacterial)
Pertussis (Whooping Cough)	Pertussis bacillus (Bacterial)

After Ramenofsky (1987: 140)

Table 1: Selected *ACIs* and their agents

A second common characteristic of *ACIs* is that they are *directly transmitted.* This means that there is no intermediate host or reservoir other than humans during the lifecycle of the causative organism. Directly transmitted diseases are thus dependent on humans for their very existence. This is in contrast to vectored diseases, such as typhus, malaria, yellow fever and bubonic plague, which exist for part of their lives within animals, arthropods[3] or even the environment. An exception to this among the diseases being studied here is influenza type A, for which avian or swine populations may act as a reservoir. However, the vast majority of transmissions of influenza A to humans are via person to person spread. Although the smallpox virus can exist for extended periods of time in a dried state on some objects under certain specific environmental conditions, it is still considered to be a directly transmitted disease.

[3]Insect vectors such as lice, mosquitoes or fleas.

Finally, these diseases are *acute*. *ACIs* are termed acute since they are of only relative brief duration. Generally, the course of the affliction is measured in days or weeks, after which the victim either recovers or dies. In many cases either total or partial immunity is conferred, meaning that additional susceptibles must be present in order for the disease to survive in a particular population. These "crowd" infections can survive only in densely populated urban communities where there is a continuous supply of susceptibles who have never contracted the disease. As a consequence, their existence will be temporary in a lightly populated region such as the Petit Nord. This is in contrast to chronic infections, such as tuberculosis or syphilis, which may persist in an infected (and infective) individual for years or even decades, during which period retransmission may occur[4]. Such chronic infections are not population density dependent, and may remain active within a very small group for years, or even permanently.

Any analysis of the diffusion of *ACIs* cannot entirely ignore other afflictions or disorders. For example, the virulence and the pathogenicity[5] of *ACIs* in the Petit Nord were no doubt affected by the presence of concurrent debilitating chronic disease or

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[4]This refers only to the main disease, and also to the period of communicability. The varicella virus, which causes chickenpox, may persist in the body in a dormant state long after the initial disease has passed, and reemerge years later as herpes-zoster, or shingles. Similarly, in very rare cases measles infection may lead to subacute sclerosing panencephalitis several years after the patient has recovered (Benenson 1995: 88, 293). Some diseases, notably typhoid, can produce carriers, people who are infected for years but show no signs of the disease, and who can infect others.

[5]The extent to which the infectious agent is able to cause overt disease in an individual (Benenson 1995: 541).

nutritional disorders, which lowered overall resistance[6]. Moreover, it is possible for chronic infections to assume epidemic proportions and mimic the rapid transmission of *ACIs* when a population suffers from concurrent nutritional or other stress (e.g. Saunders et al. 1992: 120-121). There are many instances in the historic record when the source or identity of an affliction cannot be determined, and the possibility that the disease may be chronic, vectored or even non-infectious (but triggered by some common environmental stimulus) must be taken into account. Thus, it is often possible only to note the existence of an unusual disease outbreak or suspicious sickness in the historical record where sufficient data are not available.

There is another related limitation to this and other historical studies of disease. Unlike modern medical reports and clinical studies, most historical materials are poorly suited for conducting epidemiological research. For the most part, the authors of these documents were not medically trained, making it difficult for them to describe the nature of a particular disease, even when they were motivated to do so. Moreover, until relatively recently the general level of medical knowledge was such that the distinction between certain diseases was not clear, or was not made. Nonetheless, modern researchers have been able to work around these source limitations in their examination

[6]Nutrition, age and stress can all affect how a person fights an infection, or how serious are its repercussions (Meade et al. 1988: 31-32).

of historical patterns of epidemic disease with varying levels of success[7].

The voluminous records of the Hudson's Bay Company[8], the main source of data for this study, are particularly good for this purpose compared to other historical documents. They are, according to Ray (1976: 142, fn. 4), "an excellent source of information regarding diseases and the general health of the Indians[9]." While the value of the HBC documents from the study period varies by type, date and author, on the whole the Company maintained a strong concern for the health of the natives, as well as for its employees. As a result, its personnel often wrote at length about sickness at the posts and among the natives. Thus, several scholars have made good use of the HBC records in their epidemiological analyses (e.g. Ray 1976; Ewart 1983; Boyd 1985; Decker 1989b; Hackett 1991).

THEORIES OF HISTORICAL ORIGINS AND SPREAD

The current belief concerning the origin of *ACIs* is that they arose from the

---

[7]Frequently, such problems in the sources have resulted in ambiguity about the identity of a disease or the extent of an epidemic, which has been reflected in the secondary literature. Thus, different researchers often have markedly different interpretations based on the same evidence.

[8]*The Governor and Company of Adventurers of England Tradeing into Hudson's Bay*, hereafter referred to as the HBC.

[9]Of these documents the most crucial are the post journals that normally document day to day events happening in and around the posts, including the presence of disease. Moreover, they also often contain intelligence gathered from Indians visiting from more distant regions, as well as references made by the traders as to the source and extent of the more severe epidemics. These journals have proven to be extremely useful for historical epidemiological studies. See, for example, Hackett (1991) and Decker (1989). Another valuable source is the Company's correspondence, especially the many letters written by the post managers to Governor George Simpson (HBCA D.5/ series) during the early to mid-nineteenth century.

transfer to humans of diseases that were endemic within animal populations (Boyd 1985: 14-29, 34; McNeill 1976; Cockburn 1971; 1977). These crossovers were the product of two fundamental changes in the human way of life. The first was the domestication of herd animals, or of animals that lived in large groups in the wild. As humans came into closer contact with certain animal species, they were exposed to the common diseases of these animals (their zoonoses), which eventually crossed over and became human diseases. Thus, for instance, it is thought that rhinoviruses came from horses, smallpox from cattle, measles from dogs and influenza from swine and poultry (Haggett 1992: 394; Boyd 1985: 34). The first crossovers of crowd diseases from animals to humans may have occurred shortly after the beginnings of animal domestication. However, the diseases that were so transferred were probably unstable at first, as the early groups practising animal husbandry lacked sufficient numbers to maintain them. Instead, the affliction might flare up briefly as a severe, but localised epidemic, disappearing as soon as the supply of susceptibles was exhausted (McNeill 1976: 55). This changed only with the development of large urban civilisations, when some zoonoses were permanently transferred to these populations, and thereafter emerged as distinct diseases of human cities (McNeill 1980: 29). This process of permanent transference is thought to have begun after the rise of Sumeria about 5,000 BP (McNeill 1976: 62; Boyd 1985: 27).

Urban disease pools

William McNeill (1976; 1979; 1980) was the first to examine the role that disease played in the expansion of urban civilisations. He described an historical process that

began with the development of several densely populated, separate, and epidemiologically distinct, Old World *urban disease pools* prior to about 500 BC. Over time, these urban dwellers and the crowd afflictions that emerged among them settled into a relationship of mutual toleration that saw these diseases affect only children, and with far less mortality than had earlier been the case. However, diseases that became endemic, or constantly present, within these urban pools tended to spread into the surrounding rural areas occasionally, killing many and clearing the way for further growth of urban societies. After 500 BC these pools began to exchange diseases and to expand their epidemic influence through territorial expansion, military excursions, new trading connections, and other modes of contact. As new urban civilisations emerged, and connections between them increased, there began a new process of disease homogenisation that would eventually span the entire world. This process was well underway in Europe and Asia by the late fifteenth century, when sustained contact between the eastern and western hemispheres was initiated. Following 1492, the Americas began to be included in this homogenisation through the transoceanic transfer of Old World diseases, to the great detriment of the aboriginal people of the Western Hemisphere (McNeill 1976: Chapter 5). This transfer can be seen as part of a much larger, and much older, process of disease homogenisation between urban disease pools. At the same time, it was also part of a longstanding pattern of urban to rural disease diffusion (Boyd 1985: 38).

Epidemiologically, the most significant attribute of these early urban disease pools

was their abundant populations that became sufficient to permanently maintain crowd diseases. The large number of people in a major urban community provided a continuous supply of susceptibles for these diseases in the form of children who had been born since an earlier epidemic or those who escaped earlier infection. Modern researchers have attempted to calculate the threshold population required for the continuous presence of measles. Although estimates vary considerably, it is likely that a population in excess of 250,000 to 500,000 would be sufficient to maintain this crowd disease in an isolated community (Black 1966: 207; 1980: 43; Bartlett 1957; 1960). This would provide the estimated 4,000 to 4,500 new cases per year needed to support the disease (Bartlett 1957). Although the requirements for other *ACIs* have not been investigated, it seems likely that they too required substantial urban populations for their continued survival within a single community[10].

While scholars have calculated the theoretical lower population limit for measles endemicity based on relatively isolated island communities, or by attempting to correct for communication from external sources, the threshold will no doubt be less under other conditions. Indeed, Bartlett (1957: 59) noted that the number of births required, and thus the base population, could be substantially less for "non-isolated urban areas" due to immigration of susceptibles from surrounding areas. Population density could also play a

role in determining the threshold value, in that the number needed in a less densely settled area could be far less than in a similarly sized, but denser, settlement (Black 1966: 210). This is a function of the relative frequency of contact between individuals, and so the same might also be the case with a socially stratified or physically divided community where communication between its citizens is hindered in some way. Finally, it is probable that another type of urban endemicity, a shared endemicity, could result from the regular communications between closely settled smaller cities. Black (1980: 43) concluded that, although measles cannot persist in a single pre-agricultural society, "it can sometimes spread from one community to another and thus extend its stay...." Given large enough communities, it is very possible that they could extend that stay indefinitely, acting as a communal disease pool for measles, and for other crowd diseases, that circulate among them, even though no single community exceeds the theoretical population threshold (e.g. McNeill 1976: 62). Thus, disease pools may emerge within groups of cities that have yet to pass a critical population threshold.

Once established as endemic diseases, *ACIs* settle down into a predictable pattern of infection within the urban disease pool. For most of the time they exist at a very low level of incidence, supported by new births and among children who are susceptible. In most cases, adults will have contracted the disease in their youth and, for this reason, they

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[10]An exception to this may be chickenpox, for which it has been estimated that the critical community size is only 1,000 (Black 1966: 210). This is due to the Varicella virus's ability to hide within the human body for decades following an episode of chickenpox, only to reemerge as shingles. Another exception is influenza, which may require substantially larger populations than are found in any city (Haggett 1993: 46). Due to the instability of the influenza virus the disease does not behave as a disease of childhood.

are also known as diseases of childhood. Every few years, as the number of available susceptibles reaches a critical number, an epidemic will erupt (Cliff and Haggett 1993: 245; McNeill 1980: 30). The period between these epidemics is regular if the population is a stable one, and is dependent on the size of the community (Jones and Moon 1992: 155).

Urban disease frontiers

ACIs do not always stay within the limits of the disease pool. Periodically, these diseases spread outward among other populations. Therefore, it can be said that a pool has an *urban disease frontier*, a range within which its *ACIs* may affect people beyond its immediate limits. In this way, communities with populations below that required to maintain these diseases share indirectly in the endemic status of the urban disease pool, or even of multiple disease pools. Depending on the pool's interactions with the outside world, this frontier can extend to include peoples living on other continents, and can cross oceans or other significant obstacles. As will be shown below, the distance of that frontier from the pool can change as conditions change, and is not the same for all diseases.

While different groups may be located within a pool's disease frontier, they may have very different experiences with these *ACIs*. How an *ACI* affects a given group is in large part determined by the frequency of that group's experiences with that disease. In turn, the frequency of such exposure is a function of the nature of communications

between these outlying groups and the disease pool. McNeill (1979: 95) presented a simple model describing the epidemiological relationship between an urban centre and two categories of outlying populations: *near* populations, or what McNeill called the rural peasantry, and the more distant *peripheral* populations (Figure 3). Near populations live immediately beyond the urban centre, and are well connected to it by regular interaction. This provides frequent opportunities for the introduction of the full range of *ACIs*, which regularly erupt as mild epidemics affecting only the youngest portion of the population (McNeill 1979). Although the disease disappears between epidemics, this pattern is very similar to that experienced under endemic conditions.

An example of the relationship between near populations and urban disease pools can be seen in the case of smallpox in eighteenth century Britain. Duncan *et al.* (1994) found that the disease was initially endemic only in London, but by the mid-eighteenth century was also constantly present in Glasgow and Edinburgh (Ibid.: 265). In these major communities smallpox flared up as epidemics every two to three years but was otherwise present in low levels. In the smaller towns smallpox was transient, and was only present in epidemic form. However, these epidemics occurred about every five years, a sufficient frequency to maintain smallpox as a disease of childhood despite the fact that their populations were insufficient to maintain it endemically. In this respect, their experience with an *ACI* was similar to that of communities with populations

exceeding the endemic threshold[11].

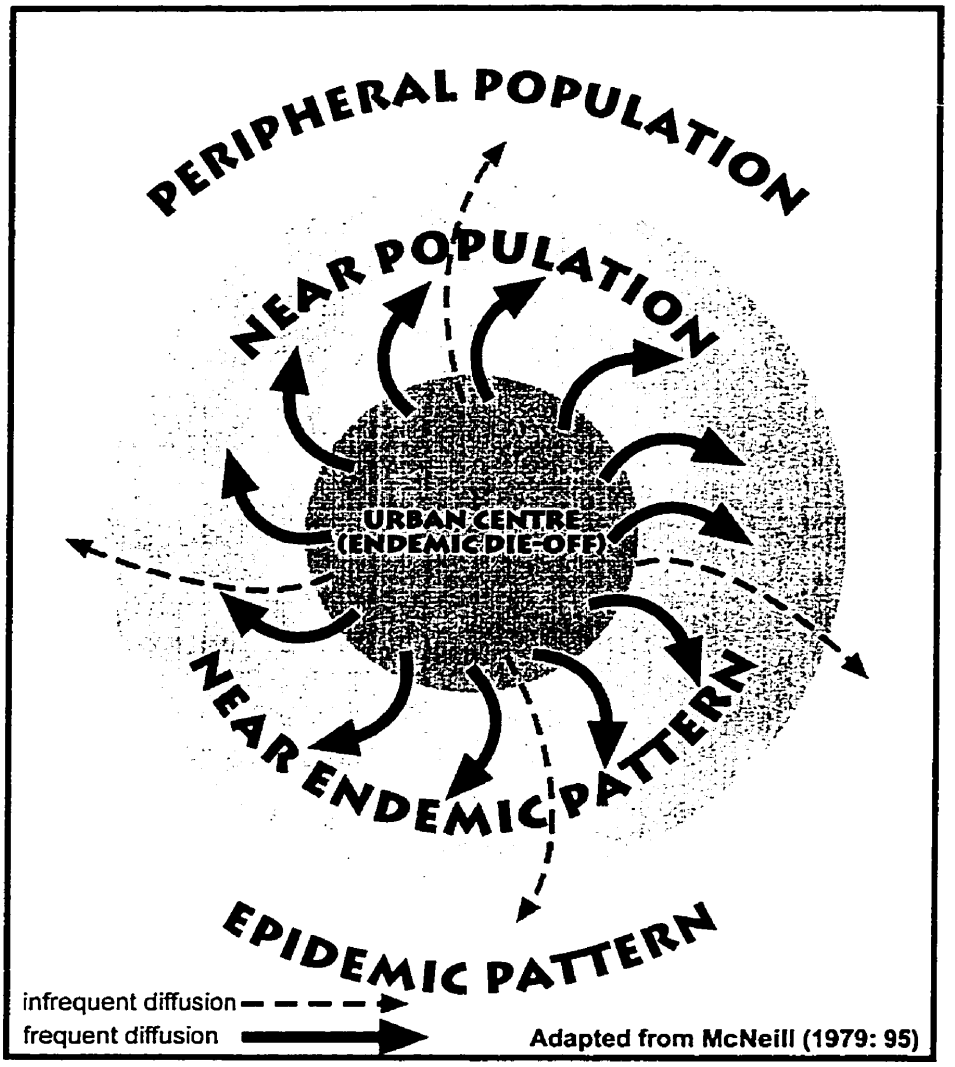

Figure 3: Epidemiological relationship between urban disease pools and outlying populations

[11]The medical historian Creighton (1965: 2: 457) observed that during the late seventeenth century and throughout the eighteenth, "a high mortality in London in a certain year meant an epidemic general in England in that or the following year...." This illustrates the relationship between that city and the smaller communities that were in close contact with it.

In contrast to the near populations are those that are "peripheral" to the urban centre (Figure 3). For these people, communication is too irregular for *ACIs* to return with sufficient regularity to become childhood diseases (McNeill 1979: 96). Instead, they reappear after longer intervals and are much more severe, affecting adults as well[12] (McNeill 1980: 31). In some cases, the geographical extent of the urban disease frontier could be immense, such that diseases carried from the pool could spread to peripheral peoples living thousands of miles away. For some extremely peripheral groups, several generations might pass before an *ACI* returned. Thus, for example, in Iceland, an island nation whose population fell well below the threshold for measles endemicity, measles outbreaks were extremely rare during the nineteenth century, with several decades passing between them (Cliff et al. 1981: 48). More importantly for the purposes of this study, this was also the case for many aboriginal groups in North America who, during the post-contact period, experienced *ACIs* such as measles and smallpox only infrequently, and who suffered severely for their isolation.

The size of the population also plays an important role in determining the frequency of return of an *ACI*, and thus its effect on a group. Bartlett (1957) identified three basic patterns of measles periodicity in British urban communities. Type I cities are

[12]In one sense, isolation from disease pools provides protection from *ACIs* for a peripheral group, since they are not generally exposed to the disease. However, each epidemic will likely take a greater toll than would be the case were the disease endemic. Haggett (1993: 47) noted the decline in the isolation of Australia, New Zealand and the South Pacific as a major factor in an increase in disease in those areas. In

those in which the population is sufficiently large that it is able to maintain measles as a disease of childhood (Figure 4). Type II cities have a population less than that required for endemicity, but greater than about 10,000. When the possibility of measles introduction is constantly present, they will experience measles epidemics at regular intervals that match the rate of the larger, endemic, cities, but the disease will be absent during the inter-epidemic periods (Figure 4). Type III communities, or those with fewer than 10,000 people, will suffer periodic measles epidemics, but will miss some since they may not have built up a sufficient supply of susceptibles to fuel an epidemic by the time the disease is reintroduced from an external source. As such, the pattern of epidemics is less regular, and adults may also be affected. The groups found in the Petit Nord throughout the study period were Type III communities, as even the largest seasonal agglomerations numbered no more than a few thousands at most.

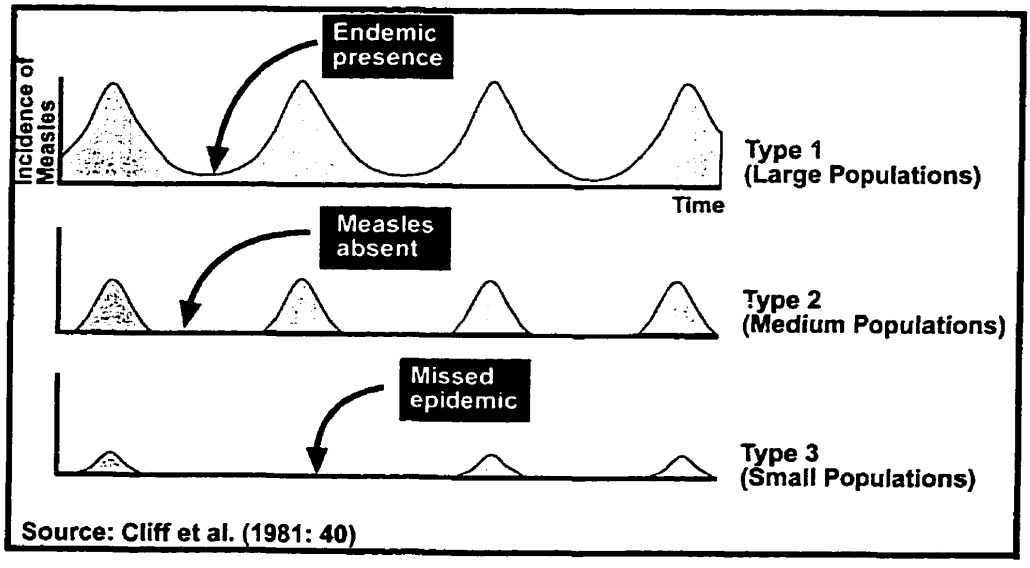

Figure 4: Relationship between community size and periodicity of measles epidemics

effect, changes in transport technology made them less peripheral than they had once been.

The epidemic transition

As changing conditions lessen the isolation between a peripheral population and an urban disease pool, it may assume a new relationship with that pool, becoming a near population. This shift in status can be thought of as an *epidemic transition*[13]. There are two basic characteristics of this transition. The first is that certain diseases return with increasing frequency and that, overall, *ACIs* become more common than before. Thus, according to Ralph Garruto (1981: 585-586), "Major epidemic diseases including measles, smallpox, influenza, pertussis, and venereal diseases[14] are constantly reintroduced [to formerly isolated groups] with shorter time intervals between epidemics as isolation continues to decrease". If they appear often enough, they cease to be "major epidemic diseases", but instead become diseases of childhood. The second characteristic is the presence of new *ACIs* that have never before appeared. While isolation persists, the range of diseases is small and limited to those with an enhanced ability to diffuse, such as smallpox (see below). As isolation declines, other diseases begin to appear in the community. Once the transition is complete, a wide range of *ACIs* will appear with some

[13]Another type of transition, called the "mortality" or "epidemiological" transition, refers to the health changes that arise from economic and social development (Meade, Florin and Gesler 1988: 108). Perhaps the most well documented manifestation of this transition is the shift from pre-industrial to modern industrial western societies which saw the decline of death and birth rates, and the rise of chronic and degenerative diseases at the expense of infectious diseases, with an upwards rise in life expectancy (*Ibid.*: 108-109).

regularity. Eventually the result will be a mutual toleration with limited mortalities as these diseases achieve childhood status. However, until that balance is achieved, the increased frequency associated with these additional diseases will likely lead to a more severe experience with these repeated *ACIs*.

There are several potential reasons for the decline in isolation that triggers this transition. First, there may be more people travelling between the two populations[15]. Larger numbers of people provide greater opportunities for *ACIs* to survive a journey to the peripheral community, especially if children are among them. Moreover, patterns of travel may be altered, exposing new populations to exogenous disease, or those diseases that are introduced from elsewhere. Throughout history, changing migration patterns have resulted in the sudden appearance of new diseases (McNeill 1980). As well, travel times may be reduced substantially, simplifying the chain of infection needed to maintain the disease during the trip (Haggett 1994: 102-103). Periodic transport innovations, such as steamships, trains and jet aircraft, have been instrumental in facilitating the long distance diffusion of *ACIs* into new areas by increasing the speed, volume and range of

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[14]Here I must differ with Garruto in that venereal diseases (sexually transmitted diseases, or STDs), as chronic infectious diseases, will not necessarily respond to decreased isolation in the way that the other, acute, diseases do. STDs are not as limited by isolation or infrequent communication, since by nature the victim may be infective for years, and they do not require large populations for endemicity. Thus, STDs and tuberculosis penetrated into the Petit Nord relatively early in the contact period, and remained within the aboriginal population despite an extremely low population density and limited contact with the urban regions.

[15]The world-wide movement of *emerging infectious diseases*, those that are successfully expanding into new ecosystems, has been made much easier by the vast increase in the mobility of the global population and the increased speed of travel in recent decades, as well as many other human-related factors (Haggett 1994; Wilson 1995; Morse 1995).

travel (e.g. Cook and Lovell 1992b: 236; Haggett 1992: 395; 1993: 46-47; Wilson 1995; Pyle 1969). It may also be that a different pool emerges in closer proximity to the peripheral population as other nearby communities grow and achieve endemic status on their own, thereby creating a closer source of infection. Likewise, new transport linkages may place the isolated region within the disease frontier of other pools or of multiple pools. Finally, the growth of new population centres that fall below endemic thresholds may still have a significant effect on long-distance patterns of diffusion by creating temporary holding bodies for *ACIs*, thereby acting as intermediate sources of infection.

Once again, one can turn to measles in Iceland as an example of this process. Cliff et al. (1981: 48) documented only two full-fledged measles epidemics for the island during the period between 1751 and 1900, and these caused considerable mortality. Between 1900 and 1945 the level of contact between Iceland and the outside world increased continuously, and there were more epidemics, all of lesser mortality (Ibid.: 48, 116). This decline in the severity of measles in Iceland after the 1882 epidemic was attributed to the increased frequency of the disease (Ibid.: 64). Following 1945, Iceland's relative epidemiological isolation disappeared, as both ship-based and air-based links increased significantly (Ibid.: 79). Thereafter, measles epidemics occurred much more frequently, were less localised, and caused fewer deaths (*Loc. Cit.*). Increasingly, the pattern of measles in Iceland came to resemble that found in the more densely populated parts of western Europe and eastern North America, although its total population remained below the endemic threshold. Iceland had experienced an epidemic transition

and was no longer a peripheral population.

Disease diffusion potential

Not all *ACIs* have equal ability to spread beyond their urban limits. In general, certain diseases diffuse earlier, faster and farther, while others are only able to travel short distances until circumstances change significantly. Put another way, this means that some diseases will appear in an isolated, or peripheral, population that others are unable to reach. As communication between the pool and the periphery intensifies and becomes more regular, and as isolation decreases, new diseases will begin to appear and earlier afflictions will return more frequently. Given sufficient decline in isolation the result will be the epidemic transition noted above. The order and timing of the introduction of specific *ACIs* reflect a combination of local contact conditions and the ability of those diseases to spread. The measure of this ability to spread is known as *diffusion potential.* Ramenofsky (1987: 167) suggested that the diffusion potential of a particular disease in a given situation varies with three factors: the length of the infectious period, the population density of the location and the surrounding region, and the contact mechanisms between communities or regions. She employed the characteristic infectious period to assign a rank to several of the major *ACIs*, thereby dividing them into three classes (Table 2). By her classification, smallpox and whooping cough were the most proficient diffusers, while colds and influenza were the least able. Such a ranking should

indicate the order of introduction of *ACIs* into a formerly isolated region. Thus, "Parasites with longer periods of communicability were introduced [to the New World] earlier and resulted in extensive spatial waves" (Ramenofsky 1987: 167). As conditions change and lessen that isolation, other diseases with lesser potential will then appear.

Class I ≥ 15 days	Class II 7 to 14 days	Class III ≤ 6 days
Whooping Cough Smallpox	Measles Scarlet Fever Chickenpox Mumps	Cold Influenza

(Ramenofsky 1987: 167)

Table 2: Diffusion Potential of Selected Infectious Diseases

While developed theoretically by Ramenofsky, this system of classification can have practical applications, enabling researchers to predict the sequence and perhaps even the timing of exogenous disease introduction in a specific historical context. Before it can be used in this way with confidence, however, it must be tested against known disease records and refined to provide a more precise division. The lengthy period covered in this study of *ACIs* in the Petit Nord provides a suitable opportunity to evaluate the proposed disease diffusion potentials.

ACIs IN THE NEW WORLD: THE COLUMBIAN EXCHANGE AND EPIDEMIC DIFFUSION IN THE HISTORICAL LITERATURE

The Columbian Exchange

Beginning in the early sixteenth century the aboriginal people of the Americas began to suffer appalling casualties from previously unknown diseases, introduced among them by those who came from the Old World. Prior to this date, the Western Hemisphere had been beyond the disease frontiers of the Old World urban pools. Due to a host of different factors, diseases that were little feared by adults in the more populous parts of Europe claimed countless lives as they found 'virgin soil' in the Americas[16]. The introduction of these new diseases to the hemisphere was not instantaneous in all parts and for all diseases but proceeded at different times in different places. This study examines a portion of that process that occurred in a region far removed in time and space from the initial introductions of the fifteenth and sixteenth centuries.

Although it has been claimed in the literature that the pre-Columbian Americas were a "relatively disease-free paradise" (Dobyns 1976b: 1), recent research has shown that this was not the case (Verano and Ubelaker 1991: 212-215; Aufderheide 1992: 165; Waldram, et al. 1995: Chapter 2). Instead, there is now reason to believe that the people of the Americas suffered from a wide variety of health conditions and diseases, some of which were infectious. For example, there is paleopathological evidence of mycobacterium tuberculosis, treponemal diseases (such as Pinta), pneumonia

[16]Virgin soil populations are those in which a particular disease organism has never before been present, or has not been present for many years (Saunders et al. 1992: 117; see also Boyd 1985: 43).

(bronchopneumonia and lobar pneumonia) and various parasitical afflictions (e.g. hookworm and roundworm) (Waldram, et al. 1995: 26, 37; Verano 1992: 17, 18; Milner 1992: 107; Powell 1992: 41). Few acute afflictions (and very few viral diseases) leave distinct markers on the human skeleton (Ortner 1992), leaving open the possibility that the list of aboriginal diseases was far longer. Others, such as streptococcal and staphylococcal infections, bacilliary and amoebic dysentery, some rickettsial and viral fevers and American leishmaniasis, may also have existed aboriginally (Boyd 1985: 39; Newman 1976; Saunders et al. 1992). Consequently, it is also likely that some pre-Columbian populations experienced periodic epidemics of some of these diseases as conditions temporarily lessened their ability to fight off endemic diseases (Saunders et al. 1992: 120-121). The disease load of specific peoples would have varied, depending on their population, their economic and social activities, the local environment, and many other factors, but it is nonetheless important to note that the hemisphere was far from disease free. Missing, however, were the *ACIs*.

While the pre-contact Americas supported a wide variety of afflictions, it is also clear that contact brought an influx of many other deadly diseases. New infections came with the explorers, traders, colonists and slaves who followed, and their impact was often devastating. Complete isolation from the Old World had left the millions of people who inhabited the hemisphere wide open to an extensive array of new pathogens (Table 3).

Smallpox	Cholera
Malaria	Pertussis
Influenza	Diphtheria
Yellow Fever	Scarlet Fever
Mumps	Rubella
Measles	Polio
Typhus	Colds
Bubonic Plague	Venereal Syphilis

Boyd (1985: 39); Ramenofsky (1993)

Table 3: Selected Old World diseases introduced to the Americas

These diseases varied considerably in the nature of the organisms that caused them, in their severity and in the ways in which they manifested themselves, but all had been absent from the Western Hemisphere prior to the late fifteenth century.

Epidemic diffusion and the New World literature

The history of the introduction, spread and effects of *ACIs* in the Americas has not always been the subject of mainstream research. Until well into the twentieth century, references to these diseases were mainly limited to broader medical histories or to brief comments in historical works[17]. There was no specialised disease-centred literature as

[17]One early and oft-cited example of medical history from Canada is John Heagerty's (1928) *Four Centuries of Medical History in Canada.* Charles Creighton (1965, but first published in 1894), in his *History of Epidemics in Britain*, commented at length on the earlier epidemics of North America. Later examples include Ashburn's (1947) *The Ranks of Death*, and Steam and Steam's (1945) *The Effect of Smallpox on the Destiny of the Amerindian.* A particularly noteworthy example of an early historical work paying close attention to the effects of epidemic disease among aboriginal people is Alfred Goldsworth Bailey's (1976, but first published 1939: 75-83) *The Conflict of European and Eastern Algonkian Cultures.* This book was far ahead of its time, both as an ethnohistorical work and in the attention given to the effects

there is today. This began to change during the 1930s as a new historical demographic emphasis emerged, one that focussed on the role of Old World disease, predominantly *ACIs*, in aboriginal depopulation. This new emphasis stemmed from dissatisfaction with earlier estimates of the size of the precontact population of the Americas (Ramenofsky 1987: 6-12). In response to a perceived underestimation of the impact of epidemic disease on the part of James Mooney, Alfred Kroeber and others, scholars at Berkeley University, including Sherburne Cook and Woodrow Borah[18], began to examine the question of precontact regional populations using historical materials (*Loc. Cit.*; Henige 1990; Dobyns 1976b). It is the work of this Berkeley School, and of its successors, that has shaped much of the subsequent research on Old World diseases in the Americas[19].

Over the next three decades, Cook, Borah and the others examined the demographic trends of Spanish America, particularly of the natives of California and Mexico. During this time they produced an array of detailed studies effectively challenging the extreme view that epidemic disease played no significant role in changing the lives of the aboriginal peoples of these areas prior to the modern era. Despite their

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of Old World diseases. Bailey cited both Heagerty's work and published primary documents from the *Jesuit Relations*. In more general studies, Diamond Jenness (1989 first published in 1932), Harold Innis (1956, but first published in 1930) and Arthur S. Morton (1939) all commented briefly upon the effects of epidemic disease on Canadian Indians. Herbert Williams (1909) provided an early study of a single epidemic in his examination of the 1616-1620 epidemic among the Indians of New England. See also Quaife's [1964, but first published 1930] study of the 1837-38 smallpox epidemic on the Upper Missouri.

[18]See, for example, Cook (1935, 1937, 1939a, 1939b; 1945; 1955; 1973b) and Borah (1964; 1976; 1991; Borah and Cook 1960; 1963). Cook and Borah have been the most prominent in terms of disease work, but Lesley Simpson and the geographer Carl Sauer were also part of this group (Henige 1990: 170; Lovell 1992b: 427).

[19]A comprehensive and highly critical analysis of the Berkeley School, and of the disease

emphasis on the depopulating effects of Old World diseases, however, the early members of the Berkeley School provided little direct information regarding disease diffusion. Neither was this subject given much consideration by other researchers until several decades later.

Greater interest in the demographic impact of Old World diseases was sparked by Henry Dobyns's (1966) "Estimating Aboriginal American Population," published in 1966. Building upon the argument of the Berkeley School that aboriginal populations declined precipitously following the introduction of *ACIs* to the New World, Dobyns estimated that approximately 100,000,000 people inhabited the hemisphere at contact. Within about a century of initial contact, he suggested, most had succumbed to disorders that had been introduced from Africa and Europe. Dobyns (1966: 404, 441-442) also briefly identified a concept that would become an integral part of his argument for much higher precontact hemispheric population estimates. This was the assumption that pandemics swept the Americas from an early date, for the most part prior to direct contact. By assuming that virtually all New World populations had declined precipitously due to epidemic diseases, even the earliest of estimates would have to have been far smaller than the pre-Columbian populations. Here, then, was an early glimpse of a view of surmised continental patterns of disease diffusion that would eventually prove both highly influential and highly controversial.

Dobyns's emphasis on massive decline due to the introduction of Old World

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literature, can be found in David Henige's (1998) *Numbers from Nowhere*. See also Denevan (1992).

disease helped to stimulate research into native historical demography, and disease in general (Dobyns 1976a: 96; Jennings 1982: 31; Trigger 1986: 259), as did subsequent publications by historians Alfred Crosby (1972; see also Crosby 1976b; 1978) and William McNeill[20] (1976). Still, studies of historical diffusion in the Americas remained comparatively rare until after the mid 1980s, and the few that were published did not address the possibility of protohistoric, continent-spanning pandemics. This part of Dobyns's argument did not immediately have a significant or widespread impact on other researchers[21].

One diffusion study that has largely been ignored by historians and ethnohistorians was Gerald Pyle's (1969) "The Diffusion of Cholera in the United States in the Nineteenth Century." Pyle, a geographer, examined the patterns and processes of diffusion of the three main cholera epidemics in the U.S. during the nineteenth century.

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[20]While Dobyns's paper undoubtedly provided considerable stimulus, equally important, if not more so, was the increasing interest in ethnohistorical research, which increased greatly following the Second World War. Ethnohistory developed as a kind of fusion between history and anthropology, and concerned itself with the study of change among aboriginal peoples (Trigger 1982: 3; Lurie 1961). Prior to the development of ethnohistory, anthropological research had virtually ignored historical change among aboriginal cultures. Some other studies of New World historical demography from this period are Cook (1973b), Ewers (1973); Jacobs (1974), Denevan (1992); Meister (1976), Miller (1976), Newman (1976) and Ubelaker (1976). At the same time, scholars began to investigate other aspects of their impact. See, for example, Lehmer (1977b), Ewers (1972; 1973), Taylor (1977), Krech (1978) and Calvin Martin (1974; 1976; 1982). Examples of epidemic inventories from this period include those by Lehmer (1977a; 1977b), Ray (1976), Taylor (1977) and Ewers (1973).

[21]See also Dobyns (1976a: 98). Dobyns was successful in influencing at least two researchers. In considering the existence of a protohistoric disease frontier based on the concepts provided by Dobyns, Wilbur Jacobs (1973: 46-47) stated that "There were, then, disease epidemics after the discovery of America which swept like wildfire through the whole Indian world, swiftly moving over the entire continent before Jamestown and Plymouth were going concerns." Crosby also appears to have been influenced by Dobyns's argument in his paper "Conquistador y Pestilencia" (Crosby 1967) and his later book "The Columbian Exchange" (Crosby 1972).

He identified a shift from a simple pattern of diffusion into the interior of North America during the initial epidemic of 1832 to a more complex hierarchical pattern for subsequent epidemics as the American settlement and transport systems evolved. This study remains one of the few to discuss the diffusion of epidemic disease within North America on a broader scale. It is also an example of the valuable contribution that can be made by geographers to the study of historical patterns of epidemic diffusion in the Americas. Similarly, another geographer, Arthur Ray (1975; 1976; 1988, but first published in 1974), documented the diffusion of the most devastating epidemics that struck the Indians of the Western Interior of Canada between 1780 and 1850. Ray's important contributions have laid the groundwork for subsequent scholarly research on disease diffusion in western Canada, but have been ignored by Dobyns[22]. Michael Trimble (1975) examined the diffusion of smallpox in the Northern Plains in his Master's thesis, leading later to his fine PhD dissertation on the same subject (Trimble 1985a) and several key publications (Trimble 1985b; 1988; 1989; 1992). Another example from this period was Clyde Dollar's (1977) study of the 1837-38 smallpox epidemic. An historian, Dollar provided an excellent analysis of the spread of smallpox from St. Louis to the Upper Missouri in 1837 via an American Fur Company ship.

Other significant research from this era concerning epidemic diffusion emerged as part of broader studies. For instance, Bruce Trigger (1987, but first published in 1976;

[22]Of these, his most significant contribution to the study of epidemic diffusion came with his 1976 paper, "Diffusion of Diseases in the Western Interior of Canada, 1830-1850." More will be said regarding this seminal work with regard to its place in the literature of the Petit Nord.

See also Trigger 1981; 1989, but first published in 1985) examined the spread of several seventeenth century epidemics in the northeastern part of the continent as part of his ethnohistory of the Huron. In his study of the sociocultural effects of the epidemics on the people of the Northern Plains, John Taylor (1977) identified three broad patterns of spread that defined different epidemic subphases on the Plains. In so doing, he distinguished both the major mechanisms of diffusion in that region and the historical changes that affected them. In his later Master's thesis, which expanded upon the same subject, Taylor (1982) described and mapped the spread of several of the epidemics that spread through the Northern Plains.

Dobyns's (1983) subsequent book, *Their Number Become Thinned*, has emerged as perhaps the most influential study of Old World disease among the aboriginal people of North America. In this work he made epidemic diffusion a central part of his argument for ever-increasing estimates of precontact aboriginal populations. Here, Dobyns reiterated and greatly elaborated upon his earlier theory of protohistoric pandemics. In a series of essays dealing with varied aspects of aboriginal population size, resource availability and use, and the diffusion and effects of introduced Old World diseases, the author linked evidence of disease episodes among distant peoples together in order to suggest broader patterns of diffusion. The implication throughout was that many epidemics struck the continent beginning with smallpox and measles in the early sixteenth century, that they included a wide range of exogenous afflictions and that they tended to spread far from their initial point of introduction, almost without limit. Thus,

he concluded, these pandemics depopulated the continent early on, in most regions long before there was historical documentation of population levels, and so "Aboriginal lifeways for the native peoples of North America clearly terminated with the large-scale depopulation caused by the initial smallpox pandemic in 1520-24" (Dobyns 1983: 25).

Dobyns's conclusions have subsequently found both approbation and criticism in the literature. This is particularly so with respect to his belief in early protohistoric pandemics. Many researchers have been persuaded (largely as a matter of faith) that at least the 1520 smallpox epidemic spread throughout large portions of the continent, if not also many other sixteenth and seventeenth century epidemics (Henige 1990: 184; e.g. Trimble 1989: 42, 48; Vehik 1989: 116-117; Milner 1980; Stannard 1992: 24; Upham 1986; McNeill 1976: 207). A recent collection of essays examining the effects of Old World disease in colonial Spanish America (Cook and Lovell 1992a) is illustrative of how this idea has captured many people's attention. In the preface, Noble David Cook, an historian, and W. George Lovell, a geographer, followed Dobyns's lead, stating that:

> Wave after wave of Old World disease swept the Americas, following well-established routes of trade and communication. Disease impact varied according to a number of factors, but by the seventeenth century aboriginal numbers had been massively reduced and native ways irrevocably altered. (Ibid.: xix)

Likewise, in his introduction to the same book, Woodrow Borah (1992: 16) described this theory as "eminently reasonable". However, attempts to verify the possibility of protohistoric pandemics using archaeological methods (e.g. Ramenofsky 1987; Campbell 1990) have proven inconclusive (Henige 1998: 148-150, 156-161, 314-315). Meanwhile,

other scholars have examined Dobyns's theories carefully and found them wanting on several grounds. In particular, he has been faulted for the questionable selection and handling of source information, and for his basic assumptions regarding diffusion (e.g. Henige 1986; 1989; 1990; 1998; Reff 1989; 1992; Snow and Lanphear 1988; 1989; Snow and Starna 1989; Thornton et al. 1992: 191). Nevertheless, such criticism has not eliminated support for Dobyns's theory, but has merely intensified the debate. With the publication of *Their Number become Thinned*, epidemic diffusion in the Americas finally became a central issue in the disease literature, if only as a theoretical concept.

Since 1983 there has been an impressive growth in the number of publications dealing with all aspects of the impact of Old World disease on aboriginal people, especially in the years leading up to, and since, 1992. This marked the quincentenary of Columbus' arrival on Hispaniola, and this anniversary sparked the publication of several important scholarly works as well as many popular articles[23]. No one discipline has dominated the literature. Instead, it has evolved into a truly interdisciplinary field with contributions by scholars from several fields, including demographers, physical and cultural anthropologists, geographers and historians[24]. In turn, this has fostered a healthy diversity of approaches and subject matters. Equally important has been the acceptance

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[23]For instance, Lovell (1992b), Cook and Lovell (1992) Verano and Ubelaker (1991; 1992), Stannard (1992), Turner and Butzer (1992) and Denevan (1992).

[24]Cook and Lovell's (1992) collection of papers on disease in colonial Spanish America, *"Secret Judgements of God"* is an excellent example of just how diverse the backgrounds of researchers has become. Contributors were educated in history, geography and anthropology, and published their findings with the assistance of medical personnel (*Ibid.*: xxii). So, too, is Verano and Ubelaker's (1992) *Disease and Demography in the Americas*, whose contributors represented an even wider range of disciplines.

of disease as an important subject matter in the more general ethnohistorical and historical literature. As the impact of exogenous diseases on aboriginal people, including non-demographic effects, has become recognised generally, scholars who do not specifically focus on disease increasingly have made contributions to the study of historical patterns of *ACIs*[25].

As a result of this growing interest in Old World disease, much more is becoming known of the presence and effects of epidemic disease among the people living in the regions surrounding the Petit Nord, although the depth of study varies considerably both in time and space. Coverage of the Northern Plains and adjacent parts of the Western Interior of Canada during the late eighteenth and early nineteenth centuries has been excellent, including several important studies of diffusion[26]. Conversely, very limited research has been undertaken among the people living to the northwest of the Petit Nord. Most notable is the work of Sheppard Krech (1978; 1981b; 1983), who has begun the task of outlining the epidemic history of the Arctic Drainage Lowlands Dene. There has been considerable study of epidemic *ACIs* among the people to the south and east of the Petit

[25] Some recent examples from those studying the aboriginal people of the Canadian Northwest include works by Paul Thistle (1986), John Milloy (1990), Dale Russell (1991), Victor Lytwyn (1993), Laura Peers (1994) and Ted Binnema (1998).

Nord, most of it focused on the period prior to the nineteenth century[27]. For example, Trigger (e.g. 1981; 1989; 1987) has done much to clarify the identity and patterns of spread of the devastating seventeenth century epidemics that spread to the interior of North America from the east, and their impact on the Huron. Of considerable importance is an ongoing debate concerning the timing of the initial afflictions, especially among the Iroquois, by which Dobyns (1983) has attempted to prove the case for hemispheric pandemics during the sixteenth century[28]. Although not neighbouring to the region, the Pacific Northwest experienced several epidemics that originated in the east, passing through the Petit Nord. Thus, the research undertaken for that region is important for establishing broader patterns of diffusion. In recent years excellent work by Robert

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[26]See, for example, studies by Taylor (1977; 1982), Lehmer (1977a; 1977b), Dollar (1977), Ramenofsky (1987) and Sunder (1965). Of note here is the work of Arthur Ray (1976; 1988) and Jody Decker (1988; 1989b; 1991), who have identified and mapped the diffusion of several epidemics that struck the Western Interior of Canada, including the western margin of the Petit Nord, between 1780 and 1850 (See below). Michael Trimble (1975; 1985; 1988; 1989) has provided excellent insights into the spread of disease among the village tribes of the Upper Missouri, and his methods provide a model for further research among smaller groups. As well, he has established a chronology of epidemics among these peoples between 1738 and 1838, which is useful for researchers studying disease episodes among nearby groups. A special issue of the *Plains Anthropologist* (Campbell 1989) dealt with issues of demography and health among the Plains Indians, and several papers relate to the people of the Northern Plains.

[27]Researchers have tended to ignore the later periods in the St. Lawrence and Upper Great Lakes regions, particularly the role played by settlers in the introduction of *ACIs*, in favour of the earlier epidemics. Peter Harstad's (1959-60; 1960a; 1960b; 1963) work sheds light on some of the later patterns of health and disease in the frontier communities of the Upper Mississippi River and Wisconsin areas. Ignored by most researchers, these publications provide another perspective on the introduction of Old World diseases among the aboriginal people of the Upper Great Lakes and Upper Mississippi. There have also been several studies of the diffusion and effects of cholera, although with little concern for the plight of the native people (e.g. Pyle 1969; Rosenberg 1962; Wylie 1983). Another theme has been the effect of *ACIs* on warfare in the Great Lakes-St. Lawrence area, both in the seventeenth and eighteenth centuries (Johnston 1982; 1987; Schlesier 1976; MacLeod 1992).

[28]Snow (1992; Snow and Lanphear 1989; Snow and Starna 1989) has taken issue with Dobyns's conclusions, while Ramenofsky (1987) attempted to verify them as part of her study of archaeological indicators of protohistoric epidemic disease.

Galois (1996), Cole Harris (1994; 1997-98) and, especially, Robert Boyd (1985; 1992; 1994a; 1995b; 1996; In Press) has gone far to clarify the disease history of the Pacific Northwest from the late eighteenth century onward.

These studies of other regions and peoples are of great benefit to the present study for several reasons. In identifying major epidemics they provide an opportunity to correlate disease episodes between regions in order to understand the broader patterns of their diffusion. Such inventories also assist in attempting to identify the nature of the diseases that struck the Petit Nord. Moreover, several of the studies identify critical mechanisms of diffusion that played an important role in either introducing *ACIs* to the Petit Nord, or in aiding in their spread within the region. Finally, by comparing the epidemic histories of different regions it becomes possible to better understand the processes and transformations that affected the presence of exogenous diseases in the Petit Nord.

The disease literature of the Petit Nord

Despite the tremendous growth in research examining the epidemic history of the New World in recent years, this trend has not held true for research in the Petit Nord. Instead, there has been little more than a handful of disease-centred studies probing the historical patterns of health of the people of this region, and very few have discussed the diffusion of these diseases. As with the overall North American literature, however, these contributions have been made by scholars from several different fields. Here again, Ray was a pioneer in this respect, following up on his earlier research on the impact of

epidemic disease on the Indians living to the west of Lake Winnipeg (Ray 1988, but first published in 1974; 1975). His article, "Diffusion of Diseases in the Western Interior of Canada, 1830-1850", looked at the spread of several major epidemics over a wide area that included the far western portion of the Petit Nord and part of the Boundary Waters (Ray 1976). Ray's paper is noteworthy for several reasons. First, it set a standard for further work by emphasising the need for a sound ethnohistorical background for such studies. Secondly, it identified many of the major characteristics of epidemic diffusion in the Northwest, including the importance of the HBC fur brigades and the Red River Settlement. Finally, most of its study period has escaped the attention of subsequent researchers, even though it was a critical one for the Northwest.

T. Kue Young, a medical specialist in community health whose study area encompassed much of the Petit Nord, followed Ray. Both in his early paper (Young 1979) and in his book (Young 1991, but first published in 1988), he provided an historical context for the present-day health patterns of the Cree and Ojibway, which topic was his primary concern. His summary of pre- twentieth century epidemic activity was sketchy, however, touching only briefly upon a few episodes in order to illustrate the severity and timing of such diseases. This does little to detract from his overall achievement, but nonetheless demonstrates the need for further detailed historical studies in order to understand the present situation (Waldram 1989: 280).

In 1983, two other researchers made contributions to the understanding of the epidemic history of the Petit Nord. Another medical doctor, William Ewart (1983),

conducted detailed archival research in order to outline the major causes of mortality at York Factory during the period 1714-1946. Ewart collated the causes of death during this period into several categories and presented the totals in a brief paper. He found that most deaths (for which the cause of death was noted) were due to infectious disease, with tuberculosis and influenza the most common causal categories noted in the archival records. In so doing, he identified only a very few specific disease episodes and said nothing about their diffusion.

That same year, Marshall Hurlich (1983), an anthropologist, wrote an ambitious paper focussing on the demographic history of the Algonquian people living within an area that is roughly coterminous with the Petit Nord. In doing so he chose a study period from the earliest observations by Europeans to the present. Of interest to the present study are his sections on disease and the fur trade (Ibid.: 151-173). Hurlich assembled a lengthy list of epidemics that affected the people of the Petit Nord and those living in surrounding regions in support of his thesis that variations in the frequency of exposure to epidemics between different groups led to differences among them in terms of dependency. However, the list of episodes was derived from secondary sources, excepting a few published primary documents, and he made no meaningful analysis of the epidemics themselves. In the end, despite the long list of epidemics, it is unclear how many of these sicknesses affected the people of the Petit Nord, how severe they were, how they spread into the region or how extensive was their diffusion within it. Without

this specific knowledge, his theory cannot be tested.

Four years after Hurlich's work, two general maps referring to epidemic disease partly in the Petit Nord were published in different historical atlases. One was a map of epidemics among the Indians of the Great Lakes during the period 1630-1880 (Tanner 1987: Map 32). However, this plate noted only four epidemics in the southern part of the Petit Nord during this entire period. Clearly this was an understatement of the presence of epidemic disease in this area. Also in 1987, Conrad Heidenreich authored a plate as part of Volume I of the *Historical Atlas of Canada*, showing the presumed diffusion of five major epidemics in eastern North America occurring between 1634 and 1650 (Heidenreich 1987b). These were devastating to the eastern Indians and spread widely in the country between Lake Huron and the Atlantic Ocean. Heidenreich concluded that four of these epidemics reached the margins of the Petit Nord, and speculated that two spread into the Lake Superior area.

Jody Decker conducted historical epidemiological research on native peoples who lived in a large part of the Northwest, including the westernmost part of the Petit Nord. In an early paper Decker (1988) examined the diffusion of the 1780-82 (her dates) smallpox epidemic to York Factory as a case study for a suggested epidemiologically informed methodology used to study the spread of historical *ACIs*. This was succeeded by a study of scurvy at York Factory (Decker 1989a) and her dissertation (Decker 1989b). The latter followed on Ray's work, and examined the diffusion and effects of several epidemics between 1774 and 1839. Decker's work leaves much room for additional

research, however. Her study area encompassed only a small portion of the Petit Nord, essentially along the narrow corridor that formed the western margins of the region between Rainy Lake and York Factory, and as far as the Severn River in the north. As well, she examined only a few of the total number of epidemics that struck the region. Her work covered only eight episodes, and one of these (the 1837-38 smallpox epidemic) did not reach the Petit Nord. There were many other epidemics that struck the region, although perhaps not with the impact of those she chose to study. Equally important, her study period does not include a century of documented epidemic activity in the Petit Nord prior to 1774. By the same token, the seven years after 1839 were critical ones in the region, and by studying this period the main regional patterns of diffusion become far clearer. Finally, in focussing on the demographic impact of the epidemics, she did not consider all of the available evidence surrounding their spread, and so some of her conclusions are open to reinterpretation with further research.

Decker's dissertation was followed by my master's thesis, which documented the diffusion of the 1819-20 measles and whooping cough epidemics within the entire Petit Nord (Hackett 1991). This study attempted to identify the origins and diffusion of these epidemics. It concluded that both spread from the east of the Petit Nord along separate paths, one through Lake Superior and one to the south of it and through the Upper Missouri. It also described their limited penetration into the region.

Finally, anthropologist Anne Herring has examined the devastating 1918-19 influenza epidemic as it struck the central subarctic, particularly at Norway House

(Herring 1993a; 1993b). As with Young's publications, her work illustrates the fact that the era of significant epidemics of *ACIs* in the Northwest did not end with midpoint of the nineteenth century, nor even with the turn of the century.

Until recently, scholars studying the history and ethnohistory of the people of the Petit Nord have largely ignored *ACIs* as a significant factor in the lives of these people. Thus, anthropologists who undertook some of the earliest ethnohistorical research, such as Ed Rogers and Charles Bishop, tended to downplay or ignore the importance of epidemic disease, including *ACIs*. For instance, Rogers doubted that epidemics had any effect on native settlement patterns in the region (Rogers 1963: 66). In later works he noted only that they "brought starvation as the normally able-bodied were rendered unable to hunt, fish, and provide the other necessities of life during the course of the disease" (Helm, Rogers and Smith 1981: 148; Rogers 1983). Bishop acknowledged the presence of several epidemics among the Northern Ojibway of the Petit Nord but suggested that such afflictions served only to counterbalance their natural increase (Bishop 1974a: 162, 267, 320, 331; see also Bishop 1972: 69; 1974b: 154; 1978). When confronted with a period of sustained population decline, such as occurred at Osnaburgh House between 1830 and 1858, Bishop explained it as a function of changes in post preference (Bishop 1974a: 157), discounting the frequent references to epidemic *ACIs* in the primary documents. However, he seems to have subsequently modified his views somewhat when pressed by a growing disease literature. In his critique of Calvin Martin's *Keepers of the Game*, Bishop (1981a: 42, 45) accepted the possibility of the

protohistoric spread of introduced *ACIs* and admitted that they may have had an effect on the natives' organisation and ecological relationships.

As new generations of ethnohistorians studying the Petit Nord have emerged, *ACIs* have gained in prominence. Thus, for instance, both Leo Waisberg (1984: 267) and Joan Lovisek (1993) have briefly identified some of the demographic consequences of epidemic disease in the Boundary Waters. Outside of the disease-centred studies, Victor Lytwyn has made the most significant contribution to our knowledge of the historical epidemiological patterns of the Petit Nord. This came in his PhD dissertation, in which he discussed at length the impact of diseases on the Lowland Cree during the period 1713-1821[29] (Lytwyn 1993: 353-373, 415-419). As part of this far-reaching and valuable study, Lytwyn traced the diffusion of the 1779-83 epidemic within the Petit Nord. As well, he identified many other outbreaks and lesser epidemics in the Hudson Bay Lowland prior to 1821, some of which had never previously been documented in the secondary literature. While Lytwyn's contribution is a significant one, there remains a need for additional historical epidemiological research on the Petit Nord. The limits to his study area and study period, the lack of a disease-specific emphasis and the limited analysis of diffusion all reinforce the need for further research. Thus, the present study will attempt to fill these and other gaps in the literature of the Petit Nord.

[29]Lytwyn also commented upon the 1779-83 smallpox and 1819-20 measles epidemics in his earlier published study, *The Fur Trade of the Little North* (Lytwyn 1986: 44, 155).

CHAPTER 3: PROTOHISTORIC *ACIs* AND THE PETIT NORD

The historical period began in the Petit Nord during the 1670s, with the initiation of a sustained fur trade in the region. Shortly thereafter, we find the first evidence of epidemic disease among its people. Nevertheless, it is possible that *ACIs* had penetrated as far as the Petit Nord prior to the beginning of historical documentation. Despite the fact that there can be no definitive answer due to the absence of supporting or contrary evidence, it is worthwhile to consider the possibility that epidemic *ACIs* affected the people of the Petit Nord during the protohistoric period. This possibility began with the initial introduction of Old World diseases to the Western Hemisphere in the sixteenth century and increased substantially during the seventeenth century. This chapter will examine the situation surrounding the introduction and spread of *ACIs* in the North America prior to the 1670s, as well as the possibility that these diseases spread to the Petit Nord.

The sixteenth century

There has been a tendency on the part of many researchers to equate contact with disease transmission, leading them to speculate that Old World diseases were introduced to the aboriginal people of the Western Hemisphere from the first meetings, no matter how fleeting[1] (Henige 1998: Chapter 11). This argument is often raised for early epidemics among the aboriginal people of northeastern North America. For instance,

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[1]Generally, this has extended only to the post-Columbian contacts. For whatever reason, few scholars have considered the possibility that the Norse settlers who arrived in Newfoundland centuries before Columbus also introduced disease. Carlson et al. (1992: 145) suggested that the Norse were too isolated and their contacts with the Indians were too limited to facilitate disease transfer.

Carlson et al. (1992: 145, 146-147) concluded that contacts between the natives and untold numbers of fishermen, explorers and traders led, at the very least, to repeated localised epidemics of Old World diseases among the Indians, perhaps as early as the 1480s (See also Jaenen 1976: 99; Martin 1982: 42-43, 51; cf. Cook 1973b: 486). If present among the eastern Indians, it is also possible that these afflictions travelled inland towards the Petit Nord.

For the most part, there is no direct evidence to support such conjecture. However, one oft-cited statement by a Jesuit missionary, Father Pierre Biard, has been interpreted to suggest that acute infectious diseases were being passed from an early date. In 1616 he noted that the Micmac:

> are astonished and often complain that, since the French mingle with and carry on trade with them, they are dying fast, and the population is thinning out. For they assert that, before this association and intercourse, all their countries were very populous, and they tell how one by one the different coasts, according as they have begun to traffic with us, have been more reduced by disease. (Thwaites 1959: III: 105)

Dobyns (1989b: 291-292) has used this passage in support of his belief in the direct introduction of contagious disorders by European sailors and traders during the sixteenth century. He implied that acute infectious diseases accompanied and were introduced by many who visited or traded with the Atlantic coastal peoples, and that they subsequently spread to the native groups of the "Great Lakes-Laurentian cultural area" (Ibid.: 292). Miller (1976) examined the early population history of the Micmac in detail, and came up with a different interpretation of this passage, one that rings true. By examining the context of the quote, and by bringing additional documentary evidence to play, she

concluded that growing reliance on European foodstuffs, foods that were often spoiled, and overindulgence in alcohol led to severe population decline among the Micmac. Indeed, there is no reason not to believe that the afflictions reported among these people (dysentery, lung, chest and intestinal diseases) were related to either dietary change, chronic infections introduced by the Europeans (e.g. tuberculosis, dysentery or intestinal parasites) or were indigenous disease exacerbated by nutritional problems. In any event, the fact that the Micmac suffered such mortality only after experiencing direct and sustained contact with the French argues strongly against the presence of waves of epidemics sweeping the interior from these contacts.

Unquestioned belief in the early transfer of the full range of acute infectious diseases fails to take into account the logistics of trans-oceanic disease diffusion during that period, at least with respect to the Old World diseases that are thought to have done most of the damage in the Americas. Snow (1980: 32-33; Snow and Lanphear 1988: 25-26) argued that it is unlikely that crowd diseases were introduced to New England prior to the seventeenth century. He based this on the belief that the early crews were too small (and contained too few susceptibles) and the pre-1600 voyages too lengthy to enable a crowd disease to survive the crossing. This does not necessarily rule out the possibility that chronic or vectored afflictions, or perhaps even *ACIs* such as influenza or colds, arrived in this manner[2]. Nevertheless, it suggests that the situation was very much against the direct importation of diseases of childhood such as smallpox and measles from

[2]Indeed, Snow and Lanphear (1989: 302) acknowledged the probability that colds and influenza were transmitted to the Indians, but that such afflictions remained localised around the point of introduction rather than becoming pandemics.

Europe to the north Atlantic coast of North America prior to the seventeenth century.

Similar arguments of contact equalling contagion have been raised for the introduction of *ACIs* to the more southern parts of the New World, beginning with Columbus's first voyage. However, there is no adequate evidence to suggest this[3]. Nevertheless, Old World diseases were early to reach Mesoamerica compared to the northern part of North America. Smallpox arrived at Santo Domingo in late 1518 or early 1519 and spread to Mexico in 1520, causing tremendous mortalities (Cook and Lovell 1992b: 219; Crosby 1972: 39). Thereafter, *ACIs* and other Old World diseases were an increasingly frequent threat in Mexico, Central America and the northern part of South America. The most significant difference between the northern and southern experiences prior to the seventeenth century was the number of susceptibles making the trip from the Old World. During the sixteenth century large numbers of slaves were brought from Africa to the Spanish colonies. These people were much more likely to have been susceptible to *ACIs* than the few adult Europeans who sailed to the north (Snow and Lanphear 1986: 26). Once this migration began in earnest, these diseases became relatively common in Mesoamerica, despite attempts at quarantine[4] (Ashburn 1947: 29, 32, 39; Cook and Lovell 1992b: 218).

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[3]The timing of the first epidemic is controversial. It has been claimed that *ACIs* were transmitted to the Indians as early as 1492 (Henige 1998: 170). Guerra (1988) argued that influenza struck the Indians of Hispaniola, having been introduced from Columbus's ships during his second voyage in 1493. Henige (1998: 169), however, noted that there is no evidence that the Indians suffered from such a disease at this time. Cf. Lovell (1992b: 428).

[4]Alden and Miller (1987) documented the relatively frequent introduction of smallpox to Brazil via the slave trade between 1560 and 1831, mainly from Africa.

Following the 1520 epidemic, *ACIs* began to appear in Mesoamerica with increasing frequency. Recent research indicates that severe epidemics struck Central Mexico on many occasions during the sixteenth century (Prem 1992). As well, according to Prem (Ibid.: 45-46), many diseases became endemic after initial introduction in Central Mexico, and thereafter flared up frequently as outbreaks affecting mainly children. Similar patterns of disease introduction appear to have occurred wherever the Spanish established bases of operation in the New World[5]. The frequent reintroduction of *ACIs* from Europe and elsewhere and their circulation through colonial Spanish territories, combined with the eventual shift to endemicity in some more populous areas, meant that Mesoamerica quickly developed into a potent disease pool. Once established, this pool carried the potential to infect the entire hemisphere.

The degree to which *ACIs* spread from Mesoamerica during the sixteenth century has been a contentious point in the recent literature. As noted, Dobyns proposed that the earliest Old World epidemics to reach the Americas spread long distances from their place of entry in Mesoamerica, including as far as the Petit Nord. For instance, he concluded that the initial 1520 smallpox epidemic spread across the present U.S., and "through all of the most densely populated portions of the Americas," probably reaching

[5]For example, at least twenty-five localised and eight widespread epidemics occurred in Guatemala between 1519-20 and 1632 (Lovell 1992a; 1992b). Similarly, Newson (1992) noted many epidemics that may have occurred in Ecuador during the sixteenth century. Alchon (1992: 159) concluded that during the sixteenth century epidemics of smallpox, measles and typhus occurred about every ten years in Quito, or slightly less frequently than under endemic conditions.

"the no man's land between Indians and Inuit[6]" (Dobyns 1983: 13, 15; 1989a: 172; 1989b: 294). Subsequent epidemics were said to have diffused similarly great distances[7]. As part of this theory, Dobyns (1983: 324) hypothesised that there was a system of diffusion that carried *ACIs* from Florida northward to the peoples of New England, before heading inland to the Iroquois tribes of the Great Lakes. In his view, "the entire Atlantic coastal region from Florida to the [Great] lakes and New England... constituted what may be termed one "epidemic region"" (*Loc. Cit.*). This led him to conclude that an Old World disease introduced among any people living within this region would likely spread throughout. Given his belief in the spread of pandemics from Mesoamerica to Florida during the sixteenth century, such a system would have had enormous consequences for the people of the Great Lakes and, therefore, those of the Petit Nord.

The evidence for this system is far from conclusive, however, and has been contested by other, more circumspect, researchers. It rests largely upon an unsubstantiated connection between a speculative inventory of protohistoric epidemics among the Indians of Florida, a statement from the 1630s that the Narragansett Indians of New England had experienced earlier epidemics in conjunction with a series of

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[6]This limit is based on the rather spurious assumption that the epidemic had to have spread as far north as it did south. There are fundamental problems with such an assumption. One is that it ignores any differences in aboriginal population density or interactions between the area to the south of Mexico and that to the north, as well as the effect of chance. Moreover, the evidence for smallpox in central Chile at this time is not convincing (Henige 1998: 124), and so Dobyns's estimate of northward diffusion based on a principal of uniformitarianism is similarly suspect. Finally, Snow and Lanphear (1988) have argued that there were buffer zones between tribes that would have stymied the northward diffusion over long distances, and Dobyns's system does not take them into account. Dobyns (1989b: 292) either misunderstood or misrepresented Snow and Lanphear's buffer zones to mean areas of non-interaction between the Indians and the whites.

earthquakes and archaeological evidence of settlement shifts by the Seneca, an Iroquoian tribe living in present-day New York State. The Florida epidemic inventory has been questioned by several scholars, especially as it relates to Dobyns's use of evidence (e.g. Thornton et al. 1992; Henige 1986a; 1986b; 1998). Of considerable interest to the present study is the possibility that early epidemics spread to the New England tribes and the Iroquois, groups with whom the native peoples of the Petit Nord had either direct or indirect communication[8].

There are little data supporting possible sixteenth century epidemics among the New England and other Atlantic coastal tribes, either from Mesoamerica or, as noted above, directly from Europe. The strongest is testimony collected by Roger Williams in the late 1630s and attributed to the Narragansett Indians (Dobyns 1983: 318). According to Williams's informants, these people had been struck by four earthquakes, in about 1568, 1574, 1584 and 1592, and each was associated with an epidemic (Snow and Lanphear 1988:20). None of these diseases was described or identified. By adjusting the dates, Dobyns (1983: 318-319) suggested that three of these were part of pandemics that spread from Florida. Snow and Lanphear (1988: 21) questioned whether the origin of the statement reflected aboriginal concepts of disease etiology and, more to the point, presented evidence suggesting that the Indians of New England remained numerous until a devastating epidemic struck them early in the seventeenth century. The fact that

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[7]Including, for example, supposed pandemics of plague in 1548, "fever" in 1585-86 and measles in 1596-97 (Dobyns 1983: 316-317).

[8]The surmised Florida epidemics are important here only if they spread northward towards the Petit Nord.

Dobyns failed to consider the possibility that these epidemics were of diseases that were aboriginal to the Americas is also a significant limitation to his conclusions. In the end, although interesting, this testimony does little to clarify the case for the existence of widespread epidemic disease in this area during the sixteenth century.

Dobyns's evidence for sixteenth century pandemics affecting the Seneca of New York State (and hence within the interior) is less compelling. He equated signs of village abandonment and subsequent resettlement of the Seneca with the presence of Old World disease (Dobyns 1983: 313-325). Drawing upon published archaeological studies, he theorised that these relocations were the result of a succession of severe epidemics, and attempted to link them to his pandemic chronology. However, there are crucial flaws in his argument. Such movements occurred for many other reasons among nearby groups such as the Mohawk and the Huron (Snow and Lanphear 1988: 21; Snow and Starna 1989: 145; Trigger 1987: 36, 147). As well, no precise date can be assigned to these relocations, rendering it impossible to correlate them with supposed epidemics (Snow and Lanphear 1988: 21). Moreover, village abandonment occurred prior to the arrival of Columbus, and so cannot simply be assumed to have been a response to the introduction of Old World disease.

Other research from the interior fails to lend support to Dobyns's theory. Ramenofsky (1987) attempted to verify the presence of protohistoric Old World epidemics among the Iroquois of New York State using indirect archaeological indicators of population levels. She was able to confirm the seventeenth century afflictions, which are documented in the historical record, but there was no evidence suggesting that such

epidemics also occurred during the previous century (Ibid.: 100-101). Snow and Starna (1989), who undertook archaeological work in the Mohawk Valley of New York, concluded that "the Mohawk nation was not significantly affected by epidemics prior to 1633" (Ibid.: 144, 147). Similarly, Trigger (1981: 22; 1987: xxxi), who has done much to clarify the ethnohistory of the nearby Huron, could find no evidence of epidemics among these people prior to the seventeenth century, despite prior penetration of European trade goods into Southern Ontario as early as the 1530s. The case of the Huron is significant to the present study for two reasons. The first is that these people provided much information about themselves to the Jesuit priests and French explorers and never mentioned that they had been decimated by epidemic disease prior to the 1630s. The other is that the Huron were closely linked by trade to the people of Lake Superior, and so evidence of early epidemics among them might suggest similar afflictions within the Petit Nord.

There is no evidence of widespread epidemics in northeastern North America during the sixteenth century but there is a single description of what appears to have been a more localised outbreak of some unidentified and deadly disease. In December of 1535 the St. Lawrence, or Laurentian, Iroquois who lived at Stadacona suffered from an unknown disorder which resulted in the deaths of more than fifty of their people (Biggar 1924: 204; Trigger 1987: 194). According to Cartier,

> In the month of December we received warning that the pestilence [*la mortalité*] had been out among the people of Stadacona to such an extent, that already, by their own confession, more than fifty persons were dead. Upon this we forbade them to come either to the fort or about us. But notwithstanding we had driven them away, the sickness broke out among

> us accompanied by most marvellous and extraordinary symptoms.... (Biggar 1924: 204)

Cartier's description of those symptoms makes it clear that the French were suffering from scurvy. The illness of the Iroquois was no doubt caused by something else, given that they knew how to treat scurvy before it became life threatening.

Dobyns (1983: 314) referred to this evidence, whereby he claimed that the French had given it to the Stadaconans. Although it is possible that a disease was transmitted from Cartier's men to the Indians (Snow and Lanphear 1988: 18), the original text suggests the opposite (Henige 1986: 302). Given what is now known about the aboriginal disease load of the Iroquois (Saunders et al. 1992; Ramenofsky 1987: 98), it could have been an epidemic of an indigenous disorder, rather than one of an Old World disorder. Moreover, there is also no indication that it spread beyond these people. Finally, Dobyns (1989b: 289) tried to connect this episode with the subsequent disappearance of the St. Lawrence Iroquois, who vacated the valley between 1542 and 1603[9] (Trigger 1987: 214). He stated that:

> The disappearance of the Laurentian Iroquois implies either that the 1535 epidemic so weakened them that they migrated not long after Cartier sailed for France, or that later sixteenth-century disease so weakened them that they moved from the river valley... (Dobyns 1989b: 289)

Here, Dobyns would consider no explanation other than Old World disease, demonstrating a preoccupation with a single causative factor or interpretation in almost every situation he encounters (Henige 1989: 306). Once again there are many possible

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[9]As well as connecting it to an epidemic in New Spain (Dobyns 1983: 314).

explanations for the movement of the St. Lawrence Iroquois, and several are far more satisfactory than hypothetical pandemics[10] (Snow and Lanphear 1989: 302; Henige 1989: 306; Trigger 1987: 194-195, 214-224).

Attempts to verify Dobyns's theory have extended to include that part of North America lying to the west of the Great Lakes. Ramenofsky (1987) also examined the archaeological record of the Upper Missouri for evidence of disease-induced change. Here, the record was ambiguous regarding sixteenth century episodes, but she found indirect evidence of population decline during the mid to late seventeenth century[11] (Ibid.: 124, 134). By that time *ACIs* had been present in the east for several decades, and by mid century critical disruptions to the eastern aboriginal groups caused by the fall of Huronia may have fostered the westward diffusion of epidemic disease. As such, this conclusion is not inconsistent with epidemics spreading from the east, rather than Mesoamerica.

There is some interesting, although by no means definitive, evidence suggesting that Old World diseases did not appear so early among at least one of the peoples living west of Lake Superior. Oral testimony by the Arikara living on the Upper Missouri indicates that one *ACI*, smallpox, had appeared only rarely among them up to the late eighteenth century. In 1794-95 the fur trader Jean Baptiste Truteau travelled to the

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[10]Henige (1989: 307) listed warfare, land exhaustion, climatic pressure and cultural practices as possible factors in the disappearance of the St. Lawrence Iroquois.

[11]Nevertheless, Trimble (1989: 42, 48) accepted Dobyns's hypothesis of sixteenth century pandemic epidemics spreading to the Northern Plains.

Arikara and, through conversation with them, was informed that these people had suffered from smallpox epidemics on three occasions prior to that time (Nasatir 1952: 299). Both Dobyns (1983: 310) and Ramenofsky (1987: 130) have referred to this statement in support of protohistoric epidemics. Without doubt, one of these was the 1779-83 smallpox epidemic that devastated many of the plains groups, including the Arikara (Trimble 1985: 75). However, there are two other eighteenth century epidemics that may very well have struck the Arikara. Taylor (1977: 79) suggested that epidemics of smallpox or a smallpox-like[12] disease struck the Arikara in the 1730s and the early 1750s and, as will be shown below, there is good reason to believe that this was the case. These would have rounded out the three epidemics that the Arikara claimed had been among them, with the first as late as the 1730s. In effect, evidence presented by proponents of early pandemics runs contrary to that proposition, at least with regard to smallpox.

As did Ramenofsky, Sarah Campbell (1990) attempted to obtain evidence of early protohistoric epidemics in North America, this time among the people of the Northern Columbia Plateau in the Pacific Northwest. Turning once again to the archaeological record, she concluded that the surrogate indicators that she had examined support a massive population decline in 1520, followed by a subsequent period of continuous population recovery until the late eighteenth century (Campbell 1990: 186-187, 190; cf. Boyd 1996a). Had this pattern been accurate, this would have been powerful evidence in

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[12]The second epidemic may have been measles rather than smallpox. However, it is improbable that the Arikara could have distinguished between smallpox and several other *ACIs*, including measles.

support of Dobyns's sixteenth century continental pandemics. There were fundamental errors in Campbell's analysis, however, errors that render her conclusions dubious (Henige 1998: 158-161). Most important, her data suggested a considerable decline in aboriginal population between 1375 and 1425, or long before the arrival of Columbus. Choosing to ignore this decline, she instead focussed on a smaller decline in some surrogates between 1475 and 1520, and claimed that her data indicated that the depopulation occurred in about 1520, as part of Dobyns's smallpox pandemic (*Loc. Cit.*). As elsewhere in northern North America, then, there is no reliable evidence for the extension of hemispheric pandemics from Mesoamerica to the Columbia Plateau during the sixteenth century. For the Petit Nord, a more probable period of protohistoric epidemics came during the following century.

The seventeenth century

Conditions changed significantly during the seventeenth century, greatly increasing the potential for direct introduction of *ACIs* to northeastern North America. After 1600 the length of a voyage from Europe was shortened from an average of six weeks to four and, as the century progressed, significant numbers of Europeans began to settle in newly founded colonies along the north Atlantic coast. This was especially so following the establishment of Plymouth in 1620 as part of the Massachusetts Bay Colony[13] (Snow and Lanphear 1988: 25-26). During the 1630s there began a mass

[13]There were a few attempts at colonisation in North America during the sixteenth century but most proved short-lived. A Portugese colony on Cape Breton Island during the 1520s and one settled by the French near Quebec in 1541 each lasted only about two years or less (Morison 1971: 228-229; Trigger 1989: 124-125, 134). The Spanish were more successful in Florida, including the establishment of St.

migration into New England including, notable as a vehicle for the transportation of crowd diseases, large numbers of children (*Loc. Cit.*). *ACIs* accompanied this migration from the outset (e.g. Duffy 1953: 43). Similar growth occurred in New France and New Netherland as well as in the other English colonies, leading to the importation of infectious diseases. At the same time, slave ships also became a significant factor in the introduction of *ACIs* to the English colonies as they had long been in Mesoamerica (Snow and Lanphear 1988: 26; Duffy 1951a: 29-30; 1953: 48). These changes led directly to an era of unprecedented epidemic activity in northeastern North America, which had become intimately connected with the disease pools of the Old World.

A common feature of the epidemics that spread in this region throughout most of the seventeenth and eighteenth centuries is that they began with the arrival of *ACIs* aboard ships from overseas sources. Unlike in Mesoamerica, these diseases did not become endemic in the northeastern colonies until over a century and a half after their initial introduction, or only beginning in the late eighteenth century. Instead, they were constantly reintroduced by ships from Europe, Africa and the West Indies (e.g. Duffy 1953: 14, 19, 43, 57, 60, 104, 137; 1951: 30; Jaenen 1976: 98; Caulfield 1943: 534, 539, 549; Creighton 1965: 2: 486), and therefore either directly from major disease pools or

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Augustine in 1566. The English first attempted to colonize North America with Roanoke in the 1580s, but were thwarted within a few years. There were reports of sickness among some of the native groups neighbouring Roanoke (Henige 1998: 150-151). The period of sustained colonization in the northern part of North America began in the early seventeenth century. Settlements at Jamestown (1607) and Quebec (1608) were followed by the arrival of the pilgrims at Plymouth in 1620 and the Dutch at New Amsterdam (New York) in 1624. There appears to have been epidemic disease among the people of Virginia from the start. Typhus, and possibly plague, may have been rampant on the ships sailing for Jamestown in 1609 (Creighton 1965: 1: 610). Earle (1992: 482; 1979) concluded that dysentery and possibly typhoid fever occurred almost annually in summertime epidemics among the Jamestown colonists between 1607 and 1624. Initially, Old World diseases were probably more often carried to Virginia than to New England

places in close contact with those pools[14]. Infectious disease was commonplace on these early transatlantic voyages, and while typhus, typhoid and dysentery were probably the greatest threats, *ACIs* such as smallpox and measles were also frequently present (Duffy 1951a; Caulfield 1943; Cook 1973b: 491). Because of the key role played by these ships in the introduction of Old World diseases, main ports including Boston, New York, Quebec and Charleston were vulnerable to repeated outbreaks of *ACIs*[15].

An important aspect of the general pattern of *ACI* diffusion during the seventeenth century is that they often passed from their initial point of introduction in the major ports to other non-native communities. Thus, from an early date epidemics raced back and forth between the colonies. For example, in 1634 smallpox diffused from the Dutch to the English, while in 1646 an influenza epidemic appeared among the French, Dutch, and English (Duffy 1953: 43-44; Creighton 1965: 1: 613). A smallpox epidemic in Boston in 1666 may have come from Canada (Duffy 1953: 45). Likewise, measles appears to have spread from Quebec to New England in 1687-88 (Caulfield 1943: 533). Other diseases spread from the English to Canada, a pattern that seems to have been more common[16].

(Creighton 1965: 1: 612), but this situation was reversed after 1630.

[14]This process began in earnest in the 1630s, but initially it may have been more common in New Netherland and the English colonies than in New France. In his *Relation* of 1647-48, the Jesuit missionary Jerome Lalement stated that "It seldom happens that sickness breaks out in the ships that come to the country..." (Thwaites 1959: XXXII: 133). French ships brought smallpox in 1639 (Marshall 1967: 18) and carried unnamed but deadly diseases in 1640 and 1648 (Thwaites 1959: VIII: 309; XXXII: 133; XXXIII: 19; LXIX: 47). However, the incidence of such introduction seems to have increased after 1650.

[15]Detailed discussions of *ACIs* in Canada and the U.S. during the colonial period can be found in Heagerty (1927), Caulfield (1943), Stearn and Stearn (1945) and Duffy (1953). See also Creighton (1965).

[16]Duffy (1953: 69) noted that the normal course of diffusion was northwest from New York into the Great Lakes region and the St. Lawrence Valley, but that epidemics also spread southward from Canada to the middle and southern English colonies.

Often, it was the Indians who served as middlemen in the diffusion between colonies, especially between the French and English (Duffy 1953: 69, 104). Such inter-colonial diffusion extended the stay of *ACIs* in the broader region, making it more likely that they would find a suitable mechanism of transport into the interior. Although *ACIs* were not endemic in northeastern North America at this time, the region nonetheless served as a temporary pool in which the diseases of more densely settled communities circulated prior to diffusion to the limits of the disease frontier[17]. Critically, this continual circulation also meant that they were carried from communities with strong external connections but where there was less contact with the Indians, such as Boston, to frontier communities where the interaction between whites and natives was routine.

These exogenous diseases spread to the aboriginal people of northeastern North America, including those living within the interior. In this way, *ACIs* that were endemic in distant pools were carried ever closer to the Petit Nord. Some researchers have blamed non-natives for the diffusion of the epidemics into the interior. For instance, Jaenen (1976: 98) stated that they were carried into the hinterland of New France by missionaries, traders and soldiers. However, more significant as a vehicle for the westward diffusion of *ACIs* during the seventeenth century was the large numbers of natives who visited places like Quebec, Fort Orange (Albany, New York), Trois Rivières and Montreal for trade or political negotiations. Certainly this was the case for the earliest round of epidemics in Canada during the 1630s, when native movements carried

[17]According to Cook (1973b: 493), following the 1630s smallpox was always present in eastern North America. In general it existed at low levels but periodically it would flare up into epidemics.

ACIs from the east towards the Upper Great Lakes or saw these people die in the vicinity of the French settlements[18] (Trigger 1987: 501, 526, 528, 588).

The period between 1600 and 1670 can be divided into three sub-periods. Between 1600 and about 1630 epidemic activity appears to have been limited in northeastern North America. It increased substantially thereafter, and *ACIs* began to spread far into the interior from the coastal colonies, at least to the doorstep of the Petit Nord. For the most part, these epidemics followed long-established lines of communication until 1649. In that year the Iroquois destroyed their Huron rivals. Thereafter followed a period of massive disruption and dislocation of aboriginal populations, as well as fundamental shifts in the patterns of native movements. These greatly affected at least the patterns of diffusion into the interior, if not also the frequency of these epidemics.

There is very little evidence for epidemics in either the Atlantic coastal area or in the interior between 1600 and about 1630. This may be due in part to a lack of documentary evidence. Nevertheless, it is also probable that Old World diseases were extremely rare in this part of North America at this time. Two outbreaks that have been identified occurred in the interior, not too distant from the Petit Nord. In 1611 many Algonkin perished due to an undifferentiated fever (Biggar 1925: 2: 207). At this time the Algonkin lived along the Ottawa and upper St. Lawrence Rivers (Trigger 1987: 279).

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[18]The primary records of the seventeenth and early eighteenth centuries contain many descriptions of epidemics spreading into the interior through native movements. Trois Rivières, a major French fur trading centre along the St. Lawrence, was an especially important locus of diffusion (Heagerty 1928: 1: 32).

Twelve years later, many of the Weskerini, an Algonquian-speaking group who lived just below Allumettes Island on the Ottawa River, died of disease and hunger (Ibid.: 499, 279). As Trigger (*Loc. Cit.*) rightly pointed out, there is nothing to suggest that either outbreak was of an Old World disease. The first widespread epidemic in northeastern North America occurred along the coast of New England, between 1616 and approximately 1619, and did not approach the Petit Nord[19]. This devastating episode caused extremely high levels of mortality, but has never been identified conclusively. Various diagnoses have been made, including smallpox, influenza, yellow fever and trichinosis (Spiess and Spiess 1987). The most likely is bubonic plague (Williams 1909), although a more recent treatment of the subject by Spiess and Spiess (1987) suggested hepatitis. It failed to spread far inland, and was also relatively constrained in its coastwise diffusion, being limited to parts of present-day Massachusetts, Maine and Rhode Island (Williams 1909).

The period following 1630 began a new era of epidemic activity in the northeastern part of the continent. Thereafter, Old World diseases routinely appeared in the eastern colonies and often among the Indians. Of particular concern is a series of four epidemics that devastated the east between 1634 and 1639, epidemics that spread at least as far to the west as Georgian Bay on Lake Huron. In 1634-35 an unidentified rash-producing disease spread westward through the St. Lawrence and Ottawa River valleys, probably by the movement of native traders. This disease reached the Huron during the

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[19]The dates of this epidemic are not certain, and various scholars have accepted end years ranging from 1619 to 1622.

summer (Trigger 1989: 230). Smallpox was active among the Iroquois and other eastern tribes at about this time, probably introduced by the English (Cook 1973b: 491; Snow 1992: 178), but it is more likely that this was another disorder such as measles that had been brought by the ships from France[20] (Trigger 1989: 230). Two years later, in the fall of 1636, the Huron were again attacked, this time by a much more severe epidemic of influenza that, once again, had spread from the St. Lawrence (*Loc. Cit.*). It was also carried by the Nipissing Indians into northern Ontario, diffusing among the Indians of the north (Trigger 1987: 527). In the summer of 1637 another unidentified epidemic, possibly scarlet fever, reached the Huron (Trigger 1989: 230; Snow 1992: 179). This time, the disease seems to have come from the Susquehannock who lived near the Dutch settlements (Ibid.: 231). It reached the Huron by way of the Wenro, who lived to the south and who traded with the Susquehannock, and it also diffused along the Ottawa River. Finally, in 1639 a virulent smallpox epidemic spread throughout the St. Lawrence valley, killing the Indians who travelled to Trois Rivières and Quebec to trade, and devastating the Huron and other eastern groups[21] (Trigger 1987: 588). There is some difference of opinion as to the origin of this epidemic. Trigger (1989: 231; 1987: 588) concluded that it came from New England, via some Algonkin (the Kichesipirini, or Island Algonkin, of the Ottawa River) who had gone to visit the Abenaki. Smallpox was

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[20]Although Snow (1992: 178) concluded that measles had reached the Huron from the Indians living to the southward.

[21]Dobyns (1993: 282) linked this smallpox epidemic to episodes in the south to suggest another pandemic, this time occurring between 1639 and 1644 and reaching from northeastern North America to Amazonia and Brazil. However, by this time smallpox was likely so common a disease in Mesoamerica, if not endemic in parts of New Spain, that it would not be difficult to find a roughly contemporaneous epidemic with which to link the Huron episode.

epidemic in New England in 1638 (Duffy 1953: 44), and so this was certainly possible. Alternatively, others have claimed that the disease was introduced to Quebec by French ships, and that it subsequently spread westward along the St. Lawrence[22] (Marshall 1967: 18, 75; Snow 1992: 179). In either case, the disease was brought among the Huron by some of their people returning from Quebec and seems to have spread along the length of that river (Trigger 1987: 588).

The presence of these diseases among the Indians of the Ottawa and St. Lawrence Rivers and, especially, the Huron, is highly significant for the Petit Nord. During the first half of the seventeenth century this region was connected to the east through important trading networks of long standing. Each year the Huron travelled east to the Canadian settlements with furs from the Upper Great Lakes to exchange for European goods[23]. They had obtained these furs (and other commodities) largely by acting as middlemen for the more distant tribes, including those of Lake Superior (Trigger 1987: 63; 1989: 205). The key to this trade was a transportation backbone that ran along the Ottawa and St. Lawrence Rivers. In turn, two of the Huron's main trading partners, the Ottawa and the Nipissing, were engaged in a more distant trade of their own. The Ottawa also traded with the Indians living on Lake Superior, while the Nipissing made annual trips to James Bay to trade among the northern peoples (Trigger 1987: 264, 353; 1989: 206). At the

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[22]Marshall (1967: 75) concluded that smallpox broke out in Quebec in August of 1639, shortly after the arrival of the ships. Thereafter it reached epidemic proportions and continued into February of 1640.

[23]They were also trading with the Susquehannoch who lived closer to the English and Dutch colonies (Trigger 1989: 206).

same time, James Bay was linked to the lower part of the St. Lawrence via other commonly used trading routes that ran between the Saguenay and Rupert Rivers (Francis and Morantz 1985: 17). Thus, aboriginal trading routes connected the eastern part of the Petit Nord to areas that were then experiencing the ravages of epidemic Old World disease.

There is a very real possibility that one or more of these epidemics reached the Petit Nord. Heidenreich (1987b) concluded that two, the influenza of 1636 and the smallpox of 1639, spread from the Ottawa River to the Moose River area of James Bay, just to the east of the Petit Nord. He also speculated that the same 1639 epidemic and the unidentified illness of 1637 might have diffused into the Lake Superior area, after having reached Georgian Bay on Lake Huron. Trigger (1987: 355) believed that at least one of these epidemics spread to the Winnebago, who then resided at Green Bay on Lake Michigan. These would have followed well-established pathways of aboriginal communication, pathways that were closely connected with lines of movement into the Petit Nord and along which *ACIs* could easily have moved. Thus, the first documented round of epidemics to spread into the interior of the northern part of North America may also have diffused into the Petit Nord[24].

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[24]Serious outbreaks of infectious disease ceased among the Huron for a decade after 1639-40, returning only in the winter of 1649-50 (Trigger 1989: 271). Nevertheless, several epidemics erupted in the Atlantic colonies and among other eastern native groups during this period, including the Iroquois (Trigger 1987: 602; Heidenreich 1987b). There is no indication that these afflictions approached the Petit Nord, and their absence among the Huron and other groups located near the region tends to suggest that they failed to reach it.

Epidemics continued to occur periodically in the east between 1650 and 1669[25]. By then, however, widespread disruptions had befallen many of the native groups of the Great Lakes area, greatly altering potential pathways of diffusion. In 1649 the Huron were attacked by the Iroquois and dispersed, many of them heading westward (Trigger 1987). This event set about a sequence of relocations that saw many of the eastern groups flee to escape the wrath of the Iroquois. The nearby Petun were scattered in 1649-50 and retreated, first to Lake Michigan and later to Chequamegon on Lake Superior (Trigger: 1989: 271). The Algonquian-speaking groups of the Upper Country also withdrew, including those of the Georgian Bay area and the Ottawa River (*Loc. Cit.*). Among them were the Ottawa and the Nipissing, many of whom ended up at Chequamegon and Lake Nipigon respectively (White 1995: 17; Thwaites 1959: IL: 69; Blair 1911: 173). As such, the shores of Lake Superior became lined with refugees (Thwaites 1959: VL: 221). Others fled northward to James Bay (Francis and Morantz 1985: 19). Within a very short span of time, southern Ontario was emptied of its inhabitants (Trigger 1989: 271). As more and more people travelled into the area to the north and west of Lake Michigan, large refugee villages emerged of mixed affiliation, including key communities along the shores of Lake Superior (White 1991). These villages proved vulnerable to the ravages of

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[25]See, for instance, Duffy (1951b: 329; 1953: 115, 186); Snow 1992: (179-180); Caulfield (1953: 532); Heagerty (1928: 1: 58); Creighton (1965: 1: 613) and Marshall (1967: 239, 265 301, 311). Some acute infectious disease is known to have appeared beyond Lake Huron between 1650 and 1670. In 1666 a severe contagious disease prevailed among the Pottawatomie, then living on Green Bay (Kellog 1959: 127; Marshall 1967: 331; Tanner 1987: Map 6). While there, Father Claude Allouez was able to baptise 340 people, of whom about 300 died shortly after converting (Marshall 1967: 331). This fits a general pattern of missionary success followed quickly by the death of the initiate, witnessed on other occasions during epidemics in northeastern North America. Father Allouez also indicated that at least one Ottawa village in the Lake Superior area was ravaged while he was there during the 1660s (White 1995: 41 fn. 71; Thwaites 1959: L: 287).

the Old World diseases (Ibid.: 41). Moreover, the flood of people travelling from the east undoubtedly posed a significant risk for the spread of such disease.

With the Huron and other eastern groups dispersed, the Iroquois pursued their raids over even greater distances, including into the Petit Nord (Long 1986: 43-44; Lytwyn 1993: 190-197). These had a significant impact on the patterns of movement in eastern North America. For many years following the fall of Huronia, the Iroquois made the Ottawa River Valley a dangerous road by which to travel for the Indians of the Upper Great Lakes. Consequently, trade was directed along routes that avoided the Ottawa, particularly along those that were connected to James Bay, for they bypassed the most dangerous areas. In 1657 Father Gabriel Druillettes learned of the existence of five major aboriginal routes of travel leading from the south to James Bay (Francis and Morantz 1985: 18; Thwaites 1959: XLIV: 241-243; Figure 5). One began at the Lower St. Laurence, near Tadoussac, and included the Saguenay River, Lake Mistassini and the Rupert River. Another ran from near Trois Rivières along the St. Maurice to the Nottaway River. Three others involved systems far to the west. A traditional route of the Nipissing followed the Ottawa River and Lake Temiscaming to Abitibi Lake. From there they proceeded by the Abitibi River which flows into James Bay. A fourth ran between the Spanish River, emptying into Lake Huron at Georgian Bay, and the Matagami River. Finally, Lake Nipigon and the Albany River connected Lake Superior to James Bay. The result was a bilateral traffic that enabled furs from the interior to be exchanged for European goods from Canada with far less risk of plunder by the Iroquois. Although the Ottawa River-St. Lawrence River route was lightly travelled at this time, if at all, the Petit

Nord nonetheless remained well connected to the east, a crucial source of *ACIs*. Consequently, Old World infections that spread from Canada into the interior with native traders stood a good chance of being directed towards James Bay, and the eastern margins of the Petit Nord.

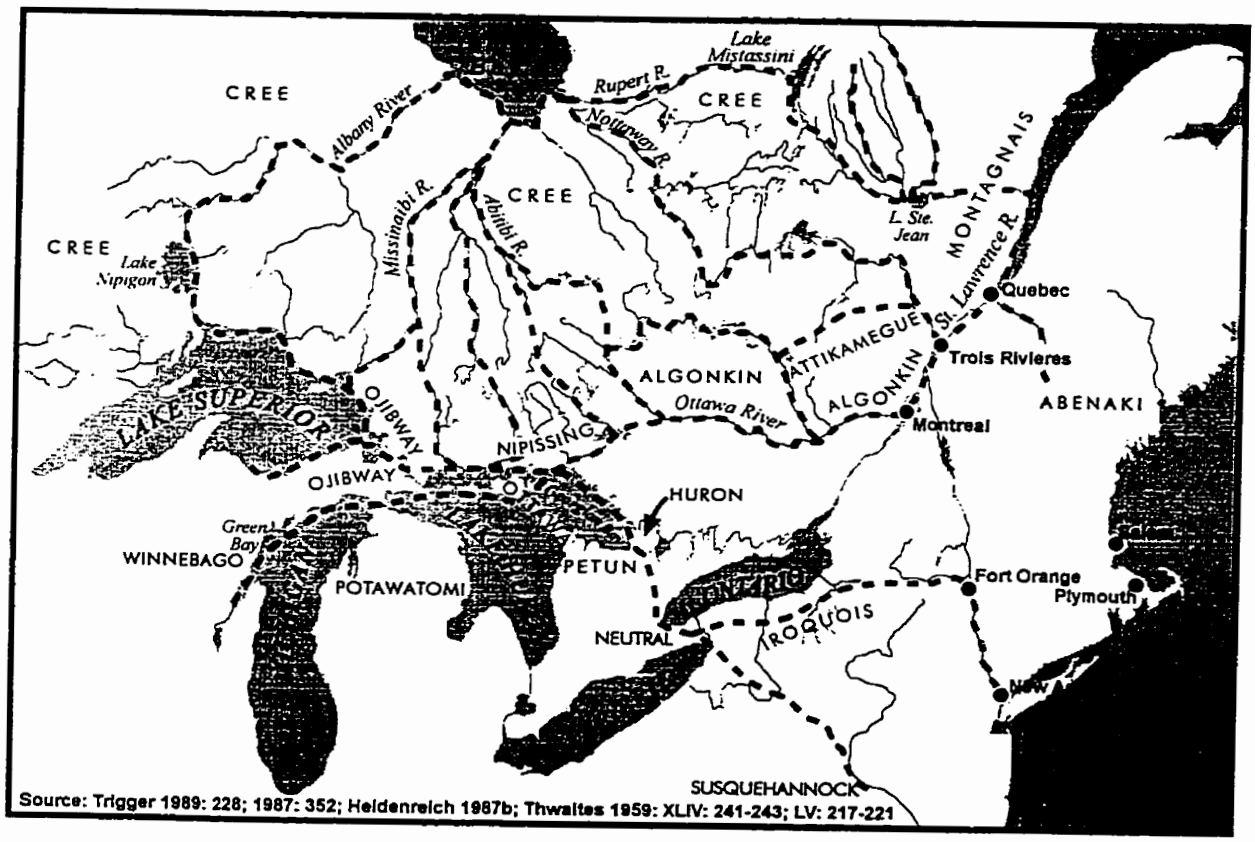

Figure 5: Seventeenth century trade routes in northeastern North America

Exploration into Hudson Bay

In addition to Mesoamerica and the European colonies of northeastern North America, there was a third potential protohistoric source of *ACIs* for the Petit Nord, one that could have seen these diseases introduced directly from Europe. Between 1610 and 1632 several European expeditions of exploration entered Hudson and James Bays, some spending time on their shores. Henry Hudson was the first European to enter this region, exploring both bodies of water in 1610 before a mutiny on June 22, 1611, saw him and a small party set adrift and left to die (Tyrrell 1968: 3-4). He was followed by Thomas Button (1612-13), Jens Munk (1619-20), Luke Foxe (1631) and Thomas James (1631-32). There is no record of any expedition to either James or Hudson Bay following Foxe and James until that of the *Nonsuch* in 1668[26] (Tyrrell 1968: 6).

While very slight, there is a possibility that the few vessels involved in these initial explorations could have introduced *ACIs* into the Petit Nord, during a period when their effects would have predated observation by European reporters[27]. Several of the parties overwintered at the Bay, and some of their crews suffered illnesses at this time. Prior to the mutiny, Hudson and his ship the *Discovery* wintered on the southeast coast of

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[26]Although Nute (1943: 286) concluded that "daring mariners from [New England] penetrated to the bay soon after English seamen gave up their interest in it" there is no evidence that such was the case. However, if there had been New Englanders at the Bay during the interim period, they would have been more likely to have carried epidemic disease to the Petit Nord than their predecessors, since such sickness was relatively common in the Atlantic colonies following 1630, and their journey was much shorter. The lack of ship-based exploration in Hudson Bay following the journeys of Foxe and James was due primarily to James' conclusion that a northwest passage did not exist south of 66°N, effectively diverting the search for the Passage to the north of Hudson Bay (Cooke 1966: 385).

[27]The English mariners of the seventeenth century suffered from a wide variety of medical complaints. Rosen (1939: 754) stated that predominant complaints in the seventeenth and eighteenth centuries were "infectious fevers, avitaminoses, pulmonary and cardiac complaints, rheumatic affections, and ulcerative conditions of the extremities." John Woodall, writing in 1617, specified fevers, scurvy,

James Bay, at or to the north of the mouth of the Rupert River, where he is thought to have traded for furs with at least one Indian of unidentified origin[28] (Morantz 1984: 176). Hudson's crew appears to have been healthy at the time of trading. Button, who went looking for Hudson with two ships, wintered at Port Nelson in 1612-13 and, although there was some lethal disorder among his men, it is not stated that they made any contact with Indians[29].

The case of Jens Munk, a Dane who spent the winter of 1619-20 in Churchill harbour, is particularly interesting as an example of the perils facing seventeenth century mariners. In 1619 he sailed from Copenhagen with two ships, the *Enhiorningen* and the *Lamprenen*, and a contingent of sixty-four men (Tyrrell 1968: 5). After reaching Hudson Bay the sudden arrival of cold weather forced them to settle at the mouth of the Churchill River until the following summer. At this time the men developed a mortal illness, and by July of 1620 only Munk and two others remained alive to guide the *Lamprenen* back to Europe. It has generally been thought that the disease was scurvy, a common complaint among seafarers of the day (*Ibid.*: 6). However, in 1973 Delbert Young put forward the alternate theory that, although scurvy may have been among Munk's men, something else was responsible for the tremendous mortality. Comparing the fate of the

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dysentery and ulcers (*Loc. Cit.*). The term "fevers" may have included several infectious diseases.

[28]Morantz (1984: 176) referred to an oral tradition among the Cree that may describe this earliest Bayside trade. There is other evidence that Hudson and the others who had been abandoned were murdered for attempting to steal corn and other items from the Indians (Trigger 1987: 275, 283-285).

[29]Button's crew was supposedly stricken with scurvy during this sojourn, such that the sailing master of the *Resolution*, Francis Nelson, succumbed and gave his surname to the river on the shore of which he was buried (Tyrrell 1968: 5). According to Young (1973: 13), some have thought that a contagious disease was passed from one of Button's ships to the Inuit living north of Churchill who were then decimated. In Young's view, this was very unlikely.

Munk expedition with that of Button and another to the north pole by three Swedes in 1897, he concluded, with some justification, that the fate of the three expeditions may have been sealed by the consumption of poorly cooked bear's meat infected with trichinae, parasitical nematode worms that cause the disease trichinosis and that are endemic in polar bear populations[30] (Young 1973: 14-15).

The explorations of 1631 by Foxe and James rounded out the early period of European exploration from Hudson Bay. Foxe, on board the pinnace *Charles*, coasted along the western shore of Hudson Bay as far south as Cape Henrietta Maria, stopping for a time at Port Nelson. There was no obvious epidemic sickness on board during this trip (Morley 1966: 32). James explored the west coast of Hudson and James Bays from the Churchill River to Charlton Island, near the Rupert River. After wintering at Charlton Island, and having scurvy break out among his crew, James and the *Henrietta Maria* sailed for England on July 1, 1632 (Cooke 1966 384-385).

In all, then, despite the presence of sickness among some of these non-native parties, there is no evidence that they carried *ACIs* to the shores of the Petit Nord, or that these diseases were transmitted to the native inhabitants, with whom they had little or no contact. It was only with the beginning of annual shipping from Europe and a steady and consistent fur trade that direct trans-oceanic shipment of *ACIs* became a reality. These

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[30]In 1682, while sailing to Port Nelson, Pierre Radisson's crew became ill after eating polar bear meat in Hudson Straight, circumstances that are similar to that of Munk and Button. The episode is interesting in that it indicates that the Inuit knew of such sickness. Radisson recorded that:

factors only fell into place after the arrival of the HBC on the scene later in the century, coinciding with the initiation of the historical period in the region.

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some of my seamen killed a white bear of extreme bigness. They eat of it to such excess that they all fell extremely sick with headaches and looseness; I thought they would have died out... We were informed by the Indians that those white bears have a poison in the liver that diffuses itself through the whole mass of the body, which occasions these distempers unto those that eat them (Adams 1961: 168-9).

CHAPTER 4 — *ACIs* IN THE PETIT NORD: 1670-1736

INTRODUCTION

This chapter examines the arrival of *ACIs* in the Petit Nord during the initial years of the historical period, that is between 1670 and 1736. As noted in Chapter 3, discussion of earlier epidemic activity in the Petit Nord is, of necessity, a theoretical exercise. Without the testimony of literate observers, and in the absence of any supporting or contrary oral tradition or archaeological evidence, it is impossible to determine when and how frequently such diseases might have struck the region during the protohistoric period. This began to change in 1668 as the HBC began its long tenure in the Canadian Northwest. With the firm's great attention to detailed record keeping documenting its trade, and the French penetrating into the region in answer to the HBC competition, the protohistoric period gradually became the historical, as direct contact was initiated with many native groups who had never before travelled eastward to trade with the French, and with those who had previously sent only a few representatives to Canada. Even so, the record of this period is extremely poor, particularly so prior to 1714, and the view throughout this period is largely a dim one from the periphery. Nevertheless, it is at this time that historical evidence for *ACIs* in the region appears, and that one finds the first accounts of epidemic sickness among the people of the Petit Nord and their nearest neighbours. Consequently, it is with the arrival of the English on James Bay that the epidemic history of the Petit Nord truly begins.

The founding of the HBC was crucial to the region's epidemic history for another reason. Although the Company played almost no role in introducing *ACIs* into the Petit

Nord prior to 1737, its very presence set the stage for such diffusion indirectly. The founding of the Company set in motion changes in the fur trade that had a profound effect on the patterns of movement of the Indian people and on their connections with the European settlements of the east. In turn, these structural changes enhanced the possibility of disease diffusion into the Petit Nord, by increasing the frequency of contact between Indians and the traders travelling from the eastern settlements, the source of much of the region's later epidemic activity. To understand the nature of *ACIs* within the Petit Nord at this time, then, it is necessary to examine the circumstances surrounding the founding of the HBC, and the changes this prompted in the spatial structure of the French fur trade.

THE FOUNDING OF THE HBC

In early June of 1668 two ships, the *Nonsuch* and the *Eaglet,* sailed from London on a trans-oceanic voyage of far reaching significance, their principal design being to trade in furs from Hudson Bay. On board the two ships were Médart Chouart, Sieur des Groseilliers, and Pierre Esprit Radisson, men already familiar with parts of the Petit Nord through their earlier explorations. Although French by birth, the two had agreed to take the English to Hudson Bay in order to set up a new fur trading enterprise. The *Eaglet,* with Radisson aboard, was damaged in a storm and forced to turn back on the outward voyage (Nute 1943: 120). Nevertheless, the expedition was a success. On September 29 the *Nonsuch* arrived at Rupert River, where the men erected Charles Fort and, prior to their departure for England on August 12, 1669, they traded with some 300 Indians (*Ibid.*:

117, 119). The fur returns from this enterprise were encouraging such that, in May of 1670, the HBC was granted a royal charter by King Charles II and a second expedition was dispatched in that year (See Morton 1939: 54-59). From then on, the HBC was to be a near constant presence in the Hudson Bay drainage basin and beyond, and within fifteen years had settled forts at the mouths of the Rupert, Moose, Severn, Albany, and Nelson Rivers (Innis 1956: 120).

Despite this presence and the annual contact with England, however, the HBC played almost no role in introducing *ACIs* into the Petit Nord prior to 1737[1]. This, however, was not the case for chronic infectious diseases, which were not as constrained by the lengthy voyage as were the acute infections, and such long term afflictions as tuberculosis and venereal diseases were quick to spread to Hudson Bay from Europe. Venereal diseases, now termed sexually transmitted diseases (STDs), were a significant problem among the HBC men almost from the start. As early as 1682, Governor John Nixon was complaining that the men were "seek of the ould disease of licentiousness" (Quoted in Rich 1976: 47). From then on, "poxed" employees appear intermittently in the HBC records (*Ibid.*: 49). For instance, in 1727, a labourer named John Murrill, who had been infected in England, was sent home "for fear he should Touch with the Natives which may prove of Ill consequence to the Company" (Davies and Johnson 1965: 124-

[1]The very few voyages of exploration to Hudson Bay prior to 1668 were unlikely to have carried *ACIs* across the Atlantic and passed them on to the people of Hudson Bay (Chapter 3). The HBC was not the only party that sent ships to Hudson Bay after 1670. Infrequently, vessels from France, New England, Canada and a few belonging to other English interests landed on the shores of Hudson and James Bay for the purposes of trade, warfare and exploration. Compared to the English ships, the others tended to be extremely rare visitors, especially following the Treaty of Utrecht in 1713. There is no evidence that any of these vessels transmitted infectious diseases once at the Bay, although Rich (1976: 44) speculated that the 1782 French expedition to Hudson Bay led by la Pérouse harboured typhus.

125). Such actions proved ineffective, as by the midpoint of the eighteenth century, venereal disease was common among the Indians who traded at Hudson Bay, passed on to them by both the HBC and Canadian traders[2] (Williams 1969: 143-144). Similarly, pulmonary tuberculosis, called consumption, was also present among both Indians and Europeans, and became common among the natives as time passed (Rich 1976: 50).

Although it may appear that the Company's transportation system was also ideal for the transoceanic diffusion of diseases such as smallpox and measles, such was not the case. Only once during this period, in 1720, is it probable that the ships carried an *ACI* to Hudson Bay. This was owing to the nature of these sicknesses and of the HBC's maritime transport system.

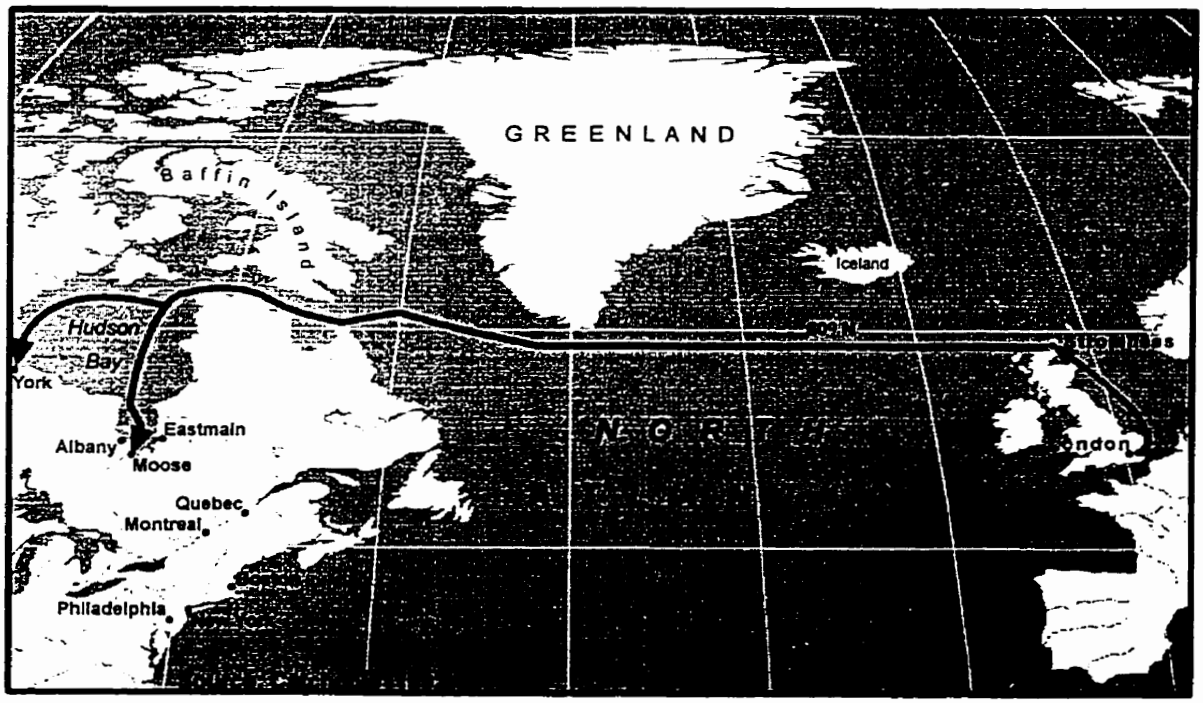

Figure 6: Sailing routes of HBC ships from England to Hudson Bay

[2]This was according to Andrew Graham, who served the HBC in a variety of capacities at Hudson Bay between 1749 and 1775 (Williams 1983: 362).

During this era, the HBC ships typically began their passage in London, departing for the port of Stromness in the Orkney Islands some time between mid-May and early June (HBCA C.4/1; Catchpole 1992: 18; Figure 6). By the time of the founding of the HBC, London was a cosmopolitan and populous city, and a potent and constant source of infectious disease. Endemic diseases were present, and those that were not might be introduced from elsewhere by the constant ship traffic. The population of the city on the eve of the HBC's first voyages was almost one half million, sufficient to maintain most, if not all, crowd diseases endemically[3] (Creighton 1965: I: 660). By this time, smallpox, for instance, had settled down into a "steady prevalence from year to year" (*Ibid.*: II: 436).

Nevertheless, it was not exclusively a disease of childhood, as the city always experienced a continuous flow of adults from rural areas where the disease was not common (*Ibid.*: II: 532). Presumably, the same was true for other crowd infections, and this might have enabled such diseases to come aboard the HBC ships, despite overwhelmingly adult crews. After a journey of generally four to seven days, the HBC ships arrived at Stromness, in the Orkney Islands. The brief layover there of between three and seven days was significant as, in addition to ballast and water, the ships took on new employees bound for Hudson Bay. These additional men could provide a new reservoir to maintain the disease on the journey to Hudson Bay.

The second phase of the journey, the voyage across the Atlantic, was the greatest factor limiting the importation of *ACIs* to Hudson Bay. The journey from Europe was

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[3]Sydenham's description of measles in London of 1670 called it "a disease mainly of young children (*infants*)..." (Creighton 1965: II: 635).

long, crews were small and no families accompanied them. Together, these circumstances worked to deter the diffusion of most infectious maladies via the Company ships. By definition, acute diseases are of brief duration, with many inducing an immune response in the victim. As such, the longer the voyage, the less likely that disease will be passed on at the destination, since at some point the ailment will have run its course among all susceptibles on board. If the transit period is decreased substantially, however, the number of consecutive cases needed to maintain the disease, expressed in generations[4] of the disease, will be reduced. This, according to Snow and Lanphear (1988: 25), was the main reason for the sudden emergence of epidemic disease in New England after 1600, when the voyage had been reduced from an average of six weeks to about four. A similar explanation was offered by Haggett (1993: esp. 46-47) in examining the introduction of smallpox, measles, influenza and rubella to Australia, New Zealand and the Southwest Pacific. Again, alterations in the trip length led directly to the introduction of "new" diseases. Compared to the voyages to New England, the journey to Hudson Bay remained of long duration during the entire eighteenth century. An analysis of the travel times from their final departure point in the British Isles to the first landing at the HBC posts during the period 1719-1737, reveals a mean duration of just over sixty-seven days, or of more than seven weeks[5]. Of thirty-two voyages during this period, only

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[4]Each generation being measured from the initial time of infection to the point when infectivity ceases.

[5]This figure was calculated from the descriptions of ships movements found in HBCA C.4/1 (*Book of Ships' Movements* Microfilm 2m135) which entries commence for 1719. The departure point from Europe, whether London or the Orkney Islands, was used as it would be the last day of contact between the crew and passengers on European soil.

two lasted less than fifty days, both in 1731 when the journey was achieved in forty-six days. Nevertheless, even the most rapid of voyages during this period was significantly longer than the four week voyages to the Atlantic seaboard which facilitated the spread of disease to that region in the early seventeenth century (Snow and Lanphear 1988: 25).

Compounding the extended duration of these voyages was the limited number of susceptibles to provide a reservoir for the transmission of acute diseases. Although no study of the HBC transport system has included an analysis of the numbers on board the ships sailing to Hudson Bay, it is safe to say that these ships were small, and carried few passengers and crew compared to the large colony ships sailing to either the Atlantic coastal colonies or to the Pacific countries studied by Haggett. The crews of these vessels were generally small, perhaps numbering less than thirty[6]. The passengers, consisting of men sent to serve in the Company's posts, were also few, especially during this period. The annual turnover of employees stationed at the Bay was minimal, and new recruits might account for only a few tens of people each year. It was only during the late eighteenth and early nineteenth centuries that intense competition with the Canadian traders forced the HBC to expand away from the Bayside and to maintain a considerable number of posts, and thus to transport greater numbers of men.

The men making these voyages were less likely to be susceptible to crowd diseases than many of the colonists. Typically, crew and passengers were adult males, although there may have been a few teenage apprentices and ship's boys as well. By

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[6]The typical English naval seaman of the sixteenth through nineteenth centuries suffered from a wide array of infectious diseases (Rosen 1939: 754). However, the complement of even a *medium* size military vessel might be 400 (*Ibid.*: 752), as compared to a much smaller number aboard the HBC ships.

contrast, the ships carrying disease to the Northeast brought entire families and, most important for the transmission of these diseases, large numbers of young children (Snow and Lanphear 1988: 27). This is a key distinction. Many of the diseases which were most destructive in the New World, among them smallpox, measles, rubella and whooping cough, were, by the founding of the HBC, largely diseases of childhood throughout much of the more populous parts of Great Britain. If the HBC men and the mariners were almost exclusively western European adults, it follows that many, even most, would have had prior exposure to these diseases by the time they reached adulthood, and thus immunity to prevent subsequent reinfection with the same disease. As such, only a small percentage of the men on board the ships would have been susceptible to *ACIs*, limiting the number able to maintain them during the voyage. When combined with the small numbers on board and the length of the journey, the possibility of HBC ships transporting *ACIs* across the Atlantic was also limited and, in this respect, they were similar to the early fishing ships sailing to eastern North America[7].

These factors do not preclude the transmission of *ACIs* but make them less likely. Given a generational period of between twenty-six and thirty days for smallpox (Snow and Lanphear 1988: 25), for instance, at least three susceptible individuals would be required to support that infection on the average voyage to Hudson Bay, with the widest spacing of cases while en route. For measles, more than three generations would be

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[7]Of course, this was not a deterrent to diseases that could exist for part of their life cycle in other species, such as typhus or plague. Typhus, a relatively common disease aboard ships, was known to have reached Hudson Bay from Europe, and it would also have been possible for plague to have been maintained during the long journey within the fleas living on the ships' rats. If the situation in 1800 was typical of HBC shipping throughout the study period, then rats were a problem on board the HBC ships (HBCA B.3/a/104: 2).

required, given a generational length of seventeen to twenty-one days (Cliff *et al.* 1981: 42). Other diseases with shorter generational periods would have required an even larger number of cases in succession. Nevertheless, while the theoretical number of cases required need not be great, it is important to note that most disease agents of this type could not remain viable in a single individual throughout the long trans-Atlantic voyage. Each additional generational link made the diffusion more complex and less likely, and with few susceptibles on board, if any, the possibility was even more remote. A significant exception, may have been the case of smallpox which, under certain circumstances, can survive for extended periods of time on inanimate objects. However, this form of transmission does not appear to have been common.

When compared to conditions found by Haggett for the southwest Pacific, it is evident how unlikely such diffusion to Hudson Bay was. In the case of measles, for example, Haggett found that among the early immigrant ships to Fiji, requiring about 70 days to complete the journey and carrying some 500 immigrants, presumably of all ages, none carried the disease to Fiji although one third had had active infections aboard when they departed (Haggett 1993: 43). In this case, the average length of the trip was almost identical to that of the HBC ships during this period, and the number of passengers was far in excess of the Company's ships' total of crew and passengers. What is more, the ships bound for Fiji carried a significant percentage of children, unlike the HBC ships. As conditions changed, however, the barrier to *ACIs* which protected the southwest Pacific broke down. Once the much faster steamships replaced the sailing vessels, one half of the ships with active measles cases aboard when they departed for Fiji remained

infective when they docked. These later vessels also carried many more passengers. Conversely, there were no corresponding changes in the HBC ships engaged in transport to Hudson Bay prior to 1850. Along the Pacific coast of North America, however, the HBC employed a steam powered vessel, the *Beaver*, and in 1847-48 this ship played a key role in transmitting measles to the aboriginal people of the Pacific coast (Galois 1996).

Although only rarely did the Company's ships carry *ACIs* into the Petit Nord, there was a more significant consequence of the founding of the HBC in the context of the epidemic history of the region. The arrival of the English fur traders in James Bay coincided with, and contributed to, the beginnings of a shift in spatial relations between the Europeans and the Indians of the Petit Nord[8]. Prior to 1666, most of the fur trade of the region was carried to the French settlements on the St. Lawrence River by Indian middleman traders on annual or semiannual trading excursions (Ray 1993a: 319-20; Morton 1939: 53). In 1666, following French military excursions against the Mohawk, a peace was made between the Iroquois Confederacy and the French and their Indian allies. This peace enabled some Ottawa and Ojibway to reoccupy their traditional homelands from which they had been forced following the fall of Huronia and, perhaps more importantly, freed the way for French traders to penetrate into the interior of the continent (Heidenreich 1987a). The entry of the HBC into direct competition for the prime furs

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[8]The progress of this movement into the Petit Nord is illustrated graphically in Heidenreich's series of plates for the *Historical Atlas of Canada* showing the expansion of the fur trade into the interior (Heidenreich 1987 plates 37-39). Innis (1956: 41-43) also identified a major shift in the French fur trade with the demise of the monopoly in 1663, which led to the emergence of the individual trader, an increase in competition in the fur trading regions, an increase in the number of traders and "a rapid expansion of trade" into the interior.

from the area north of Lake Superior, and the subsequent construction of posts at strategic river estuaries along James Bay outflanking the Indian middlemen trade, served to stir the French to further action (Nute 1943: 147; Morton 1939: 72).

The English presence at Rupert River became known to the French through Indian traders shortly after the English ships arrived. News of the 1670 voyage of the *Wivenhoe* and the *Prince Rupert* reached the French near Tadoussac in September of 1671. On the 17th of that month, at Lac St. Jean, Father Charles Albanel met:

> five canoes bearing Attikamegues, or poissons blancs, and Mistassirinins, came [who] joined us, bringing word that 2 vessels had anchored in Hutson's bay and conducted extensive trading with the Savages, having taken their station there for purposes of traffic. They showed us a hatchet and some tobacco, which they obtained from a Papinachois, who had been on a trading trip to the North sea, that very summer. (Thwaites 1959: LVI: 157)

The effect of the English trade on Hudson Bay was felt almost immediately. In May of 1673, Father Henri Nouvel at Sault Ste. Marie complained "the english have already diverted a great many of the savages who visited lake Superior" (Lytwyn 1986: 4; Thwaites 1959: LVII: 21).

In response to the English threat, the French increased exploration and direct contact with the Indians by expanding their trading posts to the north and west[9]. This

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[9]One of the first explorations undertaken after the arrival of the English was by Father Albanel, who journeyed from Tadoussac up the Saguenay River to Charles Fort in 1672 and again in 1674. This is not to say that exploration and the fur trade in the near vicinity of the Petit Nord began only after 1670. Champlain had reached Georgian Bay in 1616-17 with Brûle travelling perhaps as far as Sault Ste. Marie (Heidenreich 1976: 25). The Jesuit Fathers Charles Raymbault and Isaac Jogues reached Sault Ste. Marie in 1641 (Gigure 1966: 388). Radisson and Groseilliers explored and traded about Lake Superior during the late 1650s and the 1660s (Nute 1943). Other Jesuits, notably Claude Allouez, visited the Lake Superior region during the 1660s. Generally, however, these were isolated journeys made by a few individuals at the most, rather than the much larger number of explorers, priests and traders who came annually to the northwest after 1670 (Hickerson 1960: 86). See Lytwyn (1986: 1-3) for a brief synopsis of exploration in

brought the trading post frontier and the French into the Petit Nord for the first time (Ray 1993a: 319-20; Heidenreich 1987a). It also brought large numbers of *Coureurs de Bois,* who went to the Northwest every year, and traded and wintered with the Indians, before returning to Montreal the following year. In 1688 Baron de Lahontan wrote of Sault Ste. Marie that "this place is a great Thoroughfare for the *Coureurs de Bois* that trade with the Northern People, who usually repair to the banks of that Lake [Superior] in the Summer" (Thwaites 1905a: I: 152-3). Within two decades following the establishment of the HBC, the Petit Nord was experiencing rapid, annual and frequent traffic from Montreal, a circumstance conducive to the transporting of acute infectious disease into the region.

Also at this time, and especially in response to the English encroachment, the French began establishing permanent posts at key sites in and around the region as bases of operation for fur traders. For instance, voyageurs resided at Sault Ste. Marie shortly after the founding of the Jesuit mission in 1669 (Anick 1976: 251). Just a few years later, in 1673, a post was built on Lake Mistassini to attract Indians over a wide area, including the eastern portions of the Petit Nord (Morton 1939: 73). In 1678 Kaministiquia was settled by Duluth and in 1684 his brother, Claude Greysolon de la Tourette, established a post at Lake Nipigon (Thwaites 1905a: I: 316, fn. 1; Anick 1976: 255). Népigon, on the Nipigon River north of Lake Superior, was constructed in 1679 (Heidenreich 1987a). By 1685 the French had moved into the Albany River system and established a post at its confluence with the Kenogami (Morton 1939: 98). A post at Temiscaming was in

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the Petit Nord prior to the founding of the HBC. As well, it is very likely that the almost ubiquitous *Coureurs de Bois* had also penetrated into the region.

operation at least by 1686, and possibly as early as 1679, and another at Lake Abitibi was constructed in 1686, replacing one built in 1677 on the same lake. Both were intended to intercept furs intended for Moose Fort (Anick 1976: I: 29, 49; Heidenreich 1987a). That such posts were intended to cut off the English from the supply of furs is made perfectly clear by the intentions of Duluth, whose fort on Lake Nipigon was built to intercept the Cree and Monsoni going to the English (Margry 1876-86: 6: 20-31). In turn, the penetration of French fur trading posts had profound effects, not only on English trade[10], but also on the annual movements of the Indians who traded directly with the French. Rather than making the long journey to Canada to trade their furs, they could now wait for the Canadians within their own territories, or travel much shorter distances to the French posts. This was not always a benefit for the Indians as, by the 1680s, the Ottawa no longer made their annual trading journeys to Montreal, having been bypassed as middlemen by the inland expansion of the French (Trigger 1989: 285).

This influx of white traders into the Petit Nord carried with it the potential for the introduction of epidemic disease. The rapidly moving brigades annually penetrating into the Petit Nord from the active disease centres of the eastern seaboard must have carried acute infectious disease with them on occasion, although how often is unknown[11]. It is known that ships were regularly bringing acute infectious diseases to the eastern ports,

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[10]See Tyrrell (1968: 386-387) for evidence of the effects of such direct competition on the HBC at Moose Fort.

[11]Following the conquest, the Montreal traders of British descent adopted the personnel and much of the manner of operations of the French fur trade. There are several instances in which these later Canadian traders brought *ACIs* with them to the Northwest, and it is likely that the same had occurred during the days of the French regime.

and that the resultant epidemics often diffused widely through the Atlantic colonies and to the eastern Indians. The greater numbers of individuals annually leaving Canada for the lands north of Lake Superior and east of Lake Winnipeg following the founding of the HBC meant an increase in the number of potential vectors for transporting these diseases. Likewise, the new trading posts, visited by Indians from throughout the region, afforded ideal points for disease redistribution, just as had communities such as Quebec, Montreal and Trois Rivières before the period of expansion[12]. The disease frontier of the distant urban centres in Europe through the transport connections in North America had taken a step closer to the Petit Nord.

ACIs IN THE PETIT NORD: 1670-1736

The initial era of disease history, 1670-1736, can be further divided into periods separated by the Treaty of Utrecht, signed by France and Great Britain in 1713. Prior to the treaty, and after the initial years of westward expansion into the Petit Nord by the French, control over the Bayside posts passed back and forth between the French and English, with the HBC at times limited to the bottom of James Bay, or to York. For instance, York Fort (renamed Fort Bourbon) was captured by a French sea force in 1694, lost in 1696, and regained in 1697 by an expedition from France, thereafter remaining under French control to 1714 (Tyrrell 1968: xiii; Morton 1939: 116). Albany was captured by the French in 1686 and recovered in 1693 (HBC Post History "Albany").

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[12]In particular, the fur trading posts effectively served as nodes of diffusion by concentrating aboriginal populations in a single location, one that was directly connected to the urban disease pools of the east. These conditions were especially conducive to the diffusion of diseases that spread by droplet infection and face to face contact (Boyd 1985: 343).

These and other changes in control over the key Bayside forts influenced, and were influenced by, the course of the fur trade in the interior of the region. As long as the French controlled the more economical ship-based approach to Hudson Bay, they diverted their efforts away from the canoe-based, direct approaches to the HBC hinterlands (Morton 1939: 125-126). They could outfit strategic forts and have the Indians bring their furs to them just as did the HBC. This was especially significant in the case of York, as initial thrusts to the west of Lake Superior were quickly abandoned. In 1688, with York yet in HBC control, Jacques de Noyon first explored the Boundary Waters on behalf of the French. However, nothing came of this until the eighteenth century as only a few years later they captured York and its fur trade hinterland (*Ibid.*: 170, 125-126). Only after the treaty, beginning in 1717, did the French resume their expansion of the direct fur trade into the Boundary Waters. Unfortunately, as a result of these disruptions, documentary records are extremely limited for these years, and so the degree to which *ACIs* penetrated into the area cannot be discerned[13].

In 1714, York was returned to the English and the French abandoned Hudson Bay by the terms of the treaty. What followed was a period during which the English regained complete control of the Bay and the French resumed their expansion into the Northwest[14].

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[13]The first extant Albany journal dates from 1705-06 (Williams 1975) and the first available York journal dates from 1714-15 (HBCA B.239/a/1). Oldmixon's *History of Hudson's-Bay*, written in 1708 (Tyrrell 1968: 371-410), contains details from Thomas Gorst's journal of the early 1670s. Gorst's journal of 1671, which is in the Guildhall Library in London, was published by Grace Lee Nute (1943: Appendix 2). There are also a few extant HBC letters to London (for example Davies and Johnson 1965) and a few French memoirs written at York during the period of French control (Douglas and Wallace 1926; Tyrrell 1968).

[14]In 1715-16, for instance, the French settled one post approximately seven days' paddle up the Albany River from the Fort, and another eight days' paddle from Moose (HBCA B.3/a/9: 10d, 11).

Historical evidence becomes increasingly available for the years following the treaty, especially on the part of the HBC (Ray 1988: 51-52; Lytwyn 1986: 5; Morton 1939: 125-126). Although a more complete view of the southern part of the region emerges only during La Vérendrye's westward thrust into the Boundary Waters and onto the plains, by the 1720s the HBC was reasonably well informed of significant events in the interior, and was routinely identifying individual groups of Indian Uplanders.

1670-1713

It did not take long for an epidemic *ACI* to reach at least as far as the margins of the Petit Nord following the founding of the HBC. In the summer of 1670 smallpox diffused to Sault Ste. Marie, where it was accompanied by other sicknesses. It may also have spread into the Petit Nord. This highly mortal visitation of smallpox started in New France in 1669 and spread westward from there (Heagerty 1928: I: 28). Tanner (1987: 169) noted that in 1669-70 "epidemic disease spread widely among the tribes of Eastern Canada, New England, and upper New York, and west up the St. Lawrence River." Tanner's (1987: Map 32) map of epidemics in the Great Lakes region shows that this disease was felt as far west as the Straits of Mackinac between Lakes Huron and Michigan. Other evidence demonstrates that it extended at least as far as the Saguenay River in the east and to Sault Ste. Marie in the west.

Although there is no record of this epidemic spreading into the Petit Nord, it is nonetheless worthwhile to examine its progress. First, like the earlier Huron epidemics, it provides insights into the possible diffusion of earlier sicknesses for which there is even

less evidence. Although the English had already settled Charles Fort, the French had yet to respond by moving in force into the interior at this time. Thus, the conditions of diffusion reflected those of an earlier era, when large groups of Indian traders travelled to Canada to trade, rather than the period when the French began to move towards the Petit Nord in larger numbers. Secondly, the epidemic may have spread into the Petit Nord from Sault Ste. Marie, although there is no written evidence to confirm this. There can be no doubt, however, that some of the people of the Petit Nord were affected by this epidemic and by other diseases at Sault Ste. Marie, although not within their own territory. As such, it has to be considered, both for its impact and for the immunity that would have been acquired by the survivors.

Smallpox (*variola*) is an acute, directly transmitted, viral disease caused by the variola virus. Under some circumstances, it is capable of producing a very high case fatality rate. There are two recognised strains of smallpox, *Variola major* and *Variola minor*, these variants differing only in severity (Dixon 1962: 57). The latter causes few complications and case fatality rates are usually, although not always, low. *V. major* takes a variety of forms and, depending on the conditions of the victim and the type, would generally be fatal fifteen to forty percent of the time, although specific conditions might greatly increase the fatality rate. In cases designated as fulminating smallpox, for instance, death is almost invariably the outcome (Benenson 1985 350; Dixon 1962: 7). An attack of either *V. minor* or *V. major* usually grants full and lasting immunity to both strains (Benenson 1985: 352).

Smallpox is normally spread by respiratory emissions, or by contact with skin

lesions of infected individuals, or by contaminated materials, from which the virus enters the respiratory tract (Benenson 1985: 351; Dixon 1962: 171). Other than by the droplets emitted by the victim, sources such as corpses, clothing, flies and rags, may also spread the disease (Dixon 1962: 299-309). Following infection, the victim experiences no symptoms during an incubation period lasting ten to twelve days (Benenson 1985 352). Initial symptoms of smallpox then commence and may include headache and often backache, sudden fever, general malaise and vomiting (Dixon 1962). Two to four days later the characteristic rash generally, although not always, appears (Benenson 1985 352). It develops first on the face, and thereafter on the body and extremities, and is more common on the face and extremities (*Ibid.*: 350).

The infectious period of smallpox can be lengthy, perhaps as much as twenty-one days (Benenson 1985 352). Because of this, Ramenofsky (1987: 167) placed it in the highest category of diffusion potential. Moreover, the variola virus's ability to remain viable outside of the human body also contributes to its success at diffusion. This may explain why it was one of the first of the Old World crowd diseases to cross the Atlantic, reaching the New World in the second decade of the sixteenth century. It is thus not surprising that it was also the first recorded disease to appear on the margins of the Petit Nord.

The virus which triggered this epidemic appears to have come from Europe on French ships in the summer or early fall of 1669. In the 1740s, the historian Charlevoix wrote that it was the French who passed the disease to the Indians (Charlevoix 1866: III: 154). There can be little doubt that the epidemic started in Quebec, the major port of the

colony (Figure 7). At this time, Canada and the other European colonies along the east coast of the continent remained within the urban disease frontier of the cities of Europe, having yet to develop their own endemic pool of *ACIs*. Each epidemic was introduced from external sources via the major ports, and then passed on. In this respect, one may identify the elements of a complex system of diffusion in which the major colonial communities served to import and redistribute infectious disease to the smaller towns, such as from Quebec to Montreal, to the other colonies, and to the Indians. These colonial ports therefore acted as direct extensions of their larger counterparts in Europe, taking on some of their disease load.

From the 1630s onward, the arrival of ships heavily laden with passengers from Europe had, on many occasions, meant epidemics for the English colonies (Snow and Lanphear 1989; Cook 1973b: 491; See Chapter 3). As population increased in New France through in-migration, it also came to serve as a point of ingress for disease (Jaenen 1976: 98-99). Thus, the Ursuline Mother Marie de l'Incarnation, who lived in Quebec from 1639 to her death in 1672, described disease on board ships arriving from France on several occasions during this period (Marshall 1967). By 1668, the importation of epidemic disease by ships from France was so common an occurrence that in that atypical year she noted "the ships did not bring any maladies this year" (*Ibid.*: 345). Although she did not comment on the 1669-70 smallpox epidemic in her letters to her son, the sickness almost certainly made its way through Quebec. During the summer and fall of 1669, among the ships arriving at that port were two carrying numerous passengers

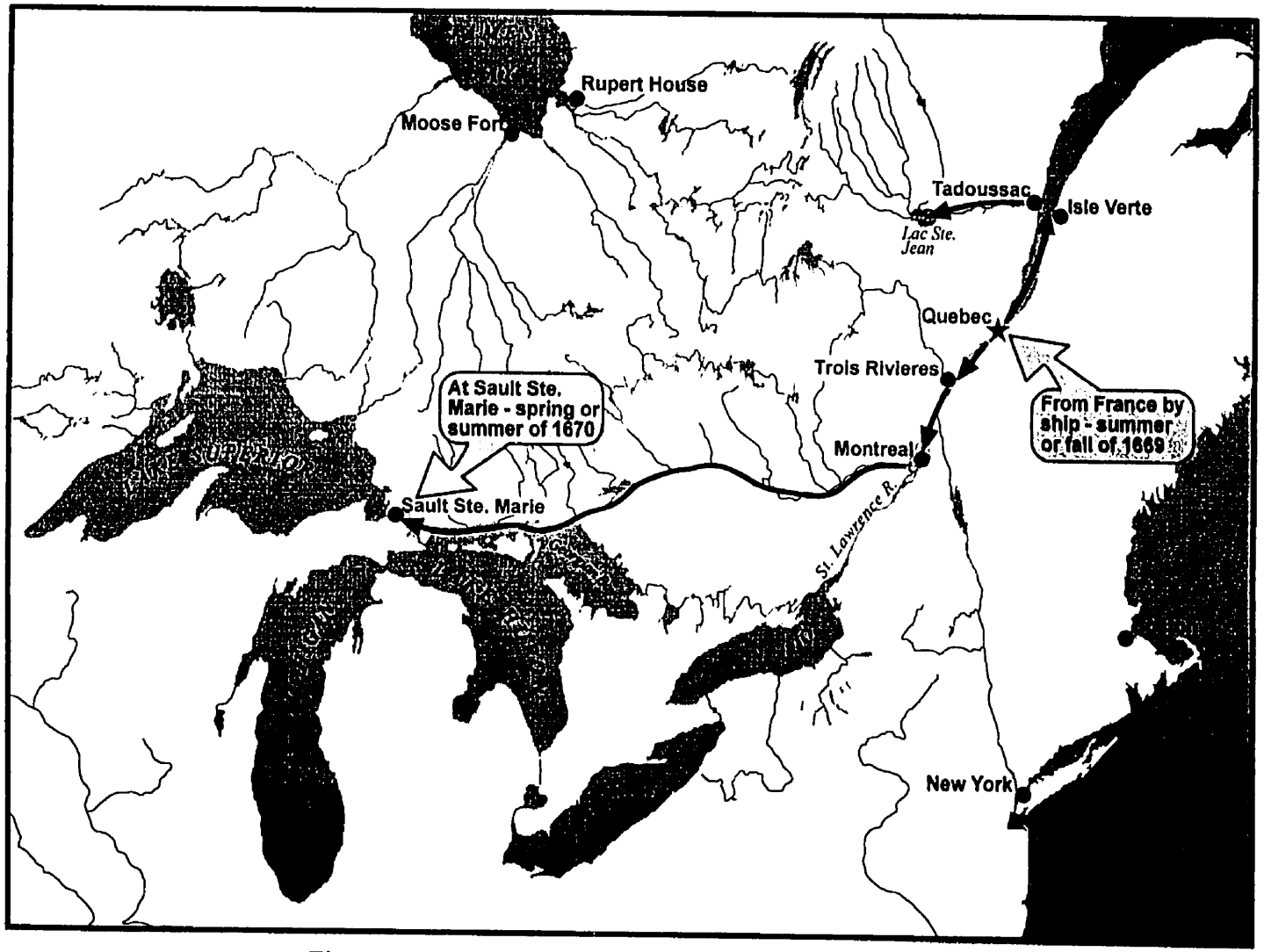

Figure 7: Diffusion of the 1669-70 smallpox epidemic

and capable of transporting the disease across the ocean[15] (*Ibid.*: 353). The first, from La Rochelle, carried men, young women and families. The second, a Norman ship, carried some 150 young women. These ships arrived sometime between late June and October (See *Ibid.*: 348, 353). Although it is not known when the epidemic broke out at Quebec it had reached Tadoussac downstream from Quebec by November (Thwaites 1959: LIII: 61-69).

Once it had broken out in the Quebec area, smallpox wreaked great havoc among the natives in this part of Canada. In the *Relation* of 1669-70 Father Chaumonot, writing from the mission of l'Annonciation de Notre-Dame, a village of converted Huron just outside of Quebec, recorded:

> A year ago, the smallpox terribly ravaged this colony; and the Montagnais and the Algonquins almost all died from it. Our Hurons, who were nearly all attacked by this disease, attribute their recovery to Our Lady of the Faith.... I lost only four persons in all the time during which this contagion lasted (Thwaites 1959: LIII: 124-125).

The Montagnais and the Algonquin of the Sillery settlement, also near Quebec, did not fare as well as the Huron. Father Albanel reported that there had been heavy mortalities at Sillery (*Loc. Cit.*). Thereafter, the Montagnais and Algonquin population was replaced as "Abénaquis were received in such numbers as to make it an Abénaquis mission..." (Charlevoix 1866: IV: 44, fn. 1, quoted in Cook 1973b: 500). Thus, when Baron de

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[15]After Colbert's ascendancy as minister to King Louis XIV, "every summer during the ensuing decade ships brought hundreds of indentured workers, settlers, and marriageable girls..." (Eccles 1972: 76). Largely because of his policies, the population of New France trebled between 1663 and 1683 (Trigger 1989: 283). A ship carrying the Jesuit priest Father François De Crespieul to New France in 1670 carried some fatal infectious disease responsible for several deaths en route (Angers 1969: 161). A ship from France may also have introduced an unnamed epidemic disease in 1685 (Morantz 1991: 219).

Lahontan visited the village in 1684, he met only the Christian Abenaki who had settled there after the epidemic (Thwaites 1905a: I: 48).

Quebec played a crucial role in the diffusion of this and other epidemics, for it was at this time a key meeting place for both the Indians and the whites. Among the major gatherings at or near Quebec in 1669 were a peace conference between the Iroquois and the Algonquian people of the upper country and a convention to choose a successor to a deceased Indian leader, attended by many groups. In both cases, it seems likely that the attendees contracted smallpox while at Quebec. Late in the month of July, Garakontié, the leader of the Onondaga Iroquois, and some of his men journeyed to Quebec to participate in peace negotiations with the upper Algonquins[16] (Thwaites 1959: LIII: 43). This was probably about the time that smallpox broke out at Quebec and the Onondaga appear to have carried the disease back to their homes, perhaps passing it on to other Iroquois tribes. Father Milet, living in the Onondaga villages in 1669-70, wrote that "contagious maladies... were making unusual ravages" there (Thwaites 1959: LIV: 43).

Also in 1669, a ceremony was held near Quebec to replace the deceased Sillery chief, Captain Noel Tekouerimat[17], either during the summer or early fall (Thwaites 1959: LII: 223-224). His successor was Negaskaouat, a Tadoussac war Captain, who was then given the deceased's name (*Loc. Cit.*). This event was of some significance, for it was attended by representatives from several peoples, including the Algonquin, the

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[16]Garakontié was baptized while on this mission, and thereafter supported good relations with the French until his death (Trigger 1989: 291).

[17]According to Morantz (1991: 216), Noel Tekouerimat was chief of the Algonquins at Quebec and at Tadoussac, and was succeeded by his sons Theodore and Charles.

Huron, the Montagnais, the Gaspesiens, the Poissons Blancs (the Attikamegue), the Abenaki, the Etechemins (the Malecite of New Brunswick) and the Nipissing (Thwaites 1959: LII: 227). Smallpox began spreading along the St. Lawrence as a direct consequence of this gathering of diverse groups[18].

From Quebec the disease moved quickly downstream towards Tadoussac, which became a local centre of diffusion of smallpox. It was brought there aboard a French shallop, which departed Quebec on November 14, 1669. This vessel carried the Jesuit Father Charles Albanel and the new Captain Tekouerimat, the former Negaskaouat, who was returning to Tadoussac from the ceremony (Thwaites 1959: LIII: 61). The latter man died shortly after arriving, having been stricken with smallpox three days after departing Quebec and, after the ship landed near Tadoussac, the disease diffused rapidly among the Indians[19] (Thwaites: 1959: LIII: 61, 63). It appeared at nearby Isle Verte, by December 5 (*Loc. Cit.*; *Ibid.*: LIII: 71) and by which time Albanel's mission at Tadoussac had become a hospital for the smallpox victims[20] (*Loc. Cit.*). Also in December, smallpox spread from Tadoussac into the Gaspe region by some Indians returning from the mission, where most succumbed (*Ibid.*: LIII: 71).

The epidemic was not restricted to the shores of the lower St. Lawrence, however,

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[18]This included a Montagnais man by the name of Iskachirini who had gone to Quebec with the French where he contracted the disease (Thwaites 1959: LIII: 93).

[19]The return of the ship eight days later brought fifteen or twenty victims in a dreadful state (Thwaites 1959: LIII: 69). Father Albanel wrote that these people "resembled monsters rather than human beings, their bodies were so hideous, emaciated, and full of corruption".

[20]The epidemic was especially devastating at Tadoussac. Whereas in normal years 1,000-1,200 would have gathered, only 100 had been seen by the spring (Thwaites 1959: LIII: 77). The mission was said to have lost greater than 120 during the previous winter (*Loc. Cit.*), although the total losses including the nearby Indians were undoubtedly much higher.

but moved up the Saguenay River from Tadoussac[21]. When Father Albanel made his journey from Tadoussac to James Bay in 1671-72, he noted that the Kakouchac (Montagnais) of Lac St. Jean had been diminished in numbers recently, "by their latest wars with the Iroquois, and by the smallpox, which is the pest[22] [*peste*] of the savages" (Thwaites 1959: LVI: 155). Significantly, the priest reported no evidence of the epidemic among the nations he met living north of Lac St. Jean, despite their strong trading ties with the people of the south. This lake, according to the priest, had been the "place whither all the Nations between the two Seas, those of the East [the Atlantic] and the North [Hudson Bay], used to repair for purposes of trade" and, at one time, he had "seen more than twenty Nations gathered there" (*Loc. Cit.*). In theory, the northeastern part of the Petit Nord was at risk of the disease diffusing through this long distance trade.

Despite these connections, diffusion to the north of Lac St. Jean seems to have been defeated by the timing of the outbreak, since the first cases did not appear at the Tadoussac area until the winter of 1669. By this time, opportunities to pass the disease on beyond Lac St. Jean were limited. The long distance trade took place during the period of open water, and this was when contact between southern and northern peoples would have occurred. Thus, the disease was spreading northward along the Saguenay only after the northern traders had returned to their homes, when further diffusion of the epidemic in this direction was no longer possible.

There may have been a second factor militating against diffusion along the

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[21]It also seems to have spread to upper New York and New England (Duffy 1951b: 330).

[22]The French term *peste* used in this context may be translated as "pestilence" or "plague", rather than "pest" in order to better convey Father Albanel's intended meaning.

Tadoussac to James Bay corridor. This was the presence of the HBC at Rupert River and, in 1670, at Moose River. By 1671 the English traders were attracting much of the trade which formerly would have gone to Lac St. Jean or to Tadoussac. The tribes which in previous years went south to trade with the Montagnais, and through them the French, were then heading north to trade directly with the HBC, much to the consternation of Father Albanel (Thwaites 1959: LVI: 177). It may be that, although the HBC did not return to the Bay until the summer of 1670 following a hiatus in 1669, the northern Indians remained near James Bay in anticipation of their return, thus defeating diffusion along one of the more frequently used routes to the Petit Nord.

The 1669-70 smallpox epidemic also diffused westward into the heart of the continent, towards the southern flank of the Petit Nord passing along the St. Lawrence from Quebec to Montreal and from there to the Upper Great Lakes (Tanner 1987: Map 32). Once again, the ceremony to resurrect Tekouerimat is implicated, for the disease thereafter appeared at Cap de la Madeleine, adjacent to Trois Rivières (Thwaites: 1959: LIII: 95). This was one of two main communities of the Attikamegue[23] who had visited Quebec to witness the ceremony. Thereafter the disease spread to Montreal, although how soon is unknown, but an Indian youth contracted the disease while there, probably during the spring or summer of 1670 (Thwaites: 1959: LIII: 95; LV: 123).

During the summer of 1670 smallpox and other acute infectious diseases made

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[23]The Attikamegue, many of whom resided near Tadoussac and at the St. Maurice River at Trois Riviere, were devastated by the disease (Charlevoix 1866: Vol. III: 153). Evidence of the disease among the Attikamegue is furnished by Father Albanel who wrote of an Attikamegue women who was "overtaken in the woods by the same disease" (Thwaites 1959: LIII: 95), presumably in her home territory. According to Charlevoix (1866: III: 153), both Tadoussac and the Trois Rivières area were almost completely abandoned by the Indians following the epidemic.

their way to Sault Ste. Marie, an Ojibway fishing and trading village on the St. Mary's River between Lakes Huron and Superior, as well as the host of a Jesuit mission (Figure 7). This was a critical location, being the gateway to Lake Superior. It was also the seasonal home to large numbers of Indians who sought its rich supply of fish. In 1670-71 it was described as "the great resort of most of the savages of these regions, and [it] lies in the almost universal route of all who go down to the French settlements[24]" (Thwaites 1959: LV: 97; See also Heidenreich 1987a). Once the rivers and lakes were free of ice, there was generally considerable traffic passing by, with large numbers of canoes travelling both ways. As this predated the buildup of the French fur trading posts within the Petit Nord and along its margins, the movement of large trading parties from the upper country to Canada was to be expected, and their return trip certainly carried with it the risk of importing epidemic disease. Typical of this period, in 1670 the fur trader Perrot accompanied more than nine hundred Ottawa from the Upper Great Lakes to Montreal in order to trade (Blair 1911: 1: 210).

Epidemic sickness appears to have accompanied Father Gabriel Druillettes to Sault Ste. Marie in the summer of 1670, for no sooner had he arrived than "a grievous disease broke out among the greater part" of those resident at the falls (Thwaites 1959: LV: 117). It is unlikely that this sickness reached this community much earlier, for Dollier de Casson and De Brehant de Galinee had been at Sault Ste. Marie between the 25th and 28th of May, 1670, and made no mention of disease among the Indians. Since they enjoyed the hospitality of Fathers Dablon and Marquette, it is unlikely that Father

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[24]See Coyne (1903: 69-75) for a brief description of the Sault and the Ottawa River route to Montreal written just prior to the arrival of the epidemic.

Druillettes had arrived yet (Coyne 1903: 69-73), and the malady may indeed have coincided with his arrival.

From the wide variety of symptoms described by the Jesuits, several sicknesses must have been extant at Sault Ste. Marie during the summer of 1670. Symptoms included: fever, inflammation of the throat, vomited blood, pain in the loins, paralysis, bloody flux, or dysentery, and the loss of both hearing and speech in one case (Thwaites 1959: LV: 119-121). The most often expressed symptom was, understandably, fever[25]. Father Dablon recorded that "the most common malady was the bloody flux [dysentery], which spread through the whole village, so infecting the atmosphere that even all the dogs were going mad with it, and dying" (*Ibid.*: LV: 123). This disease was epidemic in New York in 1669 (Duffy 1953: 215), and may have spread to Sault Ste. Marie. Although dysentery was emphasised in the *Relation*, it is certain that smallpox accompanied it to Sault Ste. Marie in the summer of 1670. It was also recorded that:

> a young Kilistinon[26], seized at Montreal with an ailment which, during the past year, swept off many Savages, was in a very feeble condition. Upon arriving here from the other side of the river [St. Mary's River], he was so low, jaundice having spread over his whole body, that he had been unable to eat a mouthful for three days; and was even left without the power to move, as if he were already dead. (Thwaites 1959: LV: 123)

It is probable that this man was one of a party of Kilistinon that de Galinee observed

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[25]Plazak (1951) suggested that meningitis was epidemic at Sault Ste. Marie, based on a few symptoms, however it is evident that several different diseases were present at Sault Ste. Marie at this time. The symptoms, which he construed as being the result of a single affliction, were probably caused by several.

[26]In the past, the name Kilistinon and several similar appellations have been treated as equivalent to the now common identifier Cree. Recent research (Greenberg and Morrison 1982; Lytwyn 1993) has put forward evidence calling such broad identification into question. As such, here I will use the terms used by the Jesuits.

preparing to depart Sault Ste. Marie for Montreal at the end of May, 1670 (Coyne 1903: 71), and they may have been part of the large Ottawa group that accompanied Perrot to Montreal[27] (Blair 1911: 1: 211). Despite the reference to jaundiced skin, which is not symptomatic of smallpox, it seems likely that this youth suffered from the effects of smallpox. The mortality which the priest referred to could only have been the smallpox epidemic to the east, which had carried off a great number of Indians living along the St. Lawrence during 1669-70.

Other Kilistinon, including children, were attacked by sickness at Sault Ste. Marie, as was a twenty-two year old Monsoni (*Monsounic*) man[28] who suffered a violent fever (Thwaites 1959: LV: 125). Apparently, the Kilistinon, although they were not normally residents of Sault Ste. Marie, thereafter arrived at Father Druillettes' church in sufficient numbers so as to cause "crowds" (*Loc. Cit.*). The Kilistinon, according to the *Relation* of 1670-71, were "dispersed through the whole Region to the North of this Lake [Superior]" (*Ibid.*: LV: 99). It is uncertain where the Monsoni made their home, but it

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[27]The name Ottawa meant trader, and was sometimes used for all of the Algonquian tribes of the Upper Great Lakes who traded with the French at Montreal via the Ottawa River. Therefore, it is not certain that the Indians accompanied by Perrot were solely of that group which had come to be identified permanently as the Ottawa. On the identity of the Ottawa Indians see the *Handbook of Indians of Canada* (1913: 374-375). On their way the group encountered some Nipissing returning from Montreal.

[28]There were other Monsoni at Sault Ste. Marie at the time of this man's malady, including his uncle who was a shaman, and who acknowledged the success of the Jesuit's religion in curing his nephew (Thwaites 1959: LV: 125-127). The Jesuits report that the ill man arrived at the village already in a state of advanced illness (*Loc. Cit.*). The question which follows, and which must be left unanswered, is whether he was coming from his own home region or from elsewhere.

was almost certainly within the Petit Nord[29]. Thus, although it is not clear whether smallpox diffused into the Petit Nord at this time, it is reasonable to conclude that, in the very least, this and other diseases struck some of the residents of this region[30].

Potentially, these diseases could have reached many of the inhabitants of the Petit Nord, both while they visited Sault Ste. Marie and through trading connections. An indication of the different nations coming to the falls in 1670 may be gathered from a description written by Father Claude Dablon in that year. At that time, the region about the rapids of the Saint Mary's River belonged to the Saulteur (or *Pahouitingwach Irini*, People of the Falls), who shared the summer resources with several other nations who

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[29]Although a roughly contemporary account by Father Albanel (Thwaites 1959: LVI: 203) placed the "Monsounik" along a great river emptying into James Bay, and described some "Kinistinons" settled near by, this may not have been correct. The Monsoni, who would be found at Rainy Lake in the Boundary Waters early in the next century, may have been there in 1670. Warren (1988: 84) learned from Ojibway oral history that "a large band [of the northern division of the Ojibway] early occupied and formed a village at Rainy Lake." Moreover, in 1684 Duluth had a post established at Lake Nipigon to intercept the Monsoni before they traded with the English on James Bay (Margry 1876-86: 6: 20-31), a move that was illogical if they resided near James Bay. This suggests that by 1684 they were living to the west of that lake. Finally, La Potherie, who was at Fort Bourbon [York Fort] later in the century, identified the Monsoni as a group who lived in that post's hinterland and who sought the role of middleman in the fur trade (Tyrrell 1968: 262-3).

[30]The effect of these diseases on the people at Sault Ste. Marie is not clear. The Jesuits claimed that in every case Father Druillettes had been successful at providing assistance and not a single person died, that 300 people were baptised by the following January, and that there was universal acceptance of Christianity, and the elimination of polygamy at Sault Ste. Marie (Thwaites 1959: LV: 125, 127, 129-131). It may be, however, that the Jesuits were not being entirely truthful, perhaps trying to provide the most appealing picture for the audience back in France. Writing in the eighteenth century, the Jesuit historian Charlevoix (1866: III: 158) provided a different perspective, stating that the Jesuits of the Sault Ste. Marie mission "in the first two years [1669-71]... baptised at least three hundred persons, most of them apparently dying children." It may be that Father Druillettes claimed to have halted the epidemic in order to increase his ability to convert the natives (Vecsey 1983: 53).

came from the Upper Great Lakes, particularly for the abundant whitefish[31] (Thwaites 1959: LIV: 133-135). Several unnamed groups, including the aforementioned Kilistinon and an unidentified group called the Ovenibigonc came from the vicinity of Hudson Bay "from time to time" (Thwaites 1959: LIV: 133-135; Hickerson 1988: 45). The ample supply of fish at Sault Ste. Marie supported as many as 2,000 people at one time (Thwaites 1959: LIV: 133-135; Hickerson 1988: 40-43; Cleland 1982: 763).

In addition to those actually residing at Sault Ste. Marie or temporarily visiting, the shoreline of Lake Superior was a meeting place for numerous groups who came long distances for food and trade. In 1665 Father Claude Allouez wrote that:

> This lake is, furthermore, the resort of twelve or fifteen distinct nations—coming, some from the north, others from the south, and still others from the west; and they all betake themselves either to the best parts of the shore for fishing, or to the islands, which are scattered in great numbers all over the lake. These peoples' motive in repairing hither is partly to obtain food by fishing, and partly to transact their petty trading with one another, when they meet. (Kellogg 1959: 105)

In the spring of 1660 Radisson and Groseilliers were met by a party of Crisitinaux at La Pointe, on the south shore of Lake Superior, and the two men subsequently travelled to the north shore, where they met a larger assembly (Nute 1943: 64-65). Despite these connections, there is no evidence that the smallpox epidemic of 1669-70 diffused into the

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[31]According to Harold Hickerson (1988: 45), these groups formed the nucleus of the Chippewa (Ojibway) tribe that began with the merger of several smaller, animal-named groups gradually absorbing many of the smaller clan-based units then living in the vicinity of Sault Ste. Marie. This expansion was supposed to have begun on a large scale in 1670 and, in Hickerson's mind, was engendered by the fur trade and for defensive purposes in light of the threat of war with the Iroquois (*Ibid.*: 50). The presence of the Monsoni, later identified by La Vérendrye as an Ojibway speaking group, may therefore be significant in terms of explaining their alliances. Also in 1670, a Feast of the Dead was hosted by the Amikwa on nearby Manitoulin Island, which involved 1,500-1,600 Indians from several different tribes, including the Saulteurs (Hickerson 1960: 89).

interior of the Petit Nord. The few HBC records from these initial years make no mention of sickness or mortality among the trading Indians although much could have gone on which escaped the notice of the first wave of HBC traders. The most that can be said is that the disease had arrived at the western margins of the Petit Nord, striking at least some of the people who lived there.

The Indians trading at Hudson Bay appear to have escaped *ACIs*, at least for a few years after the arrival of the traders. The scant HBC documents from the first four and one half years of the company's existence show an abundance of trading with nothing that can be as interpreted as indicating epidemic disease. During the inaugural voyage of 1668-69, the men traded with some 300 Indians out of Charles Fort, with no mention of disease[32] (Nute 1943: 119). The next expedition, that of 1670-71, saw Indians arriving at Charles Fort and at Moose River with no apparent significant sicknesses (*Ibid.*: 286-292). In fact, it was the HBC men on the *Wivenhoe* who suffered and died, but from the nutritional disorder of scurvy (*Ibid.*: 136, 289). In July of 1672 Father Albanel reached the mouth of the Rupert River and converted some 62 Indians, but did not mention any overt problems with infectious disease (Thwaites 1959: LVI: 189-201). After an absence between the summer of 1671 and the fall of 1672, when no HBC trader was at the Bay, the English returned. Again, the meagre company records are silent with regard to disease among the Indians, although there is very little information available regarding this year. Likewise, during the 1673-74 season there was no widespread disease worth noting. For this year, however, there is more information available about the individual

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[32]On the return of the *Nonsuch* to England it was recorded that "they report the natives to bee civil and say Beaver is very plenty" (Quoted in Rich 1942: XXV).

trading parties. In October of 1673 several Indians appeared at Charles Fort, including one from Quebec (Tyrrell 1968: 385). A group called the *Cuscididahs* arrived to trade late in March of 1674 (*Ibid.*: 386). On May 27th, 50 *Pishhapocanoes* assembled at the fort (*Ibid.*: 390). At Moose Fort in May there was a party of Abitibi Indians who had been in contact with the French (*Loc. Cit.*). By June there were Albany Lowland Cree at the same post (*Ibid.*: 391). None commented on their health. It is perhaps significant that Thomas Gorst wrote in 1671 that, at Hudson Bay, scurvy "is there the onely [*sic*] disease...[33]" (Nute 1943: 290).

That string of years in which *ACIs* were apparently absent among the Indian people trading with the HBC ended in 1674. In that year, Company servants witnessed the aftermath of a lethal epidemic among some of the Lowland Cree of James Bay. In the summer of 1674, Governor Thomas Bayley (Baily), Groseilliers and several other men explored the western coast of James Bay, and part of Hudson Bay, from Charles Fort as far north as the mouth of the Severn River during a two month voyage in July and August[34] (Bishop 1984: 28; Tyrrell 1968: 391). On July 16, the party sailed by sloop from Moose River, and two days later arrived at the mouth of the Albany River staying until the 21st (Tyrrell 1968: 391). There they met a group of local Lowland Cree. The

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[33]Early in 1674, on January 25th, three Indians arrived at Charles Fort and related having seen "some dead Bodies of Indians" as they passed Moose River (Tyrrell 1968.: 385). It was believed by the Indians that these were some of the *Onachanoes*, and had been killed by the *Nodwayes* (Nottaways). The latter were probably the Iroquois (Lytwyn 1993: 190), although Oldmixon's context suggests that they were Inuit (Tyrrell 1968: 381).There is no reason to disbelieve those who had observed the bodies as to the cause of their death.

[34]The account of this incident derives from Oldmixon's *History of Hudson's-Bay*, written in 1708 (Tyrrell 1968: 371-410), but the details were taken from Thomas Gorst's journal, of which Oldmixon had a copy (*Ibid.*: 383). Gorst had not been on the trip but remained at Moose River. Instead, he was given the account by Governor Bayley (*Ibid.*: 391).

party resumed sailing north, passing Akimiski Island (*Ibid.*: 392). On the 23rd of July they met seven "distress'd" Indians on an unidentified point whom they took on board and transported to the mouth of Ekwan River, some one hundred leagues to the southward (*Loc. Cit.*; Figure 8). At Ekwan River Bayley observed several dead and by way of explanation commented that: "There had been a great Mortality among them, and several were starv'd to Death for want of Food; this Country being such a miserable Wilderness, that it affords not sufficient Sustenance for the wretched Inhabitants" (*Loc. Cit.*). In discussing this incident, both Bishop (1984: 28) and Lytwyn (1993: 289) concluded that simple starvation was unlikely during the summer months in this area, when food resources were among their most plentiful. Lytwyn (*Loc. Cit.*) noted that "fish, waterfowl, caribou and other resources" would have been available during this season[35]. In fact, Lytwyn's research showed that during the summer nearby Akimiski Island was a reliable and plentiful source of caribou as the annual spring migration terminated on the island, where the herd spent the summer (*Ibid.*: 203, 206, 233). Obviously, some other factor must have created the conditions that resulted in the mortality and mass starvation. Bishop (1984: 28) proposed several possibilities, among which was epidemic disease "introduced by Europeans." Lytwyn's (1993: 289) work showed the wide variety of food available and so concluded that "it was more likely that the deaths were caused by a disease transmitted by the European fur traders."

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[35]Bishop (1984: 28) accepted the prevailing belief in the historical literature that the Lowlands region was one of "poverty". In 1672, Father Albanel described Akimiski (*Ouabaskou*) Island based on information gathered from the Indians (Thwaites 1959: 203-205) and praised it as "abounding in all kinds of animals" (*Loc. Cit.*).

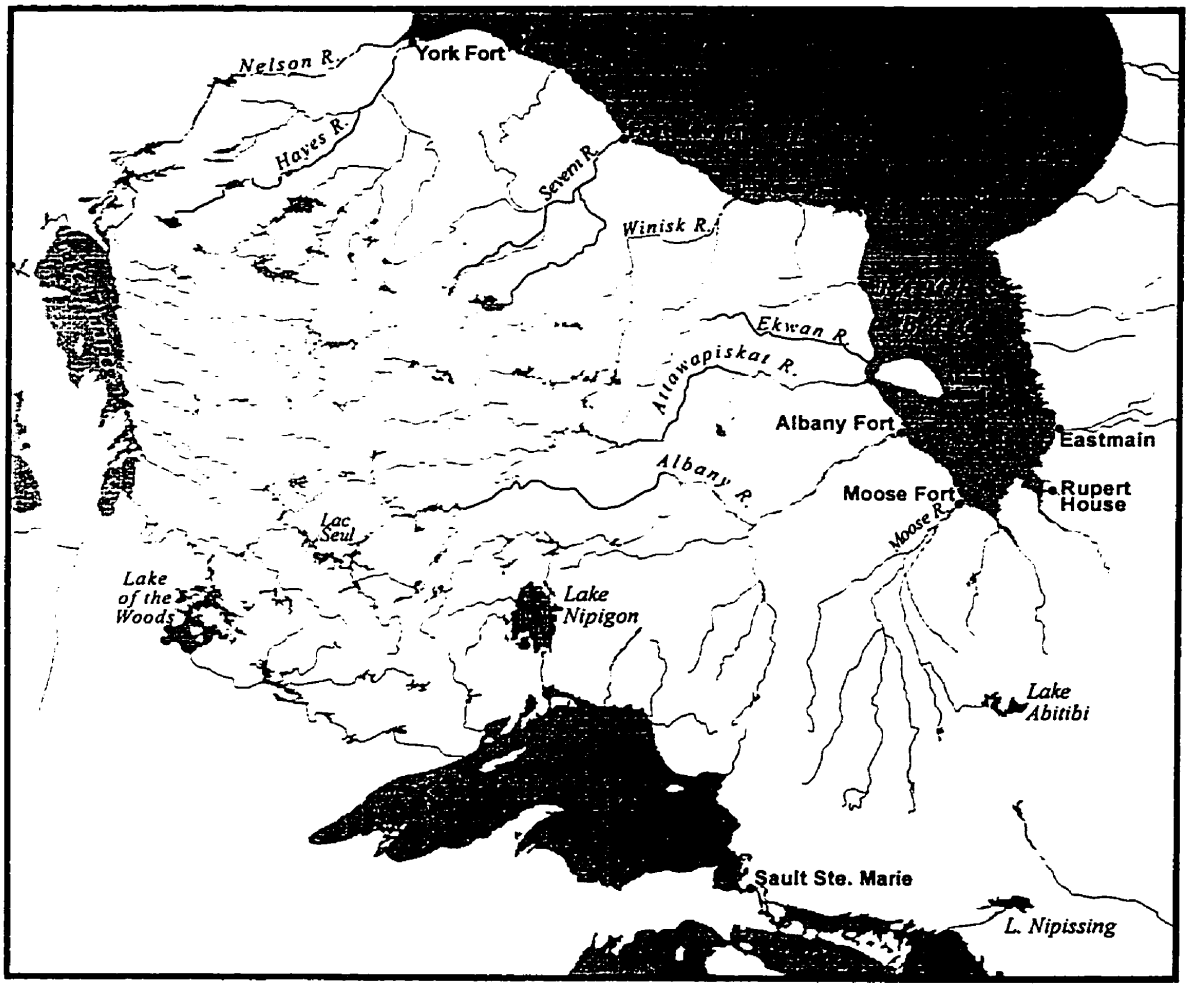

Figure 8: The Petit Nord, 1674-1736

A close examination of Bayley's statement would seem to confirm Lytwyn's conclusion that it was disease. Rather than stating that the deaths were caused by starvation, he noted a "**great** mortality" had been amongst the Indians and that "**several**", rather than all, had died of starvation. The two appear to be of different magnitudes. Given what has been observed in the wake of other virgin soil epidemics, post-epidemic

starvation among the survivors is almost to be expected, particularly among hunter-gatherers. Also significant is Bayley's use of the term "mortality" combined with the preposition "among". The *Oxford English Dictionary* provides several definitions for mortality, but the most appropriate for this context is: "loss of life on a large scale; abnormal frequency of death, as by war or pestilence", especially "a visitation of deadly plague" (*OED*: VI: 674). In the absence of any indication of warfare, it seems reasonable to conclude that Bayley was noting the effects of epidemic disease as well as epidemic induced starvation.

If indeed an epidemic struck these Indians in the summer of 1674, its origin has never been confirmed. As noted above, both Bishop and Lytwyn blamed it on Europeans, with Lytwyn specifying traders. However, it is highly unlikely that the disease was brought by the HBC ships and spread by the traders, as there is no mention of a disorder being brought over at this time. More importantly, as has been noted, there is no indication of sickness among the Indians trading at Charles Fort or Moose Fort in 1674, or during the years prior to this incident. If the illness had been brought by HBC ship, one would expect it to emerge initially among those trading at the forts and to spread from them. Instead, the disease appeared among the Indians summering near Akimiski Island, a group that does not seem to have made contact with the English before. Moreover, none of the other groups met by Bayley's party after leaving Moose River that summer was said to be suffering from the illness (Tyrrell 1968). Without mention of disease among either the traders or those they traded with, it seems likely that the sickness came from elsewhere, and the most likely alternative source is diffusion through inter-

tribal trade[36].

The general location of this epidemic, on James Bay near the Ekwan River and only a short distance to the south of Akimiski Island, may be significant, for there are indications that this area was not as sparsely populated as has perhaps been thought, and that the people who frequented this area had strong connections with those of the south. In the spring of 1659, a Nipissing named Awatanik journeyed from Lake Superior to James Bay as part of a lengthy trading expedition, and later told the Jesuits of his route and of the country he passed through (Thwaites 1959: XLV: 219-229). Having reached James Bay, Awatanik travelled to Akimiski Island, where he found an abundance of food and, nearby, remarkably large Kilistinon villages located along the coast. Some were said to contain more than 1,000 men (*Ibid.*: 225-227). These large villages were doubtless temporary assemblies for the purposes of trade and which took advantage of the seasonably plentiful food resources[37] (*Ibid.*:225-227, 231). Thus, if Governor Bayley witnessed a "great Mortality" at the Ekwan River in 1674, he must have been observing one of these temporary gatherings. Moreover, it is known that the Kilistinon were in close contact with the Indians to the southward at this time, including those of Lake Superior. Given that there is no apparent connection between this outbreak and either the

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[36]Without being able to identify the nature of the epidemic striking these people the source remains unknown. However, there was no shortage of sickness in northeastern North America at this time. For example, there are indications that a variety of ailments continued to afflict the people of the Sault Ste. Marie region into 1672-73, and many people were sick there (Thwaites 1959: LVII: 217-227). An extremely virulent outbreak of an unknown disease carried off many Mohawk living in northern New York between June and September of either 1672 or 73 (*Ibid.*: LVII: 81-83). Once again it may have come from the European colonies.

[37]The Nipissing trader suggested that the Kilistinon lived in nine different residences, some of which included as many as 1,500 men (Thwaites 1959: XLV: 227).

traders or the Indian people trading with the English, it is likely that the sickness had come from the south, and that it had been transported via intra-Indian trade.

Beyond the epidemics at Sault Ste. Marie and the Ekwan River, there is no evidence of significant disease in the Petit Nord or among its people until the second decade of the eighteenth century. Nevertheless, there is evidence at Albany Fort for this period of occasional sickness and mortality among both the HBC men and the local Cree, but none of these episodes fits the pattern expected of *ACIs*. This unhealthy state may not have been the case throughout the initial period, however. It seems that something had begun to change by the second decade of the eighteenth century. In August of 1712 Anthony Beale wrote: "Your country is very much altered to what it was formerly for we have had many sick this winter; likewise both your surgeons [David Bell and John Reed] being dead..." (Davies and Johnson 1965: 24). The following year saw several deaths and brought news of an epidemic in the interior. On June 3, 1713, a single canoe arrived at the fort from up the Albany River. The occupants informed Beale that "there was a sickness amongst them which destroyed abundance of y^{e} french..." (HBCA B.3/a/4: 34d). Nothing more is known of this reported epidemic. However, it is entirely possible that the French had brought some mortal affliction from Canada. Although limited in evidence, this episode is nonetheless highly significant in that it is the first evidence of epidemic disease in the interior of the Petit Nord to be found in the HBC records.

Indeed, it would have been remarkable had epidemic *ACIs* not reached the Petit Nord from the east during this period, for it is evident that mortal epidemics were frequent among the Indian and white populations of the east at this time, with some

approaching the margins of the Petit Nord. This included smallpox reportedly among the Nipissing living just to the east of the region in 1680 (Kellogg 1959: 331), at Mackinac in 1681-82 (Tanner 1987: Map 32) and at Detroit in 1703[38] (Drews 1939: 761). Undoubtedly, other diseases without the high profile of smallpox were also epidemic at times, but they seem to have been given less attention in the secondary sources. Dobyns identified many episodes of diseases that he concluded were not smallpox, among them several that occurred in the general vicinity of the Petit Nord during the period 1670-1713. These included: measles among the Illinois and Oneida in 1692-93, and among the

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[38]The number of major epidemics during this period was remarkable as, time and again, ships from Europe and the West Indies brought infectious diseases to the Atlantic colonies. In 1680, Duluth recorded in a memoir that the Abenakis were spreading a rumour that smallpox "was in the settlements of the French, and that it had gone up as far as Nipissinguie, where the greater part of the Nipissiriniens had died of it" (Kellogg 1959: 331). The previous year, the same sickness, brought from Albany and Manhattan, ravaged the Five Nations Iroquois and, about this time, the Iroquois living at Caughnawaga near Montreal (Heagerty 1929: I: 28-29), and so the reports may have been more than just rumours. In 1681, it was feared that smallpox would be brought to Canada by Ottawa traders, who were then suffering from it, and so they were refused permission to come to the colony (*Ibid.*: 30). Two years later, in 1683, the Indians of Green Bay, on Lake Michigan, suffered tremendous losses due to an unidentified epidemic disease, and in 1684 the French army, under de la Barre was struck by another unidentified illness while on campaign in the interior (Blair 1911-12: I: 242, 293, 354). In 1685 a highly mortal epidemic was responsible for the deaths of thirty-five sailors from France, and struck the Tadoussac region, at the mouth of the Saguenay, spreading to the Indians (Morantz 1991: 219). Cook (1973b: 494) noted that a ship from France brought both typhus and measles to Canada in 1687, and the Christian Indians suffered as a result. See also Caulfield (1943: 533) who provided evidence that the ships from France brought measles to Canada in 1687. The same disease was active in New England also in 1687-88 (*Loc. Cit.*), and no doubt the two were connected. Between 1688 and 1691 smallpox was said to have been extremely widespread, carried far afield during King William's War (Duffy 1981: 66-67), and quite possibly spread to the interior as some unnamed diseases afflicted the Illinois Indians in August of 1692 and during the winter of 1693 (Blasingham 1956: 383). Smallpox was among these same people during the period ca. 1701-1704, and so perhaps linked to the outbreak at Detroit (*Loc. Cit.*). It was also among the people of the Great Lakes region in 1701, and at Montreal during the peace conference of the summer of 1701 (White 1995: 143). Heagerty (1928: I: 33) noted that smallpox was prevalent in Canada in 1699 and again in 1702-03, when 2,000-3,000 people died. During 1702 the disease was epidemic in Boston and New York (*Ibid.*: 33-34), and Duffy (1981: 68) stated that it was carried northward to Quebec by Indians. Scarlet fever was said by Dobyns to have struck the Great Lakes tribes in 1708-10 (Dobyns 1983: 22). Finally, there may have been a widespread epidemic in the Mississippi Country in 1714. Blasingham (1956: 383-384) noted the effects of an unnamed, but severe, sickness among the Indians of Kaskaskia, in the Illinois country, during which 200-300 died, while the John K. Bear winter count of the Lower Yanktonai identifies an epidemic sickness causing cramps and convulsions for the same year (Howard 1976: 27). However, Dobyns (1983: 19) identified the Kaskaskia sickness as measles. If so, these outbreaks were not related. Caulfield (1943: 534-538) presented evidence for measles in the English colonies between 1713 and 1716.

Illinois in 1713-15[39]; influenza among the Iroquois in 1675; and scarlet fever among the Great Lakes tribes in 1708-10 (Dobyns 1983: 17, 19, 22). Nevertheless, with documentary evidence limited during this period, nothing can be concluded regarding the presence or absence of these diseases in the Petit Nord.

1714-1736

The period 1714-1736 witnessed occasional, though infrequent, epidemics at both the Bayside posts and in the interior of the Petit Nord. Although sickness appears in the records, there were also many years in which it was entirely absent. Unfortunately, the terse nature of the records makes it difficult in most cases to identify the ailments as *ACIs*, let alone to name the specific illness, and the paucity of French records generally makes it impossible to link these outbreaks with others occurring elsewhere in North America. It is conceivable that some of the incidents refer to chronic ailments aggravated by starvation. It is less likely, although not impossible, that vectored afflictions, for example typhus or even plague, had been carried into the region. Thus, it is often necessary to speak of epidemics or outbreaks in the region during this early period without identifying them as *ACIs*.

At Albany, there are very few comments in the journals regarding widespread sicknesses, although chronic and individual afflictions are noted. The ninth Albany Fort journal, covering 1715-20, is particularly interesting for what it says about the medical terms being used in the Hudson Bay country. During this period Thomas MacLeish noted

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[39]In both these cases among the Illinois, Blasingham (1956: 383-384) left these diseases unnamed, however Dobyns linked them to outbreaks that he identified as measles.

occasional instances of colds or "great colds" among the men, culminating in a major outbreak in the spring of 1720 (HBCA B.3/a/9). In the English usage of the day, a "great cold" might have meant influenza, as the latter term had yet to enter the English language (Duffy 1953: 192). Seven years later, an infectious disease was afflicting the Albany River Lowland Cree. On May 25, 1728, Joseph Myatt wrote that:

> this morn Eight Curnoes of our home Ind^{s} fitted out from here in order to goe to Warr wth the Esquomays, I Endeavored all I could to oppose it, but in vain, for severall of the Home Ind^{s} being Disordered the last winter they attribute all those things to the Mallice of their enemies... (HBCA B.3/a/16: 18)

Myatt's description rings true. According to an English seaman who overwintered at York in 1746-47, when a sickness occurred among the Lowland Cree they went to war with the Inuit, whose sorcery they blamed for their misfortunes (Drage 1748: I: 43-44). In placing the blame for their sickness on the Inuit, the Cree were demonstrating a disease etiology that was traditional among many aboriginal peoples in North America.

Two years later, in 1730, the Albany Fort journal again mentioned what may have been an *ACI,* but this time among the Indians of the interior. Early in May, thirty-eight canoes of Uplanders arrived at the fort with only a relatively small amount of furs to trade, their complaint being that "it hath been a very hard year with them, they haveing been very much afflicted wth sickness..." (HBCA B.3/a/18: 16). Two weeks later, seven canoes of Sturgeon Indians arrived from the interior and brought news that it had been a very sickly year in their home country, and that several of their people who normally traded at Albany Fort had died, including a leading Indian named Weques (*Ibid.*: 17). These Indians probably came from up the Albany River, where the French had established

a strong presence, suggesting that this disorder originated in Canada. However, neither the identity nor the source of this disease is known.

Although it is impossible to determine how widespread this epidemic was in the interior, it is possible to identify the territory of one of the afflicted groups. The Sturgeon Indians who arrived at Albany Fort on May 25 almost certainly came from the eastern part of Lake of the Woods. In the early 1740s La Vérendrye identified a group of Ojibway speaking people residing at the latter place as the *gens de la Baye d'Esturgeon* or Sturgeon Bay people[40] (Greenberg and Morrison 1982). Joseph La France, who travelled through Lake of the Woods during 1741-42, also identified these people as the Sturgeon Indians[41] (Dobbs 1744: 34). Thus, this mortal epidemic was at least among some of the people of Lake of the Woods, and probably others, since the first group arriving at Albany Fort was not identified as Sturgeon. Unfortunately, French expansion into the Lake of the Woods region under La Vérendrye came after this sickness, and there is no additional evidence to further illuminate it.

Only a few years later, the Albany Lowland Cree again suffered from another epidemic disease. During November of 1732 reports indicated that there was "a great sickness among them", although the illness was not identified (HBCA B.3/a/21: 8). It seems likely that the disease diffused to the Lowland Cree around Albany from the south,

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[40]There is a Sturgeon Lake located between Lac Seul and Lake Nipigon, in the Albany hinterland, which conceivably could have been home to "Sturgeon Indians". However, an entry in the Albany Fort journal of 1746-47 suggests that the "Catt" Indians inhabited that lake (HBCA B.3/a/38: 26). Lytwyn (1993: 123-124) noted that in 1814 the Sturgeon people still occupied a position very near Lake of the Woods, as described by HBC trader George Holdsworth.

[41]They were called this because of "the great Number of Sturgeons they take in this Lake, which is the Greatest part of their Provisions" (Dobbs 1744: 34). A map drafted from La France's information shows the Sturgeon Indians to the north of Lake of the Woods (Ray 1988: 17).

and that the Indians of the interior were also exposed at this time. Significantly, a report arrived at York in the spring of 1733 to the effect that "severall [Upland] Indians Dyed last winter with Ailements but a far greater number was famishd..." (HBCA B.239/a/15: 30d). If this was an *ACI*, the source was likely Canada. In 1732 it was reported that the French had established two posts somewhere on the Albany River (Lytwyn 1993: 195 fn. 129), and that several Canadians had wintered with the Upland Indians (Morton 1939: 178). At that time smallpox was carried into Canada from New York, where it had been active since 1731. It also travelled westward, eventually reaching the western margins of the Petit Nord several years later (See the following chapter). At the same time, the Ojibway were moving north into the lower reaches of the Albany River, where they were trading with the Uplanders, and they were also capable of transporting the virus. In a letter dated July 31, 1733, Joseph Adams of Albany Fort wrote:

> I had last May some strange Indians who informed me that the French Indians [Ojibway] have several of them wintered with the upland Indians, having some places fortified, with trading goods in them about two hundred miles from this place, intercepting the Indians that use to come here and trading with those that are unwilling by compulsive methods which must certainly be very prejudicial to your interest. (Davies and Johnson 1965: 180-181)

Many of the Ojibway and Ottawa of Lake Huron were trading with the English at Oswego despite being aligned with the Canadians (Innis 1956: 102). Oswego lay in the heart of the smallpox epidemic, and there can be no doubt that these Ojibway traders were getting their goods from the east, whether from the French or English.

The situation at York Factory during this period was somewhat different. Almost immediately after James Knight arrived in the fall of 1714, there were reports of

epidemics among nearby Indians. The following summer came news of a fatal illness among some Indians who had left York for Churchill (HBCA B.239/a/1: 46d, 50d; B.239/a/2: 21d, 22). During the summer of 1716 a great many Indians were sick and died, although this may well have been the result of starvation due to the non-arrival of the ship, rather than due to an acute infectious disease (HBCA B.239/a/2: 45d, 58; B.239/a/3: 7). A "malignant feaver", interpreted by Ewart (1983: 573) as influenza, raged among the Cree of the York region during the winter of 1716-17, and among some Chipewyan living at the post and with the Cree (HBCA B.239/a/3: 19d, 25, 25d, 28d, 32, 33). The sickness afflicted the latter Indians "one after the another" and was, according to Knight, "as mortal as if they had the Plague" (*Ibid.*: 32, 33d). Whatever its identity, this was likely an *ACI*. All of this was a prelude to the arrival, in 1720, of the first documented outbreak of smallpox in the Petit Nord.

On March 23rd, 1721, Governor Henry Kelsey[42] recorded in his journal that "2 of the Capt [Captain's] family came here for food and say he and some others are very ill altho most of the Indians that have lain here all winter have had the Small Pox which I never saw amongst the home Indians before" (HBCA B.239/a/6: 15). The smallpox which Kelsey reported among the Homeguard Cree at York Factory during the winter of 1720-21[43] may have persisted into 1722 among the remainder of the Lowland Cree living

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[42]Both Decker (1989: 59) and Lytwyn (1993: 354-356) have noted Kelsey's statement. Lytwyn tracked the outbreak's lingering effects among the Lowland Cree.

[43]Kelsey makes it clear that the plight of the Captain and his family was not due to smallpox but to some other affliction. This is borne out by the prolonged course and symptoms of the illness in the case of the Captain. Between the first mention of his illness on September 23, 1720 (HBCA B.239/a/6: 3) and his death in January of 1722 (HBCA B.239/a/7: 11d), the Captain suffered from a bewildering array of symptoms, and he had been "spitting blood" since the winter of 1718-19 (HBCA B.42/a/1: 42d). Most probably, he was suffering from an advanced case of pulmonary tuberculosis.

in the York Factory hinterland, or even among those living beyond the Homeguard (Lytwyn 1993: 356). In November of that year, Kelsey's replacement, Thomas MacLeish, recorded that: "two Indian women came here down the River having left 22 Indians young and old a coming to the Factory almost starved having mist [missed] of the Deer passing in the Fall, likewise a sickness amongst them" (HBCA B.239/a/8: 7d). Here the Cree were calling on a fur trade post in the wake of epidemic-induced starvation, a pattern that would be repeated many times throughout the fur trading lands of the Canadian Northwest over the next few centuries.

It is difficult to discern whether smallpox spread beyond the immediate York Factory hinterland at this time. In 1720-21 the HBC operated only two other permanent posts on the Westmain besides York: Churchill and Albany. The Churchill records are silent on the subject, suggesting that the disease did not diffuse there (HBCA B.42/a/1; 2). Likewise, the Albany journal leading up to August of 1720 (HBCA B.3/a/9) also makes no mention of the disease. Unfortunately, the crucial Albany journal for 1720-21 is missing. Lytwyn (1993: 356) concluded that the disease did not reach the Albany River area although some Albany Lowland Cree visiting York Factory had apparently succumbed (HBCA B.3/a/11: 19). Tentatively, then, it may be concluded that the smallpox outbreak of 1720-21 was limited to the immediate vicinity of York Factory, at least within the hinterlands of the HBC posts.

Kelsey's statement that he had yet to see smallpox among the Homeguard Cree is particularly significant given his experience at Hudson Bay. For almost 40 years, between 1684 and 1722, he was a key figure in the HBC's employ, and was rarely absent

from the Bay (Davies 1969: 308). By 1721 he had served at Albany and York, was instrumental in the resettlement of Churchill, and captained the *Knight* in a ship-based Eastmain trade out of Albany (*Ibid.*: 311, 312). As well, between 1718 and his recall in 1722, he was Governor of all the Bayside posts (*Ibid.*: 312, 313). Moreover, he had a facility with several Indian languages and a keen and curious mind, and he played a critical role in expanding the company's geographic knowledge base through his explorations and inquiries (Epp 1993; Doughty and Martin 1929; Ruggles 1991). Perhaps more than anyone in the HBC at that time, then, Henry Kelsey had an intimate and longstanding knowledge of the Indians of the Hudson Bay Lowlands. Thus, his statement can be considered key testimony regarding the epidemic history of Hudson Bay[44].

The search for the origin of this outbreak is hindered by the paucity of data from this period, and may only be inferred. There is no indication of a local source among the aboriginal people of this area although, in the summer of 1720, Richard Staunton at Churchill was informed that there had been "a great mortality" among the Inuit living far to the north during the previous year when one of the HBC sloops had visited them (HBCA B.42/a/1: 86). This statement may be interpreted as referring to epidemic disease. Nevertheless, it is unlikely that the smallpox at York Factory was connected with the Inuit mortality. There is no indication of disease among the Indians living in the

[44]Kelsey's experiences become all the more significant when it is remembered that the HBC had only begun visiting Hudson Bay in 1668, when they constructed Charles Fort. Moreover, the Company's men did not stay in the country year-round until 1673, and so observation was not possible when the ships had left for England (Morton 1939: 50, 73). Thus, between the year-round occupation of the Hudson Bay posts by the HBC men and the arrival of Henry Kelsey at York Fort in 1684, there was a period of only 11 years during which he had not been present at the Bay and when he was not in direct contact with the

Churchill hinterland, nor is there a record that the Lowland Cree had gone to war with the Inuit in the summer of 1720, as they occasionally did. It would seem that there was no contact between the two infected groups at this time[45]. This would leave the HBC supply ship from England as the most likely source of the disease. In 1720, the *Hannah* frigate left London and arrived at York Factory on September 3, after a trip of some eighty-nine days (HBCA C.4/1: 3d-4). Significantly, smallpox was "exceedingly prevalent" in London during the spring of 1720, or at the time of the *Hannah's* departure from that city (Creighton 1965: 2: 449), and it may well have carried the disease to Hudson Bay. While the length of the voyage and other factors may have precluded the survival of the virus by a series of person to person transmissions, it may have remained viable externally, perhaps on the surface of trade goods intended for Hudson Bay, or on some other fomite.

Moreover, the timing of the outbreak is consistent with a ship-based origin. Initial infection would have occurred some time during the first half of September after the ship arrived, and before the Homeguard prepared to disperse to their wintering grounds or go to the fall goose hunts. Allowing additional time for diffusion among the Homeguard Cree, a prolonged wintertime epidemic, such as was described by Kelsey, would have resulted. This would also explain the limited diffusion of the epidemic. By the time the ship arrived at Port Nelson the Uplanders had already departed, and were therefore spared

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Lowland Cree. His statement, then, covers much of this period during the early history of the HBC.

[45]Indeed, the once-common warfare between the Inuit and Lowland Cree of the York Factory area abated after the founding of Churchill Fort in 1717, as the Inuit removed themselves from the immediate area (Lytwyn 1993: 163).

the effects of the epidemic. The sickness could go nowhere but among the local Cree population. Thus, for the Lowland Cree of the York Factory area exposure to smallpox came early, the first of several outbreaks of that disease that would afflict them during the eighteenth century.

Following this outbreak, the remainder of the period to 1736 was relatively healthy at York Fort, save for a few colds among the men in June of 1724, and a single case in September of 1727 (HBCA B.239/a/8: 36; B.239/a/10: 5d). There was not much more among the Indians. In November of 1722 there was an account of starvation combined with sickness among a group of Lowland Cree who had been unable to hunt deer (HBCA B.239/a/8: 7d), and during the summer of 1727 the journal noted the suspicious death of fifty families of Upland Indians (HBCA B.239/a/10: 22d). Finally there was the sickness among some Uplanders during the winter of 1732-33. Overall, epidemic activity at York and in its hinterland was not uniform throughout this period, but occurred more frequently prior to 1723.

SUMMARY

The last three decades of the seventeenth century witnessed significant changes in the historical geography of the Petit Nord. The founding of the HBC in 1670 resulted in the establishment of several permanent fur trading posts on Hudson Bay. There is only one instance during this period that the HBC ships can be associated with introducing *ACIs* into the Petit Nord: the smallpox brought to York in 1720. More significantly for

the health of the people of the Petit Nord, the founding of the Company promoted changes in the spatial structure of the French fur trade, leading to greater expansion into the region from Canada. In turn, these movements had two further repercussions of importance in the context of this study. First, they led to an increase in the number of literate observers in the region, and of our knowledge of events occurring in the region and around its margins. Secondly, they increased the possibility of diffusion of *ACIs* to this region. In addition to the HBC supply ships, the annual fur brigades from Canada almost certainly introduced such diseases to the region on occasion, and the new posts which attracted trade from long distances could then serve as points of redistribution. Although it cannot be documented, it is probable that, following 1670, the incidence of *ACIs* in the Petit Nord increased, and that the region had taken a first step towards incorporation into the hinterland of distant urban disease pools.

Although the documentary record is sparse, two patterns of infectious disease emerged in the Petit Nord by 1736. The first is that sickness, some of it obviously infectious, was in no way rare in the region during this early period. Many of the early journals contain references to sickness among the men and Indians, although much of it was chronic and most afflicted only one or two individuals. The second suggests that *ACIs* in the form of outbreaks or larger epidemics were occasionally present, both at Hudson Bay and seemingly more frequently among the Indians of the interior. Prior to the Treaty of Utrecht, this included: a major smallpox epidemic which was probably carried from France to Canada by ship and which reached the margins of the region in 1670, afflicting some of its inhabitants; an outbreak in 1674 of some mortal affliction

along the western coast of James Bay; and a suspicious sickness among the French traders of the interior during 1712-13. Following 1714, a period for which more complete evidence is available, *ACIs* remained an occasional threat to the Indians, including unidentified epidemic complaints in the first few years at York Fort and an outbreak of smallpox in 1720, probably from the HBC ships. In 1729-30 there was a mortal epidemic among the Sturgeon Indians living at Lake of the Woods and at least one other unidentified group of Uplanders. In 1732-33, the Albany Lowland Cree were afflicted. At both Albany and York the HBC men suffered from occasional respiratory afflictions, commonly referred to as "colds" or "great colds". While by no means common, it is thus apparent that *ACIs* had begun to reach the region from external sources during this period.

CHAPTER 5: THE 1737-38 SMALLPOX EPIDEMIC

In 1737-38 smallpox struck the Indians living on the western margins of the Petit Nord. While there had been several epidemics in the interior of the region prior to 1737, in some cases causing significant mortalities, the evidence for these is limited to brief reports at the HBC's Bayside posts. The 1737-38 epidemic, in contrast, followed upon the penetration of the French into the Lake of the Woods-Red River region during the early 1730s under the charge of La Vérendrye[1], and the French records of this period are the first derived from the interior to describe epidemic activity in the Petit Nord. It is these records which provide the best evidence of the course of this epidemic, and which is only suggested in the HBC records.

Although it has been noted by several scholars, this epidemic is described only in passing in the secondary literature as a smallpox outbreak that originated among the English at Hudson Bay and spread among several groups in the Lake Winnipeg region. There has been little consensus as to when the epidemic occurred or even who was struck. Several, such as Milloy (1988: 70), Taylor (1982: 35) and Morton (1939: 18) have stated that it occurred in 1737. Others, including Morice (1910: 35), Heagerty (1928: 1: 36-37), Decker[2] (1989: 58) and Hurlich (1983: 157), specified 1738. The only common point

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[1]Pierre Gaultier de Varennes et de La Vérendrye, hereafter referred to as La Vérendrye. Note that his detailed journal for 1737-38 is missing. In its place Burpee published a translation of the summary journal which covers the major events of the period, though not in as great detail (Burpee 1927: 40).

[2]Decker (1989: 58) called it both the 1737-38 and the 1738 epidemic, the latter date based on Heagerty as well as an unidentified work by Duffy. Mandelbaum (1979: 44) mistakenly dated it to 1736. Although Ray (1988) studied several later epidemics (notably the 1779-83 smallpox epidemic) in his pioneering study of the Indians of western Canada, he did not take the 1737-38 epidemic into account. Failure to acknowledge earlier epidemic events may be a significant flaw in works which assume that epidemic disease was absent prior to 1780 and had no effect on native lifeways.

would seem to be that the disease came from Hudson Bay[3]. In large part this has been due to the difference in their sources. Those who examined a portion of the primary material concluded that it took place in 1737. Others who relied on a secondary source, generally either Morice (1910) or Heagerty (1928: 1: 36-37), have suggested 1738. A re-examination of all the primary sources, however, clarifies the matter, and shows that the epidemic occurred in both years, although it did not come from Hudson Bay.

Smallpox was first recorded by La Vérendrye during the spring of 1737. On the 26th of May, 1737, at Fort St. Charles on Lake of the Woods, he noted that:

> sixty Barrier Cree arrived and told me that the Winnipeg Cree [*les Cris du Oüinipigon*] whom I had left at fort Maurepas had all died of small-pox, which had been brought to them by those who had gone to trade with the English [*qui leur a été portée par ceux qui ont eté traiter chez les Anglois*]. I did not fail to tell them that the Master of Life had punished them for not having come to fort St. Charles as they had promised (Burpee 1927: 256-257).

These Winnipeg Cree probably lived on the Winnipeg River, towards Lake Winnipeg, although it is apparent from this statement that their neighbours on the same river, the Barrier Cree, were not affected at this time[4] (Greenberg and Morrison 1982: 93; Figure 9).

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[3]Even in this, there is not total agreement. Morton (1939: 18) acknowledged the implication of the English but speculated that it might have been linked to smallpox spreading from Canada. Dobyns (1983), in *Their Number Become Thinned,* came to a different conclusion and implied that it was part of an epidemic in 1738-39, which he believed affected native people from the Southeast to Hudson Bay, and in Texas (Dobyns 1983: 15). He provided no reference for the Hudson Bay information, although in dating it to 1738 it would appear that he made use of either Morice (1910: 35) or Heagerty (1928: 1: 36-37). Both of these noted the disease coming from Hudson Bay in 1738, a view contrary to that proposed by Dobyns.

[4]Barrier Falls is now known as Sturgeon Falls and is some forty-five miles upstream from Portage de L'Isle (Morse 1969: 90).

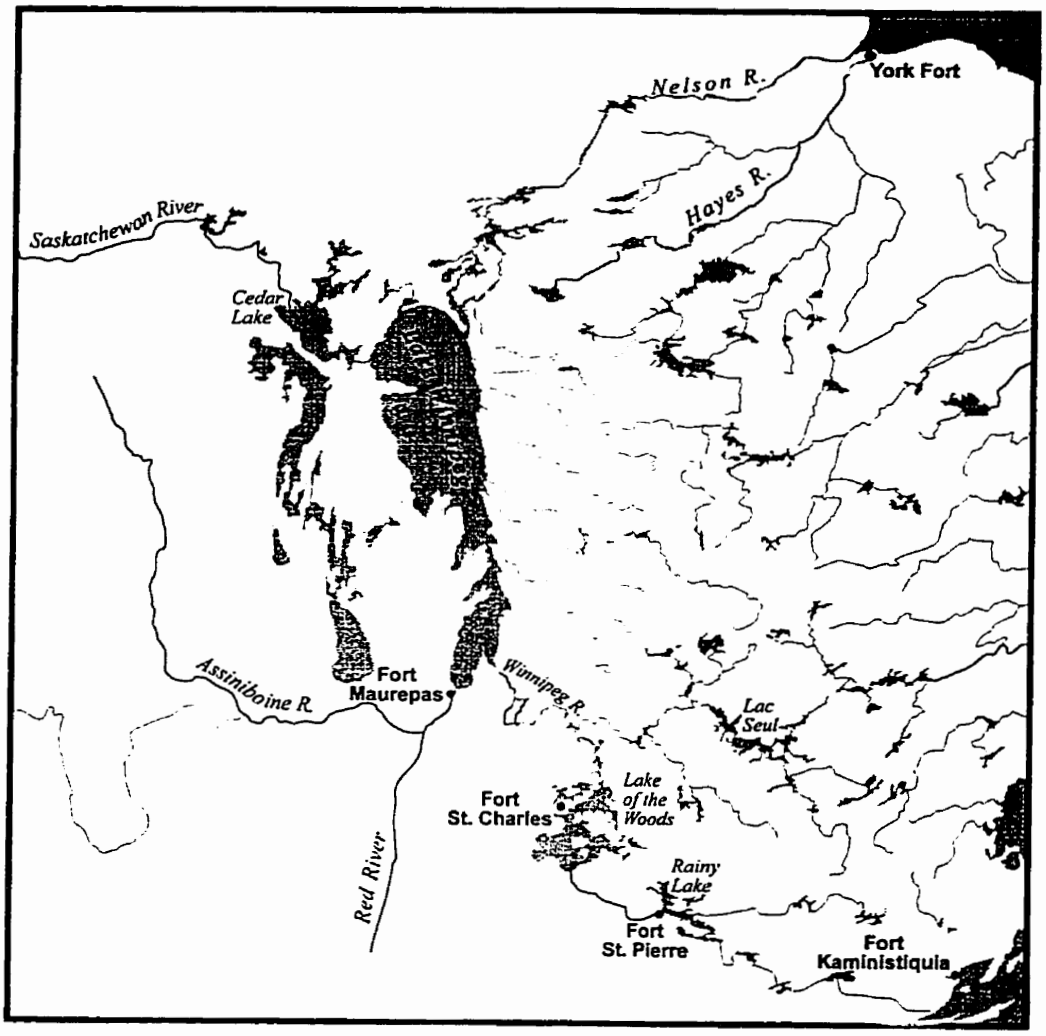

Figure 9: The 1737-38 smallpox epidemic

The reference is not to 1738, as is commonly stated in the secondary literature, but to 1737. Moreover, La Vérendrye did not claim that the disease had been contracted from the English, nor that it originated at Hudson Bay, but only that it came from those who had traded with the English. Long-distance trade to York Fort by plains tribes had been common practice since the seventeenth century, and at this time included people living not only in the Petit Nord, but as far west as the Rocky Mountains, and as far south as the Missouri River (Ray 1987a; 1988: 51-70). Indeed, trade with the HBC was common

even among the Indians with whom La Vérendrye dealt, due to chronic shortages of trade goods among the French (Burpee 1927: 208, 215, 231).

Additional news of smallpox reached Fort St. Charles two days later. On May 28, La Vérendrye's son, Louis-Joseph, arrived after a sojourn with an unnamed band of Cree. He reported that after the disease had broken out amongst these people:

> Those who escaped made a stop and threw into the river, according to their custom, all the beaver, pichoux, marten, etc., belonging to the dead as well as their own, so that the shore was lined with them and the portages full, all of which was a loss, as no one among the savages ventured to touch them. In the ten lodges that were with my son there was not one death; this was due to the remedies he gave them and the good care he took of them, which augmented their friendship for him and the French as well as their confidence in them; but they stopped like the others to succour the rest of the afflicted families. Only eighteen men came with him to join the warriors. (Burpee 1927: 258-259)

There is little doubt that La Vérendrye's description of the abandoned furs indicates widespread mortality. Similar acts of bereavement were performed by the Hudson Bay Lowland Cree on many occasions (see Lytwyn 1993: 375, especially fn. 3) and, as we shall see in Chapter 11, this was an almost universal reaction to the death of a close relative.

The afflicted Cree probably came from the north end of Lake Winnipeg. Louis-Joseph had been sent with these people in March to help choose a site for a future French post (Kavanaugh 1968: 134), a post that was later settled on the Saskatchewan River near Cedar Lake. It is not known how widespread the disease was, but the information provided to La Vérendrye implied that the epidemic had claimed many victims among

these people, other than among the ten lodges with his son[5].

Both Cree groups had attended a council with La Vérendrye, held the previous fourth and fifth of March at Fort Maurepas on the Red River (Burpee 1927: 249-255). The Council of Maurepas was a general meeting with some Cree and Assiniboine bands, attracting people from a considerable distance, although the Cree and Monsoni from the Lake of the Woods and eastward appear not to have participated (Burpee 1927: 242). The impetus for the meeting was a desire on the part of La Vérendrye to prevent retaliatory strikes by the Assiniboine, Cree and Monsoni against the Dakota Sioux, who had massacred La Vérendrye's son, Jean Baptiste, the Jesuit Father Jean-Pierre Aulneau and several voyageurs early in June of 1736 on an island on Lake of the Woods (Burpee 1903: 219; 1927: 215-216). Discussion included other subjects, notably the construction and relocation of French posts (Burpee 1927: 250, 252). Since the two afflicted Cree groups had attended, it is probable that one or both were infected during the meeting.

Rather than disappearing from the area following its outbreak in 1737, the epidemic was again active the next year. In a letter dated October 1, 1738, Beauharnois, the Governor of Canada, wrote that he had:

> received a letter which the son of [La Vérendrye] wrote me from the Lake of the Woods on the eleventh of last May, by which he informs me that the

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[5]This mortality among the Cree living at the north end of Lake Winnipeg may have triggered population movements among the Northern Ojibway. In his study of the historical geography of the Lowland Cree, Lytwyn (1993: 127) concluded that the Northern Ojibway, under the name "Bungee", did not appear in the York Factory records until 1741, after which date they appeared frequently. Consequently, it would seem that the Northern Ojibway had not traded at York until that date, at least with any regularity or in large numbers. Lytwyn further suggested that this date "may have also indicated their recent arrival in the York Factory hinterland", as they began a northward territorial expansion (*Ibid.*: 128). Given the apparent mortality among the Cree, it is possible that valuable fur producing territory in the northwestern part of the Petit Nord and along the Saskatchewan River had been opened in the wake of the 1737-38 smallpox epidemic, freeing the way for Ojibway population movements.

> Assiniboin to the number of eight hundred had left at the end of April to go and take vengeance on the Sioux for [the massacre], and that the Cree and the Monsoni had also raised war parties, though he does not give the numbers, but the small-pox broken out among them they were obliged to stop with the loss of a considerable number of their people carried off by that disease[6] (Burpee 1927: 282).

The passage seems to indicate that in late April of 1738 the Assiniboine, Cree and Monsoni had all intended to go to war against the Dakota but that the latter two groups had been prevented due to the outbreak of smallpox[7]. Most likely they fell ill somewhere between the Boundary Waters and the Dakota Sioux territory, on the way to their rendezvous with the Assiniboine.

Another Ojibway group living near the Monsoni may have been struck by the disease. The Sturgeon Indians, who lived on the east side of Lake of the Woods, were afflicted with some sickness at about this time. On June 7, 1741, nine canoes of Sturgeon Indians appeared at Albany Fort from up the Albany River. According to the journal, they "brought letele [little] goods there [*sic*] crye was that they had been all starvd + sick + some kil'd att y^{e} ware with y^{e} Poets Ind^{ns}" (HBCA B.3/a/30: 48d). With no references to the Sturgeon in the Albany journals between 1737 and this date it is possible that the Sturgeon Indians had been attacked by smallpox, had later gone to war, and had not returned to Albany Fort until three years after the epidemic. These were the same people who had suffered from an epidemic sickness in 1729-30.

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[6]This would have been the event that caused Morice to date the epidemic to 1738.

[7]The passage is not clear on that point, however. Doige (1989: 85) believed that all three groups were afflicted. Morice (1910: 35) seemed to suggest that only the Assiniboine were struck. My reading is that the Assiniboine had left but the Cree and Monsoni were prevented from joining them due to the outbreak of the disease.

More so than for any other group there is strong evidence for a significant impact of this epidemic upon the Monsoni, perhaps with disastrous long term consequences for the group. Most significant is that several of La Vérendrye's pre-epidemic estimates of Monsoni warriors gathered for war greatly exceed his estimate of total warrior strength from the post-epidemic period. For example, on May 7, 1734, nearly 400 Monsoni warriors gathered in order to go to war against the Dakota (Burpee 1927: 174). Shortly after the epidemic, the Monsoni of the Rainy Lake area claimed only 140 men in three bands, with a few more residing on the Winnipeg River[8] (Greenberg and Morrison 1982: 93). The discrepancy between the two may have been even greater in that the latter estimate would have been inclusive of all Monsoni warriors, while the earlier figure included only those gathered at that time. Given an estimate of four persons per warrior, the Monsoni population may have declined from about 1,600 to just over 560[9].

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[8]The date of the manuscript from whence this estimate is derived is crucial, since the population distributions and sizes after the epidemic may have changed considerably from the pre-epidemic period. Greenberg and Morrison (1982: 79) suggested a date of circa 1730-40. However, it refers to Fort Bourbon, on the Saskatchewan River near Lake Winnipeg, which was constructed in 1742 (Wilson 1952: 49). Thus, this document could not have been written until after 1742, and therefore after the epidemic. In all probability it was written before 1745, since La Vérendrye is identified as a Lieutenant, and he was promoted to Captain in that year. I am indebted to Patricia Kennedy and Gilles Durocher, of the National Archives in Ottawa, who provided this last information. The number of Monsoni warriors residing at the Barrier is not recorded, but the primary inhabitants of that place, the Barrier Cree, only included some 25-30 men (Greenberg and Morrison 1982: 93). Thus, one would expect that the Monsoni men numbered significantly less.

[9]The use of four individuals per warrior is merely an estimate and probably a conservative one at that. Charles McKenzie, who traded with the Mandan and who later resided at Lac Seul for a considerable period of time, employed an estimate of six individuals per warrior to calculate Indian populations (NAC MG 19, C4 Volume 38: 25). This evidence is especially significant in that the Monsoni, although forming several bands living in the region of Rainy Lake, comprised a distinct and readily identifiable people living in a relatively confined area, without the many subdivisions that were characteristic of the Cree and Assiniboine. Thus, there is not the same problem of identifying which subgroup living in what region was afflicted that is experienced when studying the latter two groups. It is fairly certain that such estimates

There is also circumstantial evidence confirming the high degree of mortality among the Monsoni caused by this epidemic. Three years later, in 1741, the prominent Monsoni chief La Colle and his people again went to war against the Sioux of the Prairie, and took back large numbers of their enemy as slaves (Burpee 1927: 380-1). Prior to this year, the capture of "slaves" had not been mentioned by La Vérendrye as a motive for Monsoni war excursions (*Ibid.*). This change in pattern may be significant. It has been noted by Johnston (1987: 23; See also Trigger 1978: 252; 1989: 260, 1987: 826-287) that following devastating epidemics some Indian groups, such as the Iroquois of the Northeast and the Piegan of the Canadian Plains, went on military expeditions against their enemies in order to capture enemy females and children to replenish those lost in the epidemic, and not only for torture. In some instances, Huron warriors were even kept alive by the Iroquois following their capture, to be adopted into their tribe (Johnston 1987: 25, 27; Trigger 1987: 826-827). If indeed La Colle was going to war as a means of acquiring enemy slaves, given the heavy losses experienced only a few years earlier, it may be that this attempt to obtain "slaves" was actually a means of trying to replenish a Monsoni population decimated by this smallpox epidemic.

Another common strategy for group survival, that of amalgamation with other surviving groups, may have been resorted to by the Monsoni following this epidemic. One of the best documented responses to severe mortality among aboriginal groups in North America has been the coalescence of the remnants of formerly autonomous peoples who had been ravaged by disease, warfare, or both, into a larger combined social group in

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include all Monsoni bands.

order to surpass a population threshold, either for defensive or subsistence purposes[10]. Prior to 1738 every indication given by La Vérendrye pictured the Monsoni as an independent people, living in a contiguous territory in the Rainy Lake-Rainy River area. This territorial coherence was reflected in the maps of the period. However, by the early 1740s a small number of Monsoni were living with the Barrier Cree along the Winnipeg River (Greenberg and Morrison 1982: 93). As late as 1736, Father Aulneau had identified the Winnipeg River as solely the domain of the Cristinaux, or Cree (Jones 1893: 73).

Similarly, the Saulteur and other Ojibway people from Lake Superior began moving into the Monsoni heartland, the Rainy Lake region, during the decades following the epidemic, so much so that the bear clan joined the moose clan (the Monsoni) as the dominant totems of the people living in the region (Hickerson 1974: 42-43; Lovisek 1993). This began in December of 1736, when large numbers of Saulteur sought refuge with the French at Vermilion River for fear of the Dakota Sioux (Burpee 1927: 238). Although this occurred prior to the appearance of the disease at Rainy Lake, every indication suggests that the process continued following the devastation of the Monsoni, and that they were increasingly joined by other peoples[11]. Thus, the group living with the

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[10]While in many cases this would involve survivors from the same linguistic and tribal divisions reforming into new arrangements, it could also lead to the emergence of "poly-ethnic" villages. Perhaps the best example is that of the Mandan who, following the 1779-83 and 1837-38 smallpox epidemics, merged with the surviving Arikara to produce new villages (Wood and Thiessen 1985: 8, 74; Lehmer 1977b: 101). As well, the diaspora following the epidemics and warfare accompanying the fall of Huronia saw the creation of major refugee villages crossing linguistic boundaries, such as the Ottawa-Huron settlement of Chequamegon. On post-epidemic amalgamation see Dobyns (1983: 302-312).

[11]In a similar vein, William Warren noted that following the almost complete decline of the Sandy Lake Ojibway of Minnesota due to the 1779-83 smallpox epidemic, their numbers were greatly augmented by additions from Lake Superior (Warren 1984: 344).

Cree following the epidemic and the movement of other Ojibway into Monsoni territory may both be symptomatic of the demographic stress that the Monsoni suffered as a result of this epidemic. As the eighteenth century progressed, the Monsoni name disappeared from common usage, perhaps suggesting the end of their identity as a distinct group.

It would seem that this smallpox epidemic made its way northward to Hudson Bay. In 1738, a fatal, although unnamed, disease appeared at York Factory. On February 26, 1739, Chief Factor James Isham noted the scourges of this sickness, writing in the journal that: "A very Remarkable Sickness and Casualtys is very much [among] our Ind^{n} hunters this year w^{ch} is y^{e} Chief occasion of y^{e} Ind^{s} drawing to y^{e} factory so soon" (HBCA B.239/a/21: 23d). These Indian hunters were the Homeguard Indians, the coastal Cree who remained near Hudson Bay all year round, and who furnished provisions and other necessaries to the HBC posts during the eighteenth century[12] (Lytwyn 1993: 57-58).

This affliction was probably introduced by infected Indians from the interior as a large number of those who traded at York during the summer of 1738 subsequently died. In June of 1739, a few months after mentioning the earlier presence of the disease among the Homeguard Cree, Isham learned from a group of inland traders that "near 50 canoes y^{t} was here [York Factory] last summ^{r}, are [?] Dead + y^{e} rest gone to war against y^{e} poets [Dakota Sioux]" (HBCA B.239/a/21: 36). Thus, some time between late May-early June of 1738, when the Indians of the interior first began to arrive at York Factory to trade, and

[12]The so-called Half-Homeguard Cree also occasionally hunted geese for York Factory but lived beyond the Homeguard territory and generally did not visit during the winter (Lytwyn 1993: 63).

June of 1739, when Isham was notified, some 50 canoes of Indians, perhaps as many as 150 people, perished for an unidentified reason[13]. Although warfare might seem a possibility at first, this is unlikely given that it was explicitly stated that "the rest" had gone to war. While other explanations are theoretically conceivable[14], death due to an epidemic disease is the most probable. If they had been infective when they reached the fort, they would have passed the disease on to the Lowland Cree on the Plantation, where both were gathered in preparation for trade. In part due to this calamity, the numbers of canoes arriving at York Factory from the interior declined severely in 1739 from the previous year[15].

It does not appear that the disease spread far into the Petit Nord. With the exception of the reference to the Sturgeon Indians at Albany Fort and those of La Vérendrye, there is no evidence of smallpox in the Albany journals, nor in the French records. It is especially noteworthy that nothing was written at Albany Fort since its hinterland included the eastern half of the Petit Nord at this time (Ray 1987a). Indians from the interior, including the so-called "French Indians" who traded with the Canadians, arrived at Albany on several occasions, without mentioning epidemic disease[16]. Presumably these people would have heard something of widespread mortality

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[13]Given an estimate of three people per canoe (Lytwyn 1993: 302). In most cases the women and children would have been left at a rendezvous spot (Ray 1993: 322), and so the total number of deaths would have been much higher, assuming the families contracted the disease as well.

[14]Starvation or some mass accident, for instance.

[15]In 1738 over 214 canoes arrived at York Factory carrying Upland traders. In 1739 the total was only 134 (HBCA B.239/a/20; 21).

[16]See, for example, HBCA B.3/a/28: 48d and HBCA B.3/a/29: 35, 35d.

among others in the region, even if they did not contract the disease themselves. Thus, it would appear that the 1737-38 smallpox epidemic spread only along the western and southwestern margins of the Petit Nord.

ORIGIN OF THE EPIDEMIC

Although it is generally stated in the secondary literature that the 1737-38 smallpox epidemic originated at Hudson Bay, based on the statement by La Vérendrye, this was not the case. There was no mention of epidemic or unusual sickness in the daily journals of the HBC posts of Eastmain, Churchill, York, Albany and Moose during the year (1736-37) leading up to the eruption in the south (HBCA B.3/a/25; B.42/a/17; B.59/a/1; B.135/a/7; B.239/a/19). In laying blame on trade with his opponent, a common tactic whenever fur trading companies or national rivals fought for native allegiances, La Vérendrye and his informants were probably politically and economically motivated. Such a statement provided La Vérendrye with the opportunity to reinforce his orders about not trading with the English at the risk of divine retribution by "the Master of Life", while demonstrating the political and economic fidelity of his informants.

Instead, the virus that triggered the 1737-38 smallpox epidemic had its origin in Europe, continuing the pattern of *ACIs* introduced to North America from external disease pools. It is possible to connect the smallpox epidemic in the Petit Nord to one that had been present in New England a few years before and which slowly spread

westward to the Great Lakes region and beyond[17]. This epidemic broke out in Boston late in 1729, having been imported once again from abroad by ship, this time from Ireland (Duffy 1953: 53). It continued raging there until the fall of 1730 (*Ibid.*: 54), before spreading throughout the British colonies. By January of the following year it appeared in Philadelphia (*Ibid.*: 78). By the end of the summer of 1731, smallpox was epidemic in New York City, where it continued into 1732 (*Ibid.*: 80). That same year it moved to the northward, striking the Six Nations Iroquois, having been brought among them by a Seneca returned from Albany (O'Callaghan 1853-61: IX: 1036). By May of 1732 it had become firmly entrenched among their villages, and was causing many deaths (Thwaites 1902-08: XVII: 172; O'Callaghan 1853-61: V: 963).

Thereafter the disease continued its northward progress towards Canada in 1732, despite an attempt by Governor Beauharnois to establish a quarantine the previous year (O'Callaghan 1853-61: IX: 1029). Initially, it struck the Christian Iroquois and Algonquin settled at Sault St. Louis (Caughnawaga) and Lake of the Two Mountains (Figure 10), afterwards extending to Montreal during the fall (Thwaites 1902-1908: XVII: 175). This order of introduction suggests that smuggling played a role in the diffusion of the epidemic since both the Mohawk at Sault St. Louis and the Nipissing at Lake of the Two Mountains acted as middlemen in the clandestine trade between Montreal and Albany (Tooker 1978: 432; Day 1978: 790; Lunn 1939: 61). The epidemic spread throughout the colony during the remainder of that year and the next. By the fall of 1733

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[17]Both A.S. Morton (1939: 18) and Taylor (1982: 35) theorised that smallpox diffused to the Plains from Canada around the time of the La Vérendrye epidemic.

it had claimed nearly two thousand Canadian lives, nine hundred in Montreal alone, and was still raging (Heagerty 1928: 72-73).

Even as smallpox subsided among the Iroquois, Beauharnois learned that it had "spread among all the [Indian] nations" of the Upper Country, the *Pays d'en Haut*[18] (Thwaites 1902-08: XVII: 173). Among those stricken were the Canadians' Indian allies living in the lands around Lake Michigan and Lake Huron, including the Miami, the Potawatomi, the Wea and the Piankeshaw[19] (*Ibid.*: 173; 175). Smallpox is thought to have been at Detroit both in 1732 and 1734 (Drews 1939: 761). It also may have reached the Illinois Indian tribes living in what is now Illinois, and the eastern portions of Missouri and Iowa (Blasingham 1956: 384, 393). By 1733-34, then, this smallpox epidemic had firmly established itself at least as far west as the lands south of Lake Michigan, and perhaps much farther. Shortly thereafter, the disease made its presence felt beyond the Mississippi River[20].

Taylor (1977; 1982) gathered evidence that places epidemic smallpox in the Upper Missouri region during the mid-1730s. Most compelling are Dakota Sioux winter counts that recall an epidemic of smallpox in 1734-35. Taylor noted three Teton counts

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[18]The area west of Lake Ontario encompassing the other Great Lakes to the Mississippi. It also would have included the region south of the lakes to the Ohio (White 1991: X).

[19]The Wea and the Piankeshaw were found along the Wabash River, while the Miami lived at the head of the Maumee River (Tanner 1987: 172). It would seem that the Miami were especially hard hit (White 1991: 217; Tanner 1987: 44).

[20]The means by which smallpox made its way from Lake Michigan to the Dakota Sioux is not known. Taylor (1982: 35) speculated that "the locus of infection may have been the Mississippi River". Of possible significance is the fact that the Dakota Rendezvous, an annual "pan-Siouan" trading fair, was connected to groups living east of the Mississippi River (Swaggerty 1988: 353). Lehmer (1977a: 106) identified this gathering as a potential source for the spread of smallpox from the Great Lakes to the Prairies.

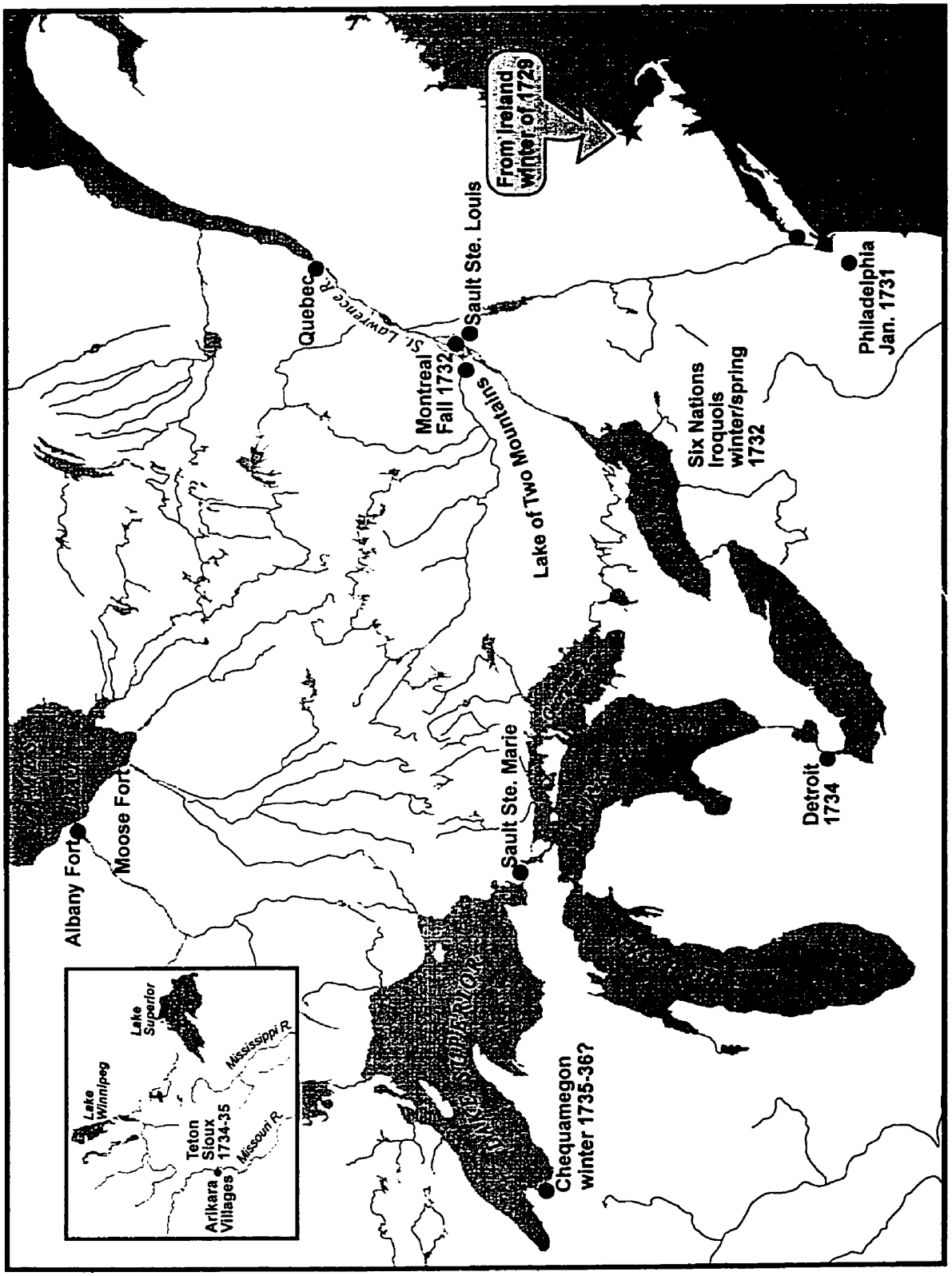

Figure 10: Origins of the 1737-38 smallpox epidemic

that depict cramps and "cutaneous eruptions," both symptoms of that disease (Taylor 1982: 35). However, a Lower Yanktonai count, the John K. Bear count, makes no mention of sickness at this time, suggesting that they had been bypassed (Howard 1976: 31). By this time the Teton, the westernmost division of the Sioux, had crossed the Mississippi and were primarily prairie inhabitants (Carver 1974; Howard 1976: 5). The Lower Yanktonai, part of the Middle, or Wiciyela, division, lived farther east, although they too were venturing out onto the prairie margins (Holzkamm 1982: 231). Despite close trading contact between the Teton and the other divisions (Swaggerty 1988: 353), the epidemic did not sweep through the entire nation. At least some of the Sioux had been spared[21].

Although Taylor (1982: 35) concluded that this could not be the same epidemic recorded by La Vérendrye, based on the perceived discrepancy of two years between the two events, they probably were. Evidence from other epidemics demonstrates that the diffusion of smallpox can be a surprisingly slow historical event[22]. As well, it is likely that the initial introduction of the disease to the Red River area occurred very early in 1737, perhaps as little as just over a year after the Sioux epidemic was at its height. Given this timing and the infrequency of smallpox on the Northern Plains at this time, it

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[21]There are no winter counts referring to the eastern (Santee) division and extending back to this period nor, apparently, is there documentary evidence. Thus it is effectively impossible to determine if the easternmost Dakota were struck by smallpox at this time.

[22]Prem (1992: 26) noted that smallpox epidemics could diffuse at a relatively slow rate, even in densely settled areas, including areas without the pronounced seasonal variations in population density and interaction found on the Northern Plains. In regions with marked seasonal variability in patterns of interaction among natives, such as was the case in much of Canada, delays in diffusion would be expected during periods of winter dispersal. Reff (1992: 266) noted a measles epidemic that had spread through central Mexico over several years, suggesting that other diseases could produce similarly lengthy epidemics.

seems unlikely that two independent epidemics could have occurred separated only by little more than a year and no great distance.

Moreover, other Indian people were probably infected following the Teton. There is evidence that smallpox had been among the Ojibway living south of Lake Superior prior to the Council of Maurepas, but after the arrival of the disease among the Dakota. This evidence revolves around the motivation for the massacre of Father Jean-Pierre Aulneau in 1736, which is noted above. Although some historians have suggested that the Aulneau massacre was an all-Dakota undertaking, the question of who participated has never been answered conclusively (See Burpee 1903). An oral tradition collected by the ethnographer John Cooper in 1928, at Lake of the Woods, makes this question pertinent to the present study. Cooper was told by Jim Eliud, an Ojibway chief, that the latter's ancestors had participated in the massacre. According to tradition:

> A certain clergyman... was a friend of the Indians, but they were "wild" then and tried to kill him, actually succeeding in their design. They believed that he was responsible for an epidemic of measles or smallpox from which many of the Indians had died. Jim mentioned Massacre Island, down the Lake, as the place where the killing had occurred. (Cooper 1936: 2)

As Cooper noted, this fragment of oral history refers to the Aulneau massacre. However, unlike Cooper (*Ibid.*: 2, fn.2), I do not believe that "the tradition is somewhat garbled." Instead, this is an important piece of evidence that does not fit the standard interpretation of the event, but is valid nonetheless. Moreover, this statement overcomes a severe limitation of oral traditions as an historical source. Often, they suffer from a disregard for standard chronology (Morantz 1984: 174) and, as such, dating the described events can be

extremely difficult or impossible. In this case the date of the event is already known. As well, knowing what took place, and in what order is equally important. Here, what is important is the identity of the participants and their motivations. There is no evidence that La Vérendrye's Ojibway allies participated in the massacre, although the Southwestern Ojibway, or Saulteur, living south of Lake Superior did. Barring the informant's appropriation of a Dakota legend, the tale suggests that the Southwestern Ojibway participated in the massacre, and in response to the epidemic[23]. The Southwestern Ojibway moved into the Boundary Waters, and to Lake of the Woods in particular, from the area south of Lake Superior during the period following the epidemic (Hickerson 1962: 2-3). This would explain why a Southwestern Ojibway tale was collected at Lake of the Woods.

This tradition suggests that the smallpox epidemic had been among some of the Ojibway before June of 1736. Further, if it is accepted that Father Aulneau had to have been present in the west when the disease first struck these Ojibway in order for those people to have cause to blame him, the timing can be pinned down even further. The priest arrived at Fort St. Charles only on October 23, 1735. In settling there he associated himself with the Southwestern Ojibway's enemies, and in the Saulteur's view he would have been in a position to aid them with his spiritual powers. An epidemic would have been readily attributed to his presence among the Cree and Monsoni. This would imply,

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[23]The concept of blaming a priest for a smallpox epidemic is reminiscent of some Huron claims during the epidemics of the 1630s (Trigger 1987: 534-538) and reflects some traditional Indian disease aetiologies blaming disease on malignant magic by the spiritually powerful (Ritzenthaler 1953; Krech 1981b, but compare Martin's (1982) *Keepers of the Game*). While with the Ojibway in 1763, Alexander Henry the Elder noted their belief that native physician/priests could inflict as well as cure disease (Bain 1901: 117).

then, that the epidemic broke out among the Ojibway who participated in the massacre sometime between October of 1735 and June of 1736, when he was killed. Given this possibility, verification of the tradition is crucial. Therefore, the composition of the war party is an important clue to the timing and diffusion of the epidemic.

An initial report by Beauharnois suggested that "a party of Prairie Sioux to the number of one hundred and thirty" was to blame (Burpee 1927: 211). However, further testimony provided by a French trader named Rene Bourassa suggests a more varied group. Bourassa had been captured by the same Dakota party on the same day, but was released before the massacre. According to his account (*Ibid.*: 262-266): "the larger portion of the savage party was composed of the Prairie Sioux, of some Lake Sioux, and some from the post of Monsieur de la Ronde. The latter appeared to be well disposed towards the French" (*Ibid.*: 264-265). Louis Denys, Sieur de la Ronde, then had charge of the French trading post at Chequamegon, on Lake Superior (*Ibid.*: 265 fn. 1; Figure 10), and the people who traded at this post were the Southwestern Ojibway. In addition, on December 23, 1736, La Vérendrye received a letter from Bourassa and his partner who were then at a post on nearby Vermilion River. He was informed that:

> a great number of Saulteur have sought refuge with them through fear of the Sioux. They questioned [the Saulteur] closely in order to ascertain how the Frenchmen had been killed, but they were unwilling to say, though amongst them there was one Saulteur who had been present at the tragedy[24]. (Burpee 1927: 238)

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[24]These are the Ojibway of the Point, that is of Chequamegon. The name Saulteur was used by the French to refer to the Ojibway living south of Lake Superior, from Sault Ste. Marie to Chequamegon (Morton 1939: 11). La Vérendrye, as did the French in general, acknowledged two divisions of Dakota, those of the Prairie and those of the River (the Mississippi) or Lake. The former group's lands lay to the west of the Mississippi River (Burpee 1927: 96, fn. 2). This is additional evidence that the Ojibway took part in the massacre since these two divisions were noted along with the Chequamegon people.

It is clear from this that the Southwestern Ojibway, who were allied with the Dakota Sioux against the Assiniboine, Cree and Northern Ojibway (Doige 1989: 79-80), were present at the massacre, but that, subsequently, a schism had developed between these peoples. The Eliud oral tradition, then, is consistent with the facts surrounding the Aulneau massacre. When evaluated given this reassessment of the identities of the attacking parties in the Aulneau massacre, it suggests that smallpox had been among the Ojibway living to the south of Lake Superior before June of 1736 and probably after October of 1735.

Finally, the disease may also have been present on the Upper Missouri as part of this epidemic. Taylor (1982: 35) presented circumstantial archaeological and historical evidence for the same epidemic among the Arikara, one of the agricultural village dwellers living along the Upper Missouri[25] (Rogers 1990: 23-24). The Arikara's traditional economy revolved around hunting, planting corn and trading with numerous Plains groups through their role as middleman in a continent-wide trading network (*Ibid.*: 42-44; Swaggerty 1988). This last activity put them at risk of participating in long distance diffusion of epidemics. According to Wood (1980: 106), "in addition to staples of the trade, most anything could... change hands at trading fairs," and "... the flow of goods - not to mention of persons and disease organisms - from one area to another was a

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[25]The Mandan and Hidatsa, both of Siouan linguistic stock, were the other sedentary, agricultural, trading societies resident along the Upper Missouri (Curtis 1970: 3: 4). It seems unlikely that they were struck by this epidemic, however. La Vérendrye visited the Mandan (or at least certain of their villages) in 1738 and made no note of the effects of disease among them (Burpee 1927: 318-353). Wood and Thiessen (1985: 6) suggested that epidemic disease first struck the Mandan and Hidatsa "perhaps as early as 1750...."

simple matter and... often exceedingly rapid." Dobyns (1992: 215) also noted the potential for epidemic disaster for Indian groups living at major trading centres, suggesting that the "intimate interpersonal interaction during trading-center fairs favored direct aerosol transmission of viruses". One of the Arikara's major trading partners was the Teton Dakota (Swaggerty 1988), who were already suffering from the disease. If the Arikara contracted smallpox during 1736, this could explain how the virus diffused between the Dakota and the tribes living nearer the Red River, and why there appears to have been a delay. The disease might have passed from the Teton through the Arikara to the Cree and Assiniboine, rather than directly from these Dakota. This would have been the final link in the diffusion of the disease from Europe to Red River.

SUMMARY

From the evidence presented here it is apparent that smallpox flared up among the people living west of Lake Superior at least twice between 1737 and 1738, although part of the same general epidemic. It appeared first between March and May of 1737 among Cree living along the Winnipeg River and those near the north end of Lake Winnipeg. In April or May of the following year it was active again among a war party of Cree and Monsoni assembling to fight the Dakota Sioux. About the same time, the disease appears to have made its way to the Homeguard Cree living in the vicinity of York Factory, but it does not seem to have penetrated any farther into the interior of the Petit Nord. Although the 1737-38 smallpox epidemic was confined to the western and southwestern margins of the Petit Nord, it was nevertheless more widespread than has heretofore been ascertained.

Contrary to the consensus in the literature, the epidemic did not come from the English at Hudson Bay. Rather it appeared first in the interior, and then did not make its way to the English post of York Factory until the following year. Instead, the virus came from Europe, thereafter spreading from the English colonies to Canada, and then to the Great Lakes region. It is clear that smallpox was epidemic from Boston to Lake Superior between 1730 and 1733. It is also certain that the disease found its way to the Dakota, the Southwestern Ojibway and perhaps the Arikara shortly thereafter and was part of the same epidemic. This is consistent with the broad pattern of epidemic activity that had emerged in the early seventeenth century and which saw *ACIs* introduced into the eastern colonies by ships from abroad, and then spread into the interior. Here, then, is strong evidence of the influence of the expanding frontier of the distant urban disease pools.

CHAPTER 6 — *ACIs* IN THE PETIT NORD: 1739-1780

In the decade following the 1737-38 smallpox epidemic, remarkably few *ACIs* were present in the Petit Nord. Thereafter, however, there was a far greater frequency of *ACIs* in the region than at any time its past. This in part may reflect the fact that the HBC records are more complete for this period. During this period there were at least two HBC posts constantly operating in the region and, by 1780, there were five. Even accounting for this, however, it is evident that, overall, *ACIs* were far more common in the Petit Nord than had previously been the case. Perhaps more significant, there was an unprecedented increase in frequency of *ACIs* generally described as "colds", and the region was struck by its first epidemic of measles. This suggests that some conditions had changed to bring the region closer to external disease pools during the period 1739-1780.

A PERIOD OF LIMITED *ACI* ACTIVITY: 1739-1750

There were relatively few *ACIs* present in the Petit Nord between 1739 and 1750. None was noted at Henley House, the HBC's first inland post, or at Albany Fort, from which it was settled, or in their hinterlands[1] (Figure 11). These were the only two HBC posts besides York settled in the Petit Nord at that time. In May of 1744 several of the York men were ill simultaneously with "violent colds" (HBCA B.239/a/25: 23). Later

[1]Henley House began operation in 1743, intended as a way station for the Indians travelling to Albany Fort to trade. Between 1755 and 1766, however, it was not in operation, save for a brief period in 1759 (HBCA Post History "Henley House").

that same year there were frequent colds both at York and at Churchill (HBCA B.239/a/26: 12d), perhaps a continuation of the earlier outbreak. Finally, in 1746-47 there was an unnamed, but highly lethal, sickness which spread among some of the Upland Indians who normally traded at York, although it cannot be discerned if this included people living in the Petit Nord. The only evidence for this is a brief mention in the annual general letter from the York Factory Council to London, dated August 18, 1747, which commented that "there has been a sickness amongst the [Upland] Natives y^{t} [that] has carried of [f] a Great many this Last year..." (HBCA A.11/114: 124). It may have been connected to an unknown illness among the Lower Yanktonai Sioux, who then wintered towards the Missouri River[2], as the entry for 1746 in the John K. Bear winter count was "They-were-struck-by-an-epidemic" (Howard 1976: 33).

INCREASED INCIDENCE OF *ACIs*: 1751-1759

While there were very few instances of epidemic activity recorded in the Petit Nord during the decade or so following 1739, the situation changed markedly after 1750 when *ACIs* began to appear with greater frequency. Initially, most were at York Factory. During the 1750s, the York post journals described many disease outbreaks, some causing considerable mortality among the Lowland Cree and Upland Indians alike. Others, however, were limited to the fur traders. The first major sickness of the decade

[2]The winter count entry for the year prior to the epidemic (1745) identified the Lower Yanktonai winter camp as being at Long Lake, in south-central present-day North Dakota, very near the Missouri River (Howard 1976: 33).

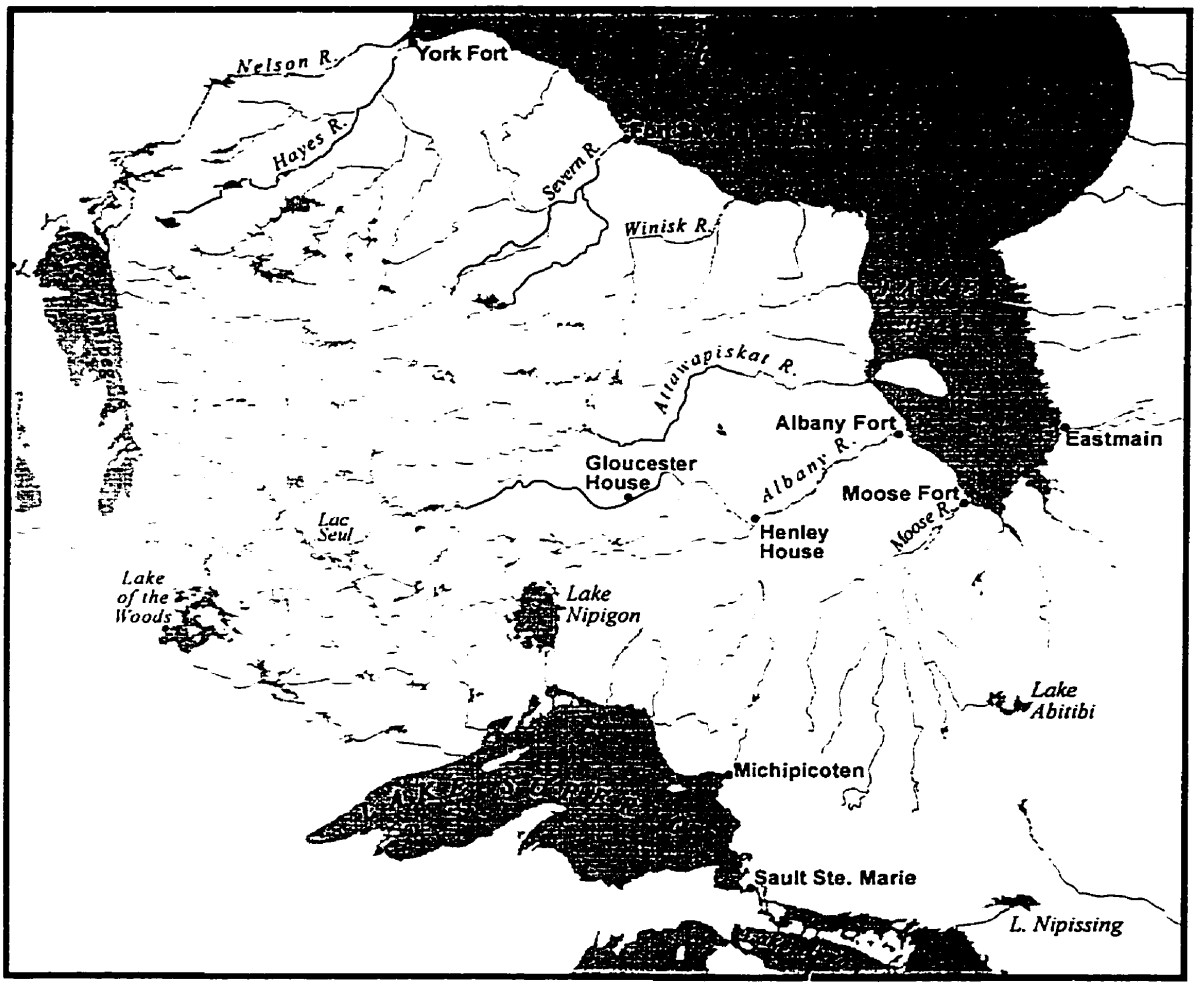

Figure 11: The Petit Nord, 1739-80

occurred when what appears to have been smallpox broke out at Michipicoten along the north shore of Lake Superior, in 1751 (Tanner 1987: Plate 32; Figure 11). This disease was said to be active in the Upper Great Lakes during 1751-52 (Blasingham 1956: 384). Indirect evidence for this comes not from the HBC, but from the French. In his "Reports from the Northern Posts" dated 17 September, 1751, La Jonquière reported that "out of forty savages at the post of Michipicotton, thirty-four have died and the remaining six

have gone in the direction of Hudson's bay" (Thwaites 1902-08: III: 85). Such a toll in human lives in this small Ojibway population and subsequent precipitous flight is strongly suggestive of a smallpox epidemic. Despite this report, there is no evidence at the HBC posts of either the epidemic or the flight of the survivors. Instead, the only "new" Indians to appear at Moose were a few who arrived with plenty to trade and another man who had once traded there but had gone elsewhere for two years (HBCA B.135/a/21: 20d, 22d; B.135/a/22: 16d). It may be that the few survivors from Michipicoten never made it to the Bay.

These people may have been infected while travelling to Canada. In 1832, Dr. Douglass Houghton collected oral testimony concerning the occurrence of smallpox among the Ojibway of Lake Superior (Schoolcraft 1973). He learned that in about 1750:

> a war-party of more than one hundred young men ... having visited Montreal for the purpose of assisting the French in their then existing troubles with the English, became infected with the disease, and but few of the party survived to reach their homes. It does not appear, although they made a precipitate retreat to their own country, that the disease was at this time communicated to any others of the tribe (*Ibid.*: 578).

Smallpox was epidemic in some eastern communities at this time, possibly including Montreal[3]. Moreover, the French were indeed having "troubles with the English" throughout the 1750s, long before the declaration of war in 1756[4]. If Houghton's

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[3]Based on this statement Taylor (1982: 37) concluded that the disease "was brought from Montreal" by the returning warriors. Heagerty (1928: I: 38; *Ibid.*: II: 319) restated the Houghton tradition, but mentioned nothing regarding smallpox in Montreal at this time. However, he noted the presence of the disease at Louisbourg and Halifax in 1749 (*Ibid.*: 74). According to Stearn and Stearn (1945: 42), Peter Kalm witnessed smallpox and its effects while travelling in New Jersey and New York in 1749 and 1750.

[4]For example, in June and July of 1750 La Jonquière summoned some of the Ottawa and Ojibway of the Upper Country to Montreal to question them regarding trade with the English (Thwaites 1902-08: III:

assessment of the timing of the Ojibway tradition is approximately correct, the Ojibway of Michipicoten may have been infected in or just before 1751 as part of the group which contracted smallpox at Montreal.

During the remainder of the 1750s, evidence for apparent *ACIs* in the Petit Nord is limited to the York Factory records, with the exception of September of 1753, when an outbreak of colds occurred among the Albany Lowland Cree shortly after the arrival of the *Seahorse* from England[5]. While the absence of such sickness in the records of Albany and Henley House is not proof of the absence of *ACIs* in the eastern part of the region, it at least demonstrates that these diseases were not penetrating to these posts, and probably not into their trading hinterlands during the 1750s[6]. Conversely, infections were comparatively frequent at York Factory through to the end of the decade.

ACIs were unusually common at York Factory during the first three years of the 1750s, as several discrete outbreaks or epidemics struck in rapid succession. The first occurred during the late spring and summer of 1751, when a deadly infectious disease broke out among the Indians living in the interior, spreading subsequently among the

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67-68).

[5]On September 21, 1753, Joseph Isbister noted that: "many of our Indians are laid up with a great Cold which has put them off from shooting and also creates a Slow fever. Several of our best Hunters not able to do us any service" (HBCA B.3/a/46: 6d). The *Seahorse* arrived at Albany on August 28 (HBCA B.3/a/45: 29). This timing is suggestive that the disease was imported from Europe by the ship.

[6]Smallpox was rampant in the Upper Great Lakes region during 1757, including at Michilimackinac, (MacLeod 1992; Jacker 1887), and tradition has it that the Southwestern Ojibway were infected while defending Quebec on the Plains of Abraham in 1759, carrying the disease back to Mackinac (Neill 1883: 198). The same disease was also active on the Ohio River and at Detroit in 1752 (Heagerty 1928: I: 38-39). It seems unlikely that none of the epidemics circulating near the eastern part of the Petit Nord managed to reach the region. According to Unrau (1984: 3) the Kansa Indians contracted smallpox in 1755-56 while at Fort Duquesne, on the Ohio River near present-day Pittsburgh. Subsequently, a few survivors carried the disease back to the Kansas River where it was passed on to others.

Lowland Cree. On June 13, Chief Factor James Isham wrote that there were "14 Ind^{s} very bad upon the plantation of a sort of mesalls, they are taking at first with violent cold, coughs sore throats swell faces, and very full of spotts" (HBCA B.239/a/34: 38d). Two days later there were upwards of twenty people ill (*Loc. Cit.*), and by June 17 "most of home Ind^{s} [were] very bad [and] not able to pitch away" (*Ibid.*: 39). On the 24th, however, Isham commented that "most of the Ind^{s} [had] recoverd" (*Ibid.*: 39d). Despite this optimism, the sickness continued among the Lowland Cree and was not as mild as Isham initially thought. Thus, on August 26, Isham noted that:

> one cannoe came down the River, brought the unwelcome news of 2 Ind^{ns} Dying, I am sorry to hear of the Death of many Ind^{ns} since last June, also one cannoe from the Baptist Creek our men has but one Cask of Geese as yet one Ind^{n} man Died there[7]. (HBCA B.239/a/35: 4).

The following day Samuel Skrimshire informed Isham of sickness among several of the Indians tenting about Flamborough House, a dependency of York Factory located just over twenty miles up the Hayes River (HBCA B.68/b/1: 10). Thereafter, all mention of this disease ceases in the York Factory records and there is no indication that the disease spread beyond the York Factory region.

The "sort of mesalls" described by Isham was probably rubeola. Rubeola (ICD-10 B05), or measles, is an acute, highly contagious, crowd disease which provokes a lasting immunity and, consequently, becomes a disease of children in larger urban populations. The measles virus has no non-human reservoir and is spread via droplet emission, through direct contact with nasal or throat secretions or, less commonly, through indirect

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[7]In a letter written the same day, Isham wrote that he was "sorry to hear by some upland Indians, down this River, of the Death of many Indians since June Last" (HBCA B.68/b/1: 9d).

contact with objects contaminated with these secretions (Benenson 1995: 294). The virus enters through the respiratory tract. After infection, there is a seven to eighteen day incubation period followed by a brief prodromal period[8], which produces fever, cough, coryza and conjunctivitis (Benenson 1995: 294; Kim-Farley 1993: 872). There is generally a fourteen-day period between exposure and the appearance of the rash (Kim-Farley 1993: 871). The macules characteristic of measles are flat, reddish brown and tend to coalesce to form large blotches (White and Fenner 1994: 577). They appear first on the head before spreading down the body and outward to the limbs, and may last four to seven days (Kim-Farley 1993: 872).

Given these symptoms, Isham was probably correct in his diagnosis[9]. The violent cold, cough and sore throat may be readily interpreted as signs of the prodromal period of measles, while the spots would be the macules of the later stage. The swollen faces are more problematic, and are not necessarily characteristic of measles. However, periorbital edema (swelling of the tissue covering the eye socket) may be experienced by some measles victims during the prodromal period (*Diseases* 1993: 169), perhaps giving the appearance of a swollen face.

Measles can cause exceptionally high case fatality rates in virgin soil populations[10]. These rates "are most likely the result of nutritional and environmental

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[8]The prodromal period is that which encompasses the earliest, premanatory, symptoms of the disease prior to the eruption of the spots.

[9]Although confusion with scarlet fever (scarlatina), a streptococcal infection, is a possibility (Creighton 1965: II: 633).

[10]For example, seem Panum's (1940) description of the 1846 epidemic on the Faeroe Islands.

factors", including malnutrition, lack of adequate, or appropriate, care and the failure to treat complications[11] (Kim-Farley 1993: 872, 874). Complications and secondary bacterial infection such as middle ear infections (otitis media), pneumonia, blindness, diarrhea and encephalitis can make the disease especially severe. This was the first recorded outbreak of measles in the York Factory area, and the Lowland Cree no doubt lacked immunity to this disease. In fact, this is the first mention of the disease in the records of the Petit Nord, despite a long history of measles epidemics in the eastern colonies and among the eastern Indians dating back to the seventeenth century (Duffy 1953: 165; Caulfield 1943). Nevertheless, the mortality rate does not seem to have been as great as observed during other virgin soil measles epidemics, except perhaps among those encamped away from the post. Presumably, the assistance and sustenance provided at the HBC post resulted in fewer mortalities than would otherwise have been the case.

The timing of the outbreak among the Lowland Cree about York Factory suggests that the disease was introduced among them by Upland Indians who had come to trade, and one trading party in particular is suspect[12]. On May 31, Isham noted the arrival of seven canoes of Keskachewan traders (HBCA B.239/a/34: 36d). These people departed York Factory on June 2, however, "at noon one cannoe return[d] the man being in a weak and Sickly condition" (HBCA B.239/a/34: 37). While not identified as such by Isham,

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[11]Geneticist James Neel (1977: 171) guessed that perhaps twenty per cent of "excess mortality" suffered by virgin soil populations during measles epidemics could be attributed to genetic factors.

[12]Certainly it could not have come from the HBC ships in this case as the sickness first broke out in June, long before the *Prince Rupert* made contact with the post (HBCA C.4/1: 6d-7). Most of the outbreaks at York Factory during the 1750s, although not all, appear to have come from the interior via traders, and it should be noted that York's trading hinterland was far more expansive than that of Albany, and the other posts.

this man was probably the first victim of measles identified in the post journal, and this fits with the pattern of subsequent disease activity. As noted above, the sickness was first described among the Lowland Cree on the York Factory plantation on June 13 (*Ibid.*: 38d). The two weeks between the arrival of the Keskachewan brigade and the appearance of the disease among the Lowlanders was long enough for the measles virus to have been transmitted and for the disease to begin to manifest itself among the latter people. It does not appear to have been among the Lowland Cree prior to this, for Isham dealt with large numbers of local Cree from March on, and made no mention of the disease until mid-June.

These Keskachewan were a group of western Cree living on the North Saskatchewan River, west of the Eagle Hills, and were found by Henday in what is now central Alberta[13] (Ray 1988: 57; Russell 1991: 147-48). They did not contract measles while in their distant home territory for, had they done so, the disease would have run its course long before they reached York Factory, or they would have been prevented from continuing somewhere along the way due to the severity of their affliction. Instead, they appear to have been infected enroute to Lake Winnipeg, only to begin to manifest their illnesses as they reached the coast.

This measles epidemic afflicted at least one other group living west of Lake Winnipeg besides the Keskachewan. On July 2, Isham noted in his journal that "38 Cannoes of Misinnipee Ind^{s} and Stone Ind^{s} came from the North River to trade, inform^{d}

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[13]According to Russell (1991: 147), Isham began the practice of identifying the Keskachewan separately from the closely allied Pegogamaw in 1751. The Pegogamaw arrived at York Factory on May 26 with another brigade of Keskachewan (HBCA B.239/a/34: 36).

me a great many cannoes of Sinepoits was oblig^{d} to turn back on acc^{t} of Sickness..." (HBCA B.239/a/34: 40d). It is impossible to say where these Assiniboine lived but there were several different bands of Assiniboine living in a relatively narrow country between the forks of the Red and Assiniboine, and the North Saskatchewan River at this time (Ray 1988: 22). Nevertheless, this is the first recorded instance of this disease among that tribe.

There is also evidence of measles on the Northern Plains about this time which, along with smallpox, was said to have ravaged the Pawnee and Witchita, located west of the Missouri in what is now Nebraska, during the period leading up to the outbreak at York Factory (Thwaites 1902-08: III: 87). In turn, there has been speculation that the village tribes of the Upper Missouri, the Mandan, Hidatsa and Arikara, all suffered from epidemic disease at this time, and perhaps measles passed northward to the Cree and Assiniboine via these agricultural traders[14] (Wood and Thiesen 1985: 6; Taylor 1982: 37). It would seem that this first measles epidemic in the Petit Nord occurred only a few years after the horse frontier reached the Northern Plains. Although plains Indians living in the southern part of the continent began acquiring horses from the Spanish as early as 1600, they were slow to spread northward (Ewers 1955: 3). Nevertheless, the Pawnee may have had horses as early as 1682, but definitely had them by 1700 (*Ibid.*: 3, 4). When La Vérendrye visited the Mandan in 1738, they had yet to acquire horses but the

[14]Wood and Thiesen (1985: 6) and Taylor (1982: 37) speculated that smallpox struck these tribes at this time, but it could have been measles, since there are no eyewitness accounts from this period. It probably did not come through the Dakota, however, as none of the extant winter counts mention disease at this time.

Arikara had them and, equally important, the Mandan were being visited by large numbers of equestrian Indians from the south each year (*Ibid.*: 4). Three years later, in 1741, when La Vérendrye's son visited the Mandan, he brought back horses. According to Ewers (*Ibid.*: 5), this year "seems to have marked the beginning of the trade in horses from nomadic tribes settled southwest of the Missouri, through the Mandan to the peoples north and east of them", especially the Assiniboine. By the date of the epidemic, then, a trade network based primarily on the exchange of horses and stretching from the Southwest to the Canadian plains was in place. On subsequent occasions the village tribes were struck by epidemics largely because of their sedentary nature and their extensive trading contacts, and perhaps this was the case in 1751. In turn, the Cree and Assiniboine to the north had long been trading with the Mandan, possibly as early as the late seventeenth century (Ray 1988: 87-89). Although there is no proof, this trading network could have carried measles from the Pawnee to the tribes of the Upper Missouri, then to the Assiniboine or Cree, and from them to the Lowland Cree via the Keskachewan.

While the origins of the measles among the Pawnee are unknown, it may have come from the east. Measles was circulating in the east over the period 1747-49 (Caulfield 1943: 539-540) and it is possible that the disease moved westward from there onto the plains. The Illinois tribes, living north of the juncture of the Mississippi and the Ohio Rivers, were at this time antagonistic to the Pawnee and to some of the Indians living farther east, and they were even being used by the French to attack the English settlements (Blasingham 1956: 379). In turn, the region east of the Mississippi was at

this time a zone of intense interaction and consequently of epidemic activity. The potential for westward transmission of disease thus was very high.

Late in June of 1752, just twelve months after measles broke out at York Factory, the Lowland Cree suffered a widespread, although less severe, epidemic of an unknown disease which caused coughs, sore throats and, according to Isham, yellow blood (HBCA B.239/a/35: 38, 39). The timing suggests that, once again, an *ACI* was brought from the interior, for this was well over a month before the ship arrived, but just after the Uplanders began arriving at the post. The sickness persisted at least into mid-July. The following month witnessed an epidemic of colds among the HBC men and the Lowland Cree. This occurred only a week after the *Prince Rupert* arrived from Europe (HBCA B.239/a/36: 3). This disease was separate from the earlier one and remained active into the winter (*Ibid.*: 14, 16d, 18d). Another, more severe, outbreak of colds broke out in July of 1753, once again brought from the interior, judging by the timing (*Ibid.*: 37). As of the 26th of July, Isham, his officers, most of the men, and all of the Indians were affected by it (*Loc. Cit.*). Unlike the previous epidemic, this one was said to be fatal to a great many Indians, particularly Uplanders. Isham was informed that "two Indian woman [are] Dead of the cold aforemention[d] which has carried a great many Uplanders of [f]" (HBCA B.239/a/36: 37). The following February brought yet another outbreak of colds among the York Factory men, although no fatalities were noted[15] (HBCA B.239/a/37: 14d).

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[15]There was also an unusual concentration of deaths due to consumption among the Lowland Cree, carrying off perhaps as many as nine throughout the winter of 1752-53 (HBCA B.239/a/37: 16d, 17, 17d, 19d, 20 25d).

For two years following the outbreak in February of 1754 no epidemic sickness appeared at York Factory. Then, in 1756, suspicious deaths began to occur among the Indians trading at that post, deaths that appear to have been the result of unidentified acute infectious disease. From the fall of 1756 to the summer of 1758, both Uplanders and Lowlanders were dying at an unusual rate from at least two different sicknesses. The first may have involved only the Lowland Cree. On March 22, 1757, two Indians from the Nelson River arrived at the fort to trade, stating that, since the previous fall, one man and two women had died, another was on the verge of death, and several others were ailing (HBCA B.239/a/42: 24d). If indeed this was the result of an infectious disease, this mortality was probably separate from that which followed, for James Isham did not include this period in a memorandum of unusual fatalities among the Indians (HBCA B.239/a/44: 41d-42).

Nothing further was said of unusual Indian deaths until the end of June and early July, when three Homeguards died in short succession, as well as an Uplander who had been left at the fort by his people (HBCA B.239/a/42: 36, 36d). Although the cause of these deaths was never explicitly stated, the pattern of mortalities noted in the journal very closely resembles that of a severe epidemic. By August 18, all of the Indians were "in a Sad condition, severall dead" (*Ibid.*: 40d). The following day, Isham noted that two more were dead, and he received "Sad news [,] Ind^{s} Inland dropping off surprizingly." (*Ibid.*: 41). Indeed, something very remarkable was happening. On the 23rd of September he counted fifteen Indians dead since August 20 (HBCA B.239/a/44: 2). People continued to die into the fall and winter, and on March 2, 1758, he wrote:

"Nothing material Dead Time of the year, Doubt a good many Ind[s] dead by reason none com'd in, as promised but hope it is otherwise having had too many Died Lately not Less then 2000 Beaver A Year" (*Ibid.*: 21). In June he learned that six men, twelve women and an unknown number of children had died up the Nelson River since the previous June (*Ibid.*: 31). The deaths continued to occur into September of 1758, although at a far slower rate.

The mortality among the Lowland Cree and Uplanders during 1757-58 greatly exceeded that of normal years, and this seems to have made an impression on Isham, for he compiled a table in his journal which identifies by name many of those who died during the peak period of mortality, July 1757, to September 1758, and which states the numbers of widows and dependants left by the men who succumbed (HBCA B.239/a/44: 41d-42; Table 4). Not only does it record that 54 Indians died[16], but it also shows the impact on the families of the victims in terms of those who died and those who were left unprovided for. Here is an unprecedented glimpse into the human suffering that these diseases were causing for some of the people of the Petit Nord. It also shows an important indirect impact of this mortality, the number of widows and orphans who survived. The number of survivors was an important consideration for the HBC who, out of humanitarian and practical motivations, had to provide for these people until other arrangements could be made. This would have been a considerable expense for the

[16]Undoubtedly an underestimate of the total number of fatalities since it does not include those who died prior to July 20, 1757, and because Isham and his sources were unaware of the total number of dead in the interior. The table refers only to those who died in the vicinity of York Factory or up the Nelson River.

concern.

MEN	WOMN	BOYS	GIRLS	MEN'S NAMES	WIDOWS & ORPHANS		
					Widows	Boys	Girls
1	1	1	2	Esinepoet, his wife, 2 girls & a boy	-	-	-
1	-	-	-	An Upland man	-	-	-
1	1	-	1	Mehegan, his wife & daughter	-	-	1
1	-	-	-	Karrakanaw	1	-	3
1	1	-	-	Wittechap & his wife Shissohon	1	-	2
-	1	-	-	Chakush's wife	-	-	-
1	1	1	1	Mistekanass, wife & 2 children	-	1	-
1	-	-	-	Wysack	-	-	-
-	1	1	-	Huhygan & brother	-	-	-
1	-	-	-	Memimetahasu—Leader of the Severn Indians	3	Several	
-	1	-	-	Athikatick	-	-	-
1	-	-	-	Makopa (or Job) a Leader	1	3	2
-	1	1	-	An old woman and a boy	-	-	-
1	-	1	-	Washeo and child	1	-	-
-	1	-	-	Athupee's wife	-	-	-
1	-	-	-	Tuckachin or Stanhope	1	1	1
-	1	-	-	Tickapotie	-	-	-
-	1	-	-	Lucas' wife, Namequean	-	-	-
7	4	4	5	From October to May died up Nelson River			
1	-	-	-	Athupee	-	-	2
-	1	1	-	Cabetekishew's old wife & boy	-	-	-
19	16	10	9	TOTAL DEATHS=54			

Source: HBCA B.239/a/44: 41d-42

Table 4: James Isham's table of deaths at York Factory from July 20, 1757 to September 14, 1758

A SHIFT IN PATTERN: 1760-1768

During the period 1760-68 the frequency of *ACIs* in the Petit Nord declined significantly at York Factory, but increased at Albany Fort. In fact, there was no mention of unusual disease activity in the York records during this period, nor at Severn, which was settled throughout this period[17]. This was a considerable change from the relatively frequent outbreaks seen at York and their near absence at Albany during the 1750s. It was only after 1768 that *ACIs* reappeared at Severn and York. In contrast, there were outbreaks of acute sickness at Albany every year between 1765 and 1768, although none between 1760 and 1764.

The first seven years of the 1760s was a period during which fur traders were prevented from travelling any distance into the Northwest from Canada, and perhaps this would partly explain this departure from the pattern of *ACI* activity witnessed during the 1750s. It thus may be that the western Indians, and especially those of the York Factory hinterland, were spared at this time due to a temporary isolation from the east. With the fall of Canada in 1760, the fur trade of the Northwest was thrown into disarray for several years, as the British traders who attempted to assume control were prevented from advancing far beyond Lake Superior by Indian people who continued to resist their presence. If, as seems likely, the Canadian fur traders were responsible for introducing *ACIs* into the Petit Nord during the French regime, then by cutting off their access to the

[17]Henley did not resume operation until 1767, after a decade of being closed, and no *ACI* appeared during the remainder of the 1760s.

Northwest the Indians were establishing an inadvertent quarantine against the diffusion of acute infectious diseases. In practice, this may have been a more significant barrier to diffusion for the western part of the Petit Nord, for Montreal-based traders continued to trade along the coast of Lake Superior during this period, and Upland Indians arrived at Albany Fort reporting the presence of numerous "French English" in their territory as early as 1765-66 (HBCA B.3/a/58: 36d). By contrast, only in 1767-68 were Canadian fur traders again able to penetrate far into the Northwest (Morton 1939: 270; Lytwyn 1986: Chapter II), after which *ACIs* returned to the western Indians[18].

In January of 1765, dysentery was among the men and many of the Indians at Albany Fort and, although the source is unknown, HBC trader Humphrey Marten thought first of the quality of the food and possible contamination of the cook's pots (HBCA B.3/a/57: 18d). There is no indication in the journal that the disease came from the Indians, and certainly it would not have come from the ships, given the timing of the outbreak. Perhaps in this case there was a chronic carrier of *Entamoeba histolytica* among the men who had inadvertently infected the others. This parasitic organism causes amoebiasis (ICD-10 A06), a common form of dysentery, and may spread through food contaminated with faecal material, a definite possibility under the poor sanitary

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[18]The opening of the trade stemmed from events in 1767, when a few traders were finally able to pass through Rainy Lake to Lake Winnipeg, and others beyond Lake Nipigon (Lytwyn 1986: 9). At Severn in May of 1768, William Tomison returned from an unsuccessful inland journey with only three canoes of Indian traders, informing Andrew Graham that:

> the English & French pedlars [were] daily trading what furs the nations catched. He said he did all he could to prevent them, but to no purpose they making him the Answer the French are nigh hand us and it is a long way to go to your factories. (HBCA B.198/a/9: 26d)

conditions present in a fur trade post. Individuals infected with *E. histolytica* may serve as a chronically ill or even asymptomatic, but still infectious, reservoir for years, making external introduction unnecessary (Benenson 1995: 10-11). Less probable is that this disease came from inland. There was an unidentified but mortal affliction which prevailed at Michilimackinac in 1765 (Johnson 1921-53: 11: 835), and which was probably the same as the "Great Death" among the Inland Indians noted in the Moose Fort journal for the winter of 1764-65 (HBCA B.135/a/: 40: 2). The latter sickness may have been within the Petit Nord, as Moose Fort typically drew Indians from as far away as Michipicoten on Lake Superior[19].

The following autumn saw a severe epidemic of colds at Albany, once again brought from England by the *Seahorse*, which arrived at Moose on September 4 (HBCA C.4/1). From Moose, the cargo and new men were transported to Albany by sloops on September 13 and 14 (HBCA B.3/a/57: 42-42d), presumably carrying the disease. On the 1st of October Marten noted that the Homeguard Indians were in very bad health, having colds and sore throats (HBCA B.3/a/58: 3, 3d, 4d). The same disease also appeared at Moose among the Indians and men that fall, shortly after the arrival of the *Seahorse* (HBCA B.135/a/40: 2, 3d-4). In the fall of 1766, sore throats were common among the Albany men, and during the following April most of the local Indians suffered from an inflammation of their eyes, perhaps conjunctivitis, as well as another unidentified

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[19]Not all of the infectious diseases then circulating around the Upper Great Lakes penetrated into Albany's hinterland, as several epidemics documented in other records do not appear in those of the HBC post. There was smallpox among the Menominee of Green Bay in 1761, an epidemic on the Wisconsin River, probably in 1766, and an unnamed epidemic sickness among the Ottawa in 1767 (Johnson 1921-53: 10: 702; 13: 467; Carver 1974: 48).

sickness (HBCA B.3/a/59: 8d, 10d, 17, 19, 17d, 2d, 36d). Finally, "plurectic [pleuritic] fevers", a non-specific term for inflammation of the lungs which can be caused by several different disorders (Miller and Keane 1983: 886), appeared among the Lowland Cree and at least one of the HBC men during the winter of 1767-68 (HBCA B.3/a/ 60: 10, 12d, 14d, 15, 15d). According to Duffy (1953: 184) pre-Revolutionary Americans employed this and similar terms as "catch-all phrases for all respiratory diseases", and no doubt a similar custom was followed by the HBC men.

THE RETURN OF *ACIs* TO THE WESTERN PETIT NORD: 1769-1780

Many instances of acute infectious disease were reported at all three of the Bayside posts in the Petit Nord and at Henley House between 1769 and 1780. When compared to previous decades, such sicknesses were once again more common than they had been, and most were colds or epidemic sore throats. This was a continuation of a pattern at Albany Fort, where *ACIs* were reported on several occasions during the previous period, but was a fundamental change for the western posts of Severn and York, and their hinterlands, which, during that period, had been free of *ACIs*. Significantly, the increase occurred in sicknesses from both the HBC ships and from the interior. Although it is not possible to conclusively identify the reasons for the re-emergence of these diseases in the western part of the region, it is nevertheless apparent that something had changed to affect the diffusion of *ACIs* to the Petit Nord.

Between 1769 and 1780 "colds" of varying descriptions were the most commonly identified acute infectious diseases appearing in the Petit Nord, and they appeared in epidemic form on several occasions. This was consistent with the overall period, 1739-80, which witnessed many similarly named outbreaks in the region. Although called colds, these Acute Respiratory Diseases (ARDs) would have included many different diseases with roughly similar symptoms. The common cold (ICD-10 J00) encompasses acute sicknesses caused by hundreds of different viruses, notably rhinoviruses and coronaviruses (Benenson 1995: 396-397). As well, there are other respiratory diseases, including those caused by adenoviruses, parainfluenza virus and respiratory syncytial virus (RSV), which cause illnesses that would undoubtedly have been included under the common term 'colds'. It is also possible that one or more of these "colds" was influenza, as the English language of the eighteenth century employed a variety of terms, including "great colds" and "epidemical colds", to identify influenza (See Duffy 1953: Chapter VI). As such, it is generally impossible to differentiate between them given the limited evidence found in the journals, other than to note that fever, which accompanied some outbreaks at Hudson Bay, is rare in adults with the common cold but is characteristic of influenza and also of some cold-like diseases[20].

These infections are not diseases of childhood. In general, adults from even

[20]Collectively these diseases are known as Acute Respiratory Diseases (ARDs), and those viral diseases that cause cold-like sicknesses and are characterised by fever are called Acute Febrile Respiratory Diseases (AFRDs) (Benenson 1995: 396-400).

densely populated regions may contract them[21] (Benenson 1995: 397, 399; Burnet and White 1972: 96). Unlike the few crowd diseases to strike the Petit Nord, ARDs often attacked the HBC men as well as the Indians, and therefore were also spread by the Europeans. This may account for the frequency of their arrival in the Petit Nord, as the white employees of the fur trade, including those from Canada, would have had no immunity to the many of these infections and were thus capable of spreading them[22].

Related to the increased prevalence of ARDs was the emergence of the HBC ships as a factor in the introduction of *ACIs*. Prior to 1739, these vessels posed little threat to the health of the people of the Petit Nord. Thereafter, this situation seems to have changed. On several occasions between 1739 and 1780, epidemics broke out at the Bayside posts shortly after the arrival of the ship from Europe, and almost all were referred to as colds[23]. Outbreaks in 1752, 1753 and 1765 have already been noted. There were several others between 1769 and 1780, and at least one resulted in fatalities.

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[21]The reasons for continued susceptibility vary. In the case of the common cold, universal susceptibility stems largely from the number of different infectious agents (Benenson 1995: 397). Others induce only a short-lived immune response, while the influenza virus is somewhat unstable, and mutations can render antibodies acquired from previous infections ineffective (*Ibid.*: 247, 399).

[22]Influenza epidemics were known to spread extremely widely in Europe and in the eastern part of North America. In the late eighteenth century Dr. William Currie of Philadelphia identified three major influenza epidemics that struck the U.S. during that century (Currie 1792: 100-101). The first occurred in the fall of 1733, when the disease arrived in North America after spreading throughout Europe, beginning with northern Germany. In the spring of 1761 the disease spread from the West Indies to Halifax, and then to Boston. From there it was said to have diffused throughout North America, although this was no doubt an exaggeration. Finally, there was an influenza epidemic in September of 1789 that was first observed in New York and subsequently spread to the West Indies and throughout the U.S. Currie was unable to learn if this last major epidemic spread to the natives. There may also have been others, as Dr. Benjamin Rush, a contemporary of Dr. Currie, identified influenza epidemics in the U.S. in 1749, 1771, the spring of 1790 and the winter of 1790-91 (*Ibid.*: 103, fn.). Several of these can be linked to major epidemics and pandemics in Europe, occurring in 1729-30, 1732-33, 1761-62 and 1788-89 (Crosby 1993: 809).

[23]The sole exception occurred in 1769 at York Factory, when "fluxes" broke out among the men

Following a violent outbreak of diarrhoea among the York Factory men, probably brought by ship in September of 1769 (HBCA B.239/a/62: 4d, 21), moderately severe colds appeared among the Homeguard Indians and the men of Albany Fort the following September, lasting into October (HBCA B.3/a/63: 4d, 5d, 7, 9). This was shortly after the sloop arrived with the trading goods from Europe. These two infections were probably localised around the HBC posts.

Conversely, when epidemic colds next struck the Petit Nord, once again brought by ship, they appeared first at York and later spread farther afield. On August 25, 1771, the *Seahorse* dropped anchor near York Factory, having taken fifty-nine days to sail from the Orkneys to Hudson Bay[24] (HBCA B.239/a/65: 50; C.4/1: 10d-11). Thereafter, violent colds began to break out among the men and Indians, continuing until the following May (HBCA B.239/a/66: 14d, 15d, 21, 25d, 45). This was an unusually long duration for an epidemic ARD at Hudson Bay, but it seems that many of those who at first suffered from this disease later developed pneumonia, even after Andrew Graham reported that the initial sickness was abating[25] (*Ibid.*: 15d, 29, 30, 39, 52). As late as March, there was "never less than five on the Doctor's list" (HBCA B.198/a/15: 21d). This disease spread to Severn in January, affecting most of the men during the following month (HBCA

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just after the ship arrived.

[24]On July 31 while en route, the *Seahorse* had taken aboard a large number of Inuit for the purposes of trade (HBCA C.1/104: 24d). It is possible, though perhaps unlikely, that the disease had been transmitted from the Inuit to the traders.

[25]The extended duration of the epidemic and the references to severe coughs, spitting blood, and "peripneumony" suggest pneumonia and perhaps bronchitis. Bacterial pneumonia is a complication of several cold-like illnesses (Benenson 1995: 398). Graham noted that the sickness did not strike all of his men at once. Rather, he stated, "as one of us gets well another turns bad" (HBCA B.239/a/66: 30d).

B.198/a/15: 16d, 18). By the spring of 1772, these colds, accompanied by sore throats and fevers, had also appeared at Fort Prince of Wales (Churchill), and four Indians and one of the HBC men died there of pneumonia (*Ibid.*: 40, 52; HBCA B.42/a/83: 26, 27d, 32, 32d, 33, 38). While it did not spread far from its initial locus at York Factory, this is the first documented instance in which an *ACI* appeared at more than two of the HBC's posts, and it was not typical of the period in this respect[26].

Although called colds, it is likely that this ARD was influenza. Influenza (ICD-10 J10, 11) is a highly contagious, acute viral disease of the respiratory tract that is spread from person to person via airborne particles. Its course in humans is brief. Following an incubation period of only one to three days, recovery usually occurs within two to seven days. The infectious period is short compared to many other *ACIs*, lasting only between three and seven days, depending upon the age of the victim (Benenson 1995: 245-247). Common symptoms include: a sore throat, cough, a runny nose, fever, chills, weakness, generalised joint and muscle pain and prostration (Crosby 1993: 807). Victims may also suffer from severe gastrointestinal problems, such as nausea, vomiting and diarrhoea (Benenson 1995: 246). Although capable of causing widespread mortality during major epidemics and pandemics, under normal circumstances and where proper medical attention is provided influenza's case fatality rate is usually low, generally one per cent or less (Crosby 1993: 807).

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[26]On at least one earlier occasion epidemic sickness had appeared at both Albany Fort and nearby Moose. As noted above, this occurred in the fall of 1765 when colds spread at both posts. Noah Webster identified an epidemic of measles in 1772-73 that he believed spread throughout North America, and possibly the whole world (Caulfield 1943: 543). There is no evidence that this epidemic spread as far as the Petit Nord, however, and it is likely that Webster was simply engaging in some hyperbole.

Influenza differs from other *ACIs* in that the virus is inherently unstable, and immunity acquired from one episode may not be effective during subsequent outbreaks. Three main types of the disease have been identified, types A, B and C, and all are antigenically distinct[27]. All three are periodically subject to antigenic *drift*, a process of genetic mutation that makes a permanent immunity impossible and renders younger adults and children susceptible to the disease (Mackenzie 1980: 144). Type A influenza can infect certain species of animals, as well as humans, and is responsible for widespread epidemics and pandemics[28]. It is also subject to infrequent antigenic *shifts*, a more severe mutation that creates an entirely new subtype to which all people are susceptible. Type B is generally associated with regional epidemics, while type C causes sporadic cases and minor localised epidemics (Benenson 1995: 246; Crosby 1993: 808).

Of considerable significance is the rapidity with which influenza can spread and form into epidemics or pandemics, and the great distances this disease can travel. This is largely a function of the instability of the virus, its short incubation period and its high rate of infectivity (Crosby 1993: 808; Ramenofsky 1987: 161; Burnet and White 1972: 125). Even before the jet age, "human movement had made the whole civilized world a single epidemiological unit as far as influenza was concerned" (Burnet and White 1972: 209). Also important is the seriousness of its complications. Sequelae, such as viral and

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[27]That is, they are immunilogically distinct, and therefore do not create cross immunity.

[28]Type A influenza exists in animal reservoirs, although transmission of the virus occurs almost exclusively between humans. It is thought that periodic antigenic shifts that create new subtypes, to which all humans are susceptible, are caused by recombination of human and animal (usually swine and duck) antigens (Benenson 1995: 247).

bacterial pneumonia, are responsible for fatalities during epidemics, particularly among the elderly[29] (Benenson 1995: 246). Subsequent bronchitis may prolong the effects of influenza for weeks or more after the disease has run its course. There is every indication from the historic record that influenza was generally a destructive disease whenever it spread among the native people of the Petit Nord.

The final *ACI* introduced by the HBC ships during this period occurred a few years later. In 1778 colds and sore throats broke out at both York Factory and Albany Fort after the arrival of the ships. They were widespread at Albany Fort in September, only a week after the sloop brought the goods from the *Prince Rupert* and brig *Charlotte*, both of which had sailed from Europe to Moose[30] (HBCA B.3/a/75: 1; C.4/1). Very likely, they moved up the Albany River, perhaps with the HBC men who were then travelling upriver as far as Gloucester House, as sickness was common among the men and some of the Indians at Henley House in November and December including at least one person with a sore throat (HBCA B.86/a/32: 16d, 21d). It did not spread much beyond Henley, for no illness of this sort appeared at Gloucester, located a short distance upriver[31] (HBCA B.78/a/4). At York Factory, colds were first noted as widely spread among the men at the beginning of October (HBCA B.239/a/76: 9, 10). It is likely that

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[29]However, occasional influenza epidemics have taken great tolls in human life, including people from all ages. Most notably, over 21 million people died during the worldwide influenza pandemic of 1918-19 (Crosby 1993: 809-810).

[30]Thomas Hutchins, Chief at Albany Fort, wrote that: "Indeed every body, both old & young, Indian European, have got a cold & sore throats" (HBCA B.3/a/75: 1).

[31]Gloucester House was established in 1777. There was no unusual sickness at this post during the period 1777 to 1780.

the disease was present immediately after the ship arrived late in the month of August, and that the epidemic built slowly thereafter.

ACIs were probably carried to the Petit Nord by the HBC ships on at least five occasions between 1769 and 1780, a span of twelve years. There were three such instances in the HBC records between 1739 and 1768, and only one prior to 1739. This change in the introduction of disease via the ships does not seem to have been the result of a major decline in travel times as, overall, there was only a modest improvement in the duration of the journey. During the period 1719-1737 the average voyage from Britain to Hudson Bay required about sixty-seven days. During the period 1738-80 the average was about fifty-nine days[32] (HBCA C.4/1). Even so, the ships that transported these sicknesses were not always among the fastest. In 1771, for instance, the *Seahorse* brought colds, or perhaps influenza, to York Factory, having taken fifty-nine days to sail from the Orkneys to Hudson Bay (*Loc. Cit.*), and other crossings implicated in the trans-oceanic diffusion of disease were similarly lengthy[33]. Neither was this a function of increased numbers of people aboard the ships providing a larger reservoir. The era of major inland expansion by the HBC, during which larger numbers of men were being imported from Europe, lay in the future, or only after 1780. If it was not the speed of the crossing nor the numbers of people on board the ships, it seems likely that something had changed in Great Britain to make these diseases more common or perhaps to increase

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[32]As in the previous chapter, these times are based on the period between the departure from European port to the arrival at Hudson Bay, using the HBC book of ships' movements.

[33]In 1753 the same ship brought "great colds" to Albany Fort and required sixty-six days to complete the voyage to Hudson Bay.

their range of circulation. Other than dysentery, all were ARDs, and the fact that these illnesses were not diseases of childhood was an important factor in their success in bridging the Atlantic, for their ship-board reservoir was not limited to children as appears to have been the case for diseases such as measles and smallpox.

This pattern of ship-based *ACI* introduction resembles that experienced by more isolated communities, although on a limited scale. Modern studies of the inhabitants of Antarctic research stations and of the remote islands of Tristan da Cunha and Spitzbergen have found that these populations tend to suffer from extremely severe epidemics of colds when their isolation is broken, generally by supply ships from more populous regions (Holmes and Allen 1973; Paul and Freese 1933; Tyrrell 1977). Under these conditions of semi-isolation, the colds behave uncharacteristically. According to Burnet and White (1972: 132) "any community which is cut off from the rest of the world for periods of a year or more will, on the arrival of visitors, suffer an epidemic of illness of the feverish cold-influenza type...." Indeed, the severity of the cold symptoms suffered by an isolated group under such conditions makes it difficult to distinguish the disease from influenza. The same phenomenon has been identified among other isolated populations, at least since the early eighteenth century (Creighton 1965: II: 431; Burnet and White 1972: 133). Such infections are short lived, however, and disappear from fully isolated populations a few weeks after the cessation of outside contact (Tyrrell 1977: 142). It is noteworthy that they may be transmitted even though the people who bring them appear healthy[34] (Burnet

[34]This pattern of ship-based infection, despite limited numbers of people on board and relatively lengthy travel times, may mean that the *ARDs* were not subject to the limitations suggested by Snow and Lanphear (1988; Snow 1980: 32) and therefore may have arrived in the east prior to the first waves of

and White 1972: 133). Although these outbreaks were nowhere near as regular at Hudson Bay due to the prolonged length of the journey, they nevertheless resemble those that occurred in the fully isolated communities, particularly with regard to the severity of the disease and the timing of the outbreak immediately following contact with the outside. It would thus seem that by 1780 the Petit Nord was at risk from another source of disease, albeit one limited to a small range of *ACIs*[35].

Although analysis is hampered by the lack of inland HBC posts, there are also several documented instances of epidemic activity inland from the Bayside posts during 1769-1780, and others undoubtedly went unreported. During the winter of 1768-69, Andrew Graham at Severn, noted that there had been "a great sickness" among some of the Upland Indians, and that two "able providers" died (HBCA B.198/a/11: 30). A suspicious mortality occurred among some of the Indians living within the York hinterland during 1773-74, although there is no evidence identifying the place of origin of those afflicted (HBCA B.239/a/70: 38d). It was also reported by Indians trading at the posts at this time that "several Indians are dead + some have gone to war" (*Ibid.*: 40). Several Indians were also ill of an unidentified complaint at Henley the following March and April, 1774 (HBCA B.86/a/21: 25d-26). Finally, in 1776-77 an unknown malady, perhaps influenza, appears to have diffused from Henley to Albany. Sickness was common at Henley House following the summer of 1776, and at one time several of the

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colonization in the seventeenth century.

[35]Boyd (1985: 340-348) documented the frequent arrival of "winter illnesses" among the people of the Columbia River area from the 1820s through the 1840s. While brought overland by the fur traders, he suggested that these ARDs were akin to the annual severe colds brought by ship to isolated locations such

men were ill with fevers (HBCA B.86/a/30: 4d, *passim*). By December of 1776, there was "an epidemic angina & Cough amongst the Europeans & Natives" at Albany Fort, and the chief complaints included fevers, coughs and pleurisy (HBCA B.3/a/71: 9, 10). In this context the latter complaint, pleurisy, suggests that pneumonia had developed as a sequel to the original affliction. As before, these symptoms are suggestive of influenza. At one point several men could only crawl (*Ibid.*: 11). Though the sickness lasted only into January and no casualties were noted, trader Thomas Hutchins was led to remark that "this year is peculiarly unlucky and unhealthy" (*Ibid.*: 10d).

There were several other outbreaks during this period that, because of their timing, probably also came from the interior[36]. Presumably, if there was no local reservoir and an *ACI* broke out other than in the early fall, then it must have come either directly or indirectly from the interior. In April of 1770, John Garbut at Severn was informed by some of the trading Indians that they had been "very sickly", although whether these people came from the Lowlands or beyond is not known (HBCA B.198/a/12: 24). In July of 1773, an epidemic of highly infectious of sore throats broke out among the people at Albany Fort. On the 24th of that month twelve of the men were ill, and two days later several of the Homeguard Indians were also badly off (HBCA B.3/a/65: 47, 47d). By the 28th Humphry Marten had very few healthy men at his

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as Spitzbergen (Ibid.: 346).

[36]The York Factory Lowland Cree suffered from a disorder of their eyes during the fall goose hunt of 1769 (HBCA B.198/a/12: 11). It is not clear what the cause was nor from whence it originated. During the spring, snowblindness was a significant problem but certainly not during the fall. If this was an acute infectious disease, then possible sources could have included the ships, the Upland Indians or even the Lowland Cree living to the south and east.

disposal, and the sick included some of the sloopers engaged in the coastal transport between posts (*Ibid.*: 48; HBCA B.59/a/44: 29d). At about this time, violent coughs struck the men of Henley House, and so the disease may have moved inland from Albany (HBCA B.86/a/19: 31-31d). It is impossible, however, to identify the origins of this disease. It was first noted at Albany on July 27. During the few weeks leading up to this mention, the Moose Sloop arrived on July 3, a sick Homeguard was brought in on the 17th and the Eastmain sloop arrived on the 22nd (HBCA B.3/a/65: 45, 46d, 47), each of which was a potential carrier of the disease. Since the outbreak at Henley House occurred after that at Albany Fort, it is conceivable that the disease diffused from the interior to either Moose or Eastmain, and was then transported to Albany by sloop or through Indian movements.

A few years later colds were again active at an HBC post, and once again the timing suggests that it did not come from the ships. During the winter and ensuing spring of 1775-76 there was a relatively mild outbreak of colds among the Indians and HBC men at York Factory (HBCA B.239/a/73: 23). The summer of 1777 saw yet another epidemic of nominal colds at York Factory, again among the men and Indians (HBCA B.239/a/74: 37). Symptoms included sore throats, violent coughs and difficulty breathing (*Ibid.*: 37, 38), and it is likely that this was no ordinary cold. Decker (1989: 156) speculated that this was a streptococcal infection, but given these symptoms it could have been influenza or some other disease. This sickness persisted into the fall and, as before, there is no record of any fatalities. In each of these cases, the timing of the sickness makes it unlikely that the ships were responsible for the initial infection.

During the twelve year period between 1769 and 1780, there were three outbreaks of *ACIs* noted in the journals at York Factory[37] and two at Severn Fort that either appeared only in their hinterlands or seem to have spread from the interior to the HBC posts. During the nine years between 1760 and 1768 there had been none. This was a small but significant increase, and may have been related to the revival of the Canadian fur trade, and its expansion into the Petit Nord. As early as the spring of 1768, there was a rush of Canadian traders into the Northwest following the reopening of the trade (Morton 1939: 270). Although little is known about this Montreal-based fur trade during its first few years following the reopening, it is clear that by the early 1770s the Canadians were well established in the hinterlands of the HBC's coastal forts. There had been a great influx of Canadian traders in the Albany hinterland in 1769-70, and by 1771 there were several Pedlars trading at the head of the Severn River (HBCA B.3/a/62: 34d; B.198/a/15: 34d). By 1779-80 the HBC's George Sutherland counted no less than seventeen Canadian houses east of Lake Winnipeg, and the country north of Lake Superior was said to be "poisoned" with Pedlars (HBCA B.211/a/1: 23d, 25). With so many Canadians trading in the region following 1768, not to mention the large numbers headed for the Grand Nord by way of the Boundary Waters, there were many more opportunities for the transmission of *ACIs* from the east than during the previous period. This was to remain the case until the union of the HBC and NWC in 1821, as each spring fur trade brigades regularly left Montreal for the Northwest.

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[37]There were three others at York Factory brought by the ships, for a total of six in the twelve year span.

SUMMARY

When compared to the initial period of the HBC's tenure at Hudson Bay *ACIs* became more frequent in the Petit Nord in the period 1739 to 1780. Of the sicknesses identified, almost all were diseases of the respiratory tract that afflicted Europeans and Indians alike, such as colds and coughs. No doubt, one or more of these was influenza. On only two occasions were crowd diseases present and these were confined to the Indian population. Whatever their type, these diseases did not occur at evenly spaced intervals during this period. Instead, distinct patterns in time and space emerged. There was only limited *ACI* activity in the years leading up to the mid-point of the eighteenth century, with only two outbreaks of colds at York in 1744-45 and an unidentified sickness among the Indians in its hinterland in 1746-47. Thereafter, acute infectious diseases, most if not all of which were undoubtedly *ACIs*, became far more common, although not always in every part of the region. Smallpox is said to have struck at Michipicoten in 1751, and colds were epidemic at Albany Fort in 1753. All other instances prior to 1760 are confined to York where epidemic sickness was frequent between 1751 and 1758. At least three were accompanied by significant mortalities: measles in 1751, colds in 1753 and an unknown mortality in 1756-57. The presence of measles is especially noteworthy, as this was the first appearance of that disease in the Petit Nord, suggesting that the Petit Nord had taken another step closer to the external disease pools. The extension of measles into the Petit Nord may well have been the result of the emergence of a trading network

involving the long distance trade in horses. As will be seen in the next chapter, this network was capable of carrying *ACIs* long distances.

During the 1760s, the pattern of disease activity changed markedly. Most of the outbreaks documented during this decade occurred at Albany Fort. Outbreaks of dysentery (1765), colds (1765), sore throats (1766), sore eyes (1767) and pleurisy (1767-68) plagued the Indians and the HBC men at Albany. Nothing appeared at York Factory or Severn prior to the latter part of the decade. This may have been due to the temporary blockade of the Montreal-based fur trade in the Northwest, which would have prevented the fur traders from introducing acute infectious diseases into the western part of the Petit Nord.

It was not until 1769, when "fluxes" struck the York men and an unidentified sickness afflicted the Indians of the Severn hinterland, that *ACIs* returned to the western part of the Petit Nord. This signalled the beginning of a new era of epidemic activity in the Petit Nord, not only around York and Severn, but also in the east near Albany and Henley. Thereafter, and throughout the 1770s, colds, coughs, sore throats and other sicknesses frequently appeared in epidemic form in the Petit Nord, both at the Bay and in the interior. In fact, the prevalence of *ACIs* in the western part of the Petit Nord during this period resembled the situation during the 1750s, suggesting that the absence of such disease during the blockade of the Montreal fur traders lasting until 1767 was a temporary anomaly. For example, seven separate outbreaks were reported at York Factory between 1768 and 1780, either among the Indians and HBC men at the Bay, or among the Indians of its hinterland. As well, for the first time there is evidence that the Bayside afflictions

were spreading inland, at least as far as Henley House. Finally, this era saw the emergence of the HBC ships as an important vector in the introduction of *ACIs* to the Petit Nord, and most of these were ARDs. In sum, very few years passed during the years between 1768 and 1780 without an outbreak of acute infectious disease somewhere in the region. This was a significant shift from the era prior to 1737 when *ACIs* were very rare visitors to the Petit Nord.

CHAPTER 7 — THE 1779-83 SMALLPOX EPIDEMIC

INTRODUCTION

Smallpox returned to the Petit Nord during the early part of the 1780s as part of what Trimble (1988: 7) has called "one of the most severe North American pandemics for Native Americans". This devastating epidemic originated in Mexico and appears to have been the most widespread in the Americas to that date. The limits to its diffusion can only be estimated, but it is thought that it spread from Mexico to Great Slave Lake in the north, and from Lake Superior to the Pacific Northwest, blanketing much of the western half of North America. The disease also spread southward into South America, as smallpox appeared in Guatemala at this time and in Colombia shortly thereafter (Lovell 1985: 154; Villamarín and Villamarín 1992: 115, 128-129). Despite suggestions by Dobyns (1983: 15) there is no evidence that it penetrated in an eastward direction beyond Lake Superior.

Research has shown that this epidemic had a profound impact upon the people it struck in the Northwest. In its wake, it left vast numbers of dead and, in some cases, it almost entirely depopulated individual bands or tribes, causing relocations that reshaped the human landscape of the regions affected[1]. By Peers' (1994: 20) estimate, one half to three quarters of the Ojibway living west of Grand Portage died. Decker (1989: 86) believed that the Woodland Cree suffered casualties amounting to up to seventy-five per cent of their total population. Although Lytwyn provided no estimate of casualties, it is

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[1]On the demographic and territorial characteristics of the epidemic in the Northwest, see, for instance: Decker (1988; 1989); Ray (1984: 105-111); Krech (1978; 1983); Lytwyn (1993: 360-373); Gunn and Tuttle (1880: 87-88); Peers (1994: 18-21); Wissler (1936: 36); and Russell (1991: 5, 161, 216).

apparent that the epidemic was destructive of human life among several of the Lowland Cree groups (Lytwyn 1993: 368-373). Other repercussions were more subtle, although no less profound, and the terrible mortality of the disease undoubtedly had a great psychological impact upon the survivors (Milloy 1990: 70-71; Peers 1994: 20; Taylor 1977). At the same time, it played a major role in shaping the emerging fur trade (Lytwyn 1986: 44; Morton 1939: 334; Glover 1952: lxii, 333; Innis 1956: 152-53, 252).

Because of its unprecedented impact in the Northwest and its conspicuous place in the primary accounts, the 1779-83 smallpox epidemic has found a significant place in western Canadian history and ethnohistory[2]. In fact, it has long since emerged as an event of considerable importance for students of both fur trade and aboriginal history. Despite this, the diffusion of this epidemic within the Petit Nord has only recently been given consideration, and then not with the detailed scrutiny it has drawn in other regions. Charles Bishop referred to the epidemic in passing in his study of the Northern Ojibway, stating that "From 1780 to 1782 a severe smallpox epidemic swept through the country greatly injuring the fur trade and drastically reducing the population in the area" (Bishop

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[2]Given its impact on the aboriginal population and the fur trade, the epidemic can arguably be called one of the most significant events in pre-confederation, western Canadian history. Among the numerous examples of works noting the epidemic's place in the history of western Canada see general histories such as: Innis (1956: 152, 192-193, 199, 252) and Morton (1939: 329, 331-334). See also Glover's introduction to the second volume of the published journals of Cumberland and Hudson Houses, an early summary of the epidemic's diffusion and effects on the Saskatchewan River (Glover 1952: lvii-lxii), and volume 1 of the *Historical Atlas of Canada* (Harris 1987: 144-145). The epidemic was a key event for several tribes living north and west of Lake Superior and this is reflected in many ethnohistorical studies, such as for the Plains Cree (Russell 1991; Milloy 1990: 12, 45, 70-71; Mandelbaum 1996: 44-45), the Lowland Cree (Lytwyn 1993: 360-373), the western Ojibway (Peers 1994: 18-21), the Assiniboine (Doige 1989) and the Indians of the western interior (Ray 1988). Both Decker (1988; 1989: 58-86) and Taylor (1977; 1982) examined the diffusion of this epidemic on the Northern Plains in some detail. On its diffusion and effects in the Pacific Northwest see Boyd (1985; 1994) and Harris (1994). See Krech (1978; 1983) concerning the impact on the Dene.

1974a: 320-321). Nevertheless, he made no attempt either to establish its extent or to assess its broader impact, despite his claim of heavy mortality[3]. Several other studies have touched on the epidemic in passing, or have noted its presence on the extreme margins of the region. Ray (1988: 105, 107) identified the disease among the Indians of Lake of the Woods and the Winnipeg River, while Decker (1988; 1989) studied its arrival at York Factory. Peers (1994: 18-21) summarised some of the evidence concerning the effects of the epidemic in her study of the Ojibway occupation of the west, including among the Ojibway living east of Lake Winnipeg. More generally, Young's *Health Care and Cultural Change* (Young 1991) remains the only substantial study of the health of the people of the region. However, it was mainly focussed on a more recent period, and thus he referred to this epidemic only briefly, while calling it "the first recorded severe epidemic in the subarctic" (Young 1991: 35). Hurlich (1983) also mentioned the epidemic in the context of the health status of the aboriginal inhabitants of northwestern Ontario.

In his recent dissertation, Lytwyn (1993: 360-373) made a more detailed study of the 1779-83 smallpox epidemic as part of a general ethnohistory of the Lowland Cree. In so doing he summarised the spread and effects of the epidemic in the Petit Nord, and is the only scholar to have done so. While Lytwyn's discussion is an important one, his study was focussed primarily on the Lowland Cree and, as such, his findings regarding the Petit Nord were not as detailed as they otherwise might have been. Consequently,

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[3]As well, Bishop (1972) failed to acknowledge this or any other epidemic in a paper on the demography of the Northern Ojibway and Swampy Cree.

some question remains as to how widespread the epidemic was, and how and when it diffused into the Petit Nord. This chapter will examine the origins and diffusion of the 1779-83 smallpox epidemic, focussing on its spread within the Petit Nord where additional evidence sheds new light upon its penetration and extent within the region.

ORIGINS OF THE 1779-83 SMALLPOX EPIDEMIC

Mexico City and the Southwest

Although there are several conflicting statements in the primary accounts regarding its origin, it is generally accepted that the 1779-83 smallpox epidemic began in Mexico City (Dobyns 1966: 441; Ramenofsky 1987: 130). Smallpox was "semi-endemic" in New Spain by this time, and Mexico City was the "major metropolis of the New World" (Cooper 1965: ix, 86). As such, smallpox was occasionally present there, although not on a permanent basis if the fear which Spanish officials had for its arrival is any indication[4]. In August of 1779 an epidemic broke out in the city. While it initially progressed slowly, in the ensuing months it increased both in virulence and the number of victims (*Ibid.*: 56). It continued into the early months of 1780, with perhaps a total of 40,000 fatalities (*Ibid.*: 68- 69; Dobyns 1966: 441). Thereafter it spread rapidly from the

[4]At the end of the eighteenth century Spanish officials had in place a set of regulations designed to prevent the disease from reaching Mexico City from elsewhere (Cooper 1965: 99-102). Had the disease been endemic in Mexico City, there would have been no need for them and, indeed, they would have been useless for combating a disease that was constantly circulating within its boundaries.

city. By late spring of 1780 it had progressed to the larger towns of what is now New Mexico, causing some loss of life throughout the summer and into autumn (Simmons 1966: 321). This was followed by a second, more deadly, wave in New Mexico the next year, which travelled up the Rio Grande. This time it afflicted the Indian population with great ferocity (*Ibid.*: 321-323). Santa Fe, the heart of the continental horse trade, was struck during the first three months of 1781 (*Ibid.*: 321; Stodder and Martin 1992: 66), and thereafter the disease spread rapidly northward, reaching the southern Canadian plains by the fall of that year.

Diffusion through the Horse Trade

During the latter decades of the eighteenth century a native trade network blanketed much of North America west of the Mississippi River, carrying trade goods between Mexico and Canada (Swagerty 1988; Ewers 1955), and it was this network that facilitated the spread of smallpox from Meso-America to the Northern Plains. According to Trimble (1985: 79), "The potential for the rapid spread of an epidemic from one region to another through this well-known trading system cannot be underestimated." Further, he concluded that "it is not unreasonable to assume that an infectious disease with a long incubation period could be transported several hundred miles and introduced into a new host population by trading parties who unknowingly were infected" (*Loc. Cit.*). Although this extensive web of exchange relationships predated contact in various forms and included many different goods (Swagerty 1988: 353), the introduction of horses to the continent during the early sixteenth century, their acquisition by Indian people beginning

in the early seventeenth century (Ewers 1955: 3), and the subsequent northward spread of the horse culture, had major implications for the diffusion of *ACIs* on the plains. The widespread adoption of the equestrian culture and the attendant horse trade "intensified the frequency and diversity of interethnic exchanges" (Swagerty 1988: 353), fostering extensive tribal contacts among different Indian groups (for example Boyd 1994b: 25). Such contacts facilitated disease transmission, as infection occurred easily while face to face trading was carried out (Dobyns 1992). Moreover, the use of horses increased the traders' rate and range of travel and allowed for longer trade expeditions in a single season (Wood 1980: 106; Roe 1968: 178, 379; Jenness 1989: 129). Consequently, fewer disease generations were required to maintain *ACIs* as the equestrian tribes journeyed from an area of frequent disease activity, New Spain, towards one of relatively infrequent activity, the Canadian Northwest. It also enabled the sick to travel greater distances before succumbing, enhancing the chances of passing the disease on as the victim journeyed. The horse trade thus provided closer connections between the Northwest and the Meso-American disease pool than had previously been the case, helping to overcome fundamental barriers to the northward diffusion of these diseases, including a paucity of navigable rivers, low population densities and great distances (Ramenofsky 1987: 167).

Not all of the people who participated in this trading network were equestrian. European firearms traded from the French and English to the more northern tribes were also important components of this trade, and some of the people who supplied them were horseless. This southward flow of guns and ammunition formed a trading system that

complemented that of the northward moving horse[5] (Secoy 1966: 2), and weapons from the northern traders found their way to the equestrian nations, either directly or indirectly. On the eastern part of the plains, European goods that had been obtained from the HBC or the French, including guns, were taken to the Mandan and traded for other commodities, as the two complementary trading frontiers converged during the mid-eighteenth century (Ray 1988: 87-89; Secoy 1966: 105). In this way, unmounted people, including the more northern Cree and the Ojibway[6], were tied into a trading network that began far to the south, and were therefore at risk to diseases that spread within that system.

Another significant aspect of the diffusion of the horse was its effect on the warfare and alliance structures of the Northwest, and this, too, would have aided in the diffusion of *ACIs*. While warfare predated the arrival of the horse on the Northern Plains, it is apparent that equestrianism did much to change its nature. For example, the need for horse-poor groups to obtain additional mounts was a powerful motivation for more frequent warfare and, in turn, led to complex alliances among different groups who banded together in order to battle the equestrian nations. During the eighteenth century in the western part of the Canadian plains, the trade in horses between the Assiniboine and Blackfoot tribes influenced the Cree and Assiniboine to join the Blackfoot in their

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[5]Secoy (1966) has traced the emergence, advancement and convergence of two complementary trading frontiers, those of the horse and gun, and their effect on military practices in the Plains region.

[6]Some of the Ojibway living on the plains probably acquired a few horses by the late eighteenth century while some of the Saskatchewan River Cree may have traded their first horses some time between 1732 and 1754, and had definitely done so prior to the epidemic (Peers 1994: 47; Milloy 1990: 25). Other Cree and Ojibway remained horseless throughout the historical period.

warfare with the horse-rich Shoshone, Kutenai and Flathead[7] (Doige 1989: 128, 132, 151). By increasing the rate of travel, horses also enabled raiders to travel greater distances in search of spoils and to gather temporarily in larger numbers than had previously been possible[8]. The Blackfoot, for instance, fought battles on many fronts and travelled great distances once they had acquired horses, in one instance raiding the Spanish in what is now the southern United States, but also crossing the Rockies and, later on, fighting the Cree and Assiniboine to the east (Ewers 1955: 171-172). Conversely, Ewers noted that Blackfoot oral tradition claims the Shoshone as their only enemy during the pre- horse period (*Ibid.*: 172). Farther east, at least by the end of the eighteenth century the desire for horses on the part of the Cree led them to begin stealing from the Mandan and Hidatsa, from whom they had long obtained horses through trade (Milloy 1988: 58). In modifying plains warfare by increasing its frequency and spatial extent, as well as perhaps the number of participants and the complexity of alliances, the horse promoted the long distance diffusion of *ACIs*, just as it did through the enhancements to the plains trading network.

In 1781, this trading network efficiently disseminated the smallpox virus over a wide area, as far-ranging equestrian nations such as the Comanche, Cheyenne, Crow, and Pawnee carried the virus northward in stages from the southwest to their trading partners throughout most of the Northern Plains (Taylor 1982: 43; Wood 1980: 100). Of the

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[7]According to Secoy (1966: 38) between the 1730s and the 1760s the Shoshone obtained horses by trading large numbers of slaves, and they obtained those slaves through warfare.

[8]For instance, Secoy (1966: 24) noted that the horse allowed the Apache to assemble several local groups into one larger, temporary, entity for the purpose of warfare.

plains Indian groups known to have been afflicted at this time, all participated in, or were linked to, this exchange at this time (Taylor 1982: 37-41; Figure 12). By the summer of 1781, two years after it first broke out in Mexico City, but only a few months after it struck Santa Fe, the disease had reached the very margins of the Northwest. Shortly thereafter it was among the Blackfoot, Cree and Assiniboine, all of whom were equestrian by this time. Over the next two years it would continue its northward diffusion, long after its spread had carried it beyond the limits of the horse frontier.

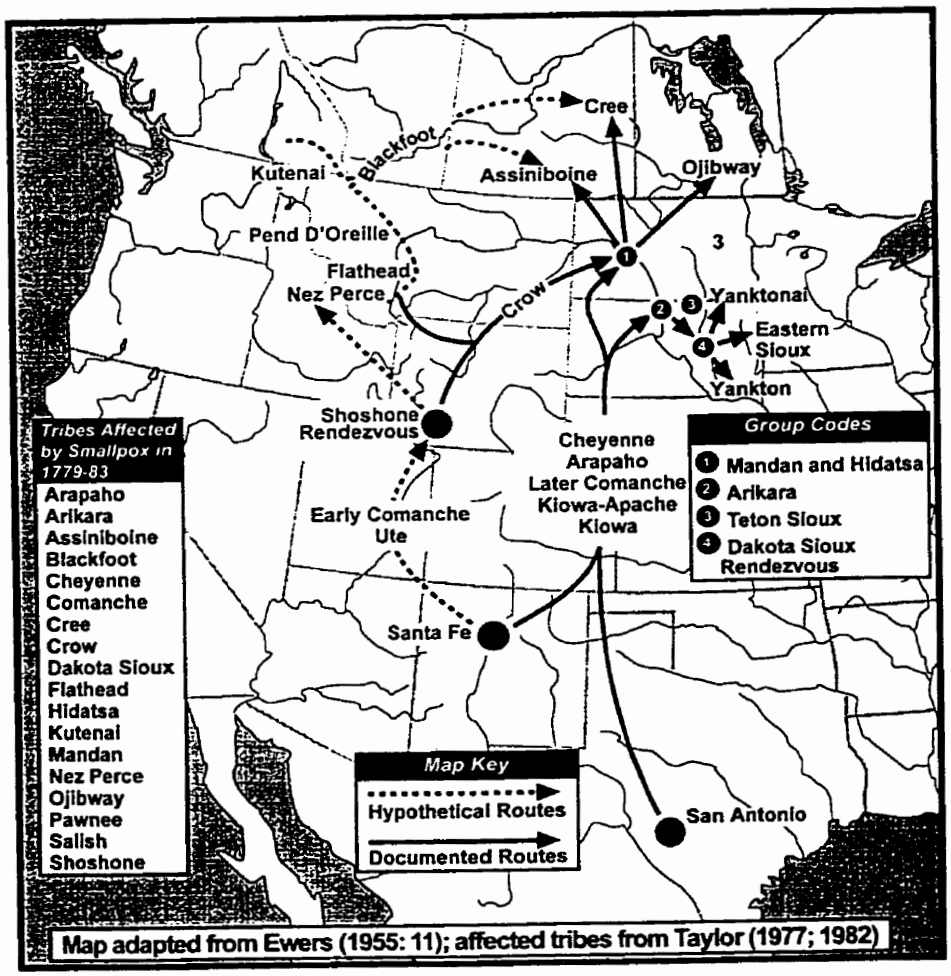

Figure 12: The pre-1805 horse trading network of the Central Plains

Although in later years epidemic disease was carried by trade between the Canadian and American plains, in this case smallpox spread to the Northwest through warfare rather than through trade. According to Edward Umfreville, a onetime HBC and NWC trader, "it was introduced among them [the Indians of the Northwest] by some of their war parties during the summer of 1781; and by the fall of the year 1782, it had diffused itself to every known part of the country" (Wallace 1954: 47-48). Two major avenues of diffusion into the Northwest may be identified. One saw the virus carried northward from the Shoshone, or Snake, Indians living towards the Rocky Mountains, into the western Canadian plains. The other derived from the village-dwelling tribes of the Upper Missouri and penetrated into the eastern Canadian plains. Both systems carried the disease into the Petit Nord, although from different directions, and both were brought northward by returning war parties as Umfreville suggested (Figure 13).

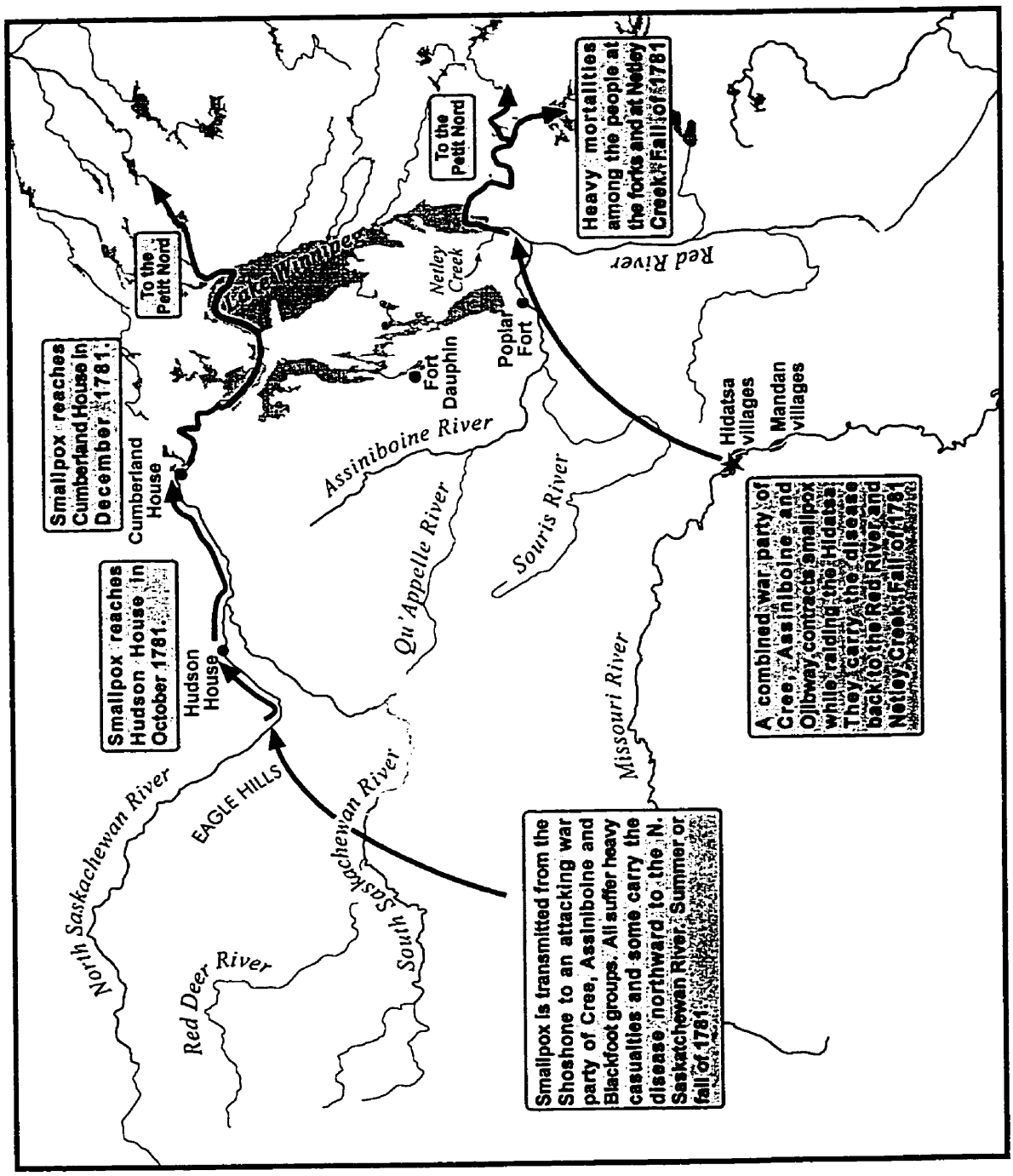

Figure 13: Approach of the 1779-83 smallpox epidemic to the Petit Nord

DIFFUSION FROM THE SHOSHONE INDIANS

One major thrust of this smallpox epidemic into the Northwest saw the virus introduced from the Shoshone Indians[9]. In the summer of 1781 a large party of Cree, Assiniboine, Piegan, Sarsi, Blood and Atsina warriors encountered their Shoshone enemies encamped near the Red Deer River (Figure 13). These people were sick and dying of smallpox, and offered no resistance. According to Saukamappee, a Cree elder who was part of the war party, and who was later interviewed by David Thompson:

> there was no one to fight with but the dead and the dying, each a mass of corruption. We did not touch them, but left the tents, and held a council on what was to be done. We all thought the Bad Spirit had made himself master of the camp and destroyed them. (Tyrrell 1916: 336-337)

Subsequently, the disease spread to the attackers, and was carried northward by those who fled in terror (Rich and Johnson 1952: 238-239, 298; HBCA B.239/a/80: 74; Williams 1975: 87).

Shortly thereafter, the disease reached the North Saskatchewan River. The diffusion process was described by Matthew Cocking at York Factory, who noted that the Cree, Assiniboine, Blackfoot, Piegan, Sarsi, Blood and Gros Ventres (Atsina):

> met with a Tent of Kanasick Athinewock (i.e.) Snake Indians who were all ill of the Small Pox (and were supposed to have received it from the Spaniards whom tis said those people trade with) killed them all and

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[9]The Shoshone were Uto-Aztecan speaking, equestrian Indians trading with several plains tribes and at war with several others. They are thought to have ranged widely in their travels and, although later on they would be most commonly found in the Great Basin region of the United States, in 1781 they had a presence in what is now southern Alberta, while trading with tribes from far to the south (Tyrrell 1916: 337-338; Shimkin 1986: 517). During the years leading up to the epidemic there was a "continual harassment" of the Shoshone and their allies by the Cree, Assiniboine and Blackfoot tribes (Doige 1989: 134-135).

> scalped them to carry away with them. By this means they received the infection and almost all of them died on their return, what few reached their own Country communicated the disorder to their Friends and it spread through the whole Country above here in some parts of which it yet rages (HBCA B.239/a/80: 74).

Here the disease passed between two major native economic systems. No longer was it among the wide-ranging and rapidly-travelling equestrian tribes of the plains, who had carried it from Santa Fe to the Northern Plains in the space of, at most, a few months. Instead, it was now spreading through the more northerly Indians of the parkland and the forests who travelled by much slower canoes. In moving from the Indians of the horse trade to those of the fur trade the epidemic's velocity declined considerably as it spread farther afield.

By October, the HBC men stationed along the North Saskatchewan were in contact with some of the smallpox victims. During the middle of the month, men from Hudson House encountered several who were just recovering at the Eagle Hills, survivors of a once larger party who had become too weak to move away from their deceased countrymen (Tyrrell 1916: 321, 322; Figure 13). According to one of the HBC men, Mitchell Oman, three-fifths of this group had died and the disease was then widespread among the Indians of this area (*Ibid.:* 322). On October 22 a party of infected Indians arrived at Hudson House (Rich and Johnson 1952: 262). On the 11th of December, 1781, the disease reached the HBC post of Cumberland House, farther down the Saskatchewan (Rich and Johnson 1952: 223-224). By Christmas Eve most, if not all, of the Indians living in the country about Cumberland House were suffering its effects (Glover 1952: lviii) and, shortly thereafter, so too were the Indians of nearby Basquia (*Loc. Cit.;* Figure

13). About the same time it began to spread into the Petit Nord.

York Factory

The epidemic devastated the people along the trade routes between Lake Winnipeg and York Factory shortly after it reached Lake Winnipeg (Figure 14). As William Tomison, the master of Cumberland House, travelled the Hayes River route in June of 1782, most of the Indians he encountered were either sick or dead of smallpox (HBCA B.49/a/12: 14d, 16, 17, 18, 19, 19d). The first infected Indians to reach York Factory were members of a brigade of sixteen canoes of Lake, or Northern Ojibway[10], who lived to the south of the Nelson River. They arrived on June 10 (HBCA B.239/a/80: 63-64; Lytwyn 1993: 361-262) and told Matthew Cocking that "a violent disorder has raged among their people which they describe as an eruption on the skin" (HBCA B.239/a/80: 63). They also informed him of widespread mortality among the Indians living to the west of Lake Winnipeg (*Ibid.* 63d). Although nothing was noted in the journal, later events make it clear that they were infected with the virus even as they told Cocking of its effects. Members of this group had first contracted the disease on a wintertime trip to Cumberland House with some Nelson River Cree (HBCA B.239/a/80: 63, 68), and it had spread among their people thereafter.

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[10]The Northern Ojibway trading at York and Severn were also called the Bungee or Pungee Indians. Entirely overshadowed by this epidemic was a mild outbreak of colds among the men of the fort the previous September which, once again, coincided with the arrival of the supply ship from England (HBCA B.239/a/80: 5).

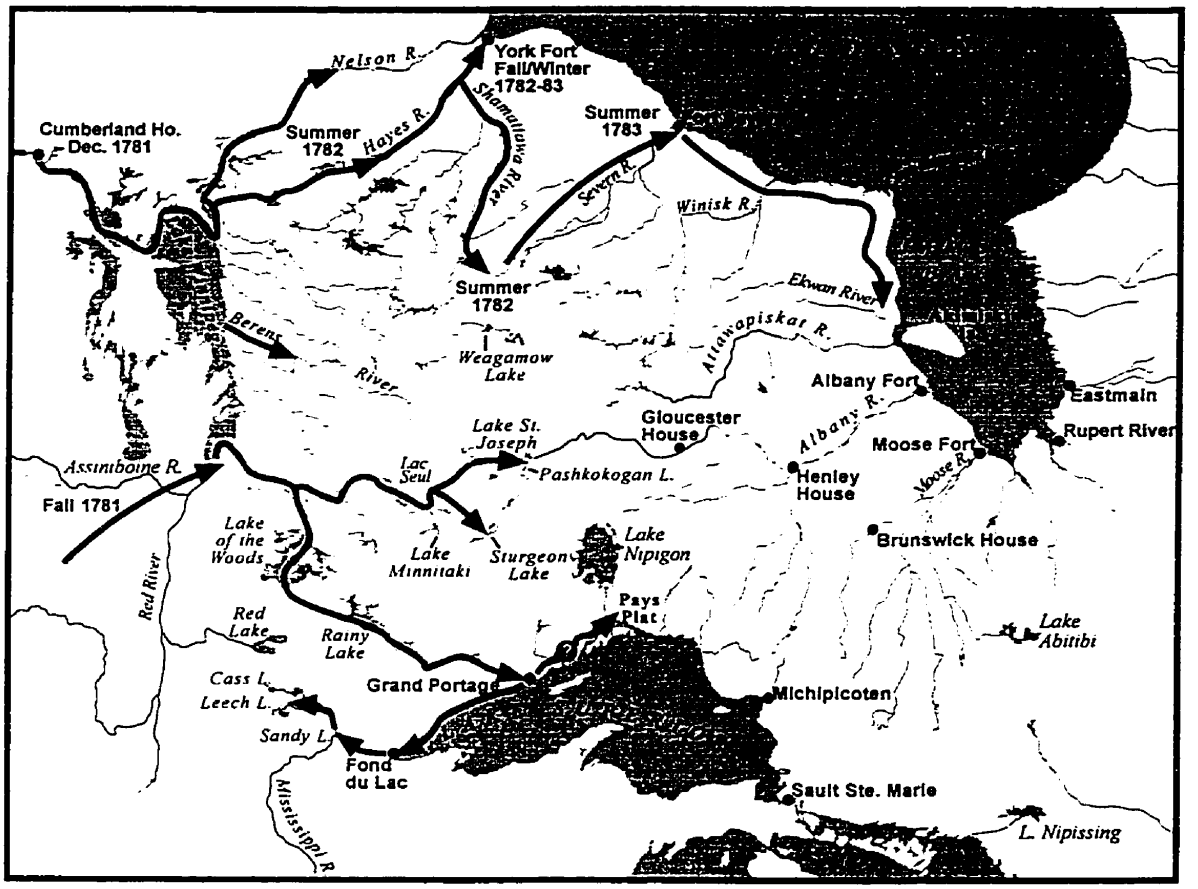

Figure 14: Diffusion of the 1779-83 smallpox epidemic in the Petit Nord

While the disease was quick to strike many of the inland Indians trading at York Factory and some of those living in the Lowlands, its spread to the Homeguard Cree was much delayed due to the desperate attempts of Matthew Cocking to shield the Homeguard and the few remaining Upland Indians who remained healthy. Thus, on July 8, 1782, with two Indians on the plantation suffering from the disease, Cocking directed the men to prevent other Indians from coming to the fort, to preclude contact with the two victims, and to tell any trading Indians that he would send men to meet them at a nearby creek (HBCA B.239/a/80: 79d). On August 6, he confined a brigade of Ojibway Indians to the creek as some of them were ill with the disease, lest "our Homeguards in the Marshes

would most probably catch the disorder either by coming to the Fort or by our peoples cloaths who must have gone over in a boat occasionally to them". He also declined to send a packet to Fort Prince of Wales by some Homeguard, fearing that the disease had already appeared among the Indians there (*Ibid.*: 90d, 91). In this, Cocking showed considerable knowledge concerning the transmission of the virus, both directly and via fomites. Subsequently, he ordered the masters of the HBC's Bayside posts to follow his lead. This policy was outlined in a letter to Peter Willdridge at Severn, dated August 12, 1782, in which the Severn master was told to:

> keep a strict look out, that none of the Homeguards come to the factory but keep them at a proper distance so that none of the Pungee's [Bungee or Ojibway] that come for debt may have any Communication with them. Should you find the disorder has attacked any of them, do all in your power for their preservation. If the Englishmen have been handling any person that may have had the small Pox, you must be careful that they shift, wash and air their Cloaths as well as themselves ere they go near one of the homeguards. (HBCA B.198/a/28: 3)

For a considerable time this policy was successful, for over two months after the arrival of the first smallpox victims at York Factory, William Falconer could write that "by this prudent precaution the homeguards here are preserved" (HBCA B.198/a/28: 3; B.239/a/80: 93-93d).

Nevertheless, eventually the York Factory Homeguard Cree, too, were stricken with smallpox (Lytwyn 1993: 366), perhaps the unwitting victims of the French destruction of York Factory. Although Cocking and his successor Humphrey Marten might do all in their power to isolate these people from the disease, after La Perouse's attack on the fort in the summer of 1782, the HBC men were dispersed and the Homeguard left to their own devices. Without Cocking's measures, the disease was

eventually passed on to these people who suffered terribly in the HBC men's absence (*Loc. Cit.*). When Humphrey Marten reoccupied York in 1783, he was visited by some of the Nelson River Homeguard who gave "a melancholy account of the havoc death hath made in the North River Indians most of whom are cutt off as are also the Churchill home guard, so that this country for some hundred of miles may too truly be said to be depopulated" (HBCA B.239/a/83: 5). Also that year, a Bungee man with his wife and child visited York, "the only survivors of about 30 that belonged to the tribe" (*Ibid.*: 36d). This was indicative of the impact of the epidemic on the natives of the York Factory area.

Severn

The disease continued its eastward diffusion, quickly spreading to the Severn Fort hinterland (Figure 14). On April 14, 1783, post master John Hodgson was visited by two men who, with their families, had travelled down the Severn River to trade. They informed him that:

> they have seen but one Indian during the Winter, that they are all dead Inland, these are very deeply marked with the small Pox, one of them has lost all his Children by it except one poor Boy, which is both blind and Lame, and they have been obliged to haul him all the Winter. (HBCA B.198/a/28: 14d-15)

Two days later Hodgson discovered that "they are intending to stay here to hunt Geese in the Spring, and as they have all been well of the small Pox since last Fall, there will be no possibility of the homeguards catching it" (*Ibid.*: 15). Given this timing, initial infection of the Indians of the Severn hinterland probably occurred during the summer of 1782, just

as the disease was spreading around York Factory. It would also seem that, as of the spring of 1783, the Severn Fort Homeguard had yet to contract the disease.

There are several routes by which the disease may have entered the Severn hinterland. Lytwyn's (1993: 372) map of the epidemic's diffusion into the Lowlands indicates that the disease had been carried from both the Albany and Hayes Rivers, the latter via Island Lake. However, a third possible route, also leading from the Hayes River, seems likely. William Tomison encountered several parties of Indians suffering the effects of smallpox as he passed down the Hayes River in June of 1782. At Oxford Lake he learned "that some of those that had been down [to York] are died on the passage since they left the Fort and several more Sick that are gone up the Shemattaway [Shamattawa] River" (HBCA B.49/a/12: 17). On July 25, 1782, Matthew Cocking met another Ojibway man who told him of the disease among his people. According to Cocking:

> The Man gives a shocking account of the ravages made by this cruel disorder among the Pungees. He says that Himself and the rest of our Deer hunters were on their way to their own parts in Shemattaway River when several of them became ill of the Small Pox most of which died before those who had not yet become ill left him and whom he expects have most of them shared the same fate by this time. (HBCA B.239/a/80: 87; see also *Ibid.*: 84)

These Ojibway, some of whom were infective, were fleeing the epidemic for their home territories.

The Shamattawa River was long used by Indians to travel between the Hayes and the Severn Rivers, into the Severn Hinterland[11]. Writing about his sojourn at Port Nelson

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[11]According to Lytwyn (1993: 128-130), some Northern Ojibway had shifted hunting territories

between 1694 and 1714, Nicholas Jérémie noted that one could ascend the Shamattawa from the Hayes, cross a portage, and descend the Beaverdam tributary of the Severn (Douglas and Wallace 1926: 35, fn. 55). This was a crossover point between the two drainage systems, and allowed the Ojibway of the Severn River region to visit and hunt for York Factory (Figure 14). It was also the route by which the infected Ojibway fled the epidemic to their territory. Once some of these infectives passed into the Severn hinterland, the virus was then passed to their countrymen who had not been to York.

It is unlikely that the epidemic spread throughout the entire Severn hinterland, however. Although Hodgson's statement seems to suggest that inland mortality was extensive, during the next two years there were no other reports of the sickness among the Severn Indians[12]. Indeed, despite fears of damage to the trade, Severn was subsequently visited by many canoes of Ojibway and Winisk River Indians[13], all with no mention of the disease. When compared to the tragic, and frequent, descriptions made at other posts, Severn Fort's hinterland seems hardly to have been touched, and it may be that only the most western of its Upland Indians were affected at this time.

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towards the north between 1749 and 1782, such that they were visiting York Factory during the winter and participating in the provision trade. By the time of the epidemic they were residing on the Nelson and Hayes Rivers within 100 miles of the fort, roughly up to the edge of the Hudson Bay Lowlands, and as far east as the Severn River.

[12]In the spring of 1784 Hodgson's successor, William Falconer, wrote that "...as to trade I foresee next Summer will produce a small share to us as well as you, the Natives being either gone to the Pedlars or out of the world but hope time will bring things to a better bearing" (HBCA B.198/a/29: 26). Of these two, the Canadians appear to have been a more pressing concern, as in a subsequent letter he claimed that "The trading natives belonging to this place have strolled away to the Pedlars..." (*Ibid.*: 44d; See also HBCA B.198/a/29: 36-36d).

[13]For examples see HBCA B.198/a/29, folios 34d, 36-36d, 37, 38-38d, 45-45d, 46-46d.

Smallpox was very slow to strike the Lowland Cree living near Severn (Lytwyn 1993: 367), perhaps partly due to the policy of isolating the local Indians from the Upland Indians at all Bayside posts. It is not known when these people were infected, but it may have occurred while the post was deserted by the HBC men, between August 20 and October 4 of 1783. There is evidence that the disease struck the Severn Cree with great force[14]. As noted by Lytwyn (*Ibid.*: 368), the goose hunt during the spring of 1784 was attended by few Indians, and William Falconer could count on very few hunters, although there remained many widows, and orphans about the place (HBCA B.198/a/29: 44d). Subsequently, attempts were made to attract replacement hunters to the fort from among those Lowlanders who traded at Albany Fort, by raising the standard of trade[15] (HBCA B.3/a/84: 32d, 52). In reaching Severn and its hinterland smallpox had penetrated a considerable distance into the Petit Nord. This was not the full extent of its diffusion, however, and others in the region were also being exposed to its deadly effects. Before it was done, this epidemic was to have a tremendous impact on the people living far to the south.

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[14]As a form of confirmation, Donald Gunn, a former HBC employee turned historian, stated that even as late as 1815 "the bleached bones of those who had become the victims of the plague, were to be seen in great quantities at several points on the shores of the Bay" (Gunn and Tuttle 1880: 87). Gunn had come to Hudson Bay in 1813 as an employee of the HBC, and for ten years served successively at York, Severn and Oxford House (Thomas 1972: 324). He was probably referring to Severn as one of the places where he saw these bones since, between 1814 and 1816, he was stationed at that post as a labourer (HBCA Biography "Donald Gunn").

[15]In May of 1785 Edward Jarvis wrote that: "My trade is about 2200 Made Beaver, and I fear will not exceed 3000, as I hear Assup's tribe, and many other Northern Indians going to Severn in consequence of an invitation and enlargement of the standard..." (Rich and Johnson 1954: 294). Captain Assup's people were in the midst of the epidemic just to the north of Albany Fort during the winter of 1783-84.

DIFFUSION FROM THE UPPER MISSOURI

At the same time that the 1779-83 smallpox epidemic spread from the Shoshone to the Saskatchewan River, and then into the Hudson Bay Lowlands, it also diffused into the Red River area from the Upper Missouri, and from there into the southern portion of the Petit Nord (Figure 13). Although this second route is frequently identified in the secondary literature, there is confusion surrounding the means by which the disease spread. Several researchers have accepted a statement written by David Thompson that, although seemingly authoritative, nonetheless is problematic[16]. In his *Narrative*, Thompson wrote that:

> From the best information this disease was caught by the Chipaways (the forest Indians) and the Sieux (of the Plains) about the same time, in the year 1780, by attacking some families of the white people, who had it, and wearing their clothes. They had no idea of the disease and its dreadful nature.
> From the Chipaways it extended over all the Indians of the forest to it's northward extremity, and by the Sieux over the Indians of the Plains and crossed the Rocky Mountains. (Tyrrell 1916: 322-323; Glover 1962: 236)

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[16]Although Taylor (1982: 41) rejected Thompson's claim, many other researchers have not. For example, A.S. Morton (1939: 329) believed it the sole means of diffusion to the Northwest, as did Ray (1988: 107), but Morton paraphrased the statement, adding that the whites were in American territory while Ray omitted the reference to whites. Dobyns (1966: 441) also summarised this passage. Doige (1989: 102) also removed the reference to infected whites. Peers (1994: 18) added a new interpretation, suggesting that "as reported to David Thompson, the epidemic spread from the south to the Sioux and Ojibwa, then to the Missouri area, and then to the Cree, Assiniboine and other plains tribes." Decker (1989: 59), also citing Thompson, wrote that "Smallpox was likely picked up by the Dakota Sioux from the trading villages along the Missouri River, and from there it was spread to the Ojibwa and Plains Natives to the north of them". Innis's belief was that the sickness "spread from the Mandans to the Assiniboines..." (Innis 1956: 192). The amateur historian George Bryce (1906: 49) claimed the same, stating that "the Assiniboines had gone on a war expedition against the Mandans of the Missouri river, and had carried back the smallpox infection which prevailed among the Mandan lodges." Typical of Bryce's historical works, there was no attribution for this statement. Glover (1952: lvii-lviii) suggested that the HBC men "believed it had started on the Mississippi and been carried northwards by returning war parties."

Despite the detail provided and the prominence of this explanation in the secondary literature, Thompson's assertion cannot be accepted as it stands. He began work on his manuscript, which was never completed, during the 1840s, long after he had received the information and while he was in his seventies, and it may be that he had become confused on this matter[17]. There are many traditions surrounding the origin and broader patterns of diffusion of this epidemic, each seemingly with a different interpretation. Indeed, in his memoirs Thompson also suggested that the sickness was brought from Canada and, as has been noted above, spread to the Northwest via the Shoshone (Tyrrell 1916: 109; 36-337). As time passed, no consensus emerged among the fur traders as to its origin, and evidently several theories circulated. This is summed up by Ross Cox, an early nineteenth century fur trader for the Pacific Fur Company and the NWC, who noted in his memoirs that:

> It is believed in the north-west that this disease was wilfully introduced by the American traders among the Indians of the Missouri .... The Americans throw the blame on the French; while they in turn deny the foul imputation, and broadly charge the Spaniards as the original delinquents[18].

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[17]Thompson's original informants were probably well informed on the subject, however. While staying at Jean Baptiste Cadotte's post at Red Lake in 1798 Thompson conversed at length with both Cadotte and Sheshepaskut, whom Thompson also called Sugar, and whom he described as principal chief of the Ojibway. This was probably "The Sweet", or "Le Sucre", the war chief of the Pillager Ojibway (Warren 1984: 376). Either man would have been familiar with the details of the epidemic and its effects upon the Ojibway.

[18]There is little doubt that the epidemic spread from the Shoshone to the northern tribes including the Blackfoot, given the corroboration of Saukamappee's testimony by the HBC men. In turn, the Shoshone may have contracted smallpox from the east, rather than from the south, if Cox was correct. He believed that:

> the disease first proceeded from the banks of the Missouri.... It travelled with destructive rapidity as far north as Athabasca and the shores of the Great Slave Lake, crossed the Rocky Mountains at the source of the Missouri, and having fastened its deadly venom on the Snake Indians, spread its devastating course to the northward and westward, until its frightful progress was arrested by the Pacific Ocean. (Cox 1857: 284)

(Cox 1832: 284; quoted in Boyd 1994: 24)

Thompson's is the only statement regarding the epidemic's origin, among many, which indicates raids on white families as the source of the disease. Noteworthy, too, is the fact that the location of these white families was never stated. Most significantly, however, the only extant Ojibway tradition of the epidemic specifies warfare with other Indians as the mechanism of diffusion, and this latter account must be considered highly reliable. While certain aspects of Thompson's tradition may be valid, such as the dual paths of diffusion through the Ojibway and the Sioux, the allusion to white families is erroneous.

Diffusion from the Hidatsa

There exists a second, more detailed, account of the spread of the disease to the Ojibway, based on testimony gathered from an Ojibway chief of considerable repute. This account claims that the disease was carried back to the Red River area through an attack on another tribe living near the Missouri River, and not from a raid on white families. This tradition, which has been mentioned by several scholars (Meyer 1977: 77; Peers 1994: 18-19; Taylor 1982: 41; Stearn and Stearn 1945: 47), has never been examined in detail, but provides a crucial second view of the entrance of the disease into the Canadian Northwest, one that explains its presence in the southern margins of the Petit Nord.

William Warren, the Ojibway historian[19], was given a detailed explanation of this

[19]That is, the chronicler of Ojibway oral history. Warren was part Ojibway, the son of Lyman Warren and Mary, the three-quarters Ojibway daughter of Michel Cadotte (Warren 1984: 9-11).

event by Esh-ke-bug-e-coshe, or Flatmouth, the noted leader of the Leech Lake Ojibway[20] (Warren 1984: 260). Flatmouth's age, his earlier life among the Assiniboine and Cree, and his status as an Ojibway spiritual leader (Schoolcraft 1969: V: 426, 427; Lovisek 1993: 293-294), and thus a keeper of tribal history, make him a very credible witness. Moreover, he was probably present when some of these events unfolded.

According to Flatmouth, shortly before the epidemic a combined party of Cree, Assiniboine and Ojibway gathered at a large Cree village located at what is now known as Netley Creek, near the mouth of the Red River, preparing to go to war against the Gi-aucth-in-ne-wug, or Gros Ventres (Warren 1984: 261; Figure 13). The warriors proceeded in a westward direction and thereafter encountered a village of their enemies, near or on the banks of the Missouri River. Upon attacking this village they found that:

> Through some cause which they could not at first account for, the resistance made to their attack was feeble. This they soon overcame, and the warriors rushing forward to secure their scalps, discovered the lodges filled with dead bodies, and they could not withstand the stench arising therefrom. The party retreated, after securing the scalps of those whom they had killed, among which was the scalp of an old man who must have been a giant in size, as his scalp is said to have been as large as a beaver

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[20]Also known as Guelle Plat. It is important to note that when Warren gathered this information Flatmouth was, at most, only one generation removed from the events. In fact, if estimates of Flatmouth's age are correct, and he was approximately 78 in 1852, he would have been alive during the 1779-83 epidemic, although of a fairly tender age (Warren 1984: 17). Flatmouth was "the head chief of the Pillager and Northern Ojibways..." according to Warren (*Ibid.*: 45-46). His father was Wa-son-aun-e-qua, or Yellow Hair, who rose to the chieftainship of the Leech Lake Ojibway in the eighteenth century (Waisberg 1984: 202). While he spent his later years at Leech Lake in present-day Minnesota, Flatmouth lived with the Assiniboine and Cree during his youth, and was also found among the Ojibway accompanying Alexander Henry (Younger) from the forks of the Assiniboine and Red Rivers up the Red in 1800 (Warren 1984: 138; Coues 1965: I: 54). In 1775, Alexander Henry, the Elder, met numbers of Pillagers at Lake of the Woods, near Rat Portage and the former site of Fort St. Charles (Bain 1901: 243-244). In 1820, he was described by Henry Schoolcraft as one of the three principal chiefs of Leech Lake and in 1832 he hosted Schoolcraft at Leech Lake (Williams 1953: 165; Schoolcraft 1973: 253). He also appeared at the HBC's Lac la Pluie post on occasion during the nineteenth century (e.g. HBCA B.105/a/12: 36d; B.105/a/13: 12d; B.105/a/14: 48). For more on Flatmouth see Waisberg (1984: 202-204).

skin. (*Loc. Cit.*)

The lack of resistance was due to the effects of smallpox among the Gros Ventres. By chance, the war party had reached the village at the height of the sickness and the inhabitants were thus unable to defend themselves. However, while the allied tribes' military success was total, they also contracted this disease during the raid and, as they made their way homeward, it began to manifest itself among their ranks with disastrous consequences. Warren further recorded that:

> On their return home, for five successive nights, this scalp [that of the giant man], which had been attached to a short stick being planted erect in the ground, was found in the morning to lean towards the west. This simple occurrence aroused the superstitious fears of the party, and when, on the fourth day, one of their number died, they threw away the fearful scalp, and proceeded homeward with quickened speed. Every day, however, their numbers decreased, as they fell sick and died. Out of the party which must have numbered a considerable body of warriors, but four survived to return home to their village at Dead River. (*Loc. Cit.*)

Thus, in a manner eerily similar to the Shoshone, the Gros Ventres had been avenged by the mechanism of their own defeat.

These Gros Ventres were the Hidatsa, one of the village tribes living roughly to the southwest of Lake Winnipeg, along the banks of the Missouri near the Knife River[21] (Kehoe and Kehoe 1974; Meyer 1977: 11, 27; Trimble 1988: 8; Wood and Thiessen 1985: 6-8 Figure 13). According to Warren, the Hidatsa often suffered attacks at the hands of these tribes, until the 1837-38 smallpox epidemic devastated the Missouri River

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[21]Their identity is not entirely clear from Warren's statement as two distinct groups were at times referred to by the latter name. Taylor (1982: 41) suggested that these were the Atsina, a tribe of bison hunting Algonkian speakers, who were also called the Gros Ventres, and who were associated with the Blackfoot living far to the west (Kehoe and Kehoe 1974: 231). This is very unlikely, however.

horticulturists. Writing in 1852, he stated that: "Till of late years the Kniste-no and Assineboins were accustomed to send their war parties against the Gros Ventres and Arickarees, and the Ojibways were often induced to join them" (Warren 1984: 179).

It may be that at about the same time that the Ojibway, Cree and Assiniboine contracted smallpox from the Hidatsa, the Sioux caught the sickness from the Mandan. Taylor (1982: 40-41) suggested that "a possible source of infection for at least some Sioux groups was the Sioux attack which destroyed the Mandan villages on the west side of the Missouri during the midst of the epidemic". It is well documented that the Mandan were dealt a severe blow by the sickness (Taylor 1977: 40; Trimble 1988: 7-9). Indeed, as Wood and Thiessen (1985: 6, 8, 72) noted, the village tribes were devastated by the epidemic, leading to fundamental changes in settlement patterns and material culture, including the creation of refugee villages composed of the survivors from different villages. Certainly it was not unheard of for the Sioux to attack the Mandan, with whom they also traded. Whether the Sioux caught the smallpox from the Mandan or from elsewhere, however, it is certain that they too fell victim to this awful disease. From the winter counts, only the westernmost Sioux were afflicted, or those who were far removed from white settlement at this time[22].

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[22]The epidemic made a prolonged stay among the Sioux, appearing as the subject for two consecutive winter count years (Stearn and Stearn 1945: 49; Taylor 1982: 40). However, it is very important to note that of the few extant winter counts that cover this period, not all note the epidemic. The *Battiste Good* (Mallery 1972) and *High Hawk* (Curtis 1970) counts from the Brule Teton, and the *Garnier* (Grange 1963) count, from the Oglala Teton, record smallpox or measles as the significant event from these years. The remaining example, the *John K. Bear* Lower Yanktonai count (Howard 1976), does not. Indeed, there is no evidence of any kind that the more eastern bands were afflicted by this epidemic. Thus, it is entirely possible that the 1779-83 smallpox epidemic struck only the westernmost of the Sioux.

The Red River Valley

While most of the war party mentioned in the Flatmouth account succumbed to smallpox on their precipitous retreat from the Missouri, a few survived to carry the virus back to the Red River region. Their destination was Netley Creek, where the disease was passed to the large camp of people awaiting the return of the war expedition[23] (Warren 1984: 261; Figure 13). The resulting massive mortality among the Cree and Assiniboine at this place was said by Warren to have given the stream the name of Ne-bo or Death River[24] (*Loc. Cit.*). Warren did not state the timing of this outbreak, but it most probably

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[23]At least in an earlier time, the village had been a place for the Cree and Assiniboine to leave the elderly and the children while those able made the long and difficult trip to trade at Hudson Bay beginning each spring (Coues 1965: I: 41). It was also undoubtedly a community that attracted Ojibway from the Boundary Waters and the Upper Mississippi who came to the west to hunt bison during certain seasons, a pattern of movement predating the epidemic and one that survived well into the nineteenth century (Peers 1994: 29; Warren 1984: 40). Netley Creek was known to the traders as "a favorite Indian encampment" (Masson 1960: I: 268). The large seasonal populations had, since La Vérendrye's Fort Maurepas, encouraged fur traders to erect posts in its vicinity, and this spot was resettled by the first Montreal traders to enter the Grand Nord following the Conquest (Champagne 1971: 23; Morton 1939: 182, 271; Masson 1960: I: 268).

[24]Also known as Dead River or *Riviére aux Morts*. Albert Thompson, the grandson of Chief Peguis whose band of Ojibway moved onto Netley Creek in the 1790s, recounted a tale of his grandfather's arrival on the Dead River after the epidemic which appears to verify Warren's statement regarding the origin of that name (Thompson 1973: 1-2). However, the tale presents logical inconsistencies that render it impossible to accept as is. Not the least of these is Thompson's conclusion that the Peguis band arrived in the latter part of the 1790s, but found the dead bodies and teepees of the victims of the 1781 smallpox epidemic, and even a surviving child, called Pockwa-now, who had been forced to live off his own abilities after the others died. This boy was said to have been in his mid-twenties in about 1812, and so born after the epidemic (*Loc. Cit.*). It is evident that Peguis gave a similar story regarding arriving at the Red River after the epidemic to Donald Gunn (for instance, see "Peguis Vindicated" *The Nor'-Wester*, April 28, 1860; see Peers 1994: 219, fn. 76). Conversely, the fur traders Alexander Henry (Younger) (Coues 1965: I: 41) and John McDonnell (Masson 1960: I: 268), and William Keating of the Long expedition of 1823 (Keating 1959: II: 77) all attributed the name to a massacre inflicted on the inhabitants by the Sioux. Keating added that this event had occurred about forty-five years before his arrival, or about 1778, and that 250 lodges of Chippewas (Ojibway) had succumbed (*Loc. Cit.*). McDonnell also included Ojibway in those who died in this massacre. With the exception of the epidemic, there is no evidence of such a degree of mortality in the York Factory records from this period. Given so many fatalities among the Indians living in that post's hinterland, it would be expected that some confirmation would appear in the HBC records. Thus, the

occurred in the fall of 1781[25].

Warren was not the only one to note this path of diffusion. Alexander Mackenzie acknowledged that "it was generally supposed to be from Missisouri [*sic*], by a war party..." (Lamb 1970: 75). Similarly, Dr. Douglass Houghton noted that this was the common belief of the Ojibway who had been afflicted by this disease. He observed that:

> The most western bands of Chippewas relate a singular allegory of the introduction of the smallpox into their country by a war-party, returning from the plains of the Missouri, as nearly as information will enable me to judge, in the year 1784. (Schoolcraft 1973: 579)

Although Houghton had mistaken the date of infection, these accounts confirm Flatmouth's statements to Warren.

There is further evidence of the impact of this epidemic in the valleys of the Red and the Assiniboine Rivers. It was common knowledge among the later fur traders and the Indians that a large Indian burial mound near the juncture of these two important rivers contained the bodies of those who died during the 1779-83 smallpox epidemic. While camped near the mass burial site in 1800, Alexander Henry the Younger was:

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explanation provided by Warren seems more likely.

[25]It is likely that the outbreak of smallpox at Netley Creek coincided with that at the forks of the Red and the Assiniboine, which was known to have occurred in the fall of 1781 (See below). According to Ray (1988: 46-7), the plains groups of southern Manitoba and Saskatchewan made annual trading ventures to the Mandan late in the summer and into autumn. If they went to war with the Hidatsa, who lived near the Mandan, then it seems logical that they would have done so during their excursions to the Missouri. Certainly, it was not earlier than the end of April, as Donald McKay, a Canadian trader stationed on the Assiniboine River, returned from a trip to the Hidatsa villages on April 24, 1781, without observing the disease (HBCA E.223/1: 20-34).

> troubled by swarms of water-snakes, which even come into our tents at midday .... They appear to lurk and breed in the old graves, of which there are many, this spot having been a place of great resort for the natives in 1781-82; and at the time the smallpox made such havoc many hundreds of men, women, and children were buried here. (Coues 1965: I: 46)

Described by Edwin Denig as "an area of several hundred yards in circumference and ten to twenty feet high, being the cemetery of nearly an entire camp of 230 lodges who died in the infection" (Ewers 1961: 115), this was actually an ancient mound that was said to predate the Cree occupancy of the Red River valley, although they employed it during the epidemic. Donald Gunn (1867: 399-400) was told by a survivor that:

> at the commencement of the mortality the Indians, for some time, buried in the mound above described, but did not erect it, and that at a later period of the disease the living did not attempt burying the dead. (*Ibid.*: 400)

It probably occurred that during the initial stages of the epidemic, when there was sufficient manpower, the living could lay the dead to rest. As the epidemic progressed and the dead overwhelmed the living, however, there was no hope of interring the deceased. The sight of unburied piles of human remains was one that would be seen in the Canadian Northwest in several places for many years after the epidemic[26] (See, for example, Gunn and Tuttle 1880: 87; Lamb 1970: 122; HBCA E.3/3: 69; Wallace 1954: 8; Taylor 1982: 39).

Other evidence of smallpox in the area concerns an attack on Canadian traders along the Assiniboine River that may have helped spread the disease. In the fall of 1781,

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[26]Denig described the graves thus: "hundreds of corpses lie beneath them, or rather the tumulé are composed of many separate burials alongside and on top of each other" (Ewers 1961: 124). Thus, it appears that it was not properly a single mound but closely packed gravesites built on and into a pre-existing burial structure.

the Canadian post of Fort des Trembles (Poplar Fort), just to the west of Portage la Prairie, was attacked by a large party of Cree, Assiniboine and Bas de la Rivière, or Ojibway[27], Indians (Gough 1988-92: I: 196; HBCA E.3/3: 64d; Figure 13). In the face of this attack the Canadian traders, including post master William Bruce and Captain James Tute[28], abandoned the fort and embarked by canoe for the forks (*Loc. Cit.*; Wallace 1934: 427, 428). As the Canadians left, they were approached by two young Indians from towards Fort Dauphin, strangers in the area who, in the words of Henry the Younger recounting the events some twenty-five years later:

> requested to embark, which was allowed them. At this time there was no mention of the Small Pox, yet but the first day they embarked, one of the lads complained of being unwell. The people gave him the loan of a blanket to cover himself with. (Gough 1988-92: I: 197)

Several of the Canadian men thereafter caught smallpox and died, probably while at the forks (Morton 1939: 329). The following June 22, while on the way to York Factory William Tomison was told by some Indians from the west side of Lake Winnipeg that "Bruce the Canadian Master has died of the Small pox also Captain Tute and many of

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[27]The term *Bas de la Rivière* was generally used by the NWC men to describe the entrance of the Winnipeg River into Lake Winnipeg, where various forts were located during the fur trade period (See Ray 1984: 127-129), and as of 1775 that area was inhabited by the Cree (Bain 1901: 246-249). However, Henry (Younger) seemed to suggest that they were not Cree, and the HBC's Peter Fidler stated that Bruce was attacked by "a band of Southern Indians joined by some Bungees" (HBCA E.3/3: 64d). Thus, they may have been Ojibway who had moved to the Winnipeg River prior to the arrival of the younger Henry, but after his uncle had passed through.

[28]Captain James Tute, one the earliest of the post-conquest British-Canadian fur traders to reach Grand Portage and the Saskatchewan River. In 1767 he met Jonathan Carver at the mouth of the Wisconsin, and subsequently led Carver's expedition to Grand Portage (Carver 1974: *Forward*). He had been dispatched by Robert Rogers on an expedition to find a northwest passage, which was in fact an excuse to settle at Fort la Prairie to trade furs, but a lack of goods prevented him from proceeding (*DCB* "Rogers, Robert": 681; Morton 1939: 261, 267). In 1773 he represented the firm of McBeath and Graham on the Red Deer River and was at Beaver Lake for Joseph Frobisher in 1776-77 (Morton 1939: 289, 318). By 1779-80 he was settled on the Saskatchewan River at Sturgeon Fort (*Ibid.*: 330).

their men has died of the like Disorder and starved for want of provisions[29]"(HBCA B.49/a/12: 16).

It may be that this unfortunate circumstance was partly the reason that David Thompson concluded that: "In the year 1782, the small pox from Canada extended to them [the plains Indians], and more than one half of them died" (Tyrrell 1916: 109). The story of the attack on Bruce's fort and the subsequent epidemic was a part of fur trade lore by the time Thompson joined the NWC, and he would no doubt have learned of it from the other traders. Moreover, William Tomison was Thompson's mentor during his term in the HBC, and he was ready to implicate the Canadian Pedlars as the source of the disease even before he heard of the deaths of Tute and Bruce[30] (Rich and Johnson 1952: 238-239). Thus, it seems that this brigade carried the virus back to the forks in the fall of 1781, and thereby contributed to the erroneous belief that the disease was brought from Canada by the Canadian fur traders.

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[29]Fur trade tradition had it that the epidemic intervened on behalf of the traders, preventing them from being murdered by the Indians. John McDonnell, a NWC trader working in the Red River valley in the mid-1790s, wrote that:

> Owing to this affair, the traders were obliged, for fear of being cut off, to reembark their canoes and return to winter at the Forks. The small pox seizing the Natives and sweeping off three fourths of them, compelled them to lay aside their intention of cutting off all the white men in the interior country. (Masson 1960: I: 270)

[30]At the time of the epidemic, at least two of the senior HBC officers independently placed the blame on the Canadian Pedlars, although this was simply speculation on their part. In February of 1782 Tomison speculated that Canadians had brought smallpox with them from Canada to the Mississippi River, infecting the Indians there, with the disease eventually spreading to the Shoshone (Rich and Johnson 1952: 238-239). Matthew Cocking at York Factory stated that "The Natives at Basquiou... probably have recieved [*sic*] it from some of the Canadian Pedlars who may have brought the disorder up with them from Canada" (HBCA B.239/a/80: 69d).

The Boundary Waters and the Upper Mississippi

While the Cree and Assiniboine suffered great mortalities at Netley Creek and at the forks of the Red and the Assiniboine rivers, the Ojibway fled the Red River valley, transporting the smallpox virus as they went. This sort of flight in the face of epidemic disease was a common response to epidemic disease among non-sedentary, aboriginal people in North America, and was often an important factor in the widespread diffusion of *ACIs*. Their eastward path carried the disease along the major line of communication between Red River and Lake Superior (Warren 1984: 262). In doing so, it passed from the territory of the Cree and Assiniboine to that of the Ojibway[31].

Smallpox quickly spread by this route from the Red River to the Petit Nord. It struck first at Rainy Lake, where the Ojibway community of this region was nearly depopulated[32] (Warren 1984: 262; Figure 14). This community probably played a key role in the diffusion of the disease for, throughout the historical period, large numbers of people from considerable distances, sometimes numbering in the thousands, gathered there at certain seasons to partake of the rich resources of Rainy Lake and Rainy River,

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[31]And so from the plains to the forest. The *Atlas of Great Lakes Indian History* (Tanner 1987: 173) presented almost the opposite view, suggesting that the disease was carried from Ojibway villages in Wisconsin and Minnesota to Grand Portage and then by the Boundary Waters to Rainy River and Lake of the Woods. However, there is no evidence that the disease even struck the Ojibway east of Fond du Lac.

[32]At this time Rainy Lake was very near the frontier between Cree and Ojibway territory within the Boundary Waters region, suggesting that the Ojibway were fleeing Netley Creek to their own territory. In a memorandum dated October 18, 1781, Alexander Henry the Elder stated that, at Rainy Lake the borders were "Inhabited by the Chipeways, being the Wester-most part of there [*sic*] Territories..." while at Lake of the Woods, "here the Natives begins, to speak the Kristinoes Language..." (NAC MG 19 A4; reprinted in Burpee 1935: II: 589). The Ojibway of this region were led by a noted chief named Nittum, or Nectam, an Ottawa Indian from Lake Michigan who was a member of the Bear clan (Lovisek 1993: 288-289), and who had lived in the area as early as 1765 (Johnson 1921-1953: 11: 806-807; Waisberg 1984: 213-214). The name Nittum signified his rank, as did another name given to him, Premier (Waisberg 1984: 213, 216; Lovisek 1993: 288). It was his grandson, also given the name Premier, who was one of the signatories to the Selkirk treaty in 1817 (Waisberg 1984: 216).

and to engage in social and religious activities (Waisberg 1984: 126-130, 133). The spring and early summer sturgeon run and the fall wild rice harvests of this area were capable of supporting very large populations (*Ibid.*: 132). Although Flatmouth did not state it, it seems likely that the Rainy Lake area served as a nexus for infection throughout the Boundary Waters region.

What testimony exists suggests that this region was almost emptied in the wake of the epidemic. In 1783, Jean-Baptiste Cadotte, a fur trader from Sault Ste. Marie working south of Lake Superior, wrote that there had been many deaths at Rainy Lake (NAC MG 21 Add. Mss. 21758: 213), while Flatmouth told William Warren that the same location was almost entirely depopulated (Warren 1984: 262). On June 22, 1782, John Kipling, master of the HBC's Gloucester House, was visited by two canoes of survivors from Rainy Lake and was told that "there is a great Mortality among the Indians and that most of the Indians in and near the raney Lake is dead; and that the assineybols country is almost Depopulated" (HBCA B.78/a/7: 24). In the 1790s, Alexander Mackenzie wrote of Basswood Lake, just to the east of Lac la Croix, "Before the smallpox ravaged this country, and completed what the Nodowasis [the Sioux], in their warfare, had gone far to accomplish, the destruction of its inhabitants, the population was very numerous..."(Lamb 1970: 103), and of Lake of the Woods, "war and small pox had diminished the inhabitants..." (*Ibid.*: 107). Finally, the disease was said to have reached Grand Portage on Lake Superior via the Pigeon River route, although its effects were not stated (Warren 1984: 262). Overall, in the 1790s, Mackenzie (Lamb 1970: 111) said of the region between the Red River and Lake Superior that it:

> was formerly very populous, but from the information I received, the aggregate of its inhabitants does not exceed three hundred warriors; and, among the few whom I saw, it appeared to me that the widows were more numerous than the men[33].

The epidemic then spread from the Boundary Waters to the Upper Mississippi region. From Grand Portage, smallpox was carried south and westward along the Lake Superior shore to Fond du Lac, where the Ojibway there suffered greatly (Warren 1984: 262). One estimate suggested that of three hundred Indians then at Fond du Lac, nearly all died[34] (Schoolcraft 1973: 579). From Fond du Lac, fleeing Ojibway carried the disease west and northward, to Sandy Lake (Warren 1984: 262; Figure 14). There, it was said, the once large village was reduced to some seven lodges (*Loc. Cit.*). According to Warren (*Ibid.*: 344), the Sandy Lake Ojibway village only recovered by augmentations from villages to the east that had escaped this scourge. Thus he learned that:

> in the year 1782, the village of Sandy Lake became nearly depopulated by the dreadful ravages of the smallpox. This band, however, gradually recovered their former strength and numbers, through accessions from the villages of their people located on Lake Superior, who were drawn to the Mississippi country by the richness of the hunting grounds, and facilities of obtaining a plentiful and easy livelihood. (Warren 1984: 344).

As was the case with Rainy Lake, the trader Cadotte stated that the residents of Sandy

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[33]Mackenzie's descriptions, and his estimate, should not be dismissed without consideration. While it is true that he relied on the testimony of others, the corridor between Grand Portage and the Winnipeg River was the established route of the Montreal-based fur traders into the region west of Lake Winnipeg, and Grand Portage had long been established as the annual summer rendezvous for those from the Northwest and from Montreal. In all probability the Montreal traders were well informed on the effects of the epidemic in the Boundary Waters, even if they had not witnessed its ravages.

[34]Although Houghton gave a date of 1770 for this outbreak, it almost certainly part of the 1779-83 epidemic.

Lake, Fond du Lac and surrounding places were all dead[35] (*Wisconsin Historical Collections* [Thwaites] 1888 XI: 170; NAC MG 21 Add. Mss. 21758: 213).

Finally, the sickness was carried up the Mississippi River to Leech Lake by some of the Pillagers, where it "somewhat lessened" their numbers (Warren 1984: 262). Arriving at Leech Lake some two years later, J.B. Perrault found a replenished population notwithstanding "*que deux ans au paravant il en avoit péri beaucoup par le picotte*" (Cormier 1978: 51). There the disease was said to have died out[36] (Warren 1984: 262). Although Perrault (Cormier 1978: 51) claimed that many had died of the smallpox, he also suggested they were still numerous in his time, and Warren's statement does not seem to reflect the same death toll found at other Ojibway communities on the Upper Mississippi. If the death rate among the Pillagers was less, especially when compared to the situation at nearby Sandy Lake and Fond du Lac, it is likely that some had been exposed at an earlier date, perhaps during the 1750s, when parties of Indians travelling to the east exposed themselves to the disease in their travels. In Houghton's version of the smallpox tradition, at least one of the Ojibway at Fond du Lac was a survivor of the earlier smallpox epidemic of 1750 that had been contracted while in Canada (Schoolcraft 1973: 579). Conceivably, some of the others from Leech Lake may have been immune at this time as well.

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[35]Perhaps not surprisingly, then, a Captain Robertson at Michilimackinac wrote to Lieutenant-Governor Haldimand in July of 1784, stating that he had not had a single Indian visitor from the Mississippi although he had had several from Lake Superior and Lake Huron (NAC MG 21 Add. Mss. 21758: 268).

[36]The disease may have hit nearby Cass Lake, as well. According to Houghton's sources, "of a large band then resident at Cass Lake, near the source of the Mississippi River, only one person, a child, escaped. The others having been attacked by the disease, died before any opportunity for dispersing was

By all accounts, the disease did not spread east from Fond du Lac along the more populated southern shore of Lake Superior, nor did it move south to the major Ojibway village at Mille Lacs. In Warren's (1984: 262) words "It did not, luckily, spread generally, over the country occupied by the tribe, and its ravages were felt almost exclusively in the section and villages which have been designated[37]". Houghton (Schoolcraft 1973: 579) learned that although "it did not extend easterly on Lake Superior, it is believed that not a single band of Chippewas north or west from Fond du Lac escaped its ravages." Moreover, there is the letter from Jean-Baptiste Cadotte, dated Sault Ste. Marie June 16, 1783, which seems to verify this limit to the epidemic (Thwaites 1888: 170). The letter states simply that "All the Indians from fond du Lac, rainy Lake, Sandy lake, and surrounding places are dead from smallpox[38]". Critically, the region mentioned in this letter as having been afflicted included only Fond du Lac and the country lying north and west. Thus, by three separate accounts, the 1779-83 smallpox epidemic diffused no further south and east among the Ojibway than the head of Lake Superior.

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offered" (Schoolcraft 1973: 579).

[37]At the time of the epidemic, Leech Lake was probably the northwestern limit of Ojibway territory in the area as they had yet to take Red Lake away from the Sioux (Warren 1984: 282). Given the villages mentioned by these sources it seems likely that the epidemic touched every Ojibway community west of Lake Superior and south of the Boundary Waters, except Mille Lacs.

[38]The original states that: "*Tous les sauvages du fond du Lac, lac de la Pluie, lac des Sables et autres endroits circonvoisine sont morts de la picotte*" (NAC MG 21 Add. Mss. 21758: 213).

Diffusion into the Interior of the Petit Nord

As smallpox was spreading through the Boundary Waters, or along the southern flank of the Petit Nord, and into the Hudson Bay Lowlands along its northern margins, it was also penetrating into the interior of the region from the south (Figure 14). Most likely, the disease was carried along the English River to Lac Seul (Lytwyn 1993: 372), which was a key part of this route into the interior. In 1784, Edward Umfreville was informed by his Indian guides that "all the Indians who trade at Albany Fort, pass by" Lac Seul (Douglas 1929: 41). Although there is no documentation of the epidemic from this period, reference was made in 1807 by the NWC fur trader Aeneas McDonell to a sickness at Lac Seul that certainly has the earmarks of this epidemic. In a letter to Roderick McKenzie dated Lac Seul, June 15, 1807, McDonell explained that in that region "the natives are by no means numerous since the general ravage made amongst them in the year _____" (NAC MG 19, C4, Volume 53: 3). In the copy of the letter found in the Masson Collection no date was supplied, merely an underline. In it, McDonell also stated that he arrived in the country in the fall of 1803 (*Loc. Cit.*). Since the date was not provided, it may very well be that the event took place before his arrival and he was leaving it to McKenzie to verify the date. If so, and since there is no record of any catastrophic event among the Lac Seul Ojibway prior to 1803 and after the 1779-83 smallpox epidemic, it would indicate that the Lac Seul people were struck by this epidemic and that population recovery was slow. Confirming this was Charles McKenzie who, over sixty years later in 1845, noted the death of a woman who may have been the

last remaining survivor of his epidemic at Lac Seul[39]. He wrote that:

> She was the only one now pocmarked in this quarter—there were a few some thirty years ago—she well remembered when that scourge, the small-pox destroyed the thousands of Indians throughout the country who will never increase to what they were in those days in all probability. (HBCA B.107/a/24: 10)

Other evidence of the epidemic's course between Lake Winnipeg and the Albany River comes from Gloucester House, at the time of the epidemic the HBC post farthest inland within the Petit Nord (Figure 14). The journals from Gloucester House do not contain any explicit statements regarding the diffusion of the disease, however, and its extent must be reconstructed from several brief statements made by Indians visiting the post to trade. At this time the degree of mobility of the inland Indians was still very high. During certain key seasons many still travelled hundreds of miles to trade[40] (Lytwyn 1986: 32), and the traders at Gloucester therefore learned of events occurring within a large part of the Petit Nord. Of the Indians bringing news of the epidemic to the post, the home territories of several can be identified, and it is possible to arrive at a rough idea of the extent of this epidemic as it passed into the interior of the region.

The first mention of the epidemic in the Gloucester House journal was on May 27, 1782, and it is evident that by then the disease had been raging inland from the post for

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[39]Bishop estimated her age at death to be about ninety years (Bishop 1974: 327).

[40]For instance, during the first half of the 1770s some of the Sturgeon Lake Indians, located to the west of Lake Nipigon, traded at Severn where Edward Umfreville was stationed (Douglas 1929: 15; HBCA Search File "Umfreville, Edward"). The family of Tinniwabino, who lived near Weagamow Lake in the Severn hinterland, traded at Gloucester, Albany and probably Severn (Rogers and Black-Rogers 1982: 163, 178, 182; Rogers and Black-Rogers 1983: 94; See also HBCA B.78/a/9: 27). Another Indian, Captain Caupermertissnewinnekee, lived on the Berens River which drained into Lake Winnipeg, and chose to trade with the HBC, first at Albany Fort and later at Gloucester House (Lytwyn 1986: 32; HBCA B.78/a/5-9).

some time, probably since the fall and winter of 1781-82. On that day, post master John Kipling "Took council with Capt[n] Abbitywabnio [*sic*] and son Concerning the Inland Trade" (HBCA B.78/a/7: 21-21d). During this meeting Abbitywabino "made a very long speech the purport of which was that the Indians Inland was very poor this year that a great Mortality had been among them, Numbers of the Principal Indians was Dead..." (*Loc. Cit.*). Abbitywabino, and his sometime companion Macatoppishnew, were from a small lake lying between Lac Seul and Lake Nipigon and southwest of Gloucester House called Sturgeon Lake[41] (Figure 14). These men, too, eventually fell to the disease, as on May 26, 1783, Kipling noted the arrival of:

> Lieut Metewisito and 2 more Indians in a Canoe with about 100 Beaver. He informs me that all the Indians on and near Lake Sturgeon are Dead in that Cruel Disorder the small Pox that out of 2 Tribes of Indians that use to resort to this place not more than 2 or 3 children are alive, and amongst them dead is 2 of the Principal Leaders that belong this (Vizit) Capt.[ns] Abbitywabnio and Macatopishnew. (HBCA B.78/a/8: 24d)

Given that Abbitywabino had been at Gloucester House, apparently healthy, as late as June 24 of 1782 (HBCA B.78/a/8: 24d), it is evident that the sickness at Sturgeon Lake must have continued to rage even as he visited the HBC post and told Kipling of the mortality.

Kipling also noted that most of Metewisito's people had died of the disease, and so the location of his country is also significant. It seems reasonable that he either came

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[41]These two Indians guided an HBC employee named George Sutherland to that lake in 1779 (HBCA B.211/a/1; see Lytwyn 1986: 33-38). According to John Long, the Indians about Sturgeon Lake were Ojibway of the "Hawoyzask or Musquash [muskrat]" nation, who were supposed to be more stationary than most of the other Ojibway (Long 1974: 62). At one time, Long met some 50 of the Nation of the Rat at Sturgeon Lake (*Ibid.*: 99). Although William Warren (1984) did not mention the rat or muskrat as a totem of the Ojibway, during the early nineteenth century Aeneas McDonell identified the muskrat as one of the clans of the Ojibway of the region (NAC MG 19, C4, Volume 53: 6).

from near Sturgeon Lake, or passed the Lake on his way to Gloucester House, since he was well acquainted with events there. Nothing further of his origin can be established with certainty. However, it may be that Metewisito was the same Uplander who threatened Gloucester House in 1778, a man named Me,ta,wiss[42]. In October of 1778, Kipling was told that Me,ta,wiss and his gang intended to kill the English in the winter of 1778-79 and had "been at Wars with the Natives on Lake St Anns [Lake Nipigon], Called by the Indians Anim me Peck, and have killed a great Number of that Indian Men, Women and Children..." (HBCA B.78/a/4: 5d). Me,ta,wiss probably belonged to the Wasses, or Ouasé (Catfish), a group of Ojibway who were consistently found to the north of Lake Superior throughout the eighteenth century, and about this time in the Pays Plat region at the mouth of the Nipigon River (Figure 14; Long 1974: 45, 97-98; *Handbook of Indians of Canada*: 379), and at Kaministiquia[43]. It was they who were engaged in conflict with the Indians of Lake Nipigon at that time, according to both John Long and the HBC men (Lytwyn 1986: 20-21, fn. 15). If Metewisito was Me,ta,wiss, this would suggest that smallpox had also struck the people living east of Grand Portage along the

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[42]Charles Bishop (1974: 277) believed that Me,ta,wiss was Captain Metweash, a noted Ojibway leader from Lac Seul. According to Robert Goodwin at Osnaburgh House in 1791, Metweash was the son of A,ba,ti,wa,bino of Sturgeon Lake (HBCA B.155/a/5: 20), which was inhabited by the muskrat people. John Long specified the "Wasses", or catfish people, of the Pays Plat region as the tribe at war with the Nipigon Indians, and that is verified in the HBC records (Lytwyn 1986: 21). Also, in 1777 Kipling used the name Metwaash when he met Metweash, and not Me,ta,wiss or any variant (HBCA B.78/a/1: 2).

[43]Chauvignerie in 1736 (O'Callaghan 1853-61: IX: 1054), La Verendrye in ca. 1742 (Greenberg and Morrison 1982), and unnamed Montreal fur traders in 1766-67 (PAM MG 10 F4, Microfilm reel M131 "A list of Indians living in the inhabited part of the Province of Quebec, as well as those residing in the South and Northwestern part of Canada as they stood in the years 1766 and 1767.") all located the Ouasés at Kaministiquia.

north shore of Lake Superior, perhaps from Grand Portage[44].

Following the council with Abbitywabino, the next news of the epidemic reached Gloucester on June 22, 1782, with the arrival of Indians from Rainy Lake, who told of the mortality at Rainy Lake and on the plains (HBCA B.78/a/7: 24). From then on, until May 24, 1783, there is no other mention of the disease in the Gloucester House records, although it continued to rage west of Gloucester House. On that date arrived "Lieut.[s] Netauahe and Sheawappennesscome and Gangs 7 canoes but brought Little or no Trade having lost some of Their Friends in Winter and they themselves have been sick most of the year so that they are unable to pay their Debts they took up last fall" (HBCA B.78/a/8: 24d). While smallpox is not definitely indicated, the combination of epidemic sickness and mortality was one which was repeated innumerable times throughout this and many other severe epidemics. Unfortunately, it is not known where either man came from, but the pattern of arrivals of the latter man indicates that he was not a local Indian. It would later be discovered that he died on his way home from Gloucester House[45].

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[44]If Metewisito came from Pays Plat and was trying to avoid travelling through the lands of his Nipigon enemies, there are back roads from the north of Lake Superior which would have taken him through Sturgeon Lake (Lytwyn 1987). In July of 1784, James Sutherland's guide from Gloucester House to Lake Nipigon refused to continue once they reached the lake since he was afraid of the Nipigon Indians, "who he says will kill him as he and his tribe kill'd some of these Indians about 7 or 8 years ago" (HBCA B.78/a/11: 12d), or roughly in 1777.

[45]Lieutenant Sheawappennesscome arrived at Gloucester House several times between 1780 and 1783, always in the summer, early fall or late spring, the periods when distant Indians would be coming to obtain debt or bring in their winter furs. He was never there during the winter unlike those who lived closer to the post. Dates of arrival included July 12, 1780; October 8, 1781; May 23, 1782; August 17, 1782; and the above date, May 24, 1783. He was of the Eagle clan of the Ojibway, and was the brother of Lieutenant Abittywescome who guided James Sutherland from Gloucester House to Pashkokogan Lake in May of 1784 (HBCA B.78/a/11: 3, 4d). On the way to the lake Sutherland observed Sheawappennesscome's grave, "who died here on his return from Glouc[r] last summer" (*Ibid.*: 4d, 8d). Evidently, he had not recovered from his sickness when he visited Gloucester House on May 24, 1783. There is no indication that he was infective with the disease when he arrived at the post, if indeed he then had smallpox.

On June 3, two other leading Uplanders arrived with similarly tragic news. Kipling learned from Captains Ca,ca,kes (Caucaukes) and Sheawaquannep (Shewequenap)[46] "that most of their young men is Dead in the Small Pox" (HBCA B.78/a/8: 25d). These men were very influential traders, and their places of origin can be deduced from the Gloucester House and later Osnaburgh House records. Shortly after the epidemic Sheawaquannep was described as one of two leaders of Pashkokogan Lake[47], located up the Albany River and near Lake St. Joseph, and having a large gang (HBCA B.78/a/10: 5; Figure 14). This lake lay along the route of all the Upland Indians to Gloucester House (HBCA B.78/a/9: 13), and so it is little wonder that the disease spread widely among the Indians living along its shores. Ca,ca,kes was also extremely influential. According to Germaine Maugenest[48], a former Solomon trader who had defected to the HBC, "he (the Capt^{n}) has great affluence [*sic*] with the Indians Inland and his gang when collect'd Carrys 900 or 1000 Made Beaver yearly to the trader and that he had his clothing sent yearly from the Commander at Machillimackinac" (HBCA B.78/a/7: 20d). Bishop (1974: 268) concluded that Ca,ca,kes's territory was Sturgeon Lake, based on later references in the HBC journals.

That the mortality among Ca,ca,kes's and Sheawaquannep's men was confined to

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[46]Sheawaquannep began trading at Osnaburgh House, located near his home territory, after the HBC established that post in 1786 (Bishop 1974: 239).

[47]When James Sutherland and a party of HBC men journeyed to this lake in May of 1784, they met several Indians, one of whom was Sheawaquannep (HBCA B.78/a/9: 21; Bishop 1974: 300, fn. 25).

[48]On Germain Maugenest (Jacques Santeron) see Lytwyn (1986: 17, ff.) and Rich and Johnson (1954: 354-365).

the young is perhaps a significant observation. It suggests that the older people of these two bands had been exposed to smallpox during a previous epidemic, and were therefore immune in 1782-83. Given virgin soil conditions and variola major, one would not expect age selective mortality, especially in such a way that the young people would be singled out for death, and there was no other recorded instance of such age selectivity in the Petit Nord. Most likely, they, along with the Pillagers of Leech Lake, had been infected with smallpox at an earlier date, once again perhaps during the westward travels of the 1750s. A similar range in immune status was probably to be found among the people of Ca,ca,kes's and Sheawaquannep's bands during this epidemic[49].

Gloucester House was unoccupied over the summer and early fall of 1783, and the next notice of smallpox received there was not until June of 1784, when an unidentified individual came to the house, being the first Uplander of the season. He told Kipling "of the Death of a great many of the Gloster Indians" (HBCA B.78/a/9: 21d). Two weeks later, two "poorly Gooded" Upland men, Captain Caupermertissnewinnekee (also called

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[49]Overall, the impact of the epidemic on Sheawaquannep's people was probably less than on other nearby bands that were universally susceptible. In 1784, James Sutherland of the HBC made a journey to Pashkokogan Lake from Gloucester House and met the two main trading captains from that lake, Sheawaquannep and Connamatchie, and their adherents, which included 15 men besides women and children (HBCA B.78/a/11: 5d). Moreover, the Canadian returns at that lake in 1783-84 amounted to 1,200 MB, not an inconsiderable amount (HBCA B.78/a/11: 6), and Pashkokogan Lake was one of only three Canadian houses in the country about Gloucester House not abandoned in 1784 (HBCA B.78/a/10: 3d). Sheawaquannep's gang has been identified as being one of the largest trading at Gloucester House and later Osnaburgh House, consistently bringing twelve to fourteen canoes to trade prior to and long after the epidemic (Bishop 1974: 268). Compare this to, for instance, the Ojibway of Sturgeon Lake where Indian report, exaggerated though it may have been, stated that all but two or three children were dead of smallpox (HBCA B.78/a/8: 24d). An excellent example of selective mortality due to prior exposure may be found among the Fall, or Atsina, Indians who, having been struck severely during the 1779-83 smallpox epidemic, suffered substantial losses among their youngest people in 1801. Thus, Peter Fidler wrote: "The smallpox the same spring [1801] also came amongst them from the Southwards towards the Mississoury river + cut off 100 principally of Children" (HBCA E.3/2: 71).

Caupmontisue Winnekee) and Lieutenant Inch, arrived at the post, informing the trader that "a great many of their Country men Died of the small pox the last summer" (HBCA B.78/a/9: 23). Unfortunately, only the first man's territory can be identified. Captain Caupermertissnewinnekee came annually to Gloucester House from a great distance inland, and his territory lay towards Lake Winnipeg, probably near the Berens River (Lytwyn 1986: 30). The Captain also lost his wife and children to the sickness which, according to Kipling, left him destitute with no one to care for him (HBCA B.78/a/12: 19d). Caupermertissnewinnekee's "country men" may have caught smallpox from a number of sources, as the Indians of the East Winnipeg Country travelled widely, and were in contact with Indians of areas where the disease had already appeared. For instance, the Indians living east of Lake Winnipeg could go to the lake to await the Canadian traders "and trade with them in there way to Canada," a practice noted by George Sutherland in June of 1778 (HBCA B.3/a/73: 40). Moreover, while at Caupermertissnewinnekee's winter camp along the Berens River in 1777-78, Sutherland was visited by some York Indians, others who traded with the HBC at Severn and still others who went to the Pedlars along the Winnipeg River[50] (HBCA B.3/a/73: 15d, 29d, 39d). The disease therefore could have spread to them through travel to any of these places, or through contact with other Indians who were infected.

This was the final reference to the 1779-83 smallpox epidemic in the Gloucester

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[50]Moreover, the Berens River Ojibway may also have been trading at the mouth of the Red River as early as the 1770s (Peers 1994: 30). The late date of their sickness suggests that infection from the Severn region was more likely, since the disease had passed through the other areas long before the summer of 1783.

House records. The full extent of its victims living upriver from Gloucester House, and its range in that direction, will probably never be known, as there are no records there from that period. The dearth of HBC records at this time was owing to the temporary closure of the inland posts. Following the destruction of York and Fort Prince of Wales by the French in the summer of 1782, and fearing further attacks, the HBC pulled their men from the inland posts, including Gloucester House, Henley House and Brunswick House. Thus, Gloucester House was empty between June 18 and October 24, 1783, a period when further news of the epidemic was filtering down the Albany River. As such, and because the Canadian men left no record of these epidemic years, the true extent of the 1779-83 smallpox epidemic's ravages upland from Gloucester House will likely never be known. It must have been considerable, however, as immediately prior to abandoning Gloucester House, Kipling wrote that "there has hardly been an Indian in but what has lost some part of their family in that Cruel Disorder which I fear will greatly hurt the trade here" (HBCA B.78/a/8: 25d).

Despite frequent references in the Gloucester journals, the epidemic did not spread throughout the interior of the Petit Nord. Although evidence is limited and inferential, there is reason to believe that the disease did not reach Lake Nipigon[51]. On at least two occasions during the epidemic, Gloucester House was visited by Indians from Lake Nipigon (or Lake St. Anns), both times without mention of the sickness. The first occurred early on, but after the arrival of the news of the epidemic from Lac la Pluie. On

[51]Lytwyn's map (1993: 372), however, suggested that the disease had passed from Lac Seul to Lake Nipigon.

July 12, 1782, John Kipling wrote that:

> Came here a Capt[n] Antikiskickwescam and Brothers in 2 Canoes, Sons to the Late Capt[n] Meetwass (formerly an Albany Leader) from Lake st Anns those is the first Indians that has been here from that Lake, the Distance is not more then [*sic*] 150 Miles from this place.... (HBCA B.78/a/7: 25d-26)

The second visit took place long after the epidemic had begun to rage throughout the Boundary Waters and in the upper Albany River area, again with no mention of disease among the Nipigon Indians. On April 5, 1783, Lieutenant Quesip and another man arrived from that lake, bringing only 70 MB[52] in furs due to the fact that their tent had burned down with all their furs (HBCA B.78/a/8: 25d-26). Significantly, of the seven Canadian posts in the Gloucester House area in 1783, Lake Nipigon was one of three that were not abandoned following the epidemic[53] (HBCA B.78/a/10: 3d). Perhaps the constant animosity between the Nipigon Indians and many of their neighbours prevented the disease from spreading to Lake Nipigon[54].

A statement made to Kipling by an Indian named Newitchicanescume in June of

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[52]Made Beavers, a trading standard employed by the HBC to convert the value of furs into trading goods.

[53]It is not known how many Indians lived on Lake Nipigon. James Sutherland of the HBC was told in 1784 that there were about 30 hunting Indians belonging to this lake, who were serviced by this one post (HBCA B.78/a/11: 6d), however John Long, who travelled through the area several times and whose company had a post there, believed that the Nipigon Indians totalled ten times that number (Long 1974: 50). Sutherland's guide was not on good terms with the Nipigon Indians, and he may have been trying to dissuade the HBC man from recommending an HBC post for that Lake by deliberately underestimating the number of hunters.

[54]In fact, there was a buffer area that separated the Lands of the Nipigon Indians from those of the Albany River Indians. In 1784, while on a trip from Gloucester House to Lake Nipigon, James Sutherland found the lands about one of the carrying places on the route to be teeming with beaver, "the Gloc[r] Indians and the St Ann Indians being at variance is afraid to approach so near each others teretorys" (HBCA B.78/a/11: 13).

1783 raises other doubts about complete devastation inland. In a conference at Gloucester House he was told of the Manatie Country inhabited by a great many "Jeppoys," and lying southwest of Pashkokogan Lake (HBCA B.78/a/10: 3). There, resources were said to be plentiful and there were no Canadian traders. Lytwyn determined that this area was the Monontague country lying about Lake Minnitaki (Figure 14), to the southeast of Lac Seul, and that the "Jeppoys" were in fact Ojibway Indians[55] (Lytwyn 1986: 48). This description of numerous Indians hardly seems to fit a post-epidemic population, or one in the midst of devastating smallpox. This suggests that, at least as late as June of 1783, the epidemic had yet to reach Lake Minnitaki, although the Indians living nearby at Sturgeon Lake and Lac Seul were both hit hard by the disease by that time.

Lytwyn (1993: 364, 372) concluded that the epidemic spread from Lake of the Woods to the Albany River, and then downstream to Albany Fort. Further examination of the evidence suggests that this was not the case. Although it is clear that smallpox did indeed reach the upper waters of the Albany, at least as far as Pashkokogan Lake, there is no evidence that the disease ever spread to those who lived in the vicinity of Gloucester House. While Uplanders arrived at the post with news of the epidemic on several occasions between 1781 and 1784, no individual was ever identified as having smallpox when they arrived at the post, nor were the Indians living in the vicinity of the post ever

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[55]The fur trader John Long, who was there a few years prior to the epidemic, stated that there were about "three hundred of the Chippeway nation who resort to it" (Long 1974: 76). Long also commended the resources of "Lake Manontoye", which abounded "with excellent fish and wild fowl; and oats, rice, and cranberries... (*Loc. Cit.*)." A few years later, in 1786-87, a "great sickness" struck the Indians of the Monontague, causing an unknown number of deaths (HBCA B.78/a/16: 31d).

mentioned as suffering from the disease[56]. In fact, a letter written by Edward Jarvis at Albany Fort dated July 5, 1783, stated specifically that "... small Pox had made its way **above** Gloucester" (HBCA B.198/a/28: 24) [emphasis mine], without suggesting that the disease had progressed to the post. As well, while the Upland Indians were suffering from smallpox during the summer and fall of 1783, and unable to proceed to Gloucester House, Kipling gained his trade chiefly from the home Indians and the "Northward Indians" (HBCA B.78/a/9: 13d). The latter people were reported to be starving but were never noted as suffering from sickness (*Ibid.*: 7, 12d), and both groups seem to have escaped infection even after the post's temporary closure. It would therefore appear that the 1779-83 smallpox epidemic did not spread down the Albany River as far as Gloucester House[57].

Neither did it reach Henley House, either from up the Albany or from elsewhere. Between 1779 and 1784 the post was continuously occupied, except for the summer of 1783 when the men were withdrawn to Albany Fort for fear of French attack. During this period there was no reference either to the epidemic or to sick Indians, beyond a very few individuals with unidentified ailments. Neither was there any indication by the local Indians that anything untoward had happened during the HBC men's absence, unlike the

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[56]One Uplander who arrived at the post on May 24, 1783, Lieutenant Sheawappennesscome, died on his return up river (HBCA B.78/a/8: 24d). However, there is no indication that he succumbed to smallpox, and not some other affliction or even an accident.

[57]Curiously, rumours of smallpox inland arrived at Gloucester House for several years after. In 1785, an inland Captain, Notonwescome, informed John Kipling that "that Cruel Disorder the small pox is again raging inland but had not reached his country when he left it" (HBCA B.78/a/12: 21d). In July of 1786 two other Captains brought news from the Canadians that the disease continued to rage among the Indians on the Churchill River (HBCA B.78/a/14: 8).

case at some of the Bayside posts that were abandoned. If the post records are indicative of the situation, the men at Henley House remained unaware of the epidemic even as it devastated the Indians living farther up the Albany River and to the northwest, in the Severn hinterland.

Albany Fort

Although Lytwyn (1993: 364) believed that this smallpox epidemic spread down the Albany River to Albany Fort at least by May of 1782, this was not the case. To be sure, diverse sicknesses were present among the Indians visiting Albany during the period 1782 to 1783, and also among the HBC men, but smallpox did not reach this post. However, a severe sickness appeared among the Albany Indians in May of 1782, which Lytwyn interpreted as smallpox. During that month, Thomas Hutchins, Chief of Albany Fort, wrote to Edward Jarvis telling him that "there is a great Sickness and Mortality amongst [the Lowland Cree] particularly the Children which quite disheartens the whole" (HBCA B.135/b/12: 26d-27). On the 24th, Hutchins wrote in his journal that "a poor child died on the plantation, this is the 7th of the Natives who have died this spring and several more are still very ill" (HBCA B.3/a/80: 20d). By June 5, five more Indians had died at Albany (*Ibid.*: 21). At one time, twenty-five Indians, mainly children, were ill on the plantation (HBCA B.86/a/36: 52d).

Despite the widespread sickness and a higher than normal death rate among the Lowland Cree at this time, this probably was not smallpox. Significantly, this disease was never identified. It is not likely that Hutchins, who was trained as a surgeon and had

prior experience in treating men under similar conditions at York Factory (Williams 1979), failed to identify an epidemic of smallpox, or misdiagnosed it, while distinguishing lesser complaints and sicknesses such as rheumatism, quinsy, fever and "intermitting fever" (HBCA B.3/a/80: 3d, 29d). Moreover, in contrast to the situation at York, Severn and Fort Prince of Wales, where smallpox killed many, and probably most, of the natives who contracted it, the case fatality rate among the Albany Lowland Cree was not of a similar magnitude, and was concentrated among the children[58]. If a highly virulent strain of smallpox was raging amongst them one might expect to see many more deaths than were noted by Hutchins.

Another mortal sickness appeared among the Albany Indians and the HBC men during September of 1782, after Edward Jarvis took over from Hutchins. On September 12, one day into his tenure, Jarvis noted that "two of the home Indians Died this Day, several others much afflicted with a Consumption, many of the Englishmen are also ailing" (HBCA B.3/a/81: 1-1d). Three days later he noted that, since his arrival at the post, three Indians had died, "and several more had taken sick" (HBCA B.3/a/81: 2d). Although not identified, there is reason to believe that these sicknesses were of the same disease. Sick Indians continued to arrive at the fort seeking medicines throughout 1782-83, and in November of 1783 there was an outbreak among the HBC men of an unspecified disease that caused sore throats and swelled necks[59] (HBCA B.3/a/82: 9d-

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[58]Unlike the case at Leech Lake, there was no recent smallpox epidemic here that would have influenced the age of its victims.

[59]On November 29, 1782, Jarvis wrote, "such a universal sickness is uncommon in the Country" (HBCA B.3/a/82: 9d).

10). Other named illnesses included "Phthisis Pulmonalis", consumption, "sues venerea", and "the gripes".

There was a pandemic of influenza in Europe in 1781-82 (Crosby 1993: 809), and it is probable that this was one of the sicknesses that so affected the people of the Albany area at this time. This influenza epidemic was considered by Crosby to have been "one of the greatest manifestations of disease of all history," both in its virulence and its geographic spread[60] (*Loc. Cit.*). Once again, the timing of the outbreak, in early September of 1782, suggests introduction from Europe, a pattern of diffusion that was well established during the preceding half century. The disease was rampant in Scotland, England and Ireland late in the spring of 1782, and appeared in London in mid-May, striking adults in very large numbers (Creighton 1965: II: 363, 364). At about this time the *Seahorse* (II), under Captain Joseph Richards, was preparing to sail for Hudson Bay. The ship set sail for the Orkneys with 59 men aboard, including the Captain (HBCA C.1/1047; C.4/1). On June 19 Joseph Richards died of "an inflammatory sore throat and fever", almost certainly influenza, while docked at Stromness in the Orkneys (HBCA C.1/1047: 7). Thereafter, the *Seahorse* took nine passengers on board, and continued on to James Bay, arriving at Moose on August 24 (*Loc. Cit.*; C.4/1). On the 30th, seven of the passengers from the Orkneys were dispatched by sloop for Albany Fort (HBCA C.1/1047: 33d). Within two weeks, the disease had assumed epidemic proportions

[60]Crosby may have overstated the case for its virulence but this epidemic was widespread. It appeared in Asia in the autumn of 1781 and spread from Siberia to Russia in December. It was in Finland and Germany by February, and reached Denmark, Sweden and England in April. By early summer of 1782 it had broken out in France, Italy, and most of the rest of Europe (Burnet and White 1972: 203).

among the HBC men and Indians at Albany. Most likely, then, the outbreak at Albany Fort was part of a more widespread influenza pandemic from Europe.

Although it is tempting to conclude that smallpox had arrived at Albany Fort in the spring of 1782, based on the mortality there and the presence of the disease elsewhere, this was not the case. As demonstrated in the previous chapter, disease outbreaks were not unusual at Albany during the period leading up to the 1779-83 smallpox epidemic, and on occasion they proved to be mortal. Moreover, there is nothing in the records to suggest that the sicknesses noted by Hutchins and Jarvis were the same. Indeed, there were long gaps in time between outbreaks, and the traders named several distinct afflictions. Finally, when news of smallpox among the Indians living in the Hudson Bay Lowlands near Albany Fort reached the Fort in August of 1784, Jarvis's comments regarding the outbreak of the disease reflected an entirely new disease to be dealt with, rather than a continuation of an existing one (HBCA B.3/a/82: 46; See below). In short, 1782-83 was a period of diverse sickness at Albany, but one that was not entirely dissimilar to the years preceding it.

When the smallpox epidemic finally reached the Lowland Cree of the Albany region, Jarvis learned of it only after the fact. Thus, on August 9, 1784, he wrote that:

> In the afternoon Capt[n] Assup came in so poor that many of his young fellows could not even pay their debts, they tell of numerous deaths among the Indians around them by an epidemical disorder which from their description should seem to be the small pox, which I fear has made its way from the northward. (HBCA B.3/a/82: 46)

This is the only direct reference to smallpox among the Indians who traded at Albany Fort, and there is no evidence that it spread any farther than these people. Captain Assup

was an influential leader who, along with four other leaders of the Albany Fort Lowland Cree, Questach, Pusquothecot, Saquot, and Archekishick, claimed specific hunting grounds to the north of Albany Fort along the Ekwan and Attawapiskat Rivers (Lytwyn 1993: 47-48, Figure 14). Presumably, Assup was referring to the bands led by these men when he noted that the disease was killing the Indians living around his people. It would seem that Assup's gang also suffered, however, as in 1786 Jarvis recalled that "My Great Northern Capt[n] Assup has lost so many of his Gang..." (HBCA B.198/a/33: 39).

Given that the disease had failed to spread down the Albany River to the Lowlands, Jarvis's statement that it had come from the northward is reasonable. It will be recalled that the epidemic was late in arriving in the Severn River area, afflicting the Lowland Cree perhaps during the late summer or early fall of 1783, and this timing is roughly consistent with what little is known of the outbreak of the disease to the north of Albany Fort. As well, there was contact between the Severn and Albany Cree at this time. There were Severn River Indians at Albany Fort in June of 1783 (HBCA B.3/a/81: 29), and there is evidence that some of the Albany Cree were travelling in the opposite direction. Assup and his gang were known to trade at Severn Fort (HBCA B.198/a/33: 34, 34d), and perhaps they or some of the others had done so during the epidemic. As well, 1783 was one of a string of years during which the goose hunts failed and hunger was common in the Lowlands around Albany Fort. In response, the Cree turned to the caribou in order to survive, and in the fall of 1783 Jarvis was told by the goose hunters:

> Do not urge us to hunt geese when there are really none to kill, but let us have our debt and get as far as we can from the Factory to the northward where deer are plenty and we can live as we too plainly see should we be distressed you cannot assist us. (HBCA B.3/a/83: 2)

Two prime summer hunting grounds for caribou were at Akimiski Island and near Cape Henrietta Maria, to the east of the Severn River (Lytwyn 1993: 201, 206-207, 232). Both destinations were not far from the territory of the "Eastward Home guards" of Severn Fort[61]. Regardless of the means of diffusion, however, this extension to the people of the Attawapiskat and Ekwan Rivers was to be the northeastern limit of the 1779-83 smallpox epidemic.

Further Diffusion

The 1779-83 smallpox epidemic did not spread in an eastward direction beyond the areas noted above. In the Lowlands, the disease never reached Moose Fort, nor beyond it to the east coast of James Bay. The Moose Fort journals of the period make no mention of smallpox, although other sickness occasionally appeared among the men and Indians (HBCA B.135/a/65-69). At Eastmain and Rupert River (Figure 14), both the HBC men and the Indians remained relatively free of sickness, compared to the more western HBC posts (HBCA B.59/a/58-59; B.186/a/4). Although the records for these posts contain some gaps[62], there is no reason to believe that the epidemic spread eastward

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[61]The Lowland Cree who hunted caribou to the east of the Severn River (Lytwyn 1993: 232).

[62]Eastmain journals cover the periods September 28, 1782, to July 19, 1783, and October 6, 1783, to August 7, 1784, (HBCA B.59/a/58, 59). The Rupert House journal covers only October 15, 1783, to June 11, 1784, (HBCA B.186/a/4).

of the Attawapiskat and Ekwan Rivers in the Hudson Bay Lowlands.

Similarly, none of the Upland Indians arriving at Moose during the period of the epidemic complained of sickness, suggesting that smallpox did not penetrate east of the Albany River watershed. The Abitibi Indians, the "Uplanders" and the "Eastern Indians[63]," continued to bring their furs to Moose Fort without mentioning the disease. This is significant in that, during this period, Moose Fort was visited by Indians from great distances, particularly from along the rivers and lakes draining into the Moose River (Figure 14). In fact, this period was a banner era of trade at Moose and its dependencies, with returns rising from just over 9,000 MB in 1780 and 1781, to greater than 10,000 MB in 1783 and 1784, and then to over 12,000 MB in 1785 and 1786 (Morton 1939: 424).

Inferences regarding the heavily travelled Moose-Michipicoten corridor and the eastern part of Lake Superior may be drawn from the records of the only inland dependency from Moose Fort in operation at the time of the epidemic, Brunswick House (or Wapiscogamy), which occupied a strategic position in this corridor. The journals of the years 1781-1784 (HBCA B.23/a/5-7) record the arrival of many Indians from throughout the drainage basin, several from as far south as Michipicoten on Lake Superior, but are silent on the subject of smallpox (For example, HBCA B.23/a/5: 16d, 20d, B.23/a/6: 23). Far from the heavy losses in trade experienced farther west due to the smallpox epidemic, Philip Turnor reported an increase in trade for Brunswick House for

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[63]Between the spring of 1784 and the fall of 1785 Indians identified using only the generic term "Uplanders" arrived on May 13, 18 and 29 of 1784, and May 11, June 22 and September 6 of 1785 (Rich and Johnson 1954: 46, 47, 49, 114, 121, 140). "Eastern Indians" arrived at Moose Fort on April 26, June 3 and 11, September 17 and October 5 of 1784, and June 7 of 1785 (*Ibid.*: 42, 50, 51, 77, 80, 118).

1782-83 over the previous outfit year, suggesting that there was nothing disrupting the Indians' hunts for that year at least (HBCA A.11/44: 162d). Although Brunswick House was temporarily abandoned during the summer of 1783, only to be reopened towards the end of October[64] (Rich and Johnson 1954: 155), the Indians traded at Michipicoten and returned to Brunswick the following spring (HBCA B.23/a/7: 11, 13, 13d, 14d; Rich and Johnson 1954: 194-195). In no case did the Indians ever mention smallpox. Thus, as late as the spring of 1784 smallpox had not yet penetrated into the Moose- Michipicoten corridor (HBCA B.23/a/7: 14d), and by that time the epidemic had extinguished itself elsewhere in the Petit Nord.

SUMMARY

Between 1781 and 1783 smallpox once again penetrated into the Petit Nord, this time as part of the northern extension of a devastating pandemic that began in Mexico City in 1779. This was the first documented epidemic to strike the region from the southern part of the continent, and it appears to have been transported along the continental horse trading network, which only a few decades before had expanded to include the village tribes of the Upper Missouri and some of the people of the Northwest. The arrival of the horse on the plains marked a significant improvement over other

[64]It was left unoccupied between June 29 and October 20 of 1783. During the summer of 1783, George Donald was ordered to proceed to Michipicoten from Brunswick House (HBCA B.23/a/6: 25). While there, he met Indians from Wapiscogamy and Lake Superior, all of whom promised to trade with the HBC in the spring (HBCA A.11/44: 162d).

aboriginal means of transportation, in that mounted travel greatly increased the rate and range of movement of both groups and individuals. It is quite likely that this innovation in transport was crucial to overcoming substantial barriers that had previously prevented the spread of smallpox from Mexico to the Northwest. Nevertheless, there is no evidence that the 1779-83 smallpox epidemic was followed by other epidemics from Meso-America to the northern part of the continent, except, possibly, the smallpox epidemic of 1801 that barely penetrated into Canadian territory[65].

Once on the Northern Plains, the disease spread to the Northwest in the summer and fall of 1781 by the return of two different war parties, which had gone to war against the Hidatsa and the Shoshone. As a result, the epidemic entered the Petit Nord by two different routes: from the Saskatchewan River into the northwestern part of the region, and by the Red River to the Boundary Waters. In both cases, diffusion along the main routes of travel occurred first, followed by much slower spread along the less travelled rivers.

In the north, the epidemic spread quickly from the Saskatchewan River to York Factory, killing many of the native people along the Nelson and Hayes River corridors, and also many of the Lowland Cree in the vicinity of the post. The Homeguard Cree of York Factory were kept isolated from the disease for several months, due to the efforts of the HBC men, but eventually they too were infected. At about the same time as smallpox

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[65]Smallpox was epidemic in Mexico City in 1797-98 (Cooper 1965). In 1798, the disease was present at St. Louis, thereafter spreading north and westward as it diffused through several mounted Indian tribes, and from the Arapaho to the Atsina living near the South Saskatchewan River (Taylor 1982: 45; Decker 1989: 86-89).

first reached York Factory, it was brought to the Severn River area by Ojibway hunters who had been infected at York. Sometime thereafter, it spread down river to the Lowland Cree about Severn. Although the people of the Severn River contracted the disease early, smallpox was very slow to reach the coast, arriving many months later. Finally, in 1783 the disease reached some of the Cree living along the Attawapiskat and Ekwan Rivers, to the north of the Albany River. They contracted it from the Lowland Cree living towards Severn Fort. This marked the northeastern limits of the epidemic's diffusion within the Petit Nord.

In the south, smallpox spread rapidly throughout the Boundary Waters. This began during the fall and winter of 1781. Subsequently, the disease expanded into the Upper Mississippi, as the Ojibway fled the mortality at Netley Creek. It also penetrated into the interior of the Petit Nord from the Boundary Waters, as word of the epidemic's impact at Sturgeon Lake arrived at Gloucester House in May of 1782. Once in the interior its progress slowed dramatically, and the disease persisted in a relatively small area for a considerable period of time[66]. The epidemic did not diffuse throughout the region, however. Reports from the Indians arriving at Gloucester House suggest that the disease was widespread and highly virulent towards the headwaters of the Albany River, particularly at Lac Seul, Pashkokogan Lake and Sturgeon Lake. It is also likely that the sickness appeared in the East Winnipeg country, around Berens River. However, the source of infection there is not known. It may also have reached the north shore of Lake

[66]This is not unexpected, as Lovell (1985: 154-155) found that this epidemic persisted in some Guatemalan communities for up to eight months.

Superior, towards Pays Plat. Conversely, there is no evidence that it spread among the Indians living farther down the Albany than Pashkokogan Lake. Negative evidence suggests that the epidemic did not reach Lake Nipigon, nor the eastern part of the Petit Nord that lay within the Moose hinterland. Due to the scarcity of HBC posts in the interior, it is unknown if the epidemic spread elsewhere, particularly to the north of the Albany River in the heart of the Petit Nord.

Although the precise extent of this epidemic is unknown, it is important to note that the 1779-83 smallpox epidemic had varying effects among the aboriginal people of the Petit Nord, and that there were limits to its diffusion. In many cases, well documented elsewhere in the Northwest but undoubtedly similar in parts of the Petit Nord, entire bands were devastated, losing a significant portion of their population to the disease. Other bands were untouched by this epidemic. In fact, it is likely that many of the people living in the eastern half of the region were spared the disease at this time. Between these extremes were perhaps a few bands whose older members were immune due to prior exposure, but whose young people caught the disease and died.

Similar varied experiences may have been the case among the Athapaskan tribes of the Arctic Drainage Lowlands, but a lack of evidence renders it impossible to prove. It is known that the epidemic ravaged the Chipewyan who traded directly with the English and Canadian fur traders. However, there is some question as to whether it struck the more northerly tribes through these middlemen (Krech 1978: 712). Alexander Mackenzie's observations of some of those northern people, dating to 1789, do not suggest that they were coping with the after-effects of a disastrous smallpox epidemic

(*Loc. Cit.*). If so, it is to be expected. Krech's (1983: 130) study of these people demonstrated that discontinuities in diffusion were common when later epidemics struck this region, and frequently only a few of the several different tribes of the area were affected by a particular sickness.

Such discontinuities may be typical of the smaller, more isolated aboriginal populations found in the northern forests, rather than the people living in other regions where favourable resources and transport technology supported larger populations that interacted more frequently, and in larger groups[67]. In contrast to the Petit Nord, the 1779-83 epidemic had a more uniform effect on the plains, where the disease seems to have afflicted most, if not all, of the native people, and to a similar degree. Taylor (1977: 78) concluded that all of the Northern Plains people he studied suffered a uniformly high rate of mortality, exceeding twenty-five percent of the total population in each case[68]. He determined that "The pattern of distribution of the 1780-1782 smallpox epidemic... indicates a very high degree of interaction among the ethnic groups on the Northern Plains..." (Taylor 1977: 59). Trimble (1988: 8) noted that the epidemic spread to all of the tribes of the Great Plains. Much of this is attributable to the influence of the horse,

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[67]There is some debate between Boyd and Harris over the extent of the epidemic in the coastal region of the Pacific Northwest, where densely populated, sedentary societies subsisted primarily on the rich marine resources. Boyd (1994: 17-18) concluded that the epidemic "must have spread to the limits of settlement" given the extensive social and economic networks then operating among these people. Conversely, Harris (1994: 605, 606, 615) emphasised their "geographically circumscribed lives" that led to significant levels of isolation between groups, concluding that there were definite limits to the epidemic's diffusion, and that several peoples were not affected at this time. Lovell (1985: 156-159), a geographer, found a wide variation in mortality rates experienced by the Indians of the Cuchumatán Highlands of Guatemala who became infected during this epidemic.

[68]The exception may have been a large group of Crow who fortunately escaped infection (Taylor 1982: 38-39).

which enabled diseases to spread quickly over long distances[69] (Harris 1994: 615), something that was not available to the people of the forests.

The variations in the epidemic experiences both between the people living within the Petit Nord, and between them and the people of other regions, are significant in themselves. It is to be expected, for instance, that sudden shifts in relative population levels would have implications for military balances of power between rivals (Taylor 1977: 64-65; Ray 1988: 98). As well, the loss of population might lead to the amalgamation of survivors from different bands, extending even to people of different tribal affiliations (Peers 1994: 20-21; Taylor 1977; Dobyns 1983: 302), or to the relaxation of taboos concerning marriage partners. In the context of the 1779-83 epidemic, however, perhaps the most fundamental change deriving from differential impact was the movement of Ojibway people into the Red River Valley. Evidence suggests that the Ojibway, and to a lesser extent the Ottawa, began to move onto the plains permanently and in larger numbers after the epidemic decimated the Cree and Assiniboine living in the valley[70] (Ray 1988: 104; Peers 1994: 18-21). On a smaller scale, similar movements probably occurred wherever the epidemic provided opportunities for territorial expansion, just as they did at Sandy Lake in the Upper

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[69]As such, depending on the degree to which the horse enhanced the movements of the plains people, and assuming these diseases were introduced at that time, the pattern of epidemic diffusion on the plains during the prehorse period might have resembled that of the Petit Nord, with marked discontinuities in the spread of *ACIs* as local populations escaped infection through isolation and buffer zones dampened the progress of epidemics. This may have served as a barrier to the diffusion of disease from the south until the relatively late date when the people of the Northern Plains acquired horses.

[70]A clue to the origins of these Ojibway is that the epidemic also devastated the people living to the northwest of Lake Superior, suggesting that the Ojibway who moved into the west more likely came from the country to the northeast or south of the lake.

Mississippi. Overall, this reinforces the need to study these epidemics and their spatial variation in detail, for a more thorough understanding of their effects on particular people, and thus their full impact.

CHAPTER 8 — *ACIs* IN THE PETIT NORD: 1784-1818

INTRODUCTION

The late eighteenth and early nineteenth centuries witnessed changes within the Petit Nord and elsewhere that had a significant impact on the presence and spread of *ACIs* in the Petit Nord. Between 1784 and 1818 the fur trade of the Petit Nord underwent fundamental changes that played a role in both the diffusion and reporting of acute infectious diseases in the region. During the initial period of its tenure in the Petit Nord, the HBC remained tied to Hudson and James Bay, content to have the Indians carry their furs to the coastal factories. As its Canadian rivals increasingly intercepted their supply of furs, however, the Company was forced to move inland, beginning in earnest in the 1780s. Thus began a period of unprecedented expansion by the English company within the Petit Nord[1]. At first, this thrust was modest. Between 1786 and 1795 Albany was alone among the Company's Bayside posts to expand into the Petit Nord. By 1795 it controlled a loose network of posts stretching from James Bay westward to Lake Winnipeg, and from the Boundary Waters north to the Bloodvein River, flowing into the

[1]Although expansion into the interior of the region by the HBC had its start in the 1740s with the construction of Henley House, followed three decades later by Gloucester House, the vast majority of new post construction came after the 1779-83 smallpox epidemic (Moodie 1987), and so these first settlements were merely a prelude to what was to come. With the widespread construction of inland posts, there was no doubt a decline in the distances some Indian people travelled each year to trade. While Bishop (1974: 262) believed that the era of intense fur trade competition fostered increased mobility on the part of the Indians as they moved from post to post in order to play one trader off against another, more likely the opposite was true. With a large number of posts located near their home territories, it was no longer necessary for them to make the long, tedious, and sometimes dangerous, trips to the HBC Bayside posts to trade. By 1795, there could be several HBC and Canadian posts with a few days journey of their lands, particularly in the southern part of the Petit Nord.

east side of Lake Winnipeg[2] (Lytwyn 1986; Figure 15). There was no similar inland expansion within the Petit Nord from York or Severn to that point[3], and Albany's hinterland quickly grew to envelope the entire southern half of the region, parts of which it once had shared with York Factory (*Ibid.*: 84; cf. Ray 1987a). It was the people living in what was then Albany's hinterland who were most at risk to being exposed to *ACIs* in the Petit Nord, not only because posts were established among them, but also because they lived close to the trade routes of the plains and to the heavily travelled canoe routes from the east which became corridors for the diffusion of disease. In some cases, the traders contracted these ailments as they journeyed to and from their posts in the interior. Consequently, not only were the Albany men learning first hand of the diseases striking the interior following 1784 but, by travelling far inland, they also began to transport these diseases over long distances.

Following 1795, and until the early nineteenth century, competition between the Canadians and the HBC intensified, spurring greater exploration of the region and additional post construction by both groups of traders (Lytwyn 1986: Chapter VI). During this period, the number of posts in operation within the Petit Nord reached its

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[2]Expansion into the country inland from Gloucester House began in 1786, and included the construction of posts at Lake St. Joseph (Osnaburgh House in 1786), Cat Lake (1788), Red Lake (1790), Escabitchewan (1792), Lake Nipigon (1793), on the English River (Portage de l'Isle 1793), Rainy River (1793), the Bloodvein River (along the eastern shore of Lake Winnipeg 1794) and at the mouth of the Winnipeg River (1795) (Lytwyn 1986: 53-80). In 1793, the Albany men also settled a post along the Assiniboine River, called Brandon House.

[3]Of course, men from York Factory had long since begun to settle posts far up the Saskatchewan River within the Grand Nord. Cumberland House, often considered the first of the HBC's inland posts, was established in 1774, followed by posts located farther and farther up both the north and south branches of the river.

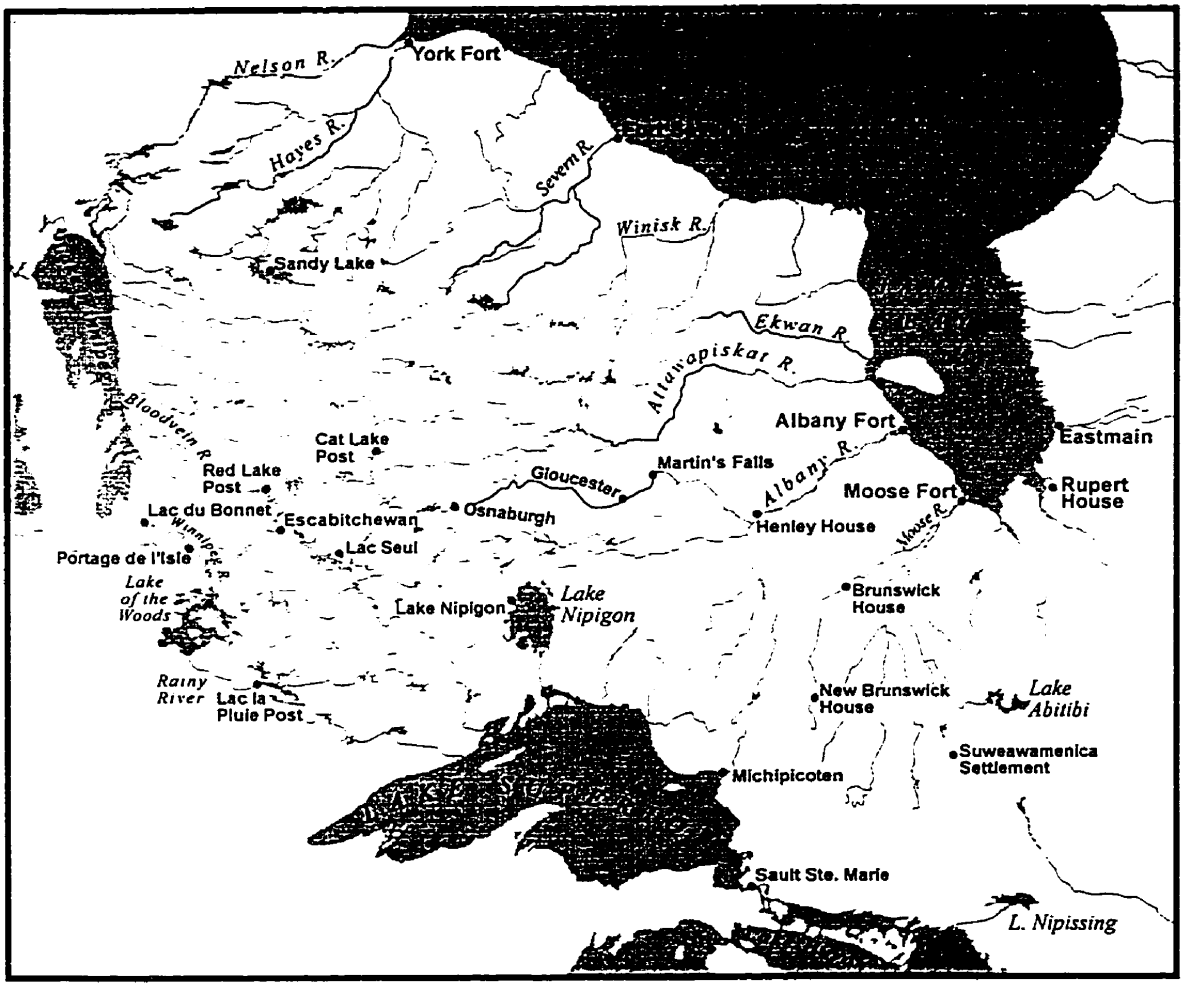

Figure 15: The Petit Nord, 1784-1818

peak, and no longer were the inland posts limited to a few major sites[4]. Instead, many smaller outposts were also settled and, in some cases, men were sent to winter with the

[4]The numbers of HBC posts operating in the Petit Nord peaked by about 1800 (Moodie, Lytwyn and Kaye 1987; Moodie 1987). Whereas there had only been two HBC posts in the interior of the Petit Nord during the 1779-83 epidemic, Henley House and Gloucester House, by the turn of the century there were over twenty-five (Moodie 1987: 364, 368; Lytwyn 1986: 115; Lytwyn 1987). Despite fluctuations in the numbers of HBC posts in the Petit Nord between 1804 and 1821 (Moodie 1987: 370), this relatively dense system of posts continued even after the merger of the HBC and NWC in 1821.

trappers, trading the furs as they were taken (Lytwyn 1986: 104). Consequently, contact between the traders and the natives occurred with unprecedented frequency and, equally importantly, it occurred throughout much of the year, rather than in a tightly constrained trading season[5]. Given this increased contact, the opportunities for transmission of *ACIs* to the Indian people in the Petit Nord also increased, as did the likelihood that such afflictions would be reported in the HBC records. It was also during this period that York Factory began to establish a presence within the Petit Nord. By the turn of the century, York Factory's trading area extended farther south than it had since the beginning of inland expansion, although it was still limited to the northwestern corner of the region (Lytwyn 1986: 98, 101-102). It would be another decade before the York men again traded for the furs of the western part of the Boundary Waters region.

Finally, the fur trade of the Petit Nord changed fundamentally after 1810, particularly that of the HBC. As the NWC scaled back its operations within parts of the region[6], the HBC radically reorganised its fur trade in the entire Northwest in 1810, as part of the "New System" or the "Retrenching System", in order to reduce costs and to more effectively compete with the Canadians[7] (Rich 1959-60: II: 291). Fundamental to

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[5]For a brief period, between 1799 and 1805, a second major Canadian company called the XY or New Northwest Company traded in the Northwest from Montreal (Lytwyn 1986: 82). This heightened the competition for furs, and contributed to the tremendous growth in the number of posts in both the Grand Nord and the Petit Nord at this time (Moodie 1987). Following the merger of the two Canadian companies in 1805, the number of posts located in the Petit Nord declined significantly for several years, until increasing once again about a decade later (*Ibid.*: 366).

[6]For example, the NWC limited its involvement in the East Winnipeg Country after 1813-14, and in its Lac Ouinipique Department as a whole, due to the decline of fur bearing animals in that area (Lytwyn 1986: 137-139, 141, 149).

[7]Another facet of the retrenching system that was to play a major role in the diffusion of *ACIs* into

this system was the creation of two massive fur-trading departments: the Northern, controlled and outfitted from York Factory; and the Southern, controlled and outfitted from Moose Factory. No longer was Albany a key administrative centre. In fact, Albany was stripped of many of its inland posts. A district that had once stretched as far as the Assiniboine River was reduced to only Osnaburgh, Gloucester House, Martin's Falls and Henley House (Rich 1959-60: II: 314, 315). In contrast, the trade at York Factory derived from a huge territory that covered large parts of the Petit Nord, including most of the western part of the region and much of the Boundary Waters. This reversed the situation in place before 1810, as it was York Factory whose hinterland now included the more southern lands, while Albany's was severely cut back.

The period following 1790 saw a significant increase in the presence of non-natives on the Upper Missouri (Trimble 1988: 10; 1985: 36-37). Among these visitors were increasing numbers of fur traders and explorers who visited the village tribes from the south. Such men were potential disease vectors by virtue of their interaction with both the native and non-native spheres (Trimble 1985: 37). Trimble (1985: 39) concluded that, thereafter, "a regularized mechanism existed for the diffusion of epidemic diseases to the villages." Of significance to the Northwest was a similar increase in the number of traders from the north, as both HBC and NWC men began to make annual visits to the village tribes (*Loc. Cit.*). While it is true that these men might transmit *ACIs* from the Northwest to the village tribes, they also could bring disease with them on their

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the Northwest was the settlement of the Selkirk Colony, on the Red River, in 1812. However, this settlement does not seem to have had any effect on the introduction of *ACIs* to the Petit Nord before 1819.

return to the north, as could the native people who continued to visit the Upper Missouri with regularity. If Trimble's assessment of the possible impact of these visits is correct, then there was likely an increase in the number of *ACIs* reaching the Petit Nord through these movements.

Of greater impact on the Petit Nord, at least in the long term, was the emergence of endemic disease pools in the larger American cities along the Atlantic coast. Although *ACIs* had spread from the east into the interior of the continent since the first half of the seventeenth century, it was toward the end of the eighteenth century that the American urban communities located along the Atlantic coast had grown sufficiently large to permanently host the crowd diseases[8] (Duffy 1953: 178). By the late eighteenth century the disease load of the larger cities and their surrounding hinterlands was substantial, and included afflictions such as colds, erysipelas, hepatitis, smallpox, measles, whooping cough and mumps, among many others (Currie 1792: 4-5, 94, 96, 9). These new pools led to new patterns in disease diffusion in the interior of the continent. For a century and a half, the European colonies along the Atlantic coast of North America had served only as extensions of distant disease pools. On occasion, ships from Europe or the West Indies

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[8] Initially, some of these diseases may have been endemic within the eastern United States as a whole, rather than in each major city. As late as 1792, measles was not uncommon in Philadelphia, although seemingly not yet endemic, while smallpox was endemic (Currie 1792: 65, 99). At that time its population was only 42,400 (*Ibid.*: 196), or far less than the population threshold for measles endemicity that has been calculated by modern theoreticians. Dr. William Currie, a physician living in that city, stated that "the measles has been less constant" than smallpox, and that measles could flare up at any season of the year (*Ibid.*: 99). This suggests that the disease, while not endemic, did occur with considerable frequency, and the lack of a temporal pattern suggests that it was not being reintroduced from Europe on each occasion. However, following 1795 the disease was endemic in the city, with occasional epidemics, possibly on a seven-year cycle, and about the same time in Boston (Caulfield 1943: 548, 555). As of 1790, the American population numbered nearly four million, located mainly along the eastern seaboard (Borchert 1967: 314, 315). There was no dominant city at this time; instead the three major cities, Philadelphia, New

introduced *ACIs* into the major port cities, and these afflictions sometimes flowed through the colonies to other non-native communities, and to the aboriginal people of the continent. However, between epidemics, these diseases disappeared and several years or even decades could pass before they returned[9]. During these intervening periods, generations might reach adulthood without being exposed to a particular affliction. With the creation of local disease pools, however, these sicknesses became a constant threat to spread beyond the limits of non-native settlement, and into the interior.

Moreover, *ACIs* continued to arrive in the east from foreign ports even after they became endemic in the eastern cities. This reinforced their already substantial disease loads (e.g. Caulfield 1943: 549). In fact, the circulation of many diseases on a global scale was enhanced during the late eighteenth century by accelerated population growth, urbanisation and improved transportation (Crosby 1993: 809). Thus, the post-Revolutionary period can be seen as a new era of *ACIs* in the east.

ALBANY EXPANDS INLAND: 1784-1795

ACIs broke out regularly in the Petit Nord between 1784 and 1795, as almost every year saw at least one outbreak or epidemic in the region (Appendix 1). Although

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York and Boston, all had similar populations (*Ibid.*: 314), among which these diseases circulated.

[9] See, for example, Duffy (1953: 106, 108, 111) regarding smallpox. In some communities with closer connections to the external (overseas) disease pools the period between epidemics was less. Duffy (*Ibid*: 49-68) identified eighteenth century smallpox epidemics in Boston, the colonial city most frequently visited by this disease, in 1702, 1721, 1730, 1751, 1763-64 and 1774. During the same period, measles epidemics struck Boston in 1713, 1729, 1739, 1749, 1759, 1772, 1783, 1790 and 1797 (Caulfield 1943:

there appears to have been an increase in the frequency of such sicknesses in the interior over the previous period based on the HBC records, it is not clear whether this apparent escalation was genuine or an artefact of the more complete written record. At any rate, not all parts of the Petit Nord suffered *ACIs* with the same regularity at this time. In fact, there was a marked difference between the experiences of the people living in the southern part of the region and those living more to the north. Almost all of the disease outbreaks during this period occurred in the southern half of the Petit Nord. There is evidence for only one outbreak among the people of the interior living to the north of the Albany River, and there is no reason to believe that this was not representative of the actual situation[10]. Most likely, the far greater frequency of disease activity among the people of the south was due to the fact that they lived on or near the major routes connecting the region with the east and the trading routes of the plains; *ACIs* could travel rapidly along these pathways from the endemic regions.

Elsewhere, *ACIs* broke out more often among the Indians and the HBC men of the Albany Fort area than among those living at the other Bayside posts of Severn and York (Figure 15). As during 1770-1780, these afflictions continued to be relatively common at Albany, with at least seven recorded outbreaks in its vicinity between 1784 and 1795.

552-553). See also Currie (1792: 36).

[10]The sole exception was a sickness among the Indians living in the Severn hinterland during the winter of 1788-89 (HBCA B.198/a/39: 34), people who would have lived to the north of the Albany River. There was no report of epidemic activity among the people of the York Factory hinterland between 1784 and 1795.

Most were described as colds, or what may be classified generally as ARDs[11]. Conversely, widespread sicknesses were less common at Severn, with three outbreaks at the post. Only two occurred at York Factory[12]. During this period, the HBC ships played only a minor role in introducing acute infectious diseases to the Petit Nord. Between 1784 and 1795 only two, a severe disorder among the York Homeguard Cree in the fall of 1793 (HBCA B.239/a/96: 25-25d), and an epidemic that broke out amongst the Albany Indians the following fall (HBCA B.3/a/96: 5, 6d, 26d, 27d), fit the general temporal pattern for having been introduced from Europe[13]. Instead, most of these afflictions came from continental sources. Consequently, Albany, whose trading hinterland encompassed most of the southern half of the Petit Nord, was more exposed to the spread of *ACIs* than either Severn or York, through the movements of its inland men who travelled down the Albany River from the interior each summer. This was an unfortunate and unforeseen consequence of an increasingly complex transport system and the expansion of the fur trade into the region.

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[11]This included "colds" in 1784, 1785, 1787 and 1792. There was also an outbreak of "sore throats" in 1787. Of the twelve years between 1784 and 1795, at least six witnessed an acute infectious disease in the Albany area, for a total of seven separate sicknesses.

[12]The sicknesses at York included: bowel complaints among the men in August of 1786 and an epidemic disorder among the Lowland Cree in the fall of 1793 (HBCA B.239/a/86: 52d, 53d; B.239/a/96: 25-25d). Neither of these appears to have been severe. The illnesses at Severn were also minor, and included: a widespread ailment that struck the Homeguard Cree and the Winisk River Indians during the winter of 1789-90, some unidentified ailment forcing the Indians to remain on the Severn plantation in December of 1794 and a sickness among both the Indians and men at Severn in August of 1795 (HBCA B.198/a/40: 33d, 34d, 35; B.198/a/46: 18; B.198/a/47: 5). As at York Factory, these do not seem to have been mortal in any case.

[13]Others may have had a local origin, perhaps with a chronic carrier or with a non-human reservoir, depending on the disease. As well, some may have come from the east, through connections with Moose Fort, a post that traded with some Indians who travelled between Canada and Hudson Bay.

Most of the epidemics noted during the first few years after 1784 were minor complaints, and many were outbreaks of relatively mild ARDs. Nevertheless, there were a few exceptions. In 1785, a rumour circulated at Gloucester House that smallpox raged again, somewhere to the west of the post. This rumour may have been spurious, as the Indian who reported it had not observed it himself, and there is no other evidence of this disease at this time[14]. Far more significant was a severe sickness among the Indians living to the southwest of Gloucester House during the following year. According to Captain Metweash, whose lands were in the Lac Seul area, there was a highly mortal epidemic during the fall and winter of 1786-87 in the Monontague country immediately to the south of Lac Seul (HBCA B.78/a/16: 27d, 28, 29, 31d). This was one of the areas untouched by the 1779-83 smallpox epidemic. This unnamed disease did not spread to the Albany River area as the Osnaburgh House journal does not mention any affliction among the Indians who traded there (HBCA B.155/a/1; 2; Figure 15).

The next major epidemic in the Petit Nord occurred in 1788-89, and marked the return of smallpox, only five years after the previous epidemic. This time, however, it was limited to the eastern part of the region, an area that had been unaffected during the earlier visitation. Between 1787 and 1789 smallpox spread from Lake Erie to the northeastern shore of Lake Superior, and then into the interior towards Moose Fort. It

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[14]This was Notonnescome, who indicated that: "that Cruel Disorder the small pox is again raging inland but had not reached his Country when he left it." (HBCA B.78/a/12: 21d). His territory is unknown, but he also stated that his young men had traded their furs in the Fire Country, possibly meaning the prairies (Lytwyn 1986: 58, fn. 33). A day later, a stranger arrived from the Fire Country and asserted that there were many Indians there, but made no mention of this epidemic. In fact, no one ever corroborated this rumour at Gloucester House or at any of the other posts in the Petit Nord. Another rumour was spread by the NWC in 1786 to the effect that smallpox was still among the Indians of the Churchill River, leaving few alive (HBCA B.78/a/14: 8).

broke out first in 1787, near the southern end of Lake Huron and the western part of Lake Erie. There is evidence of the disease among the Wyandot and Munsee living near the southwestern shore of Lake Erie and at Detroit, as well as perhaps among the Piankeshaw [Miami] living on the Wabash (Steam and Steam 1945: 49; Tanner 1987: 173, map 32). No doubt, it originated in the east, unlike the previous smallpox epidemic, perhaps introduced by settlers, as the Wyandot blamed the white people for their suffering (Steam and Steam 1945: 49). At this time, substantial numbers of Americans were moving into the Ohio country from the east. Between October 10, 1786, and December 9, 1787, nearly six thousand American settlers travelled along the Ohio River from the settled parts of the United States (White 1995: 418), and their route lay only a short distance to the south of these native people.

This smallpox epidemic reached Michipicoten, on the north shore of Lake Superior, probably during the spring or summer of 1788, and subsequently spread to the northward (Figure 15). News of the mortality reached the nearest HBC post, New Brunswick (or Micabanish) House, late in the summer of that year. On August 28, 1788, Captain Nimica arrived at the post and informed Post Master William Bolland that "Cap[t] Mumim both his wives, and several of his Children, died a few days ago, on their return from Mishipicoote" (HBCA B.145/a/2: 4d-5). Early in September, Captain Sackawabish[15], who often aided the HBC and who had been scouting out Indians for Bolland, returned from Michipicoten and commented that "a great mortality prevails

[15]Captain Sackawabish fell ill a short time later, and was sick throughout the winter (HBCA B.145/a/2: 26).

there" (*Ibid.*: 6). By this time, however, smallpox had already spread to the New Brunswick area. The following day came news that Captain Nimica and his wife were dead of the same disease, and several more in their family were sick, having been taken ill only a few days after they departed the post late in August (*Ibid.*: 6-6d). Throughout the fall and into the following spring, Indians in the New Brunswick House area continued to fall ill and many perished, including several who had gone to Michipicoten (e.g. *Ibid.*: 5, 15d, 24d-25, 26). On June 3, Bolland, anticipating a disastrous year for trade, learned that:

> three more of the Indians (belonging the family) that was here in Winter (vide 22nd Jan) died very suddenly this spring and that several others, which I expected in here, to Trade, died in y^{e} Winter, so that there is not now above one fifth, of the Natives, living that I had been inform'd would be in here, which makes me much afraid the Trade will be but very small .... (HBCA B.145/a/2: 24d-25)

Bolland's fears proved true, as even those who survived trapped very few furs (*Ibid.*: 26).

Although there was considerable mobility on the part of the Indian people living north and west of Lake Superior at this time, this smallpox epidemic had only a limited penetration into the Petit Nord, likely only between the Michipicoten and New Brunswick House areas. It did not reach as far as Brunswick (or Wapiscogamy) House, located just to the north of New Brunswick House, nor to Henley House on the Albany River (Figure 15). Neither is there any mention in the Osnaburgh and Gloucester House journals, the other HBC posts in the interior of the Petit Nord at this time. Both of these posts attracted natives from wide areas, including the western part of Lake Superior, and had the disease struck some of the people in their trading hinterlands it is to be expected that news of the sickness would have reached the post.

In spite of its restricted diffusion, there is evidence that this sickness had some effect beyond the deaths it caused. For instance, at least one Indian avoided New Brunswick House temporarily for fear of catching the disease, and traded at another post[16] (HBCA B.145/a/2: 29d). Another example of the extended impact of this epidemic comes from Osnaburgh House, far to the northwest of Michipicoten. On June 11, 1790, a stranger from a distant land visited postmaster Robert Goodwin. Goodwin wrote in the journal that:

> at Noon a strange Captain from the Utchepoy [Ojibway] Country came in to see the place & know the track he says all the indians in his Country are dying with the small pox & he intends to leave it and come here to trade y^{e} next year if I use him well.... (HBCA B.155/a/4: 26d)

While this may be seen as an isolated incident, it may also have been part of the motivation for a general westward Ojibway migration that was taking place at this time, one that has been linked to the 1779-83 epidemic (Peers 1994: 18-21).

Following the 1788-89 smallpox epidemic, acute infectious diseases continued to appear in the Petit Nord at a steady rate for the remainder of the period to 1795 (Appendix 1). Several of these were severe or spread widely. For instance, there was widespread sickness among the local Gloucester House Indians and the HBC men in the winter and spring of 1789-90, followed by an outbreak among the men and Indians at Henley House that spring (HBCA B.78/a/18: passim, 18d, 19, 20d; B.86/a/43: 16, 45,

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[16]On August 12, 1789, William Bolland noted that "O'tash'a'way'kee'shick' came in with a few Furrs, he informs me he would have been in early in the spring, but hearing such a mortality prevail'd about here, he was afraid to come, he therefore went with his Furrs to the Sowe'way'minica Settlemt ..." (HBCA B.145/a/2: 29d). The Suweawamenica Settlement, also called Langue de Terre, was located on the west branch of the Montreal River, to the north of Lake Huron (HBCA File "Suweawamenica Settlement"; Moodie, Lytwyn and Kaye 1987).

47d). These were probably the same disease. The following fall and winter saw an outbreak of sore throats and colds among the Indians and some of the men at Henley House, accompanied by violent pain in the chest of some[17] (HBCA B.86/a/45: 3, 8, 9, 12d, 15d, 32). At least four of the Henley House Indians died during this episode. Although it is impcssible tell for certain, this disease may have been influenza. There had been a European influenza pandemic in 1788-89 (Crosby 1993: 809), and the disease had appeared in New York City in September of 1789, before spreading widely in the east and to the West Indies (Currie 1792: 2). The symptoms experienced at Gloucester and Henley were similar to those caused by influenza. It may be that this disease had spread into the interior of the continent as well, reaching at least as far as the Albany River.

Also during this period came the first reports of sickness and disease-induced mortality in the southwestern part of the Petit Nord, the first such written accounts by non-natives from that quarter since the days of La Vérendrye. As the HBC men settled posts throughout more and more of the Petit Nord, they began to observe for themselves the effects of epidemics that before would have been known to them only by (often vague) report. Still, not all were eyewitness accounts, especially during the initial years. In September of 1793, some Escabitchewan Indians told James Sutherland that there had been a "great death" among the Indians at Lac la Pluie and Portage de l'Isle (HBCA B.166/a/1: 2d; Figure 15). Sutherland suspected that this was merely a ploy designed to keep him from travelling beyond Escabitchewan. More certain is a "great mortality" documented among the Indians of the Lac la Pluie area in the winter of 1795-96. Eleven

native people of diverse ages died, including several prominent men[18] (HBCA B.105/a/3: 1d, 17d, 32). This was the first of many reports of sickness and mortality that would strike the people living within the southwestern part of the Petit Nord over the next two decades.

Finally, there was a severe and unusual sickness among the Albany Fort Indians in April of 1795 (HBCA B.3/a/96: 26d, 27, 27d). This latter affliction, which caused a "great and Uncommon Mortality" among the Albany Cree, was unknown to the HBC surgeon, "either by Observation or Tradition". There were said to have been six fatalities among the Cree between April 1 and 21 alone (*Ibid.*: 27d). Its symptoms included a swollen and inflamed tongue that made breathing difficult, and a painful swelling that extended from the lower jaw, towards the ears and down to the sternum, or breastbone (*Ibid.*: 26d, 27d). Curiously, it seems to have bypassed the HBC men, despite the presence of several sick natives at the post. Possibly, this was a disease that the Europeans had had in childhood and that behaved atypically when among the Indian people[19]. Of the many sicknesses that broke out at Albany between 1784 and 1795, this

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[17]This was perhaps symptomatic of pneumonia.

[18]Several of the HBC men also fell sick during January, perhaps with the same disease as was then affecting the natives. There was also an epidemic of inflammatory fevers among the HBC men of Swan River, to the west of Lake Winnipeg in the fall of 1795, and a suspicious sickness among the men and the Indians at Severn in August of the same year (HBCA B.239/a/99: 15d; B.198/a/47: 5).

[19]Two possibilities are scarlet fever and diphtheria, both acute infectious diseases that can affect the throat and make breathing difficult (Creighton 1965: II: 678-679). During the eighteenth century these diseases caused severe epidemics in Europe and eastern North America, and diphtheria, in particular, was only poorly understood (*Loc. Cit.*). Both may become diseases of childhood in larger populations, and the conditions under which this outbreak occurred were unlike those with which the Albany surgeon was familiar. An epidemic of diphtheria in New England in 1735-36 was particularly devastating (Duffy 1953: 131-134).

was the only one that was responsible for deaths among the natives or traders.

COMPETITION PEAKS: 1796-1809

Following 1795, both English and Canadian traders entered the Petit Nord in larger numbers than ever before. While the escalation in competition for furs had a fundamental impact on the fur trade in the region, it seems to have had little effect on the importation of *ACIs*. The overall frequency of acute infectious diseases in the region remained relatively constant compared to the previous period. Outbreaks remained a regular occurrence at Albany, with at least ten at the post or in its vicinity between 1796 and 1809. As before, most were ARD's that broke out in the summer or fall[20] (Appendix 1). *ACIs* continued to be rare at the other Bayside posts in the Petit Nord, with only three at York Factory and none at Severn. In contrast, there were many reports of sickness of varying extent in the interior of the region, all within the southern half. This was consistent with the pattern of epidemic activity of 1784-1795. However, this era also witnessed the first recorded epidemic of another crowd disease, whooping cough, in the Petit Nord. This suggests that the disease frontier of the urban pools had moved closer to the Petit Nord.

Even though there is no evidence that the rapid augmentation in the number of fur traders in the Petit Nord between 1796 and 1809 led to an increase in the number of *ACIs*,

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[20]This included outbreaks of "colds" or "catarrh" in 1796, 1804, 1805, 1806, 1807 and 1809 (Appendix 1). Some sicknesses that broke out in the Albany region during this period do not clearly indicate a source by their timing. For example, there was an unknown disease circulating among the Indians to the north of the Factory in the spring of 1798 and epidemic colds among the Albany men and Indians in February of 1800 (HBCA B.3/a/101: 18d; B.3/a/103: 13d, 21d).

it is clear that these men sometimes participated in the diffusion of these diseases within the region. For instance, all three of the outbreaks at York Factory occurred in June or July, the months when the inland crews arrived at the post[21]. While this post had a very limited hinterland within the Petit Nord at this time, its trading network extended far into the Grand Nord, along both branches of the Saskatchewan River (e.g. Moodie, Lytwyn, Kaye and Ray 1987), and each summer saw the furs of its inland trading posts carried to the Factory. As such, it was vulnerable to diseases from the plains and parkland to the southwest, carried by the returning brigades. On several occasions, HBC vessels appear to have introduced sicknesses to Albany. In 1805 and again in 1806, epidemic colds broke out at the Factory in September shortly after the arrival of the *Prince of Wales* from the British Isles (HBCA B.3/a/108: 3d; B.3/a/109: 18; C.4/1: 16d-17). On two other occasions, the Sloop brought respiratory disease to Albany from Moose. This occurred in August of 1796 and again in August of 1807 (HBCA B.3/a/97: 32, 33d, 34; HBCA B.3/a/109: 17d, 18).

In several instances during this period, HBC employees also helped spread acute infectious diseases within the Petit Nord. For example, outbreaks occurred at Martin's Falls in July of 1801 and July of 1804, just as the men of the Albany Inland District brought their furs to this post to be sent to Albany (HBCA B.3/a/104: 20, 20d, 21, 21d; HBCA B.3/a/106: 25d, 43d; B.123/a/8: 32). In both cases, the diseases subsequently

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[21]These included a sickness among the HBC men in July of 1798, another among the crews from the inland posts in June-July of 1800, and colds and sore throats among the York men and some Indians in July of 1802 (HBCA B.239/a/102: 50, 52; B.239/a/104: 45, 45d, 46, passim; B.239/a/106: 37d, 38d, 39d).

appeared at Albany after men returned from Martin's Falls[22]. *ACIs* also flowed in the other direction, as in 1796, when a virulent respiratory disease spread from Moose to Albany and was also carried up the Albany River by HBC men, perhaps as far as Lac la Pluie[23]. Remarkably, while it is probable that the Montreal-based traders also participated in the introduction of *ACIs* into the Petit Nord and in their spread within the region, there is only one instance during this period, in 1797, when they were implicated in the HBC records. Most likely, this was due to the limitations of the HBC documents and the paucity of the Canadian records, rather than to their lack of participation in such diffusion.

As during the previous period, there were many disease episodes in the Petit Nord during 1796-1809, but only a few were severe. The first came in 1796 and was among the most widespread of epidemics to that date. During the summer of that year a particularly severe respiratory disease appeared at Moose, and subsequently spread to the Petit Nord. It had not come from Europe, however, as it was well entrenched among the HBC men and the Indians at Moose by August 19, the day before the ship arrived from England[24] (HBCA B.135/a/83: 28, 28d). On that day, trader John Thomas noted in his journal that: "We have now and for some time past, had several Natives under the

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[22]As well, in September-August of 1808 sickness broke out among many of the HBC men travelling from Osnaburgh House to Sandy Lake, and those at Albany, and subsequently among the Albany Indians (HBCA B.193/a/3: 2d, 3, 3d; B.3/a/111: 5, 12, 18; B.123/a/13: 2d).

[23]See below for more on this epidemic.

[24]Although no doubt the new men from Europe who accompanied the ship served to prolong the presence of the epidemic at Moose beyond the point at which it otherwise would have died out.

Surgeon's care with severe colds +c which is afflicting several in the Factory" (*Ibid.*: 28). Here the disease proved fatal to many of the natives, as well as one of the HBC men (HBCA B.198/a/49: 44b). Shortly after, it spread to Albany Fort. On August 25, 1796, the Moosé sloop arrived at Albany bringing several new men (HBCA B.3/a/97: 32), as well as this virulent disease. By the 5th of September an "epidemical catarrh" raged among the HBC men and some 200 Indians who were tenting around the post (*Ibid.*: 33d, 34). Within a few days, several of the Indians were dead, while others were dangerously ill, and the sickness was almost universal (*Ibid.*: 34d). By the 24th, many of the Indians had succumbed, and the fate of others was doubtful (HBCA B.135/a/84: 44). It also spread amongst the Indians living to the northwest of the post (HBCA B.3/a/98: 11). The disease lingered at Albany long into the winter, preventing the Homeguard Cree from participating in the goose hunt. It also kept many from departing for their winter quarters (*Ibid.*: 3d, 4d, 6, 12d; B.123/a/3: 14d). These factors would have exacerbated the mortality caused by the epidemic. In the end, more than twenty of the Albany Indians died during the epidemic (HBCA B.135/a/84: 47d), and so this disease took a significant toll on the Indian people at Albany as well as at Moose. Coming as it did on the heels of the mortality of 1795 among the Albany natives, it must have been especially distressing to these people.

The disorder moved inland from Albany during the fall, as several infected HBC men travelled up the Albany River to the posts of the interior. Two men who were bound for Osnaburgh battled the disease from Albany at least as far as Henley House (HBCA B.86/a/52: 4d; Figure 15). Others continued farther upriver despite being ill. On

September 10, Peter Laughlin arrived at Martin's Falls while still sick (HBCA B.123/a/3: 10). Donald McKay and his men reached the post from Albany having been much delayed by sickness (HBCA B.3/b/34: 5d-6). Shortly after these arrivals, most of the Martin's Falls men fell ill of the same disease (*Ibid.*: 8d). Martin's Falls was the main transhipment post of the Albany Inland brigades, where furs from the interior were exchanged for goods from Hudson Bay. Thus, this situation was potentially disastrous for the people living up the Albany River, for the men returning to the upstream posts were all potential vectors for its diffusion.

Additional evidence suggests that this ARD continued to spread to the southwest of Martin's Falls, perhaps reaching as far as the Boundary Waters. Three of the chiefs living in the Escabitchewan area died in the period September, 1796, to May, 1797 (HBCA B.64/a/2: 14d), most likely of the prevailing pestilence. There is also evidence of a severe ARD in the Boundary Waters about this time. In April of 1797, John McKay at Lac la Pluie noted that "The Indians here are all of them very bad with a Cold", as was one of his men (HBCA B.105/a/4: 20, 20d). If these were indeed linked to the epidemic at Albany, then this disease had travelled farther than any other during this period, having spread across the Petit Nord from James Bay almost to Lake Winnipeg. In this case, the long-distance diffusion was aided by the inland movement of the HBC men during the fall, which was itself a product of the expanding fur trade of the latter part of the eighteenth century. It is thus significant that this disease was an ARD, a class of ailments to which most of the HBC men would have been susceptible, rather than a crowd disease that would have found far fewer potential victims among the Company's ranks.

Thereafter followed an outbreak of another severe respiratory disease near Escabitchewan during the summer and early fall of 1797, one that was responsible for a few deaths (HBCA B.64/a/3: 7d, 8, 16). Described as a cold accompanied by a sore throat and headache, it also disabled the HBC men at the post. This disease was said by the Indians to have been introduced by the "Frenchmen," or the Canadians (HBCA B.64/a/3: 3). Early in August an Indian Captain arrived at the post and brought word of the death of a prominent Indian (One Arm) and his wife, of "a distemper that the Frenchmen has brought among them" (HBCA B.64/a/3: 3). The next significant disease episode occurred in 1798. An especially deadly, febrile disease broke out at Sandy Lake, near the headwaters of the Albany River, during the fall of 1798 (HBCA B.192/a/1: 4d, 5, 5d, 7). Numerous fatalities were reported among the Indians trading at the HBC post, while many others were incapacitated by this affliction (*Ibid.*: 5, 7, 7d, 8d, 9). It also struck at Lac Seul, and one of the NWC men and three Indians died there about the same time (*Ibid.*: 8), although it does not appear to have travelled farther east into the Petit Nord[25].

Several additional acute infectious diseases appeared in the upper part of the Albany River and in the western part of the Boundary Waters during the first few years of the nineteenth century. In 1801, HBC fur traders carried a severe diarrhoeal disorder that first broke out at Martin's Falls. From there, it was transported down to Albany, and upstream at least as far as Osnaburgh House. At Albany, it struck the men and Indians

[25]There is no evidence of this sickness at the HBC's Osnaburgh House, nor at the posts farther down the Albany River, including Martin's Falls and Henley House (HBCA B.155/a/14; B.123/a/5; B.86/a/54).

with great force[26] (HBCA B.3/a/104: 20, 20d, 21, 21d). This "epidemical disorder" persisted at Albany Fort from mid-July at least until September[27], and incapacitated most of the men and Indians, causing at least one death (HBCA B.3/a/104: 20, 20d, 21; B.135/a/88[a]: 78). It was also carried up the Albany River from Martin's Falls, beyond Osnaburgh, by some of the HBC men bound for Brandon House[28] (HBCA B.23/a/8: 5d). That same year, or possibly in 1802, smallpox struck the aboriginal people of Rainy Lake and Lake of the Woods (Tanner 1987: 173, Map 32). Its impact is unknown, but it was part of a much larger smallpox epidemic that ravaged Indian people in the Upper Great Lakes and Upper Mississippi regions, one that was perhaps connected to a smallpox epidemic that struck the plains at about the same time[29] (*Ibid.*: 173). There is no evidence that it penetrated any farther into the Petit Nord, however, nor is it known how

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[26]The identity of this disease is not clear. Although referred to by John Hodgson and others as a bowel disease (and as the bloody flux), it seems to have been much more. At one point Hodgson suggested that the symptoms were similar to those of yellow fever (ICD-10 A95), an extremely severe, acute infectious, viral disease transmitted by mosquitoes, generally *Aedes aegypti* (Benenson 1995: 519). However, victims of yellow fever do not typically experience problems with excessive loose bowel movements, but instead suffer from a myriad of other symptoms including fever and headache, both of which plagued the HBC trader (HBCA B.3/a/23, 24). Identification of this very serious affliction based upon these few symptoms is impossible.

[27]A separate disease may have first broken out in the spring, as there was a reference to widespread, but unidentified, sickness among the Albany men in April (HBCA B.3/a/104: 11). Thereafter, the next mention of sickness was not until mid-July. There is no journal between September of 1801 and August of 1802, and so it is not possible to tell if it continued to rage after the beginning of September.

[28]Unfortunately, the lack of journals for Martin's Falls and Osnaburgh from this period means that nothing else can be said regarding its diffusion.

[29]The evidence for the presence of smallpox at Lake of the Woods was not cited, but there is certainly sufficient evidence of the disease among the Sioux at this time (Taylor 1977, 1982). Taylor (1982: 45-47) suggested that at least three major epidemics may have been circulating on the Northern Plains between 1800 and 1803, including undifferentiated respiratory infections, smallpox and a possibly concurrent (with the smallpox) epidemic of measles.

great was its extent in the Lake of the Woods area.

In 1804, another severe ARD diffused from the interior to Albany[30]. Again, it appeared at Martin's Falls in July, at the same time that the Inland men arrived (HBCA B.3/a/106: 25d; B.123/a/8: 32). Thereafter, it spread widely among the post's men (HBCA B.135/a/91: 91: 39). By the end of the month, several of the men from the upland posts had been left behind, being too sick to return to their assigned stations. There was also great difficulty in dispatching boats for Albany Fort due to sickness among the crews (HBCA B.123/a/8: 33). Nevertheless, John Hodgson, the master of Albany, and several of his men were able to make their return from Martin's Falls at this time, despite being infected with this disease (HBCA B.3/a/106: 43d). Following their arrival at Albany on July 25th, he and two other men were incapacitated due to the affliction, which subsequently became almost universal at the Factory (*Ibid.*: 25d; B.135/a/91: 38d, 39). The timing of the outbreak at Martin's Falls suggests that it had been brought by the inland crews. As well, Tanner's map of epidemics in the Great Lakes region (Tanner 1987: Map 32) indicates that an unidentified epidemic struck the people of the Lake of the Woods area in 1804, possibly the same one. As such, it is likely that the disease had come from somewhere in the southern part of the Petit Nord.

Two years later, in 1806, some of the aboriginal people living within the extreme southwestern part of the Petit Nord were exposed to an especially mortal disorder that had never before been recorded in this region, a virgin soil whooping cough epidemic. The only evidence for this outbreak occurs in a brief entry in the journal of George Nelson, a

NWC trader who traded at Lac du Bonnet, on the Winnipeg River (Figure 15). On January 15, 1806, Nelson received news from Dominique Ducharme, a fellow NWC trader who was at nearby Portage de l'Isle (Lytwyn 1986: 113), informing him that, there, the "indians [were] almost continually dying/&/ as if a contagious sickness prevailed in that quarter his only child now about 1 year old is very bad with it" (Metropolitan Toronto Reference Library, Baldwin Collection, George Nelson MS, Journal, 1805-06: 16). Although the disease was not identified by Ducharme, this outbreak was almost certainly part of a whooping cough epidemic that was at this time active a short distance to the west of the Petit Nord.

In 1806-07 whooping cough spread at least as far as from the Red River to the Columbia, and from the Missouri River to the Saskatchewan. According to the NWC's Alexander Henry, the younger, the affliction "appeared all along the Red and Assiniboine rivers, on the Saskatchewan even to Fort des Prairies[31], and in several other parts of the North-west, carrying off many people" (Coues 1965: 1: 343). The sickness was also among the Mandan late in the spring of 1806, and both Alexander Henry and Charles McKenzie witnessed its effects (Trimble 1985: 52-53). There, according to the latter trader, the disease killed 130 people in less than a month (Masson 1960: 1: 371). The epidemic was also noted in at least one Sioux winter count[32] (Cohen 1942b: 19). Finally,

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[30]This followed a brief, but severe, illness among the Osnaburgh men in March of 1803 that does not appear to have spread very far afield (HBCA B.155/a/17: 16).

[31]Fort des Prairies was an alternate name given by the Canadian traders to several different posts. Given the context of Henry's statement, the most likely is Fort Augustus, located on the Saskatchewan River at the site of present-day Edmonton, Alberta (Voorhis 1930: 60).

[32]This was the Swift Bear count of the Brule Teton. The year given by Cohen was 1807-08 but

it spread almost to the Pacific Ocean as, in September of 1807, David Thompson, another NWC trader, learned that this disease was devastating the Kutenai who were then located near Fort Kutenai, north of the Columbia River in the Pacific Northwest[33] (NAC MG 19 A8, Volume 8 (Part III) — David Thompson Papers: 278, 279). Given the prevalence of whooping cough on the Red River and its wide geographic spread, it is very likely that the outbreak on the Winnipeg River was of the same disease.

Whooping cough, or pertussis (ICD-10 A37.0), is an acute, directly transmitted[34], bacterial disease that primarily affects the respiratory tract. It is highly infective, and survivors gain a lasting immunity, although not to parapertussis (ICD-10 A37.1), a clinically similar disease[35]. Following infection, there is a relatively lengthy incubation period of six to twenty days leading to a brief catarrhal period, during which the victim experiences minor respiratory symptoms, including a runny nose, a persistent cough and a slight fever. The victim subsequently develops an irritating cough that may become paroxysmal within a few weeks, and is characterised by a series of violent coughs that are followed by high-pitched "whoops" or crowing upon inhalation. Adults, adolescents and infants under six months of age may not present with the paroxysmal cough. The infectious period lasts approximately twenty-eight days, and some symptoms may persist

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this may have been an error. Many other Sioux counts do not identify the epidemic, and several, such as those of Lone Dog (Mallery 1972) and John K. Bear (Howard 1976: 43), note a later whooping cough epidemic dated about 1814 or 1815.

[33]Thompson wrote that the Kutenai were "3 days march hence + that many y them are very sick + several children dead, it seems by the Hooping Cough."

[34]By droplet emission during coughing.

[35]Nor does infection with parapertussis produce immunity to pertussis.

for up to two months or longer, distinguishing whooping cough from many other ARDs. In larger communities, it is a disease of childhood and it is capable of causing significant mortalities in virgin soil populations, particularly due to a sequel of pneumonia. Convalescence is prolonged, and may require four months to two years for complete recovery (Benenson 1995: 347-348; Ramenofsky 1987: 149-151; Miller and Keane 1983: 1213-1214).

This was the first time that whooping cough was noted in the Petit Nord. This is significant in that, unlike many other *ACIs*, whooping cough can be readily identified by its characteristic whoop and by its prolonged duration. Also called chincough, it was rare in colonial America until the middle of the eighteenth century, when it began to appear with greater frequency[36] (Duffy 1953: 179). As well, it was a disease of childhood in England by the mid-eighteenth century (Creighton 1965: II: 670), and so there would have been few aboard the HBC ships who were susceptible to the disease. This would partly explain why the disease does not seem to have penetrated to the Petit Nord until the nineteenth century. Whether because of the increased presence of the disease in the east or because of some other factor that assisted its diffusion to the Northwest, the epidemic in 1806 signalled a new era in the disease history of the Petit Nord during which whooping cough appeared repeatedly following progressively shorter inter-epidemic intervals. This was also the final *ACI* of any significance to appear in the Petit Nord

[36]By the 1790s, whooping cough was apparently a relatively common disease in the U.S. (Currie 1792: 6, 99).

before the reorganisation of the HBC in 1810[37].

THE FUR TRADE REORGANIZED: 1810-1818

Following 1810, the fur trade of the Petit Nord underwent considerable change as the NWC withdrew many of its men from the region and the HBC restructured its operations. Coinciding with these changes was a period of limited *ACI* activity in the region. There were only seven recorded incidents during the nine years 1810 to 1818, a considerable decline over the previous two periods (Appendix 1). There is no obvious explanation for this decline, although it may in part have been due to the limited number of journals that have survived from this period. Nevertheless, even at some posts with complete (or near complete) journal records, such as Albany and Osnaburgh, there is a dearth of comments regarding disease outbreaks. None was noted at Albany, while there was only one brief period of suspicious sickness at Osnaburgh House over the winter and spring of 1814-15 (HBCA B.155/a/27: 6d, 8, 10, 13). It thus appears that fewer *ACIs*

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[37]A debilitating disease attacked the HBC men at Osnaburgh House during the fall and winter of 1807-08, however this was probably not an *ACI*. This affliction, which John Sutherland called an "intermitting fever," caused light-headedness, headaches, looseness of the bowels, thirst, shivering and violent shaking. Some of the victims were ill over the course of several months (HBCA B.123/a/12: 7). The term intermitting fever (or intermittent fever) was often used to describe malaria, which itself consists of four similar, and chronic, parasitic diseases capable of causing symptoms such as were described by Sutherland (Benenson 1995: 283; Harstad 1963: 110). Although popularly considered to be a tropical disease, malaria was nonetheless a significant problem in the Upper Mississippi country during the eighteenth and nineteenth centuries, and may have appeared to the north of the Boundary Waters at this time. See Harstad (1959-60; 1963) and Ackerknecht (1945) on malaria in the Upper Mississippi. Daniel Harmon, a NWC fur trader, noted in his memoirs that he suffered an attack of fever and ague, another term for malaria, while on his way to Grand Portage, and another while at the post, in June of 1801 (Lamb 1957: 20, 21).

were managing to penetrate into the Petit Nord between 1810 and 1818.

This remarkable turnaround at Albany contrasts with the situation at York, where there were four outbreaks in this nine-year span. During the previous nineteen years, there had been only three. This may be explained partly by the structural changes made to the HBC's operations in 1810. With the loss of its interior posts beyond Osnaburgh House, Albany's men were no longer travelling through or trading in the southernmost part of region, and therefore were not exposed to the diseases that circulated along the main travelling routes. Conversely, the posts in this area were now settled and supplied from York, which may then have been exposed to more diseases through the movements of its own people.

None of the sicknesses that broke out during this period was more than a temporary inconvenience to the HBC men or the Indian people. Two were localised outbreaks of some diarrhoeal disease referred to as dysentery (Appendix 1). The first of these, an "obstinant dysentery" among the employees at York Factory during the fall of 1811, is noteworthy as it was introduced by the European ships. This sickness was still widespread as of mid-December, with a few cases even as late as the following July[38] (HBCA B.239/a/118: 1d, 2, 3d, 5, 19). Unlike the previous two periods, there was no major epidemic in the Petit Nord during these nine years. Thus, the years between 1810 and 1818 were relatively uneventful ones in terms of *ACIs*.

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[38]No such sickness appeared at Oxford House (HBCA B.156/a/5). York Factory was spared a deadly typhus epidemic in 1813, brought to Hudson Bay from Europe by settlers bound for the Selkirk Colony, when the Captain of the *Prince of Wales* refused to land his passengers at York but instead continued to Churchill (Bryce 1910: 92, Appendix: 321; Morton 1957: 50).

SUMMARY

ACIs appeared in the Petit Nord with considerable frequency during the period from 1784 to1818, and many of them were very severe. Nevertheless, it is not clear that this was entirely the result of an increase in epidemic activity over previous periods, since it coincided with an era of inland expansion by the HBC, and therefore of increased documentary evidence. Two broad trends in the pattern of acute infectious disease activity emerged during the initial years of this period, between 1784 and 1795. The first was that these sicknesses were much more common at Albany than at Severn or York, the other two HBC Bayside posts in the Petit Nord. This was no doubt due to Albany's rapid expansion into the interior of the region, which placed several major lines of communication, and therefore of disease diffusion, within its hinterland. Because of this, the HBC traders played an increasing role in the spread of these diseases within the Petit Nord as they settled additional inland posts and travelled through the interior. The other trend was the greatly increased number of outbreaks in the interior of the Petit Nord, almost all of which occurred in the southern part of the region. Initially, most struck in the area between Henley House and Gloucester House but, as the Albany men expanded farther west and south in the 1780s and 1790s, reports of *ACIs* in this direction became more common. These trends continued for over a decade, between 1796 and 1809, with increasing epidemic activity in the southwestern part of the region. It may be significant that the number of posts in the region, as well as the number of traders, peaked at this

time, but without an equivalent rise in the frequency of *ACIs*.

Much changed between 1810 and 1818 with respect to *ACIs*. There was a significant decline in the frequency of *ACIs* in the Petit Nord during these years. For unknown reasons, they were far rarer during this period than they had been since 1784. As well, none of these episodes was very severe. Instead, they were minor complaints with little lasting impact on either the fur traders or the natives. Another fundamental shift was the complete absence of such diseases from Albany during these years, while they appeared more regularly at York Factory than they had for several decades. No doubt, this was caused by the restructuring of the HBC fur trading system in 1810 that severed much of Albany's inland district, giving it to York. Access to the interior of the Petit Nord, particularly to the southern part, enhanced the likelihood that *ACIs* would be carried back to York which now administered and outfitted the trade of that area, rather than to Albany, which was now isolated from the major routes of travel along the southern flank of the Petit Nord.

While it is not certain to what degree there was an increase in the number of *ACIs* striking the Petit Nord between 1784 and 1818 over the previous period, there is evidence that there had been a shift towards the external urban disease pools. The presence of whooping cough, a crowd disease that seems never before to have reached the region, suggests some degree of breakdown in the isolation between this region and the endemic pools. Moreover, the repeated attacks of smallpox, in 1788-89, 1802 and perhaps in 1785, so soon after the 1779-83 epidemic also indicates a significant shift. Never before had that disease appeared in the region with so few years between attacks. Still, this was

little more than a prelude to the next three decades, when the decline in the isolation of the Petit Nord far exceeded all that come before it. This era of rapid change began with concurrent measles and whooping cough epidemics that ravaged much of the Petit Nord in 1819-20, and these epidemics will be the subject of the following chapter.

CHAPTER 9 — THE MEASLES AND WHOOPING COUGH EPIDEMICS OF 1819-20

It was almost four decades after the smallpox epidemic of 1779-83 before a disease of comparable severity and extent struck the Petit Nord. Between 1819 and 1820 at least two *ACIs* were epidemic in the region. The first was measles, a disease that had been absent for almost seventy years, and that probably had never before afflicted many of the people of the region. As such, the stage was set for a virgin soil epidemic in the Petit Nord and, as the disease spread, it took on proportions consistent with virgin soil measles epidemics generally. In this case, the disease afflicted, not only the people of the Petit Nord, but many throughout the Northwest, extending from the western shores of Lake Superior westward to the Rockies, and from the Upper Missouri north to Great Slave Lake. There is also some evidence that it crossed the Rockies into the Pacific Northwest (Boyd 1994a: 9).

The second *ACI* was whooping cough, a disease that had been present in the Petit Nord in 1806, at which time it was limited to a minor outbreak on the Winnipeg River. This singular outbreak in the Petit Nord, however, was part of a much larger epidemic that had spread westward from the Red River and the Upper Missouri to the Rocky Mountains. For the people of the Petit Nord, in consequence, the whooping cough epidemic, like the measles with which many people were concurrently afflicted, also was a virgin soil epidemic.

Although different diseases had struck different parts of the Petit Nord at the same time in the past, these synchronous occurrences were highly localised episodes compared to the measles and whooping cough epidemics of 1819-20. Thus, for the first time there

is evidence of widespread multiple, or compound[1], epidemics in the Petit Nord, and in this case both were virgin soil in nature. In addition, other minor diseases were also operative in the region at this time. For example, dysentery was identified at Norway House and Lac la Pluie (HBCA B.154/a/8: 6; B.105/a/7: 73). Elsewhere in the Petit Nord, there was a frequently mentioned combination of symptoms that involved pain of the "breast" or "belly", and often of the head as well. This set of symptoms was particularly common at Martin's Falls during the winter of 1819-20 and the following spring, where several men complained of pain in the breast and belly, an ailment to which one man, James Graham, eventually succumbed (HBCA B.123/a/18; B.135/b/40: 55). Another HBC man, Robert Cock, died of a sickness with the same symptoms about the same time at Attawapiscat, a post with which Martin's Falls had contact immediately prior to its own outbreak (*Ibid.*: 18). It may be that the two afflictions were of the same disease[2], and part of an unidentified epidemic among the HBC men in the areas noted.

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[1]According to Borah (1992: 7), compound epidemics are outbreaks of sickness that contain several diseases. In essence, they are the intersection in time and space of two or more separate epidemics. The concurrent epidemics that struck different parts of the Petit Nord during its history would not be considered compound epidemics, as they did not strike the same populations.

[2]Some of the HBC men at Osnaburgh also began complaining of pain in the breast shortly after the arrival of William McKay from Martin's Falls in December of 1819 (HBCA B.155/a/32: 11, 14, 14d, 18). It is possible that some disease was spreading westward from Attawapiscat to Martin's Falls, and then to Osnaburgh at this time. As well, several of the Long Lake men, including post master John Train, were ill of an unidentified disease during the fall of 1819, possibly brought with them from Albany (HBCA B.117/a/4: 5, 6).

MEASLES

While it may not have had the virulence of the 1779-83 smallpox epidemic, the 1819-20 measles epidemic rivalled the former in its northward extent on the continent and, in different parts of its range, was accompanied by heavy mortalities. For instance, Taylor (1977: 78) estimated that 10-25% of the Sioux and greater than 25% of the Assiniboine, Blackfoot, Cree and Gros Ventres succumbed to this epidemic. Ray (1988: 106-110), in contrast, suggested mortality rates approaching 40-50%. These estimates are consistent with losses experienced by other populations during virgin soil measles epidemics where medical or subsistence aid was not provided. In 1875, 30% of the population of Fiji died in a single measles epidemic (Morley 1980: 117) while about 27% of the population of the Faeroe Islands succumbed in an 1846 epidemic (Panum 1940; Ramenofsky 1987: 161). Finally, there was a case fatality rate of 27% among those who did not receive aid during an epidemic in Brazil in 1954 (Black *et al.* 1977: 120). Within the Petit Nord accurate estimates are lacking, but in one case the mortality due to measles exceeded two-thirds of the local population (Hackett 1991: 132).

The first evidence of the measles epidemic comes from August of 1818, on the north shore of Lake Superior (Figure 16). On August 17 two canoes of freemen arrived at the HBC post at Michipicoten from Sault Ste. Marie, and some were infected with measles (HBCA B.129/a/9: 6d; Hackett 1991: 28-29). After a week's delay, several of these men and a few of the HBC employees headed north, towards Matagami (HBCA B.129/a/9: 7d). That same day two of the men who stayed behind were noted in the HBC journal as being "unwell at present with the measles" (*Ibid.*: 8d), while others among the

canoe bound for Matagami also fell ill. On September 3, post master Andrew Stewart wrote that:

> Mr. Monin and party that departed from here for Mataugumie [*sic*] on the 24th ULT were still at the first portage about two hours walk from here ... They had not even got their canoe over the portage owing as they say to two of the men being unwell (HBCA B.129/a/9: 9d).

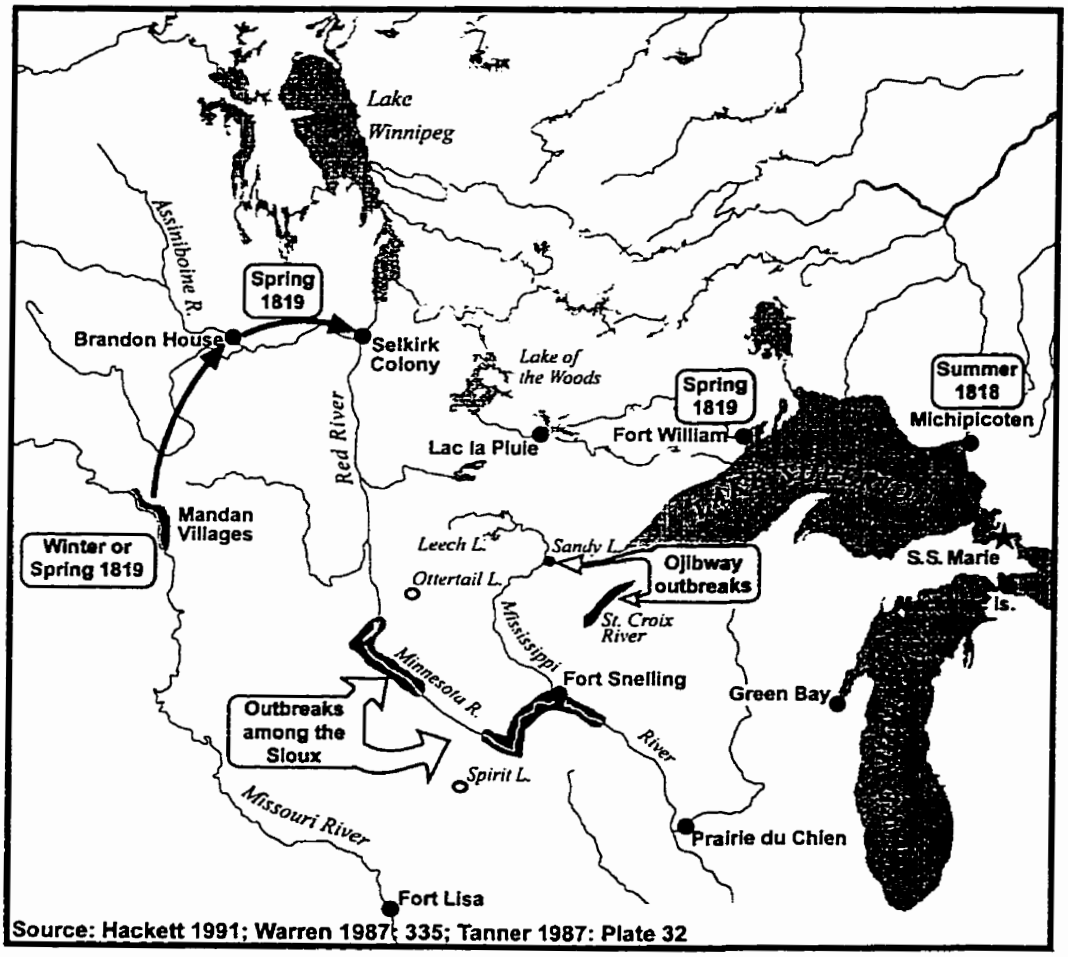

Figure 16: Locations of initial outbreaks of measles, 1818-19

This particular outbreak did not lead to further diffusion in the Petit Nord. Because of the delay at Michipicoten, measles went no farther than the portage. While there were no fatalities and this episode produced only minor, and temporary inconvenience, this

outbreak nonetheless suggests an eastern origin for the affliction that would strike the Petit Nord the following year. The freemen had come from Sault Ste. Marie and had been infected there or somewhere in its vicinity (Hackett 1991: 29). Sault Ste. Marie could not have been the source of the virus, however, as its population was far too small to support measles endemically. The fact that the outbreak occurred in the Great Lakes region, however, suggests an eastern origin, where the disease was endemic at this time.

Although diseases of eastern origin may have spread along many routes, the evidence suggests that the measles that reappeared in the Petit Nord in 1819 spread from the east along two distinct pathways. The first of these led directly into the Petit Nord from Lake Superior. The second followed a more circuitous route, spreading south of the lake to the Upper Missouri, and from there north and east into the Petit Nord.

Diffusion from Fort William

According to Peter Fidler, writing in June of 1820, measles was "... brought up last Summer by the NW [i.e. the NWC brigades] from Canada" (HBCA B.51/a/3: 2). The following August, Fidler commented more generally on the extent of the disease and observed that measles (together with whooping cough) had been introduced into the Northwest by the NWC traders at Fort William, their major administrative post on the

northwestern shore of Lake Superior[3] (Figure 16). In the context of the decline of the fur returns as a result of these afflictions, Fidler noted that: "The deficiency of trade all thro' the Northern Department I am sorry to inform you - principally by the Measles + Chincough being introduced last spring 12 month from the Grand Depot of theirs [the NWC's Fort William] on Lake Superior..." (HBCA A.10/2:242). While there are no records from Fort William for this period, there is evidence of the measles at the nearby HBC post, Point Meuron, in late May. At that time, an employee named Antoine Pacquette began presenting visible symptoms of the disease (HBCA B.231/a/4: 27d), probably having contracted the disease on a visit to Fort William (Hackett 1991: 57).

The disease then spread from Fort William to the Ojibway of this region. In July of 1821 Nicholas Garry was at Fort William in order to supervise the rationalisation of the newly merged HBC and NWC. While there, he observed a ceremony involving the chiefs of the post. Garry recorded in his diary that:

> We had today the Ceremony of Two Chiefs offering their presents in the great Hall and Receiving a return. The Chiefs preceeded [*sic*] by an English Flag marched into the Hall accompanied by all the tribe ... One of the Chiefs then rose and really in a very graceful manner made a speech ... He said his Tribe had been afflicted with the Measles and this would a little account for the few People he had brought with him ... (Garry 1900: 116)

From Fort William, the NWC brigades carried the disease north and westward into the

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[3]Following the abandonment of Grand Portage early in the nineteenth century in favour of Fort William, each June and July the population of this post swelled greatly. As the winterers arrived with their furs from the interior, and voyageurs arrived from Montreal with the trade goods, the NWC's headquarters in the Northwest became more a town than a post, hosting over three thousand people within the post and its environs (Davidson 1918: 204; Wallace 1934: 1). Within the fort were a large house, the council house, the doctor's residence, store houses, a prison, apartments for mechanics and several other buildings (Davidson 1918: 238). Outside of the pickets were the temporary quarters of the voyageurs, Indians and others (*Loc. Cit.*).

Petit Nord.

Some of the brigades that left Fort William during the summer of 1819 travelled through the Boundary Waters, bound for the Grand Nord, and were responsible for passing on the measles along their way. By early September, the disease was among the Indians at Portage de l'Isle, brought among them by the NWC men (Hackett 1991: 94; HBCA B.105/a/27: 28). At Garden Island, on Lake of the Woods, measles and whooping cough were proving fatal to the Indian people by late August (Hackett 1991: 94; HBCA B.105/a/7: 29d; James 1830: 255). At Sabaskong Bay, at the southeastern corner of Lake of the Woods, the HBC trader sent from Lac la Pluie was forced to pay a premium for wild rice as

> the Indians ... were violently enraged against us, the NW [the NWC] having circulated a Report among the Indians that the measles which has been so prevalent among them + of which many of them died had been brought into the country by us. (HBCA B.105/a/7: 36d)

In all probability, measles spread widely among the Indian people living on Lake of the Woods and along the Winnipeg River, since it was introduced at about the time of the wild rice harvest, during which there was considerable interaction between these people (Waisberg 1984: 127; Vennum 1988: 143, 163). Nevertheless, not everyone in this vicinity suffered during the epidemic. At the HBC's Whitefish Lake outpost on Lake of the Woods, returns for 1819-20 exceeded previous years (HBCA B.105/a/7: 86d). This suggests that the Ojibway wintering there remained free of sickness (Figure 17).

It is not known precisely when measles reached Lac la Pluie, but it devastated the people of this area during the fall and winter of 1819-20. Roderick McKenzie, the master

of the HBC's Lac la Pluie post, was late in assuming charge of his assigned post, arriving on September 14[4], and the first mention of the disease in his journal is not until early October, by which date it seems to have spread widely. On October 3, McKenzie noted that he "endeavoured to get the Indians to Seine but they had all refused Saying how could I expect them to Seine when they are all dying of the measles" (HBCA B.105/a/7: 38d). Later in the month he was informed that "all the Indians towards Vermilion + Sturgeon lake were Starving + many of them dieing [*sic*] of Sickness which we suppose to be the measles which is [raging] among them all over this part of the country" (*Ibid.*: 43). Throughout the winter of 1819-20, sickness and starvation went hand in hand among the Indians of this area, exacerbating the effects of the epidemic, and on many occasions ill and starving natives arrived at one of the posts in need of sustenance (HBCA B.105/a/7: 49d, 50, 51d, 54d). Only by the spring did the health of the Indians in the Lac la Pluie district return.

[4]McKenzie had passed through Lac la Pluie early in July on his way to York Factory, before the outbreak of the disease, but did not return to occupy the post until mid-September (HBCA B.105/a/7: 15d, 30d).

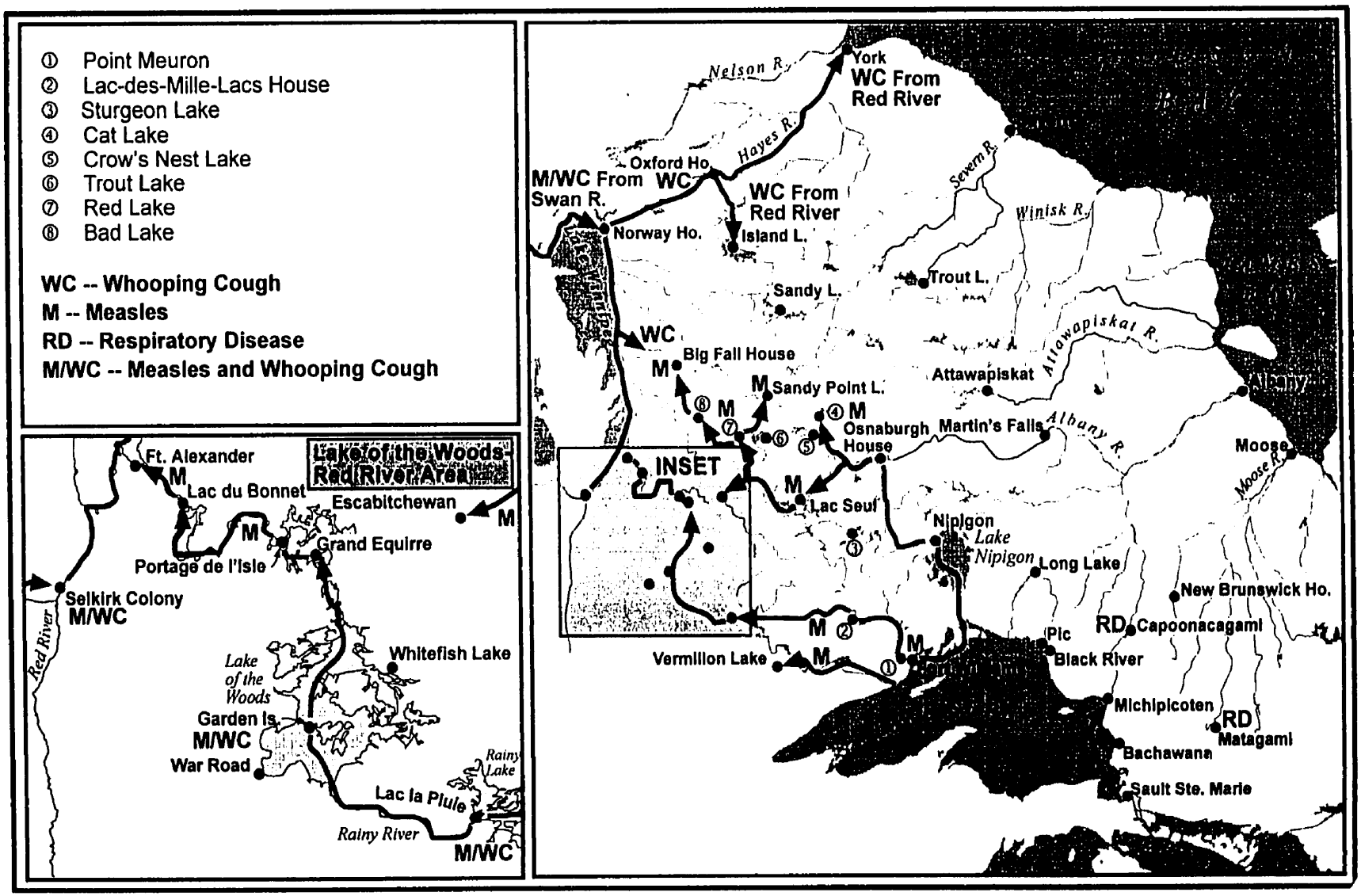

Figure 17: Diffusion of the 1819-20 measles and whooping cough epidemics in the Petit Nord

Other NWC canoes carried measles into the interior of the Petit Nord as they headed northward from Fort William. In mid-August of 1819, George Atkinson, an HBC trader stationed at Osnaburgh House, learned of the tragic events then unfolding in the country to the south of his post. At the nearby Pedlar's Path, on the NWC's route to the interior of the Petit Nord, he met a Canadian clerk whose men were sick. Atkinson grew uneasy when he:

> saw the servants of the N.W. Mr McKinzey [*sic*] (chief clerk) with 10 men in two Large Canoes who informed me of a Distemper raging amoungst the Indians to the Southward, and have destroyed numbers of them - and I much fear that great numbers of these Indians in this quarter will feel the Horror of this malady as they [*sic*] are already numbers of People in a most distressing situation belonging to the NW Servants who are now proceeding to the Northward. He informed me that two of his men were [prevented] from walking and that six more were ill of the same disorder (HBCA B.155/a/32: 4d).

The clerk, Charles McKenzie, was headed first to Crow's Nest Lake and, thereafter, to Lac Seul. By the southward, Atkinson probably meant the region near Fort William, and possibly the Lake Nipigon area, through which this brigade had travelled. Ironically, despite Atkinson's fears and the passing of these infected men, measles did not appear among the native people of the Osnaburgh House area, nor did it ever spread to Sturgeon Lake, where the Canadians had a post outfitted by the same Charles McKenzie (Hackett 1991: 98-99; HBCA B.123/b/2: 20; B.155/a/32; B.155/e/8; HBCA B.107/a/7: 13; Anick 1976: 410).

From Osnaburgh, McKenzie proceeded to Crow's Nest Lake and Cat Lake where he obtained new men, three of his men being in no condition to continue (Anick 1976:

410; Figure 17). With no records from the NWC posts of that area, and no HBC posts in the vicinity, it is impossible to know whether or not the Indians trading at the Cat Lake and Crow's Nest Lake posts contracted measles from the infected men who had been left there. After outfitting his replenished brigade, McKenzie headed for Lac Seul.

McKenzie arrived at Lac Seul on September 4 with two canoes and ten men (HBCA B.64/a/8: 4d), passing measles on to the Ojibway of the area. According to the Lac Seul Ojibway who survived the epidemic, it was "the Canadians at Lac Sall [who] had brought the Mesals to that place..." (HBCA B.64/e/2: 2). Once introduced, the disease attacked the Lac Seul people with a ferocity perhaps unequalled in the Northwest. Following the epidemic, reliable estimates stated that about two thirds of the Lac Seul people succumbed to measles (HBCA B.64/a/7: 20). Most died within four months of the appearance of the disease and some of the few survivors temporarily fled the area[5] (HBCA B.105/a/7: 56d). As James Slater passed through the lake in June of 1820, he was told by some of the Indians that twenty-three Indian hunters and a total of seventy men, women and children had died (HBCA B.64/a/8: 20). In his district report he added that there were only five young hunters still alive at Lac Seul who were capable of hunting anything (HBCA B.64/e/2: 4d), whereas the previous year there had been thirty hunters attached to the NWC's Lac Seul post (Anick 1976: 404). In later years, Charles McKenzie stated that two thirds of the Lac Seul Ojibway died during this epidemic, for a

[5]On Christmas Day of 1819, Roderick McKenzie at Lac la Pluie was visited by some of the surviving Lac Seul Indians who informed him that "there were no less than Sixty Indians Men, Women, + Children, dead of the Measles +c at Lake Saul since the Fall set in, all that were in that Department excepting three" (HBCA B.105/a/7: 56d).

total of seventy-six[6] (HBCA B.107/e/3: 2; HBCA B.107/a/25: 12). Thus, it is evident that a tremendous death had occurred among the Indians at Lac Seul. In the words of one HBC trader, they "were almost intirely swept away" (HBCA B. 123/e/32d).

Despite this mortality and the small numbers of survivors, within a few years of the epidemic the number of Indians trading at the Lac Seul post rebounded considerably, exceeding the pre-epidemic figure. In 1826-27 there were 60 Indian hunters attached to Lac Seul and the smaller Cedar Lake outpost (HBCA B.107/e/3: 2). In 1827-28 there were 51 hunters, about one tenth of whom were youths (HBCA B.107/a/6: 26). A more complete description of the population attached to Lac Seul and outposts was provided in the Albany District Report for 1828-29, and included fifty men, forty-eight women, fifty boys, fifty girls and seventeen widows and orphans[7] (HBCA B.3/e/15: 3). By 1837, McKenzie counted 339 Indians trading at Lac Seul (HBCA B.107/a/16: 28). Seemingly, then, the number of people trading at Lac Seul had increased incredibly over the space of seven years, an increase that continued to at least 1837.

This apparent rebound led Bishop (1974a: 159-62) to emphasise the rapid growth of the Lac Seul population during this era, and to suggest an overall gradual population increase during the century, despite also acknowledging the epidemic of 1819-20 and the "levelling" impact of other epidemics. The implication, although not stated, was that the

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[6]In 1837-38 he noted that the population of Lac Seul "is greatly on the Increase since 1821.... At the above quoted date the population was small the Measles having carried off 7/10 of the natives a few years before which is the cause that there is not a very old man among them" (HBCA B.107/a/16: 28).

[7]The report attached to the 1828-29 Lac Seul journal provided a slightly different breakdown and total with: fifty men and six boys (hunters); sixty-two married women and widows, fifty-seven male and female children, for a total of 218 (HBCA B.107/a/7: 21d).

survivors had managed to recoup their numbers to a point where they exceeded those of the pre-epidemic period. Clearly, however, if both the post-epidemic population estimates and the later tallies are correct, then the level of fertility at Lac Seul would have had to have been unbelievably high during this period. Moreover, given the apparent rise in the numbers of hunters, generally adult males, from perhaps five to approximately fifty, recruitment from the sub-adult male cohort would have exceeded the total reported surviving population. As such, it is evident that something was incorrect with these estimates or, more accurately, with the way in which they are used.

The answer lies in the fact that none of these are truly censuses. The early figures are estimates of the mortality effects of the epidemic, and of the number of survivors. These provide reasonably accurate indications of the post-epidemic population of the Lac Seul Indians. The later counts, however, are enumerations of those Indians trading at the Lac Seul post and include many more than the local people. Following the merger of 1821, many of the posts in the Petit Nord were closed, such that, by 1825-26, there were far fewer posts operating in the area under the HBC banner and none was settled by the NWC (Lytwyn 1986: 159). For instance, Red Lake[8] was not settled after 1821, and some of the hunters thereafter traded at Lac Seul, as did the Indians of Escabitchewan, which was abandoned in 1824 (HBCA B.107/e/2: 2d; HBCA Post Histories "Escabitchewan", "Red Lake"). Cat Lake closed about the same time, and some of the Indians went to live or trade at Lac Seul (HBCA B.107/e/2: 3). The Kingfishers, a group of Ojibway who had

[8]In August of 1822 McKenzie wrote that "The Red Lake Ind[s] are expecting a house on their lands this year - but if not, the most of them will take their Debts at this place" (HBCA B.107/a/2: 6).

traded in the Berens River area during the epidemic, also began to frequent the Lac Seul post at this time (for example HBCA B.107/a/2: 5, B.107/a/9: 24d). Several Lac la Pluie people also traded at Lac Seul following 1821 (HBCA B.105/a/9: 68). Moreover, by 1833, the Sturgeon Lake Indians had overrun the lands of the Lac Seul Indians, forced there by a fear of the Lake Nipigon Indians who had in turn encroached on their lands (HBCA B.107/a/12: 10d; B.107/a/13: 4). In 1819-20 they would have traded at the NWC's Sturgeon Lake post or at the HBC's Osnaburgh House. Following the merger, then, the Lac Seul post attracted hunters from a large territory, including many who traded at other posts in 1819-20, all of whom were being included in McKenzie's post-merger "censuses" of the Lac Seul Indians. Thus, comparison of these early and later post-epidemic estimates does not provide evidence of a significant rebound of the Lac Seul population following the 1819-20 measles epidemic, nor does it support the hypothesis that there was continuity of population levels in the area, other than through migration from other areas. This suggests that care must be taken when using simple post-based estimates as indicators of demographic trends for, except in rare circumstances during the fur trade period, none of these was intended for such purposes.

In 1819, McKenzie settled two more outposts to the west of Lac Seul, at Red Lake and at Bad Lake (Lytwyn 1986: 155; Figure 17), and his men introduced the measles to the Indians trading at those posts. As the NWC began to give advances to the Ojibway of the Red Lake area in September, they also spread the disease (Hackett 1991: 103). Thereafter, and until the spring, measles circulated among these people and among the HBC men (HBCA B.64/e/2: 3). Here, four of the five HBC hunters died, for a total of

twelve of all ages, male ands female (*Loc. Cit.*), and the number of total casualties would have been extremely high had not the HBC's Marcus Calder fed the ill who remained at the house (*Loc. Cit.*).

The NWC did not settle at Escabitchewan in 1819-20, and measles was later to arrive in this area than it did at Lac Seul or Red Lake, where it had been carried by the Canadian traders. Instead, the disease was introduced from Lac Seul by some Ojibway who arrived at the HBC post on October 10, having earlier traded with Charles McKenzie (Hackett 1991: 104). Other Indians at Escabitchewan soon contracted this sickness, as did the HBC trader's children (HBCA B.64/a/8: 6d, 7, 7d) and thereafter it spread widely among the local Indians. Within two weeks it had broken out at nearby Cut Lake, and on the 24th a family of Indians from Cut Lake arrived at the HBC post suffering from measles (HBCA B.64/a/8: 6d). Most of the twenty-four Indians attached to the Escabitchewan post contracted measles and thirteen died (HBCA B.64/e/2: 4).

In 1819-20, the HBC also settled an outpost at Big Lake, a two-day walk from Portage de l'Isle on the Winnipeg River[9], again unopposed by the NWC. Despite being in the vicinity of the Winnipeg River and close to Lake of the Woods, where measles and whooping cough broke out during the summer, measles appeared at Big Lake only at the time of the fall freeze-up, or in mid to late October (HBCA B.64/a/8: 3). This suggests that they were not part of the initial Boundary Waters thrust which swept through early on, but contracted the disease at a later date, perhaps from the north or by trading later

[9]The location of Big Lake is not clear, beyond the fact that it was about two days walking from Portage de l'Isle.

with the NWC on the Winnipeg River (Hackett 1991: 106). The number of mortalities, or even the degree of sickness at Big Lake, is unknown.

Charles McKenzie's men also introduced measles into the East Winnipeg Country from Bad Lake[10]. William Harris, who had been left at Lac Seul during the summer, departed with four men for the Bad Lake outpost on the Bloodvein River shortly after McKenzie's arrival. Here again they infected the Indians during the fall trading, who began dying shortly thereafter (Hackett 1991: 108; HBCA B.16/e/3: 2, 10d, 18). According to Donald Sutherland of the HBC, the Bad Lake area was inhabited by the Kingfishers, and during this epidemic they lost their Chief, his eldest daughter and five other men (HBCA B.16/e/3: 2; HBCA B.16/a/3: 2). Despite this, the level of mortality was low among the Indians trading with the NWC at Bad Lake, at least compared to that at Lac Seul. On January 1, 1847, Charles McKenzie recalled in his Lac Seul journal that he had

> passed many new year days at this place [Lac Seul] and never seen our store so very empty of furs as at present on such a day, save in 1819, when the Measles passed before and carried off 76 souls from this post alone - and left us but seven hunters - with plenty widows and orphans - But I had then another post at Bad Lake where the Measles made less havoc. (HBCA B.107/a/25: 12)

Thereafter, measles spread widely through the area to the east of Lake Winnipeg, and resulted in what HBC trader Donald Sutherland called "the Dreadful Death" (HBCA B.16/a/3: 11d). The Indians who traded at Sutherland's Big Fall House on Berens River were probably exposed either directly or indirectly from Bad Lake, and only after they

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[10]Bad Lake, or Lake St. Charles, was said to be sixteen days by canoe or ten days walking from Lac Seul (HBCA B.107/a/12: 3).

had traded with the HBC (Hackett 1991: 110; HBCA B.64/a/7: 7: 13). Others in the East Winnipeg Country were infected elsewhere. Those who lived upstream along the Berens River at Sandy Point Lake received debt at the HBC's Red Lake House (HBCA B.64/a/8: 16), and it is probable that that is where they contracted the disease (Hackett 1991: 110).

Diffusion from the Upper Missouri

While measles spread into the Northwest from Fort William, on Lake Superior, another branch of the same epidemic entered the region from the Upper Missouri (Figure 16). Writing in the summer of 1820, HBC Governor William Williams observed that:

> The diseases [whooping cough and measles] has been introduced from some of the American out Posts on the River Missouri, and first shewed themselves at the Mandan villages and have from them spread all over the Country like contagions with a rapidity almost beyond belief. (HBCA D.1/2: 11d)

The Mandan Indians' location on the Upper Missouri was central in terms of transportation and trade and made them vulnerable to such epidemics, as was seen in the context of the 1779-83 smallpox epidemic. In 1819 they served as a bridge between the American Plains and the Canadian Northwest for the measles and whooping cough epidemics.

Measles reached the Mandan early in 1819. In December of 1818, a large party of HBC men travelled from Brandon House to trade with the Mandan, before returning to their post after Christmas. They did not observe measles among these Indians. However "they learned from the Mandan that the nearest American was 300 miles below but that

the next spring they were intended to go up [the Missouri River] and settle at the Mandan villages" (HBCA B.22/a/21: 40). This was probably Fort Lisa, at Council Bluffs on the Missouri, which was the supply depot of the Missouri Fur Company. At this time, the post consisted "of a few huts in which he [Manuel Lisa] deposits his goods, and from which he sends out his traders with outfits" (Nichols 1969: 41). One of these traders may have gone to the Mandan early in 1819 carrying the disease[11], for the next northern visitors to the Mandan contracted measles while there.

Peter Fidler, who identified Fort William as a source of measles during this epidemic, was also certain that measles spread from the Upper Missouri. In June of 1819 he wrote that "Grant + party brot it [measles] from the Mandans last month when they visited them" (HBCA B.51/a/2: 6d). This Grant was an Ojibway named Captain Grant, also called Oo ke mow es cume, who lived with his people in a small native settlement located on the Assiniboine River half way between Brandon House and Portage la Prairie (Moodie and Kaye 1986: 175; HBCA B.51/e/1: 18d). They left for the Missouri in April, and by the next month had carried measles back to the Assiniboine River on their return[12]. The disease then spread to the HBC's Brandon House, and from there

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[11]According to a contemporary letter describing the trade of the Upper Missouri in 1819 (Wishart 1979: 47), the Missouri Fur Company had several traders among the Sioux, and two were trading with the Arikara. Although there were no permanent American traders at the Mandan villages at this time, it would have been a relatively short journey from the Arikara to their northern neighbours along the Missouri. In April of 1820, John Gale, at Council Bluffs, noted the downward progress of three boats from the Mandan trade laden with buffalo robes (Nichols 1969: 84). As late as the summer of 1817, Manuel Lisa was still proposing to settle a fort among the village tribes of the Upper Missouri, the Arikara, Mandan and Hidatsa (Chittenden 1954: 901-902).

[12]On October 7, 1818, Fidler, then at Brandon House, noted the arrival of four Indians who traded dry provisions and informed him that they intended to go to the Mandan villages to purchase horses, "as they are friend" (HBCA B.22/a/21: 34). In April he wrote that Captain Grant and fifteen Ojibway had left

downstream to the Selkirk Colony[13]. By late June, measles reached the juncture of the Red and the Assiniboine. Fidler, then at Fort Douglas, wrote that:

> the colony men came from Brandon Ho. where they were left by Mr. Laidlaw enclosing the Ground- most of our people at Brandon badly with the Whooping Cough + Measles - as also the Indians - 5 Crees + 4 Stone [Assiniboine] Indians already dead of it (HBCA B.51/a/2: 7).

Nearly two weeks later several of his own children and others in the colony were ill with the measles, and at least two people had succumbed (HBCA B.51/a/2: 8). From July through the remainder of the summer, the colonists' children and the native population of the area suffered greatly from both measles and whooping cough[14].

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for the Mandan "as friends" (*Ibid.*: 51). A large company of Assiniboine contracted measles when they went south to make war on the Mandan, although slightly after Grant's group. They began collecting in early April and headed south in June (HBCA B.27/a/8: 31; B.51/a/2: 4). On August 27 Fidler noted the return of what remained of the party. "... Only 7 returned, all died by the way, on their return supposed by the Indians to be Smallpox" (*Ibid.*: 13; See also PAM MG 7 D13 Microfilm reel 1 *Belleau Collection*: Provencher to Amable Dionne, St. Boniface, 1 September, 1819).

[13]The newly-formed village of Birsay, located just to the west of the Red River Settlement, was struck by both measles and whooping cough, and was abandoned in the summer of 1819 partly due to the ravages of these diseases (Kaye 1981: 21). There was some confusion in the Northwest surrounding the role of the Red River Settlement in introducing measles into the region, largely the result of attempts by the NWC to use the epidemic to their advantage. The Canadians took every opportunity to blame its effects on the HBC, and on the Colony that threatened their trade. For instance, near Edmonton House they:

> tried every means to [exasperate] the natives against us [the HBC], as well as against the Settlers at Red River, by impressing upon their mind that it was the Setters who first brought those diseases to Red River, and that it was the English (as we are called) who brought them from there to this part of the country .... (HBCA B.60/e/3: 7).

See also Masson (1889: I: 130); Esau (1986: 100); HBCA (B.51/a/2: 11; B.105/a/7: 29d).

[14]On July 27 the Roman Catholic Father J.-N. Provencher wrote that "La cocqueluche [whooping cough] et la rougeole [measles] ont regné cet été parici et ont fait mourir beaucoup d'enfants" (PAM MG 7 D13 Microfilm reel 1 *Belleau Collection*: Provencher to Bishop Plessis, St. Boniface, 27 July, 1819). See also Provencher to Dionne (PAM MG 7 D13 Microfilm reel 1 *Belleau Collection*: Provencher to Amable Dionne, St. Boniface, 1 September, 1819). Writing in October of 1819, Midshipman Robert Hood, with the Franklin Expedition then at Norway House, noted that "the inhabitants [of the Red River Settlement] were swept away by the measles and hooping cough" (Houston 1974: 36).

As Governor Williams suggested, once the measles entered the Northwest from the Mandan it diffused widely and rapidly, reaching as far as Lake Athabasca by the fall of 1819 (Decker 1989: 90; Krech 1983). Nevertheless, while this branch of the epidemic spread through much of the Grand Nord, it merely touched the western margins of the Petit Nord, appearing only at Norway House, and only in mid-November. It was carried there (with whooping cough) by two native men from Swan River, to the west of Lake Winnipeg. They arrived at the post on November 13 and reported that measles and whooping cough were "carrying off a great number of [the] Indians" in the Swan River area (HBCA B.154/a/8: 6). A week later several of the children at the post fell ill of a disease which soon proved to be measles (*Ibid.*: 7). By November 27, both whooping cough and measles were spreading among the people at Norway House, some of whom were very ill (*Ibid.*: 8). The references to these diseases cease after November, and neither appears to have spread among the aboriginal people of the area, no doubt because the outbreak occurred after the Indians had dispersed to their winter quarters. As at Michipicoten, the outbreak at Norway House did not lead to further diffusion in the Petit Nord.

WHOOPING COUGH

There may be a tendency to consider whooping cough the lesser of the two diseases sweeping the Northwest in 1819-20, since, under circumstances more familiar to western observers, it would produce considerably lower mortalities than measles with its severe complications. Nevertheless, it, too, was capable of interfering with the

subsistence quest of the hunters of the forest, and thus causing starvation, as the severity of the cough experienced by its victims could make hunting game impossible. During the 1819-20 epidemic, it was observed at Fort Wedderburn, in the Athabasca country, that whooping cough was a threat to the lives of the Chipewyan as "a disease particularly distressing among the Indians as well for its long continuance as its depriving them of the means of subsistence the whole of their caution in approaching an animal being rendered abortive by a single cough" (HBCA B.39/a/15: 8). Despite its severity, in 1819-20 whooping cough was overshadowed in the minds of observers by the concurrent measles, and the evidence regarding its diffusion is thus far more limited. As such, it cannot be studied in the same detail as the measles epidemic.

It appears that the whooping cough, like the measles, had an eastern origin. This disease was prevalent in Detroit during the fall of 1817 (Drews 1939: 762), and it is likely that this was an early manifestation of the same epidemic. Other sicknesses spreading westward from the Upper Great Lakes, including smallpox in 1737-38 and some later whooping cough epidemics, arrived in the Red River region after a considerable delay, in several cases years. Whooping cough appeared in the Red River Settlement in the spring of 1819, and so its pattern of diffusion was similar to that of other, better documented, epidemics[15].

As was the case with measles, the statements made by HBC employees pointed to the Upper Missouri and to Fort William as local sources of whooping cough for the

[15]These better documented epidemics occurred in the post-merger period and will be addressed in the following chapter.

Northwest (HBCA A.10/2: 242; D.1 /2: 11d). In this case, however, there is no supporting evidence of the disease in the eastern part of the Boundary Waters or among the brigades heading northward from Fort William, and so it may not have been present with measles at the NWC administrative headquarters, as Peter Fidler claimed. Nevertheless, whooping cough was found in at least two locations in the western part of the Boundary Waters. In September it was noted at Garden Island, on Lake of the Woods, and during the fall it afflicted the Indians at Rapid River, near Lac la Pluie (HBCA B.105/a/7: 29d, 71d). No doubt it was present in other Indian communities in this area, although not documented by the HBC, but there is no indication whatsoever that it was present towards Fort William. Rather than arriving from Lake Superior, it is more likely that these outbreaks were part of the epidemic that had come from the Upper Missouri.

There is good evidence regarding the disease's progress from the Upper Missouri. As noted above, whooping cough spread from the Mandan to the HBC's Brandon House on the Assiniboine River in the spring of 1819, and was at the Red River Settlement by late June (HBCA B.51/a/2: 7). At about the same time it diffused northward. On July 12, Roderick McKenzie met a band of Indians suffering from whooping cough at the mouth of the Red River (HBCA B.105/a/7: 18). It seems also to have appeared in the Berens River area at this time. During the summer, what appears to have been an epidemic disease struck the Indians then located along the eastern shore of Lake Winnipeg. Donald Sutherland, the master of the HBC's Berens River post, noted that the Indians he expected to meet at his post in August were then "at Pigeon River all lying badly and near

to death" (HBC B.16/a/2: 7). Likewise, he later learned that the people of Little Bloodvein River "were all very badly the Course of the Summer" (*Ibid.*: 8). This was prior to the arrival of measles, which only penetrated to the East Winnipeg Country in the fall, and so the affliction was probably whooping cough.

Over the course of the summer and fall of 1819, whooping cough spread from Red River to York Factory, and to the east (Figure 17). At Island Lake, and throughout the country surrounding this post, the Indians suffered from whooping cough during the summer and fall of 1819, and into the ensuing winter, though with few fatalities (HBCA B.93/a/2). It was said to have been "brought from Red River this summer by some of the half breeds in the Service" (*Ibid.*: 3d). Although the number of fatalities was not great, the epidemic nonetheless had a significant impact on the trade of this area. In the Island Lake District report for 1819-20 it was noted that:

> the trade would have been much better had not the Indians been severely afflicted with a disorder which prevailed among them last summer in all parts of the country[.] it proved mortal to a few at this place and brought whole families to the verge of distraction for want of food, as it was not an uncommon sight to see a good hunter with three wives (as many of them have) and a number of children reduced to a state of perfect misery all from his being in a condition so as to be unable to give assistance either to himself or his family - at this place two Indian Men who were both good hunters and a woman died of the distemper, many of them were never perfectly recovered during the most part of the winter, consequently could not exert themselves ... (HBCA B.93/e/1: 2d-3)

Whooping cough was carried to York Factory during the early part of the summer

of 1819, again from the Red River area[16]. On July 7 James Swain noted in the journal that there were: "a number of the Natives and Children afflicted with the Hooping Cough. It has been brought from Red River + seems highly contagious" (HBCA B.239/a/126: 32). The disease remained active among the Indians and the HBC men's families into August, and for a considerable time after, disrupting the fall goose hunt (HBCA B.239/a/126: 33, 35; B.239/a/127: 23). Sick Indians continued to arrive at the post as late as March of the following year, and Swain's replacement, Adam Snodie, was made aware of the recent deaths of two more people in February of 1820 (HBCA B.239/a/127: 24, 26). The trade at York Factory suffered as several of the best trappers were carried off by whooping cough (HBCA B.239/e/2: 2).

Nearby Oxford House remained free of whooping cough until late August or September. It is impossible to identify the date of its arrival with any more precision, however, as there is a gap in the HBC's Oxford House records between August 20, 1819,

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[16]Although the Arctic explorer, Sir John Franklin, was at York Factory in late August and wrote in his memoirs that the Lowland Cree about York Factory "had a squalid look, and were suffering under the combined affliction of hooping-cough and measles..." (Franklin 1823: 25), it is likely that only whooping cough struck the Indians of that area in 1819-20. There are several reasons why it is unlikely that measles ever reached York Factory in 1819-20. Firstly, the disease was never mentioned in the York Factory records at this time. Secondly, during a subsequent measles epidemic in 1846, Lettitia Hargrave, wife of Chief Factor James Hargrave, stated that measles affected all age groups of Indians at York Factory (Macleod 1947: 221-222), suggesting that there was no age selectivity among its victims. Had there been a prior measles epidemic in 1819-20, age selectivity would have resulted due to the immunity of the survivors of the earlier epidemic. Finally, Franklin's account was not written at the time, unlike the HBC journals of Swain and Snodie. He lost his journal of the expedition and relied on journals belonging to three other members of his party to later reconstruct the events in his published narrative (Houston 1984: xi). None of the journals written by John Richardson, Robert Hood or George Back (Houston 1974; 1984; 1994) describe the Indians at York Factory, however. Thus, Franklin's description of the Cree, written some years after the fact, probably came from his memory, and his statement that measles was then at York Factory may reflect his faulty recollection. There can be no such doubt about the presence of whooping cough. It was Franklin's misfortune to contract the disease even as he observed the York Factory Cree. Along the Hayes River, Hood (Houston 1974: 33) wrote that "Since our departure from York, Mr. Franklin had been afflicted so severely with the hooping cough, that it threatened strangulation."

and October 1, 1820. There was no mention of the disease in the HBC journals prior to this gap in the record. Sir John Franklin and his Arctic expedition arrived at the post on September 27, and he later noted in his published narrative that the Oxford House Indians were afflicted with the combined scourges of whooping cough and measles (Franklin 1823: 37). In this he was in agreement with Midshipman Robert Hood, also of the expedition[17] (Houston 1974: 31). Nevertheless, the identification of measles among the Oxford House Indians is questionable. There is no other evidence of the disease in the Hayes River area until the following November, when it spread to Norway House from the west. Moreover, George Back, another member of the Franklin Expedition, identified whooping cough alone as their affliction, and his record seems the most precise of the three with regard to the state of health of the Indians[18] (Houston 1994: 17). Thus, it would appear that whooping cough reached Oxford House some time between August 20 and September 27, and was not accompanied by measles. The source of the disease is not

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[17]Since Franklin's memoir of the expedition was written making use of the journals of the other men on the expedition, he probably got this information from Hood's journal. Hood's description of their state is of some interest, in that it identifies both the psychological impact and alterations to their subsistence practices caused by their sickness. He noted that:

> a number of Indian tents were pitched near the house, and the dreadful ravages of the hooping cough and the measles had filled them with lamentation and despair. The poor creatures felt so deeply the loss of their relations, that they forsook their hunting occupations and starvation brought them to the border of the lake, where without much trouble they obtained fish. (Houston 1974: 31)

[18]Back wrote that "near the lake, were several tents of Indians, who were all ill of the hooping cough". The fact that he failed to mention measles is probably more significant than the fact that the other two did, for they were consistent in mentioning both diseases together. Franklin seems to have linked the two diseases in his mind, for they are generally mentioned in conjunction. This was the case at York Factory, at Oxford House, to the west of Lake Winnipeg along the Saskatchewan River, and again still farther up that river (Franklin 1823: 25, 37, 47, 49). Conversely, Back noted only whooping cough at Oxford House but both diseases at Muddy Lake, along the Saskatchewan River, suggesting that he was giving their plight more consideration. The situation at York Factory is especially noteworthy, in that Franklin's suggestion of measles is not born out by the testimony of the HBC men.

known, but it had already reached York Factory and Island Lake before it broke out at Oxford House.

Whooping cough's appearance in the Norway House area was considerably delayed, compared to the rest of the Red River-York Factory corridor. Along with measles, it reached the post only in mid-November, or long after the disease had spread to Oxford House, York Factory and Island Lake[19]. It was likely brought by the same party of Indians who introduced measles from the country to the west of Lake Winnipeg (HBCA B.154/a/8: 6, 8) and, like measles, did not spread beyond the post's confines.

While there is evidence of one or both diseases at all of the HBC posts between Norway House and York Factory, and also at Island Lake, neither disease spread eastward to the Indians of the Severn district. These people, including the Cree living in the Hudson Bay Lowlands about Severn, and the Indians of the interior who hunted along the Severn River and eastward, were not subjected to epidemic sickness in 1819-20. Neither the post journals (HBCA B.198/a/58[a]; B.198/a/59), nor the Severn district report for 1819-20, which includes data on the Trout Lake and Winisk River outposts (HBCA B.198/e/3), nor John Work's journal of an expedition up the Winisk River (HBCA B.198/a/58[b]), make mention of disease among the Indians (Figure 17). In the northwestern part of the Petit Nord, whooping cough was confined to the country to the

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[19]There is a gap in the Norway House record between May 30 and October 11, 1819, but the first mention of the disease was not until November, and so it is highly unlikely that either whooping cough or measles broke out in that area during that period. The Franklin expedition arrived at Norway House on October 6, and none of their journals make mention of any disease. Two days earlier, at Robinson Lake, located midway between Norway House and Oxford House, they had met a party of "Bois-brulées" who also seem to have been free of epidemic sickness (Houston 1994: 19, 20; Houston 1974: 35).

west of the Severn River, primarily along the western margin of the Petit Nord.

OTHER SICKNESSES

Although both measles and whooping cough failed to penetrate to the north and east of Lake Nipigon, following the initial outbreak of measles at Michipicoten (Hackett 1991: 112-115), other disorders were present. At Long Lake, for instance, an unknown malady struck HBC post master John Train and four of his men in September of 1819. This disease may have been brought by them to their post (HBCA B.117/a/5: 5, 5d). As noted above, at Martin's Falls the men experienced an unidentified sickness with generalised symptoms including pain in the breast, perhaps suggestive of pneumonia. At Michipicoten and Albany, the 1819-20 outfit year was relatively uneventful with regard to sickness (HBCA B.129/a/10; B.3/a/124). In the New Brunswick House area, to the east of the Albany River, there were reports of a widespread but unidentified respiratory disease that was epidemic among both Indians and traders alike. In late May of 1819, Charles McCormick noted in his journal that "there is an epidemical catarrh at present prevailing which affects almost everyone in this quarter" (HBCA B.145/a/39: 49). It is likely that this was influenza, which was then prevalent in the Great Lakes basin (Heagerty 1928: 1: 212). The following January, Jacob Truthwaite at Capoonacagami, an outpost to the north and west of Michipicoten, wrote to McCormick's successor, John Murphy, and informed him that he and the Indians had had a "terrible hard cough all the fall and are not quite clear of it" (HBCA B.145/a/41). Murphy responded a few days later, saying that "there are few parts of the Indian Country in which it [the cough] has not

been felt this year" (*Ibid.*: 14). Judging from the fur traders' few entries, and the excellent returns at New Brunswick House (HBCA B.145/e/9: 10), there were probably few, if any, fatalities owing to this epidemic.

SUMMARY

In 1819-20 measles, whooping cough and other unidentified diseases struck the people of the Petit Nord. Measles originated in the east, and passed through Sault Ste. Marie to the Petit Nord by way of the NWC's main post, Fort William, and by the Mandan villages of the Upper Missouri. From the Upper Missouri, this disease spread throughout much of the Grand Nord, yet it barely touched the Petit Nord. After moving northward from the Assiniboine River to the Swan River, it was introduced among the people of Norway House in November of 1819. It made no further progress here, being limited to the people of that post. Almost certainly, subsequent diffusion from the post was prevented by the delay in its introduction, which came after the local Indian people had dispersed for their winter territories.

In contrast, measles penetrated deeply into the Petit Nord from Fort William, initially transported by NWC canoes, and later by aboriginal people through more localised movements. The broader pattern of diffusion was hierarchical, reflecting the transport network of the NWC and the organization of its posts. During the summer of 1819, measles spread westward from Lake Superior through the Boundary Waters. It was passed to the Indians around Fort William and reached the Winnipeg River by late August, eventually diffusing throughout the western part of the Boundary Waters. As the

NWC men traded with the Indians, obtained provisions and departed for their outposts, they infected the Indians with this disease. Farther north, measles was carried by NWC men who had contracted the disease at Fort William, first to the Cat Lake area, also in late August and, later, westward as far as Lake Winnipeg, as subsequent posts were in turn manned by infected employees. The northern part of the Petit Nord escaped the measles epidemic, however. Although some NWC brigades travelled from Fort William to distant posts such as Trout Lake, Island Lake and Sandy Lake, there was almost no chance that they would introduce measles at their destinations. Average travel times from Fort William to these posts exceeded 50 days (Lytwyn 1987), and if crewmembers were infected with measles travel times would have been much longer, if indeed the brigades could complete their journeys. By the time they reached their posts, they would no longer have been infective.

The fate of the Lake Nipigon Indians is unknown, but it seems likely that they, too, were struck by measles at this time. The disease was brought to Lac Seul early in September, by a NWC brigade that had been remanned at Cat Lake. Later in the month, NWC men transmitted the virus from Lac Seul to outposts at Red and Bad Lakes. Thereafter, Indian people distributed it throughout this broad country as they interacted with the traders and with each other. Consequently, measles arrived at Escabitchewan late in October with some Ojibway who had traded with the NWC at Lac Seul. It was also passed on to the Indians of Sandy Point Lake as they travelled to Red Lake to trade, probably in September, and it arrived at Big Lake late in the same month. Finally, it also spread through the East Winnipeg Country from the NWC post at Bad Lake. Once again,

the onset of winter probably limited further diffusion of the virus.

Whooping cough also struck the Petit Nord in 1819-20. However its pattern of diffusion did not correspond precisely to that of measles, and it seems to have had less impact on the people of the region. It, too, originated in the east, and spread to the Northwest from the Upper Missouri, but beyond this the two diseases travelled by different means. Whooping cough diffused early through the Red River-York Factory corridor, and was carried by HBC employees. It struck first in the Island Lake area and at York Factory, and probably in the Berens River area, early in the summer of 1819. Subsequently, it broke out at Oxford House. The disease reached Norway House only in mid-November, having been introduced from the west of Lake Winnipeg. Whooping cough did not penetrate any farther into the northwestern part of the Petit Nord than Island Lake, however, and it did not reach the Severn area. Where it did strike the people of this quarter, casualties do not seem to have been great.

Whooping cough also appeared among some of the people of the Boundary Waters. It was present along the Winnipeg River and near Lac la Pluie, and almost certainly elsewhere in the western part of this heavily travelled transport corridor. As both diseases overlapped in this relatively densely populated area, whooping cough would have had more of an impact than where it appeared alone. Unlike measles, it was unable to penetrate into the interior of the Petit Nord from the Boundary Waters.

There were other sicknesses present in the Petit Nord in 1819-20, and at least one was an *ACI*. As well as various complaints experienced by the HBC men, there was an ARD circulating in the western part of the region between the spring and fall of 1819 that

affected both the Indians and the traders. Although the descriptions of this widespread sickness identify only a very hard cough and "an epidemical catarrh" it is likely that this was an extension of an influenza epidemic then circulating in the Great Lakes region.

Part of the significance of the 1819-20 epidemics lies in the fact that at least two widespread sicknesses appeared in the region concurrently, and overlapped in the most densely populated part of the region. It is also noteworthy that both *ACIs* were virgin soil in nature. Nevertheless, they did not sweep throughout the region. Beyond the resultant mortality, which was considerable in some areas, it is important to note that, for the first time in its history, the region was struck by compound epidemics, in which more than one widespread disease appeared at the same time and among the same people. This new phenomenon would be repeated in subsequent years, however, such that the 1819-20 epidemics heralded a new era in the epidemic history of the Petit Nord in which *ACIs* appeared with far greater frequency than before. During the quarter century following these measles and whooping cough epidemics, the region was buffeted by repeated, at times overlapping, epidemics that offered little respite for the native people. This crucial period will be examined in the following chapter.

CHAPTER 10 —*ACIs* IN THE PETIT NORD: 1821-1845

INTRODUCTION

Significant changes occurred in the patterns of non-native settlement and movement in northeastern North America following 1821, both within the Petit Nord and beyond its limits. These developments led to changes in the frequency and range of *ACIs* within the region. They also resulted in alterations to the patterns of epidemic diffusion within the Petit Nord. The most significant of these changes derived from the merger of the HBC and NWC in 1821, and from the further penetration of non-natives into the interior of the continent. As a result, the Petit Nord's long period of relative isolation from external urban disease pools came to an end, and the people of the region experienced epidemic diseases with unprecedented frequency.

In 1821, the two rival fur trading companies in the Northwest, the NWC and the HBC, merged under the banner of the English company, transforming both the fur trade of the region and the epidemic fortunes of the people of the Petit Nord. The overseas functions of the newly constituted HBC shifted to York Factory, and the merger all but eliminated the use of the St. Lawrence-Ottawa River route to the Northwest, a transport thoroughfare that had served as a corridor for disease diffusion for over a century. Instead, the vast majority of goods and men reached the Northwest aboard HBC vessels from Europe, via the Bayside Factories (Innis 1956: 289). Following the merger, a few men annually travelled from Canada to the Northwest via the Ottawa River or the Great Lakes, but this flow was insignificant compared to the NWC brigades of the pre-merger days. More importantly in the long term, the merger also increased the likelihood that the

employees of the Company would act as vectors in the long-distance diffusion of *ACIs*. Prior to 1821, both companies hired principally from populations in relatively close proximity to larger urban areas, the British Isles for the HBC and Canada for the NWC (Moodie, Kaye, Lytwyn and Ray 1987). Following the merger, however, the HBC's men came increasingly from those born in the Northwest (Moodie, Kaye and Lytwyn 1993), and especially from those from the mixed-blood population. Unlike the Europeans and Canadians, these men had grown up in relative isolation from major disease pools, with little exposure to crowd diseases.

Also contributing to the increased frequency of disease was the rapid post-merger growth of the Red River Settlement. From its inception in 1812 to the merger, the Settlement had been a small, relatively insular and slowly growing colony, whose population was almost entirely European in origin (Kaye 1967: 38). Following 1821 it was transformed into a rapidly expanding, dynamic regional community, increasingly comprised of people born in the Northwest (Kaye, Moodie and Sprague 1993; Kaye 1981: 164). This growth in the settlement's population made it an effective temporary holding body for *ACIs*, not large enough to host crowd diseases endemically, but sufficiently so to prolong an epidemic's stay in the Red River region. Significantly, these diseases appeared with considerable frequency due to increased mobility and interaction with the outside world on the part of the people of the post merger Red River Settlement. Moreover, the settlement increasingly served to meet the seasonal voyaging demands of the HBC. These men travelled with the brigades for only a brief period during the spring and summer, but could carry diseases from the colony great distances into the fur trading territories.

Beyond the HBC's territories, the most important factor affecting the diffusion of *ACIs* into the Petit Nord was the movement of increasingly larger numbers of Americans into the interior of the continent. Each year, from the mid-1820s on, thousands of settlers moved westward into the lands bordering the Upper Great Lakes, bringing the settlement frontier closer to the borders of the Petit Nord. These eastern immigrants travelled in larger numbers than ever before, and with greater speed, due to improvements in transport and travel afforded by the widespread adoption of the steamboat and the construction of canals. Together, these factors led to an increase in the number and range of *ACIs* that circulated in the interior, and thereby led to an increase of the disease load of the Petit Nord.

A NEW ERA OF *ACIs*

The epidemic record of the Petit Nord after 1821 indicates that there was a long-term shift in the disease load of the region. Throughout most of this period *ACIs* appeared with increasing frequency such that, by the mid-1830s, observers were beginning to comment upon the deteriorating health of the country. For example, at Lac Seul the veteran trader Charles McKenzie noted with concern in 1836 that:

> this country certainly is getting very unhealthy of late years... yet some 30, or 20 years ago there were none of these Coughs or Colds in the country either among the natives or the whites[1]. (HBCA B.107/a/14: 7d)

In 1833 George Keith, a former NWC and then HBC trader, commented that "pestilent... colds... often appear at" Michipicoten (HBCA B.129/a/18: 7). The situation worsened during the following decade. By 1841 the Petit Nord had been exposed to many of the infections of the urban disease pools on a regular basis. Once again, this frequent disease activity was reflected in comments made by contemporary observers. In 1843 Donald Ross noted that "every breeze from that quarter [the country to the south of Norway House] ... blows some foul disease or other amongst us" (Glazebrook 1938: 461). Two years later, Ross wrote to Governor Simpson, informing him that "as to the propriety, or I may add, in the unhealthy state of the country for some years back, the necessity of having a medical man stationed at this place (Norway House), a Doctor at York Factory is simply useful for that Establishment alone..." (HBCA D.5/14: 242d). Finally, in 1845 two British army officers, Lieutenants Henry Warre and Mervyn Vavasour, travelled along the north shore of Lake Superior and through the Boundary Waters. They concluded that "The Indians on the route by which we have lately passed, are so reduced in numbers by their intercourse with the white traders, by sickness and other causes, that they may be considered as perfectly harmless" (HBCA A.12/3: 137). While their conclusion that the Indians living in this border region were "perfectly harmless" was

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[1]In his report for 1839-40, McKenzie noted that: "The Indians were entirely free from any flying [acute epidemic] disease, to which they have been so much subject of late years..." (HBCA B.107/a/18: 19d).

naive, there can be no doubt that these native people, like many others, had been hit hard by epidemic disease.

The increased variety of diseases within the region during this period also suggests a shift in disease load. Of the many afflictions that appeared, several had never before been described in the region. These included chickenpox, which struck in 1835, and mumps, which appeared first in 1841 and again in 1845 (Appendix 2). Other diseases that had been rare prior to 1821 appeared with greater frequency in the ensuing twenty-five years. For instance, whooping cough, which had been unknown in the Petit Nord prior to the nineteenth century, appeared on at least three occasions during this period, and the earliest of these epidemics, in 1825-27, came on the heels of that of 1819-20. More significantly, this era saw the emergence of influenza as the most common *ACI* in the Petit Nord, appearing both as small, localised outbreaks and epidemics, and as spatially extensive epidemics[2]. Following an epidemic in the Great Lakes area in 1819 and an outbreak at Norway House in 1822, the disease appeared twice in the Red River Settlement in the space of less than a year, in March-April of 1827 and January of the following year (Appendix 2). There were also outbreaks in the Albany-Martin's Falls area in 1833, to the north of Lake Superior in the winter of 1833-34, and at Fort William in September of 1835 (*Loc. Cit.*). These were followed by a series of widespread, often

[2]It is to be expected that a disease such as influenza (or colds) would emerge as the dominant *ACI* in the region following the transition. There was a limit to the frequency with which crowd diseases could flare up in the region. No matter how constant the opportunity for infection they could not progress among a given population unless sufficient numbers of susceptibles were available (Snow 1992: 184). Until the number of susceptibles had been replenished, usually by births, a crowd disease that was introduced would spread farther. Thus, in smaller populations the inter-epidemic period for a particular disease like smallpox might not be lessened significantly by an increase in the frequency of its appearance among nearby populations.

devastating, epidemics that swept through the Northwest between 1835 and 1846 (Ray 1976). All of this suggests that conditions had been altered in a way that favoured the diffusion of "new" diseases, as well as the other *ACIs* that previously had been only rarely present in the region. The Petit Nord was undergoing an epidemic transition, one that was underway elsewhere in the continent, and for the same reasons.

The Epidemic Transition and the Petit Nord

As noted in Chapter 2, the erosion of isolation between a lightly populated region and an urban region with its own disease pool will generally lead to an increase in the frequency and variety of acute infectious diseases present in the formerly isolated area. This shift from isolation to inclusion within the disease frontier of such pools can be considered an epidemic transition. When the degree of isolation was considerable and the decline abrupt, this transition could be most pronounced. For example, in analysing the epidemic history of the Pacific Northwest, Boyd (1985: 348-352; 1990; In Press) identified the rapid breakdown of isolation between that region and the outside world in the 1840s as the precipitating factor in the emergence of a new era of diseases. As settlers and their children flooded the region from the east, its disease load both increased and became far more varied. By the mid-1840s nearly every large party of settlers that travelled through the Columbia River corridor brought infectious diseases, most of which spread among the Indians of the Pacific Northwest[3] (Boyd In Press). Thus, the previously

[3]These settlers travelled along the various overland trails from the Missouri River to the Pacific coast beginning in 1841, the most famous being the Oregon Trail (Schlissel 1982). In doing so, they acted as the moving edge of the American frontier, the outer limits of the influence of the urban east and growing

isolated Pacific Northwest was, in a decade or less, integrated into what Boyd called the larger Euroamerican disease pool[4] (*Loc. Cit.*). While separated from the Pacific Northwest by nearly 2,000 kilometres, a similar transition occurred in the Petit Nord, but at a slightly earlier date due to its proximity to the eastern disease pools. Here, the transition was also fuelled by the wholesale expansion of non-native settlement into the interior. Unlike the Pacific Northwest, there was no significant immigration into the Petit Nord by non-natives prior to 1850. Instead, it was the flood of settlers into the territory beyond its borders that led to the vast increase in *ACIs* in this region.

Nevertheless, as the frontier threatened to make its way through the Petit Nord

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Midwest. In a manner reminiscent of the introduction of *ACIs* to the Atlantic colonies by ships during the seventeenth century, settler caravans brought with them from the east diseases such as measles, scarlet fever, typhoid fever, mumps, dysentery, smallpox and cholera (Reed 1944: 269; Schlissel 1982: 10-11, 13, 15, 47, 60). Along the way, they passed on these diseases to the Indians (Schlissel 1982: 53; 118, 131, 182; Taylor 1977: 60). On the impact of the Oregon Trail on some of the Plains Indians, see comments made by the AFC trader Edwin Denig (Ewers 1961: 19, 36, 38, 185, 186 fn. 35). Taylor (1977: 60) considered the trails to be a critical mechanism in the spread of *ACIs* to the Northern Plains, and beyond. Stoffle *et al.* (1995) blamed the Mormons, who travelled along a similar trail, for the introduction of epidemic diseases to the Numic Indians living in Utah.

[4]There are other examples of such a transition. Taylor (1977: 59-60) noted a fundamental shift in the diseases striking the Northern Plains that corresponds to the breakdown of isolation. Prior to the nineteenth century, he suggested, only smallpox was present on the Northern Plains. Subsequently, diseases with less ability to diffuse than smallpox such as whooping cough, influenza and streptococcal infections began to appear. Taylor (1977: 56) also concluded that, for the northern plains, "after 1849, epidemics of one sort or another occurred almost yearly, due to the intensification of Native American and Euroamerican interaction...." A more recent example comes from the Yukon. In the spring of 1942 work began on the Alaska Highway and, as large numbers of outsiders arrived from the settled parts of the south during the first two years of construction, the Indians of Teslin Lake were exposed to an array of infectious diseases that was unprecedented in their history (Marchand 1943: 1019). These strangers brought with them, and passed on, many infections that were normally found in cities. Measles appeared in September of 1942, infecting 129 of a total population of 135, and was followed in rapid succession by outbreaks of dysentery, a catarrhal jaundice, whooping cough, German measles, mumps, tonsillitis and upper respiratory complaints. Finally, there was a severe outbreak of meningococcic meningitis during the summer of 1943 that claimed the lives of four children. In this short time, then, the Indians of Teslin Lake had suddenly been exposed to many of the ills of urban life (*Ibid.*: 1020) after previously being isolated from these infections. The sudden increase in *ACIs* during this period at Teslin Lake corresponds to the more lasting transition that occurred in the Petit Nord during the period 1821-45.

during the 1850s, the Indians grew very wary. When Henry Yule Hind and his "Red River Exploring Expedition" reached Lake of the Woods in 1857 they did so as the advance guard of white settlement, and the Ojibway were not co-operative. Asked to provide a guide for the route to Red River, the Indians refused, stating that: "We do not want the white man; when the white man comes he brings disease, and our people perish; we do not wish to die. Many white men would bring death to us, and our people would pass away" (Hind 1971: 100). Their response was dictated by years of frequent exposure to epidemic disease and by the fate of those living elsewhere. They knew that with such settlement came devastating diseases, and by impeding the progress of its agents they sought to prevent the epidemics that were sure to follow.

The movement into the interior by significant numbers of Americans began shortly after the American Revolution. Initially, these settlers took advantage of the natural waterways and moved along the Ohio and St. Lawrence Rivers and towards Lakes Erie and Ontario[5] (White 1995; Smith 1985: 162). In 1800, the United States was primarily a coastal nation with only a few pockets of settlement in the interior (Figure 18). Thereafter expansion into the interior increased, particularly after the War of 1812[6], and by 1820 the settlement frontier extended in a thin ribbon along the Ohio River to the Mississippi (Figure 18; Smith 1985: 162; Brown 1948: 195). There remained little in the

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[5]St. Louis, founded from New Orleans in 1763 by the French, Pittsburgh, Cincinnati, Lexington and Louisville were all settled on or near the Ohio River during the late eighteenth century, but all had small populations by the turn of the century (Wade 1959: 35).

[6]The end of the war saw a new era in westward movement by the Americans, largely due to the destruction of much of the Indian resistance that had previously prevented their movements into the area to the north of the Ohio River (Meinig 1993: 223).

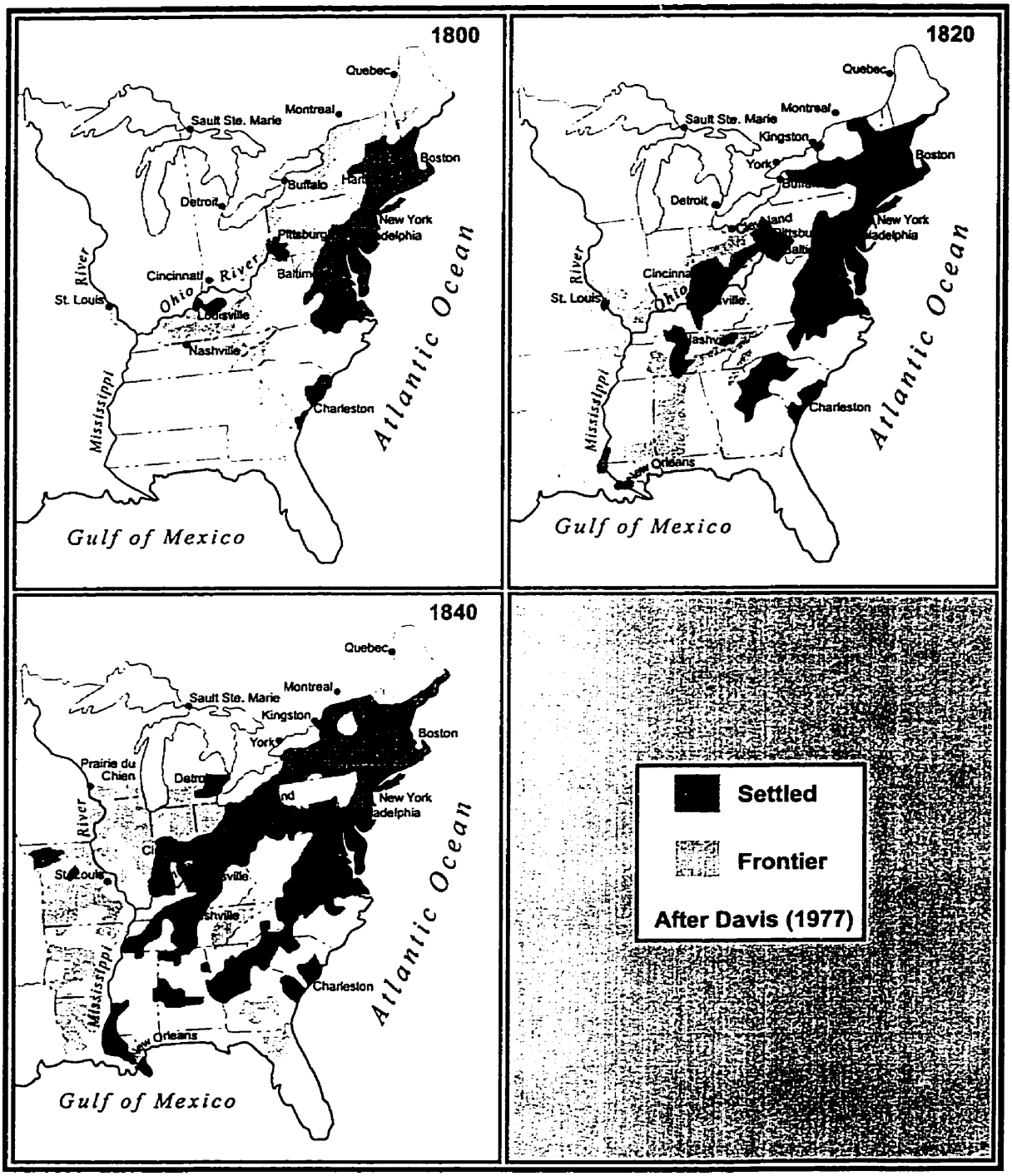

Figure 18: Changing patterns of settlement in the United States, 1800-1840

way of migration into the Upper Great Lakes until later in the decade, when it began to penetrate to the south and east of the Petit Nord[7] (Tanner 1987: 11).

In 1825 the Erie Canal was completed, connecting New York City with Lake Erie and bypassing Niagara Falls. In providing a through waterway from the Atlantic coast to the Great Lakes, this canal immediately facilitated migration into the interior. Not only did its completion lead to an overall increase in the numbers moving westward from the U.S., but it also contributed, for the first time, to the rapid influx of immigrants from the eastern states into the lands bordering the Upper Great Lakes (Vance 1986: 125; Tanner 1987: 122; Harstad 1963: vi). New York City, its starting point, landed the greatest share of immigrants to the United States. Many of these new Americans moved directly westward via the canal in the years following 1825[8] (Shaw 1966: 274). By 1826, as many as 1,200 people arrived at Buffalo, the terminus of the canal system, in a single day, bound for the west (Shaw 1966: 273). In 1835, a total of 5,126 boats passed Buffalo heading westward (Vance 1986: 126). The Welland Canal, which connected Lakes Ontario with Lake Erie and also bypassed Niagara Falls, was built as a Canadian alternative shortly thereafter, and was also heavily used (Glazebrook 1964: 83). By 1830 vessels had a through communication from New York City and Montreal as far as Sault

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[7]There was no significant movement into the area to the west of the Great Lakes until after the mid-point of the century. For instance, the non-native population of Minnesota was less than 5,000 in 1849 but rose to nearly 170,000 by 1860 (Larsen 1930: 387).

[8]Migration to the U.S. from overseas was substantial over the next two decades, with 538,000 people arriving during the 1830s and 1,427,000 during the 1840s (Birdsall and Florin 1992: 51). By contrast, only 129,000 people came to America during the 1820s (*Loc. Cit.*). Between 1800 and 1850 the U.S. population grew remarkably, from 5,306,000 to 23,192,000 (Meinig 1993: 222). Most of this was due to natural increase.

Ste. Marie, and beyond into Lake Michigan. Other canals soon followed, creating a network of internal waterways that carried goods and people between the east and the interior[9] (Vance 1986; Glazebrook 1964; Legget 1955, 1976; Scheiber 1969; Shaw 1966).

A second improvement in transportation, the steamboat, also had an effect on the westward flow of American settlers during the 1830s and 1840s. According to Borchert (1967: 303), the era of the steamboat on the Ohio-Mississippi-Missouri began in the 1830s, although they had first appeared in significant numbers during the previous decade. The same was true of the Great Lakes, and the prevalence of these faster vessels enabled the movement of large numbers of people into the interior. Thus, "The introduction of steam power created major transportation corridors on the western rivers and the Great Lakes and resulted in enlargement of the hinterlands of ports on both the inland waterways and the Atlantic" (*Loc. Cit.*). This contributed to the tremendous growth in the non-native population in the interior. According to Hunter and Hunter (1949: 29):

> During the half century following the introduction of the steamboat the West experienced an extraordinary growth. The mass movement of settlers from the older states, supplemented by a mounting stream of immigration from abroad, assumed phenomenal proportions in the second quarter of the nineteenth century. ... In the face of this mass onslaught the frontier retreated steadily; [and] the Indians were thrust back....

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[9]Meinig (1993: 223) identified the post 1830 period as an important phase in American expansion, during which westward migrations proceeded on a large scale due to "the complete removal of the Indians, the full impact of the canal age, and the emergence of the railroad from an experimental supplement to a primary medium of transport". The late 1830s saw the beginnings of the creation of a railroad infrastructure in the U.S. that would eventually replace the combination of canals and steamboat as the main means of transport into the interior (Meinig 1993: 324-327; Vance 1986: 286-289). Even before a real infrastructure was complete, the use of discrete railroads decreased the travel times for journeys from the eastern cities into the interior of the continent.

With the widespread adoption of the steamboat countless settlements sprang up along the Ohio and the Mississippi, and what were once small communities, such as Cincinnati, New Orleans and St. Louis, grew greatly in size[10] (Hunter and Hunter 1949: 30; Wade 1959: 70). Although Hunter and Hunter were describing the effects of the steamboat on settlement in the west, the same was true of the Great Lakes states.

By 1840 the American settlement frontier had expanded far to the west of the Mississippi (Figure 18). At the same time, there had also been significant infilling of the area to the east of this line including towards the Upper Great Lakes[11]. As the 1840s progressed, increasing numbers of people travelled into this area by way of the St. Lawrence River or the Erie Canal, and the Great Lakes. Many communities grew rapidly because of their situation. Thus, for instance, Detroit grew from only 2,222 people in 1830 to 21,019 by 1850 (Pyle 1969: 62, 67). Buffalo grew from 8,653 to 42,261 and Chicago from 4,470 to 29,963 over the same period (*Loc. Cit.*). Similar growth was evident in Canada, although of a smaller magnitude. As settlement in the province of Upper Canada accelerated during the late 1820s and the 1830s, the population rose rapidly, from 177,174 in 1827, to 321,145 in 1834 (Goheen 1973: 48, 50) and to 432,159 in 1840 (Glazebrook 1964: 60). By 1837, the settled area of Upper Canada extended as far north and west as Penetanguishene, on the southeastern end of Georgian Bay, and to

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[10]Cincinnati expanded from 24,831 to 115,435 people between 1830 and 1850 (Pyle 1969: 62; Hunter and Hunter 1949: 30). St. Louis, a gateway to both the Upper Missouri and the Upper Mississippi, experienced considerable population growth. During the 1830s and 1840s the city grew by more than tenfold, from 6,694 in 1830 to 77,860 in 1850 (St. Louis City Plan Commission 1969).

[11]There was considerable immigration into the Wisconsin part of the Upper Mississippi during the 1830s and 1840s, and its non-native population grew from 3,245 in 1830 to about 200,000 by 1847 (Barton

Amherstburg, opposite the Detroit shore of the St. Clair River, in the southwest (Burghardt 1993). As well, regular boat traffic connected Penetanguishene with Sault Ste. Marie (*Loc. Cit.*), and large numbers of American vessels regularly stopped at the Sault and passed through Mackinac Strait, immediately to the south, bound for the frontier communities of Lake Michigan[12]. By the 1840s the settlement frontier in both Canada and the U.S. had extended almost to the Petit Nord.

This combination of rapid settlement expansion from the east and improved transportation infrastructure stripped away the isolation of the Petit Nord, exposing it to more disease than ever before. The northwestward shift of the settlement frontier was especially significant as frontier communities acted as conduits through which many of the diseases of the urban pools spread to more remote peoples[13]. Even the smallest of towns was exposed to the diseases of civilisation at times, brought to them by the new arrivals from the east. On the frontier "Common diseases of childhood, such as measles, scarlet fever, whooping cough, chicken pox, and mumps, came as a matter of course..." (Harstad 1963: 209). Those natives who ventured into these communities risked

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1847: 55; Pritchett 1970: 249). Most of this growth occurred after 1842 (Barton 1847: 55).

[12]In August of 1843 the HBC's John Ballenden noted that "This has been rather a gay Summer at the Sault [Ste. Marie] - Steamboats every fortnight, and often weekly" (HBCA D.5/8: 412d). One of the steamboats brought 200 passengers to the town (*Loc. Cit.*), again a potential source of disease. James Hargrave had been stationed in the village by the NWC prior to the merger, and when he returned in 1851 he found it "changed by the advance of settlers" (Glazebrook 1938: xviii-xix). In 1854 Governor Simpson stated that: "This [Sault Ste. Marie] district may now be considered as within the limits of the "civilised world", the shores & islands being studded with growing villages & settlements while it is traversed in all directions by steamers..." (HBCA D.4/74: 449-450).

[13]George Catlin, an artist who travelled through the American west in the early 1830s, referred to it as the "*blasting frontier*" which, as it moved westward, destroyed all whom it passed over (Catlin 1973: 1: 60-61).

contracting *ACIs*, and carrying these diseases back to their homes (e.g. Harstad 1960b: 254; HBCA D.5/14: 138), while fur traders and others who visited the frontier each year, but worked beyond it, also risked spreading diseases to the native people (e.g. Taylor 1977: 59). Moreover, the larger frontier communities could support these crowd diseases until the season when communication with the aboriginal people was most likely. This meant that a disease introduced in the fall or winter, when contact with native people was often minimal, could still be passed on in the ensuing spring or summer.

The advances in transportation afforded by the construction of canals and the steamboat in themselves were also significant in carrying *ACIs* into the interior. Although Pyle (1969) has argued that the internal transportation network of the early 1830s was crude when compared to that of 1849 and 1866, based as it was on the use of steamboats on inland waterways, it nevertheless was a significant advancement over that which had preceded it. Two improvements afforded by this new transportation technology were particularly conducive to the spread of *ACIs*: the increased speed of travel and the larger number of passengers that could travel in a single vessel[14].

[14] Improvements in speed and passenger capacity have been cited in the emergence of new patterns of disease diffusion in other contexts (Haggett 1992: 395; Haggett 1993: 46-47; Haggett 1994: 102-103).

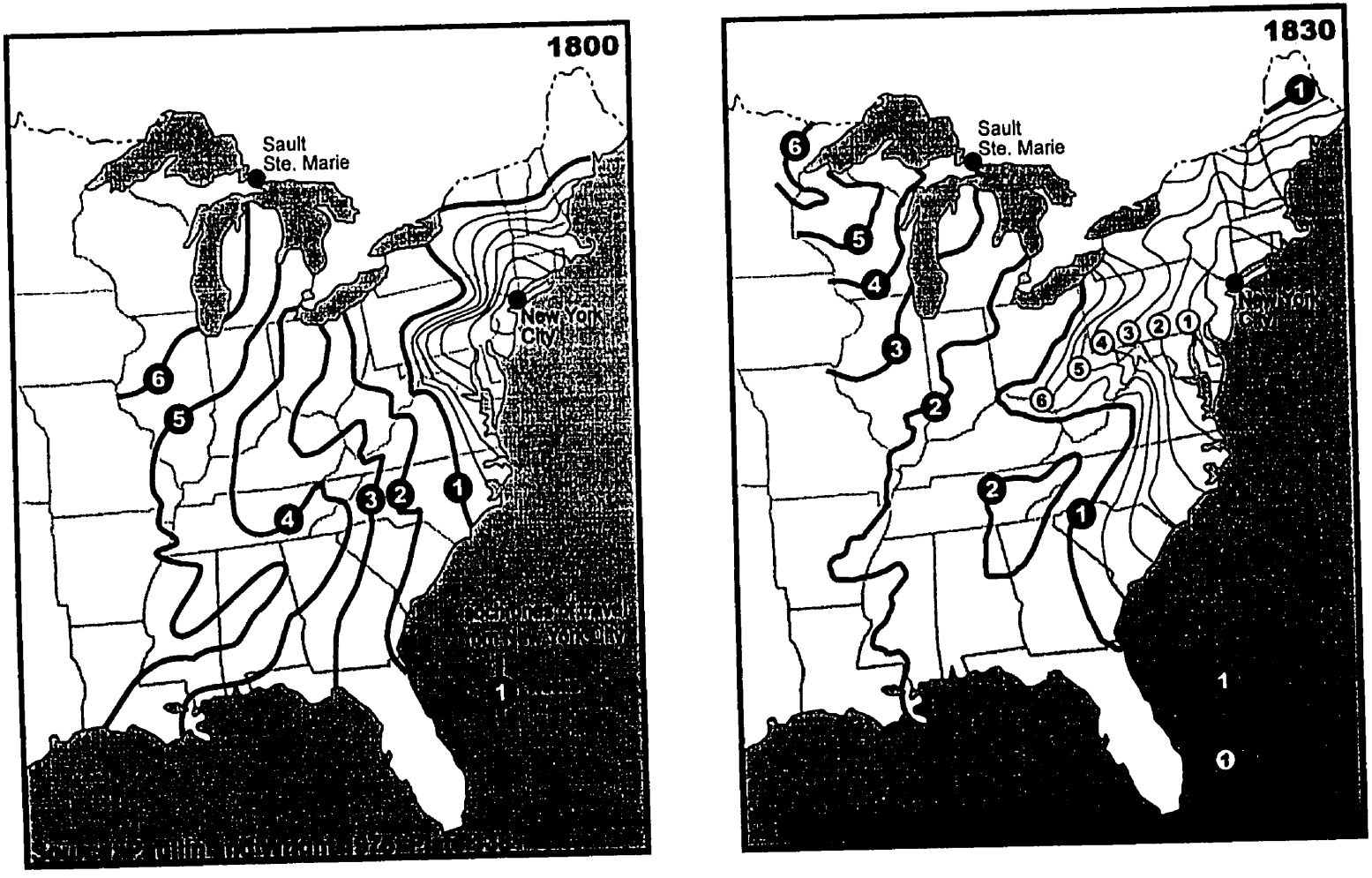

Figure 19: Travel times into the interior of North America from New York City, 1800 and 1830

The combination of the steamboat and canals made for a much quicker passage from the eastern cities. For example, the trip from New York City to Sault Ste. Marie that took six weeks in 1800 took only three in 1830 (Paullin and Wright 1975: Plate 138; Figure 19). The trip from Montreal could be done in even less time, in one case as little as ten days[15]. As such, the chain of infection required to maintain an *ACI* from the eastern cities was much shorter. Given the incubation and infective periods of some acute infectious diseases, an individual who was exposed to a disease in the east could now be infective after completing the journey to the threshold of the Petit Nord. Compounding this was the growth of the new interior settlements. With dozens of communities established along the major transport routes, it was not necessary for a single person, or even vessel, to transport a disease the entire way. Instead, an affliction might be introduced at a town by a passing ship, erupt into an epidemic, and then be passed to other ships to be carried farther into the interior. This meant that, despite being many weeks away from the eastern disease pools, the western flank of the Petit Nord was now at risk to the epidemics of eastern provenance.

Steamboats also offered improvements in size over most sailing vessels plying the interior waterways. Many steamboats were packed with immigrant families with children. Consequently, there was a tendency for these ships to carry acute infectious

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[15]In 1848 Sir John Richardson (1981: I: 51) passed from Montreal to Sault Ste. Marie by steamer in only about ten days. Almost a decade later, in 1857, Henry Youle Hind departed Toronto on July 23, passing through the new locks at Sault Ste. Marie on the 27th, before reaching Fort William on the 31st (Hind 1971: I: 9, 14, 75). By September 4 he was at the Red River Settlement, only forty days after leaving Toronto. In 1858 Hind returned to the Settlement by way of Detroit and Grand Portage and arrived after only 28 days (*Ibid.*: 75). By then, larger vessels could travel without interruption from Europe to Fort William (*Ibid.*: 14).

diseases, especially among the poorer classes (Hunter and Hunter 1949: 430-435). Larger numbers of passengers (and of susceptibles) meant that, in general, these diseases would remain viable for a longer journey. This, combined with the practice of abandoning victims along the way (and attempting to avoid quarantine) (e.g. *Loc. Cit.*) made these vessels ideal for the widespread diffusion of *ACIs* in the interior of North America.

An example of the capabilities of the transport and settlement infrastructures of the 1830s to facilitate the diffusion of *ACIs* into the interior may be found in the rapid and wide spread of the 1832 cholera epidemic. This epidemic was part of the second devastating cholera pandemic that began in India in 1826 and spread through much of Asia, and subsequently to Europe, North Africa and the Americas. It was brought to North America aboard crowded immigrant ships from Europe (Cliff and Haggett 1993: 4). This epidemic first appeared in some eastern Canadian and American cities in June, and during the summer spread into the interior of the continent from New York City and Montreal on ships crammed with immigrants, and on troop vessels (Godfrey 1968: 12, 14; Bilson 1975: 17; Pyle 1969: 61; Rosenberg 1962: 23, 25 fn. 19). The disease travelled along the major waterways, and spread by the St. Lawrence to the Great Lakes, and by the Hudson River, the Erie Canal, Lake Erie, the Ohio Canal and the Ohio River to the Mississippi (Bilson 1975: 18; Pyle 1969: 61, 64; Godfrey 1968: 14-15; Marks and Beatty 1976: 203). It then moved down the Mississippi to New Orleans before returning up the river to southern Minnesota and Wisconsin (Rosenberg 1962: 36, 37; Hunter and Hunter 1949: 431; Pyle 1969: 63). It also spread to northern Michigan, via the Great Lakes, and into northern Wisconsin and Illinois (Rosenberg 1962: 36; Hunter and Hunter

1949: 431). By the time it died out, this cholera epidemic had spared few Canadian or American communities, reaching to the very limits of the settlement frontier. While it did not reach the Petit Nord, its pathways reflected the routes and mechanisms that would carry other diseases from the urban disease pools into the interior.

Although cholera did not penetrate to the Northwest during this first North American pandemic, reaching only so far as Mackinac[16] (Marks and Beatty 1976: 204; HBCA B.105/a/17: 3d), news, accompanied by anticipation and fear, travelled quickly through the Northwest (e.g. Glazebrook 1938: 96, 102; HBCA B.129/a/17: 3d, 6d, 7, 9; B.149/a/16: 2d; B.4/a/7: 2d; PAM MG 7 D13 Microfilm reel 1 *Belleau Collection*: Bishop Panet to J.-N. Provencher, Quebec, 14 April, 1832). This advance wave of fear of the epidemic suggests that the fur traders, Indians and missionaries of the Northwest were well aware that they were at risk to the diseases arising in the urban east. This was especially the case with those in the Petit Nord, which lay only a short distance from the settlement frontier. Thus, in December of 1832, Donald Ross at Norway House wrote that "that awful calamity the Cholera is it seems raging with deadly violence in Canada & the States — it will reach us of course next Summer if not sooner — God help us all...." (Glazebrook 1938: 102). Ross was wrong, but his comments illustrate the degree to which the Petit Nord was becoming integrated into the external urban disease pools by

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[16]It was brought to Mackinac from Buffalo by the troop carrier *Sheldon Thompson*, and resulted in the death of almost one third of the troops stationed there (Marks and Beatty 1976: 204). One AFC employee died of the disease as he travelled from Mackinac towards Sault Ste. Marie, bound for Lake Superior (HBCA B.107/a/17: 3d). There was a quarantine at Sault Ste. Marie (*Loc. Cit.*), but more effective were the dire warnings travelling westward from that town (HBCA B.129/a/17: 3d). A bowel complaint afflicted the people at Pic in August of 1832 (HBCA B.162/a/6: 8, 9d , 11), but it lacked the severity and mortality associated with cholera. Cholera was again epidemic in 1834, and may have spread to the Mandan villages (Trimble 1988: 23), however once again it did not penetrate into the Petit Nord.

the early 1830s[17].

Extra-Regional Patterns of Diffusion

Most of the epidemics entering the Petit Nord during the period 1821-1845 followed one of two basic patterns of diffusion, both of which were tied into the expanding settlement frontier. Many came to the region from the east, via Lake Huron and Sault Ste. Marie, and were relatively localised in their spread. Others came from the southwest, and diffused much farther. The eastern pattern was relatively simple, with the main threat being the nearby American and Canadian frontier communities, as well as the frequent summertime passenger traffic that arrived at Sault Ste. Marie or passed through Mackinac Strait. The immediate cause of many of the epidemics of this period was the movement of non-natives and aboriginal people between Lake Superior and Sault Ste. Marie or Lake Huron. During the 1830s, George Keith explained the increased frequency of disease at Michipicoten by referring to Sault Ste. Marie, which he called "a hot-bed which often sends its pestilential effluvia hither[18]," (HBCA B.129/a/16: 18d). Other epidemics can be traced to Lake Huron. In fact, the area between Manitoulin Island and Sault Ste. Marie annually attracted many Indians from Lake Superior, often exposing

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[17]Similarly, in September Chief Factor Colin Robertson tried frantically to send word to the Red River Settlement where his family was, presumably in order to warn them of its approach (HBCA B.129/a/17: 6d-7, 9). At Fort Alexander, at the mouth of the Winnipeg River, a trader wrote in June of 1833 that: "Some of the Indians at Rapeds unwell I am learned - Should it be the Cholera gad helpe us in this country" (HBCA B.4/a/7: 2d).

[18]In his Michipicoten District Report for 1833-34, George Keith identified easy access to Upper Canada as an advantage of the District, and close proximity to the United States and Canada as a disadvantage (HBCA B.129/e/9: 2, 2d).

them to *ACIs*[19] (HBCA D.5/10: 219). Once carried back to the north shore of Lake Superior, however, none of these epidemics seems to have spread far inland. Instead, they remained among the people who frequented the vicinity of this lake, without being passed to the people who lived farther west or north.

The second basic pattern of diffusion into the Petit Nord saw epidemics enter the region from the southwest, thereafter spreading east to the Boundary Waters, and north towards York Factory. The key to this pattern was the Red River Settlement, which emerged during this period as a nexus for disease diffusion between the U.S. and the Canadian Northwest[20]. Its role in these epidemics was multi-faceted. On occasion it served as a temporary holding body, a sort of limited disease pool, sometimes hosting these diseases for months on end. With a population in the thousands, many of whom were born in the Northwest, the Settlement provided a ready supply of susceptibles for the

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[19]Perhaps the main threat to the Lake Superior Indians was the annual ceremony held by the British on Lake Huron, where they provided presents to loyal Indians (See below). Nevertheless, there were many reasons for the Lake Superior Indians to travel to this area, thereby exposing themselves to epidemic disease. American annuity payments of the period required Indian people to collect at one of several central offices, or agencies, thus increasing the risk of spreading disease farther afield. In 1848 J. Elliot Cabot noted that at Mackinac Island "In August and September (the time for distributing the "presents") there are generally several hundreds of them on the Island" (Cabot 1850: 23). Speaking of the period circa 1830, Tanner (1987: 130) noted that "During the summer, visiting Indians from the west and south often increased the population temporarily to around 3,000 [from 980] at "the Sault" and to 6,000 or more at Michilimackinac [from about 1,200]." These temporary communities grew at the same time of the year that the ships from the east were travelling to the Upper Great Lakes. Treaty negotiations were a significant health risk for the same reason (Dobyns 1992: 215).

[20] No detailed study of the epidemic history of the Red River Settlement has as yet been completed, and so the full extent of the *ACIs* is unknown. However, both Young (1988: 37) and Decker (1989: 145) have suggested that the establishment of this community was a factor in the introduction of *ACIs* during the nineteenth century. The outbreaks and epidemics within the settlement noted in the Appendix are based on a reading of only a portion of the relevant primary documents.

epidemics that passed through it[21]. Moreover, its social geography was also conducive to the temporary maintenance of *ACIs*. Rather than being a single community, Red River was formed of several spatially discrete, and ethnically distinct, communities organised around local churches or parishes that were established along the Red and the Assiniboine Rivers[22]. As such, and with no central core, the communication of infectious disease between these communities was often slow, prolonging the presence of disease in the colony.

The extended duration of these epidemics played an important role in the diffusion of *ACIs* throughout the Northwest, as it enabled these diseases to spread much farther than they might otherwise have done. The Red River Settlement supplied a large part of the seasonal labour force required to man the Northwest brigades. Every spring many of the men departed Red River for the north to serve on the transport brigades (Ens 1996: 43-46; Kaye, Moodie and Sprague 1993; Innis 1956: 309-310). On many occasions, *ACIs* that were epidemic in the area at the time of their departure were subsequently spread far beyond its limits with these brigades (Ray 1976: 156). In prolonging the stay of these diseases until the brigades left, the Red River Settlement greatly enhanced the possibility that an epidemic would continue to spread within the Northwest.

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[21] After the merger, the Red River Settlement grew quickly not only by natural increase, but also by an influx of métis and of former fur traders with their largely mixed-blood families from throughout the Northwest (Kaye 1967: 38, 42, 119; Pannekoek 1976: 83; Innis 1956: 288). By 1824 its population numbered about 2,000. By 1840 it had more than doubled to nearly 5,000 (Kaye 1967: 43, 119).

[22] By the 1830s, according to Ens (1989: 74), Red River was "an amalgam of small, largely métis, peasant communities of varying ethnic and religious orientations." At its peak, the Settlement extended as far as from Pembina, North Dakota, to Lake Winnipeg and from the forks to Portage la Prairie

Perhaps the most important role played by the Red River Settlement during these epidemics was that of gateway to the Northwest. Between 1821 and 1845 many of the major epidemics that spread widely through the Northwest entered the region through the Settlement. This was due to its far-reaching connections. During the first few years of its existence, a period when its inhabitants' connections were fairly limited, *ACIs* did not trouble the Red River Settlement on a frequent basis. Aside from the epidemics of 1819-20, it does not seem to have been subject to an epidemic *ACI* until well after the merger. It is not clear when this changed, but by the late 1820s these diseases had become far more common[23]. This corresponds to a period when its non-fur trade connections had become far more complex and the interests and economic activities of its people took many of them far away from Red River. Commercial ties between its agricultural citizens and the American frontier communities developed very early in the settlement's existence and, within a few years of the merger, there were occasional livestock drives from, and then to, the south (Kaye 1967: 90, 105, 217-218; Kaye 1981). By the 1840s the settlers were driving horses and cattle to St. Paul (Minnesota), with some regularity (Kaye 1967: 218), while the Red River métis were also making annual trading ventures to Pembina,

[23]Whooping cough returned to Red River in 1827, and influenza was epidemic early in the same year and the following January. During the winter of 1828-29 there was yet another epidemic, possibly of an ARD. Measles was said to have been brought from Red River to the Lac Seul area in the fall of 1828 (Appendix 2). By 1845 it was not uncommon for two or more afflictions to appear in the Red River Settlement in a given year, or even at the same time. In December of 1834 John Charles wrote to James Hargrave, stating that "the Colony generally speaking is not so healthy as of late years" (Glazebrook 1938: 148). In 1830 Alexander Ross wrote to Hargrave informing him that there was "No sickness in the Colony this year" (*Ibid.*: 60), seemingly a noteworthy occurrence.

the Upper Missouri, St. Paul and nearby Mendota[24] (Gluek 1965: 47; Ens 1996: 76; Pritchett 1970: 254).

The Red River Settlement was also a significant crossroads for Indian people from near and far. It was a source of seasonal employment for the Indians of the Winnipeg, Swan River and Cumberland districts, and attracted a steady flow of natives in search of trade, or to settle permanently (Ray 1988: 218-219; Peers 1994: 103). There was also a significant Cree and Ojibway population resident in the Red River area who, as part of their seasonal cycle, regularly travelled to Pembina, Turtle Mountain or the Upper Missouri to trade furs with the Americans (Peers 1994: 103, 126). In her study of the Ojibway of western Canada, Peers (1994: 123-124) characterised the Red River Settlement as a "centre for communication among Ojibwa in the entire Northwest and Boundary Waters region." People from Red Lake (Minnesota), Lake of the Woods and Rainy Lake periodically arrived to visit their relatives and renew ties along with the more established local groups, such as those from Lake Manitoba and Pembina (*Loc. Cit.*). As well, the Assiniboine, who by this time were living in southern Saskatchewan and the northern U.S., traded buffalo robes at the posts of the Upper Missouri (Ray 1988: 183-184), as well as trading with the aboriginal people of the area (Wood and Thiessen 1985: 8, fn. 9), but continued to visit Red River (e.g. HBCA B. D.5/17: 287). These broad connections, especially those with the U.S., put the Red River Settlement increasingly at risk of *ACIs*.

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[24]Peter Garrioch, one of the more commercially active of the Red River métis, had a very wide range of travel in the 1840s. He was based out of Red River and travelled to St. Paul, the Souris River, Pembina, and Forts Union and Clark on the Upper Missouri (PAM MG2 C38 Journal of Peter Garrioch 1843-47, Typescript Copy).

One source of disease for the Red River Settlement was the fur trading posts of the Upper Missouri that annually attracted Indians from the Northwest. Taylor (1977: 60) considered the era beginning with the founding of American fur posts on the Upper Missouri to be a distinct sub-phase in the epidemic history of the Northern Plains. He concluded that the establishment of American posts on the Upper Missouri in the 1830s increased contact between the natives of the region and the Euroamericans, thereby facilitating the transmission of epidemic disease to the Northern Plains[25]. Beginning in the mid to late 1820s there was a flurry of American post construction along the Upper Missouri after a temporary absence of a few years (Trimble 1988: 21; Wishart 1979: 47, 55). This new period of post construction began in 1825-26 when the Americans built a "strong fort" among the Mandan that attracted the Ojibway and Assiniboine from north of the border who normally traded at Red River[26] (HBCA B.235/a/7: 25). Other posts soon followed on the Missouri, including Fort Union in 1829 and Fort McKenzie in 1834[27] (Wishart 1979: 55), and travel to these posts was incorporated into the seasonal cycle of the Cree, Assiniboine and Ojibway (Ray 1976: 140). As the "outliers of the Euro-American settlement frontier" (Wishart 1979:87), it is to be expected that they were

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[25]Wishart (1979: 67) also considered the fur trade to be a major carrier of smallpox onto the Northern Plains during the 1790s and into the nineteenth century.

[26]In 1825-26, the Americans also sent men among the Brandon House, Pembina and Beaver Creek Indians (HBCA B.235/a/7: 25).

[27]Other American traders were operating to the east and north. In 1826 the Americans were in full force at the Grand Forks of the Red, to the south of the Red River Settlement (HBCA B.235/a/8: 13), and by September of 1827 there were American traders at nearby Lac Travers and Turtle Mountain as well (HBCA B.235/a/9: 7d). The AFC operated posts along the international frontier within the Boundary Waters until an arrangement negotiated with the HBC in 1832 saw them abandon those frontier posts in return for an annual payment (see HBCA B.105/a/17: 21).

sources of the diseases from the settled area with which they were in communication[28]. Indeed, Trimble (1985: 187) identified Fort Clark, a post built by the AFC among the Mandan, "as a repository for epidemic-disease hazards," diseases that were carried up the Missouri River by the traders themselves (Trimble 1989: 57). As many of these posts attracted Indians from British territory, the risk of infection was not limited to those in American territory, and especially the Red River Settlement with its developing connections to the south.

As elsewhere, the steamboat no doubt played a crucial role in the diffusion of *ACIs* upriver to the posts of the Upper Missouri. Following 1831, steam-powered vessels replaced the much slower keelboats for transporting goods and men up the Missouri River from St. Louis[29] (Wishart 1979: 83-86; St. Louis City Planning Commission 1969: 10). From an epidemiological perspective, this was a shift of considerable significance, as it brought more people into the area from the settlement frontier, and carried them there in less time than previously possible. In Sunder's (1965: 19) words "Steam unsealed the upper river: unsealed its isolation, kept the seal broken...." In doing so, the steamboat greatly lessened the time of the journey from St. Louis to the upper posts, in

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[28]When Prince Maximilian of Wied-Neuwied visited Fort Clark, located among the Mandan, in the winter of 1833-34, he commented that these Indians frequently suffered disorders, especially in the spring and fall, that catarrhs were very common among them, and that there were several distinct sicknesses affecting the Mandan during that winter (Thwaites 1906: XXIII: 236-237). Trimble (1988) noted that there were several infectious diseases among the village tribe of the Upper Missouri during the 1830s, but the lack of earlier documentation makes it impossible to judge if an increase had occurred over earlier periods.

[29]Steamboats had been employed on the Upper Missouri as early as the spring of 1819. However, the first efforts were largely unsuccessful (McLarty 1957; Wishart 1979: 86) and were not followed up for many years. Instead, regular traffic was instituted only after 1831 when the AFC's *Yellowstone* travelled to Fort Tecumseh, just above the mouth of the Teton River (Wishart 1979: 86).

effect compressing "time-distance[30]" (Wishart 1979: 86). This had the effect of limiting spatial friction for diseases travelling upriver, increasing the potential range of *ACIs* carried from the settlement frontier. In this respect, the use of the steamboat on the Missouri River resembled the adoption of the horse on the plains. However, unlike the horse, these large vessels often carried additional passengers on the upstream voyage, making the pool of susceptibles larger than it might otherwise have been[31].

Not all of the epidemics at the Red River Settlement came from St. Louis and the settlement frontier to the south. It is evident that the Settlement was also tied into a system of disease diffusion that included the Upper Great Lakes. Several of the post-merger epidemics that entered the Petit Nord via the Red River Settlement can be traced to the Lake Huron-Lake Superior area, similar in this respect to the 1819-20 epidemics[32]. These epidemics travelled westward through American territory, and their diffusion is indicative of the strong east-west connections that had developed to the south of the Boundary Waters, both by the natives and the fur traders. For instance, the fur trade of the Upper Mississippi region was supplied from Mackinac (Williams 1953: 466; Brown 1948: 301), unlike that of the Upper Missouri, and, every fall, brigades from the Lake Superior-Lake Huron area entered the country to the southwest of the Red River

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[30]In 1832, after the first successful steamboat voyage from St. Louis to Fort Union, Ramsey Crooks expressed this sentiment to Pierre Chouteau, telling the trader that "you have brought the Falls of the Missouri as near comparatively as the River Platte was in my younger days" (quoted in Wishart 1979: 86).

[31]Thus, the 1837 and 1856 smallpox epidemics were transmitted first to the Upper Missouri by fur company steamships (Dollar 1977; United States 1856: 86), and later to the Canadian Northwest.

[32]Such as whooping cough in 1825-27, Scarlet Fever and whooping cough in 1843 and measles in 1845-46.

Settlement. There were also assemblies in the Lake Superior and Lake Huron areas that attracted Indians from the area to the west of Lake Superior, as for annuity payments from both the Americans and the British[33]. These and other connections carried *ACIs* westward from the Lake Superior area through American territory, diseases that eventually found their way north to the Red River Settlement and east into the Petit Nord.

HBC Transport and the Spread of ACIs *within the Petit Nord*

Once within the Petit Nord, *ACIs* spread mainly through the movements of the aboriginal people and of the HBC men. The latter were often responsible for transporting these sicknesses long distances. This was an intensification of a pattern of diffusion that first appeared in the region during the latter part of the eighteenth century. Whereas there were only a few instances of the HBC brigades transporting *ACIs* prior to 1821, this became much more common during the post-merger period. In his study of the Western Interior during the period 1830-1850, Ray (1976: 157) identified the Company's brigades as the fundamental mechanism in the diffusion of several major epidemic diseases. Other evidence from this period indicates that the brigades spread many sicknesses before Ray's period of study, and many minor afflictions in addition to those he noted. In 1841 a Dr. Hodgkin, a critic of the HBC, summed up the role of the fur traders in the diffusion of *ACIs* in the Northwest, stating that:

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[33]Prior to 1843 the Sandy Lake Indians travelled to Drummond's Island to receive presents from the British (Williams 1953: 430), while the seasonal cycle of the Red Lake (Minnesota) Ojibway included trips to Red River, Lapointe and Sault Ste. Marie (Moodie and Kaye 1986: 180). The Robinson Treaties for the Lake Superior and Lake Huron Indians living in British territory were not signed until 1850.

> Parts of the country [the HBC territory] so traversed [by traders] must, at some seasons of the year, produce disease + thus occussion [*sic*] endemics [*sic*] of a more or less destructive character. The diseases of the whites, + more especially small-pox + measles will at times be introduced, + the havoc which they almost inevitably make amongst the people unprepared to meet or treat them, is awfully great. (HBCA D.5/6: 2d).

There were several factors leading to the expansion of the HBC's role in the transport of *ACIs*. One was the increased employment of métis and Indian men on the brigades following 1821[34]. Unlike most of the pre-merger HBC workers, these men had been born in the Northwest, where *ACIs* circulated with far less frequency than in the more densely populated areas. Consequently, they were more likely to be susceptible to the crowd diseases, in addition to the frequent ARDs and diarrhoeal diseases, than were the Company's European employees. The season of open water in the Northwest was short and the distances to be covered were often considerable, and so these brigades had to travel quickly. Within the brigades, any disease having a significant incubation period could travel substantial distances before the men were immobilised or ceased to be infective. Further, the hierarchical nature of the transport network facilitated the spread of *ACIs* by collecting and distributing disease at key posts. Furs from throughout the Northwest were gathered for shipment to market at major posts, requiring men from distant areas to travel to the same destination at about the same time before returning to their winter stations. *ACIs* introduced at one of these pivotal posts could be passed to the

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[34]Most of the brigades were manned by men of mixed Indian and white ancestry (Ray 1976: 141), but the contribution made by Indian men should not be underestimated. Local Indians were employed extensively between Norway House and York, as were men from Island Lake District (Innis 1956: 308), between Severn and York on the Severn brigade, as well as on the Albany Inland brigades from Lac Seul. Some Berens River Indians also worked on the freight brigades between Norway House and York Factory.

men of different brigades and could thereby be carried towards the limits of the transport network, and far from the initial source of infection. As a result, many epidemics reflected the movements of the HBC brigades, both in their timing and in their routes of diffusion.

Following the merger, the Petit Nord was served by three basic HBC transport systems, covering the western, southeastern and central parts of the region. Each played a distinct role in the diffusion of *ACIs*. The western portion of the Petit Nord was part of the Northern Department and of a far-flung transport system that, at its height, ran from just to the east of Rainy Lake to the western Arctic (Ray 1976: 141, 143). Each summer, canoe and boat brigades from throughout the Department arrived at either Norway House or York Factory, exchanging the returns of the previous outfit for trading goods for the following year[35]. Brigades from the Lac la Pluie, Winnipeg, and Island Districts reached these posts in June or July, arriving at about the same time as those from the Red River Settlement (Innis 1956: 290, 291; Ray 1976: 141; Figure 20). As well, the Berens River outpost was outfitted from Norway House[36], and Severn and its sometime outpost of Trout Lake were supplied directly from York Factory. Another brigade from the Red River Settlement was sent to Lac la Pluie during the spring. As a result, the posts of the western Petit Nord were tied into a vast, interconnected transport system that was readily

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[35]Norway House was established as a second depot for the brigades from the more distant part of the Northern Department (Innis 1956: 290-291). Freight brigades were employed to transport furs and goods between Norway House and York Factory.

[36]Berens River was initially part of Norway House District. It was transferred to Winnipeg District between 1825 and 1832, before returning to Norway House District until the twentieth century, with the exception of 1834-37 when it was part of Jack River District (HBCA File "Berens River").

suited to transporting *ACIs*. Of particular importance was the fact that the Red River Settlement was linked to this system, annually supplying a significant portion of the voyaging labour (Innis 1956: 310).

This relationship between the major transport routes and the spread of disease was not lost on the Indians of the Northwest. In 1823 the fur trader George Nelson interviewed an Indian man at Lac la Ronge, to the west of the Petit Nord, whose spirit guardian was "Sickness" personified. According to this man, in 1819 this spirit had warned him to stay away from the main travel routes and to "pitch off immediately into the woods," as it was by those "large waters" that he was set to travel among the Indians spreading measles (Brown and Brightman 1988: 49-50). By the 1830s, the aboriginal people knew generally to get away from the "large waters" if possible when epidemic diseases were abroad[37] (HBCA B.49/a/49: 35d-36).

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[37]In effect, to practise self-quarantine. In May of 1838 a large group of Indians collected near Cumberland House, preparing to leave the post (HBCA B.49/a/49: 35d). They were:

> off to pass the Summer in the thick woods a considerable distance from hence, so that they may not come in contact with the Saskatchewan Brigade on its way downwards. They are one + all dreadfully alarmed at the idea of catching the small Pox which has induced them to leave off hunting rather earlier than usual so as to give them time to get out of the way before the People of the infected part of the Country pass. (HBCA B.49/a/49: 35d-36)

A similar movement occurred during the influenza epidemic of 1835 as the Indians of the Northern Department of the HBC fled from the vicinity of the posts "with a view to escaping it" (HBCA D.4/103: 11).

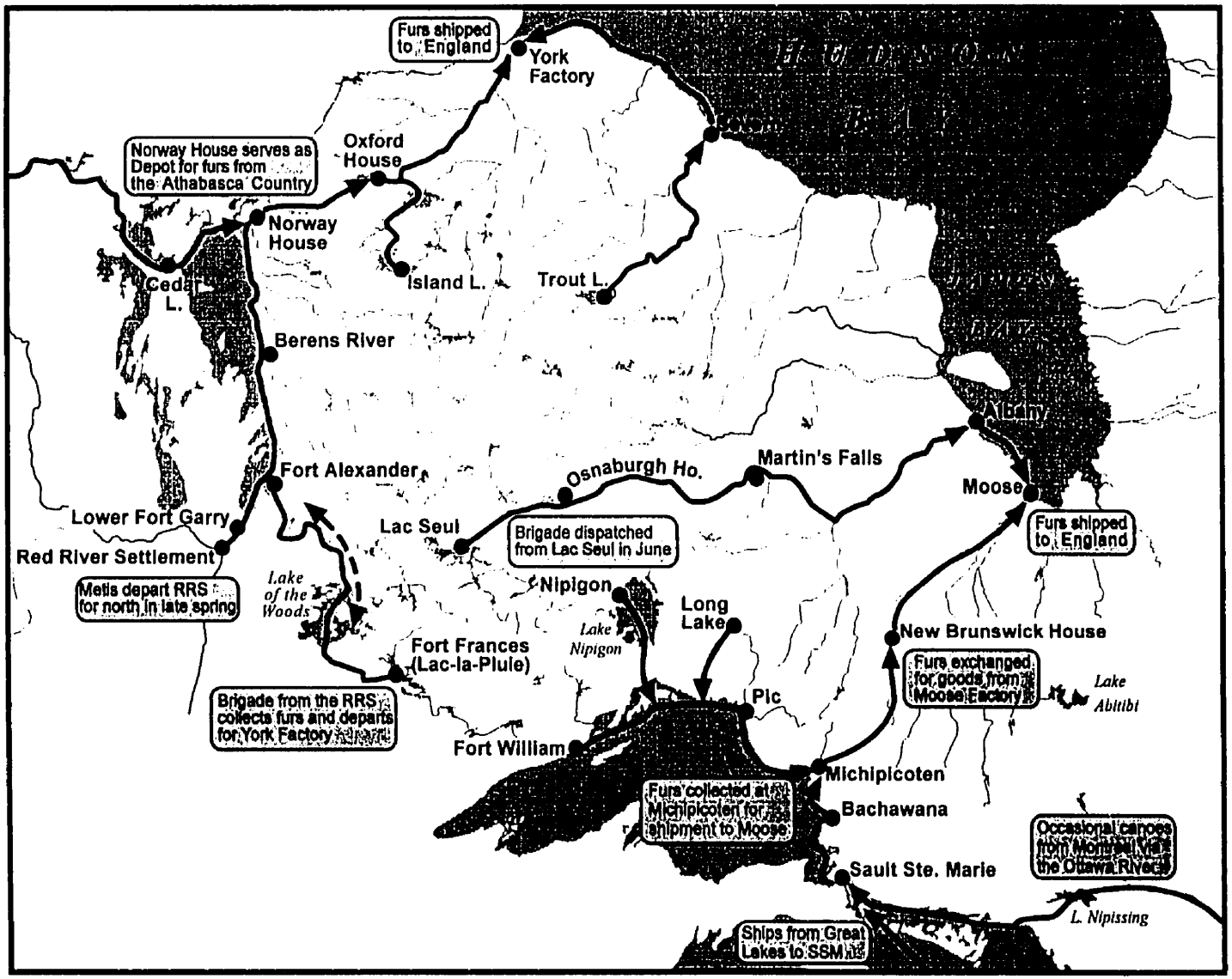

Figure 20: Post-merger HBC brigade routes in the Petit Nord

The southeastern part of the Petit Nord, including the Lake Superior District, was served by a separate transport system. Following 1821, the HBC established a supply route connecting the posts on the north side of Lake Superior: Sault Ste. Marie, Bachawana, Michipicoten, Pic, Long Lake, Nipigon and Fort William[38] (Weiler 1973: 5; Figure 20). Michipicoten was the entrepôt, and during the spring each post sent its returns there in order to be shipped to England, at the same time receiving trade goods and supplies for the coming year (*Ibid.*: 18). Other goods travelled from Canada and the United States to Michipicoten via Sault Ste. Marie (*Loc. Cit.*). Another set of boats and canoes worked in conjunction with the Lake Superior brigades to transport goods and furs between Lake Superior and James Bay[39] (Figure 20). Each June, brigades were dispatched from Michipicoten via the Michipicoten and New Brunswick Rivers to a transfer point at Long Portage (*Ibid.*: 20), although some canoes continued on to Moose Factory. Other crews headed southward from Moose with the ensuing year's trade goods, and the brigades exchanged cargoes at Long Portage before returning. In this case, connections with Sault Ste. Marie and the eastern settlement frontier, as well as its hierarchical structure, made this transport system another potent vehicle for spreading *ACIs*.

There is also evidence of considerable mobility on the part of the natives living

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[38]The post of Sturgeon Lake, to the west of Lake Nipigon, was initially supplied from Lake Superior, transferred to Albany Inland and supplied via Lac Seul, and later transferred back to Lake Superior District and outfitted from Lake Nipigon (HBCA B.107/a/6: 1).

along the north shore of Lake Superior at this time, which also was an important means of spreading epidemic disease. For instance, in 1827-28 Fort William was visited by Indians from Lake Nipigon, Sault Ste. Marie and Fond du Lac (HBCA B.231/a/7: 4, 7, 12, 23). In the 1830s, the Pic Indians traded with both the HBC and the AFC, and thus south of the international border (King 1836: I: 28). Such movements were common during the seasons when canoe traffic was possible. As we shall see, annual trips by the Lake Superior Indians to Lake Huron were also responsible for the transmission of *ACIs* to Lake Superior on many occasions.

The remainder of the Petit Nord was part of Albany Inland District that stretched from Albany to Lac Seul, and included the posts of Martin's Falls, Osnaburgh House and Lac Seul[40]. As part of the Southern Department, this transport system was detached from that of the Lake Superior-Moose Factory system, with the only connection being the limited coastal contact between Albany and Moose. This part of the Petit Nord was thus somewhat of a backwater, with only limited HBC traffic moving up and down the Albany River. Each June a few canoes left Lac Seul for Fort Albany. At Osnaburgh House this brigade picked up more men and furs, and continued to Albany before returning to Lac Seul by mid to late August. Perhaps because of the greater distance between posts, its regional isolation and the small numbers of men needed for the transport, fewer diseases

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[39]In 1837, a schooner, the *Whitefish*, was put into operation on Lake Superior to aid in transportation (Weiler 1973: 23). According to Arthur (1973: 69), the journey from Lake Superior to Hudson Bay required only sixteen days travel.

[40]Escabitchewan and Red Lake were maintained as outposts of Lac Seul for a few years after the merger, but these were closed when it became obvious that the HBC posts were competing with themselves (HBCA Files "Escabitchewan"; "Red Lake"). Cat Lake and Cedar Lake were other Lac Seul outposts that seem to have endured for a much longer period.

were spread by the Company through this system than others.

In addition to these transport systems, there was some travel by HBC employees between these systems. For example, sloops were used to connect some of the HBC's coastal factories, including Albany and Moose. As well, express canoes and a few transport canoes still ran occasionally between Canada and the Northwest, passing through Sault Ste. Marie and Lake Superior, and along the Boundary Waters. Other than these, however, contact between the posts located within the different systems was rare. Instead, the transmission of disease between them was more often the result of the movements of the aboriginal people, if it occurred at all. Such was the case at Lac Seul of Albany Inland District, located only a short distance from the Northern Department's posts in the Boundary Waters. Although there was almost no contact between the men stationed at these posts in these two districts, *ACIs* spread to Lac Seul from the Boundary Waters and the Red River Settlement on many occasions during this period. These afflictions were usually transported by the Lac Seul natives themselves, people who had substantial contact with those from the Boundary Waters during the spring and summer, as well as with the Red River Settlement.

The Decline of Smallpox

While most *ACIs* became more frequent visitors to the Petit Nord after 1821, one important disease did not. Although smallpox had appeared in the region several times

during the eighteenth century, it did not return between 1821 and 1845[41]. Its absence during this period is remarkable given that this feared disease appeared nearby on many occasions during this era. Although once rare in the interior of North America, it was far more common by the 1830s. Indeed, with the rapid westward advancement of the settlement frontier, this disease emerged as an almost constant threat. Nowhere was this more apparent than to the south and the east of the Petit Nord, where the settlement frontier approached the margins of the region[42]. Epidemics reached Detroit in 1813, 1829, 1831 and 1837 (Drews 1939: 762, 764, 766, 774). According to Harstad (1960b: 254), "By the late 1830s the white population was so widespread in Wisconsin that the Indian continually came into contact with them; thus there were unlimited opportunities for exposure to smallpox." Moreover, "Throughout the 1840s and 1850s, [smallpox] was present somewhere in the [Upper Mississippi] and few communities were spared an epidemic" (Harstad 1963: 218). The same was no doubt true of the adjacent country to the south of Lake Superior. Thus, this disease broke out in epidemic form in the area between Lake Superior and the Upper Mississippi in 1842, 1843, 1846, 1847, 1849 and 1851 (HBCA D.5/7: 229d; Harstad 1963: 219, 220; United States 1847: 91-92; United

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[41] Charles McKenzie at Lac Seul was told of a highly mortal visitation of smallpox at the Lake Nipigon post in the spring and summer of 1843, but this was merely an unfounded rumour (HBCA B.107/a/21: 16d; B.107/a/22: 1d). James Anderson, the Nipigon post manager, made no mention of the disease in his letters to George Simpson (HBCA D.5/8: 59, 321-321d), and the only severe epidemic at that post in 1843 was dysentery in the fall (HBCA D.5/10: 84). There is no other record of the disease in the Petit Nord prior to the end of the study period.

[42]More than likely, the disease was also a frequent visitor to the southwest of the region, near the Missouri River. For instance, an epidemic that spread from St. Louis and devastated many of the plains tribes in 1837-38 was preceded on the Lower Missouri by an outbreak in 1830 and an epidemic in 1831 (Trimble 1988: 18, 20). See also Innes (1993: Maps VII.8.1 and VII.8.2).

States 1848: 558; United States 1849: 111; United States 1851: 162, 168).

The absence of smallpox in the Petit Nord at the same time was due in large part to the introduction of vaccination by the HBC, which created an artificial barrier to the spread of this disease. Although the most feared of the Old World diseases, smallpox was the only one for which a simple and safe procedure, vaccination, could effectively prevent the spread of the disease. This followed the demonstration by Edward Jenner in 1796 that infection with cowpox, or what he termed vaccinia (ICD-10 B08.0), a comparatively mild disease, had the beneficial side effect of providing immunity to the far more deadly smallpox[43] (*DNB* 1921-22: X: 759; Dixon 1962: 261). Once publicised by Jenner, the practice spread widely in a short time. By the early nineteenth century it was common practice throughout Spanish America, including Mexico (Cooper 1965: 154-155; Lovell 1985: 161), and was met with much support in the United States (Dixon 1962: 276; *DNB* 1921-22: X: 760; Stearn and Stearn 1945: 56). Among the first beneficiaries of the introduction of vaccination to the New World were its aboriginal people[44].

The HBC was quick to consider the possibility of vaccinating the Indians of the

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[43]Prior to the late 1790s the only widely known preventative measure against smallpox was variolation, an ancient technique by which susceptible individuals are purposefully infected with smallpox using infective matter from recovering victims to induce a milder form of the disease. Variolation had several drawbacks including the fact that the patient actually contracted the disease, was infectious and could easily suffer the full effects of smallpox, including death, if not done properly (Stearn and Stearn 1945: 53).

[44]The Americans began to vaccinate the eastern Indians as early as 1801. However, plans to vaccinate the western peoples on the Lewis and Clark expedition and the Long expedition of 1820 were unsuccessful (Taylor 1982: 54; Stearn and Stearn 1945: 56-57, 62). Instead, large-scale vaccination of the Indians living in American territory west of Lake Superior had to wait until the 1830s.

Northwest. In 1811, Lord Selkirk, the founder of the Red River Settlement and a major shareholder in the Company, suggested using vaccinations as a tool to gain the support of the Red River Indians in preparation for the establishment of the colony. He noted that:

> A boon of immense consequence may be held out in the communication of the vaccine. On this point it may be necessary to proceed cautiously to avoid misapprehension, but time and patience will convince them, both of the value and the beneficence of the gift. Perhaps by judicious management on the part of the interpreters, they may be made to entertain very high ideas of the power of those who have such a command over nature[45]. (PAM MG 2 A1 Selkirk Papers, "Instructions to Miles Macdonell,"1811, p. 178)

Two years later, in October of 1813, Thomas Thomas vaccinated three of his children at York Factory with cowpox matter that had been supplied by the Company[46] (HBCA B.239/a/124: 73d). A vaccination program was implemented a few years later. This was during the measles epidemic in 1820, in response to rumours of smallpox spreading from the south. This saw the HBC people vaccinate many of the people of the Red River region (PAM MG 7 D13 Microfilm reel 1 *Belleau Collection*, Provencher to Dionne, 1 Sept. 1819; HBCA B.51/a/2: 13; HBCA A.10/2: 242; HBCA B.51/a/3: 5d). There were a few other small scale efforts by HBC employees over the next fifteen years, such as in the summer of 1824, when a few people were vaccinated at Cumberland House and at

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[45]The possibility of preventing smallpox would have come down strongly in the Company's, and the Colony's, favour, since Peguis and many of the Indians of the Red River area had entered the region in the wake of the devastating 1779-83 epidemic (Thompson 1973; PAM MG12 A1 Archibald Papers #780 "Draft Notes": 5).

[46]Until additional evidence emerges, this can be considered the first documented instance of vaccination in western Canada. Cf. Waldram, Herring and Young (1995: 123), who point to William Todd's efforts during the 1837 epidemic as the first example.

Norway House (Thistle 1986: 62; HBCA B.49/a/40: 8d-10), and in 1826, when Thomas Vincent had the Albany Indians and his family vaccinated using a supply obtained from a friend in Canada (HBCA B.3/e/8: 3; B.135/a/128: 30; B.3/a/130: 20, 20d, 21, 21d-23d). While these efforts would have hindered the spread of smallpox locally, there was no company-wide endeavour until the late 1830s, when another terrible smallpox epidemic struck the Northwest and spurred the Company to further action.

During the summer of 1837 smallpox spread up the Missouri and ravaged many of the peoples of the Northern Plains (Trimble 1988: 20-21; Dollar 1977; Ray 1975). The AFC steamboat, *St. Peter's*, transmitted the disease from St. Louis, as it made its stops on the long journey up the Missouri to Fort Union. The steamboat introduced the disease in succession at the Sioux agency, and at the AFC Forts, Pierre, Clark, and Union (Dollar 1977). A longboat carried it from Fort Union to Fort McKenzie (Ray 1975: 9). At each stop it was transmitted to the Indians and it was eventually carried back into the Northwest (Ray 1988: 188).

Although the Assiniboine, Blood, Blackfoot and Piegan suffered severely during this epidemic, its diffusion among the Cree and Ojibway was blocked by the efforts of the HBC men, especially William Todd at Fort Pelly, who vaccinated all of the Indians he met once reports reached him of the devastation on the Missouri River (Ray 1975; Ray 1988: 188-191). Todd also distributed vaccine to other posts, and to the Indians, and in so doing established a barrier to further diffusion. Elsewhere, such as at York Factory, Severn, Cumberland House and at Lac la Pluie, traders followed Todd's lead when word reached them of the devastation along the Saskatchewan through the winter packet

(HBCA B.198/a/77[a]: 33, 33d; B.49/a/49: 24, 24d, 25d, 28, 31; B.105/a/20: 15d, 16). At least partly due to these actions, and in part due to good fortune, the 1837-38 smallpox epidemic did not reach the Petit Nord[47].

This smallpox epidemic became the catalyst for an HBC program that attempted to vaccinate every native person within range of its posts in the region from James Bay to the Pacific, and from the Red River Settlement north to the Arctic. On June 1, 1838, the Governor and Committee in London sent a letter to Sir George Simpson informing him of their plan to prevent any further diffusion of the epidemic (HBCA D.5/5: 49-49d; see also A.6/2: 158d). Thus, they wrote:

> We now forward to each of the Factories Packets of vaccine matter, and we desire that it be distributed throughout the Country, and that the Gentlemen in charge of Posts exert their utmost influence among the different tribes to induce them to submit to inoculation which we have no doubt they will readily do if pains be taken to impress on their minds the great benefit they will derive therefrom. (HBCA D.5/5: 49-49d)

The mechanism of distribution was the Company's supply network and it was uniquely suited to the task. Every year, goods, men and letters were sent out from London or from Canada for the benefit of each post. The system was hierarchical, so instructions to the Factors, Traders and Clerks in charge of posts, and in this case vaccine as well, could be sent to a few main supply depots and from there to lesser posts and then to outposts. Part

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[47]As smallpox spread in the west in 1837-38, an entirely separate smallpox epidemic was among the Indians of southeastern Michigan (Schoolcraft 1978: 567, 575, 576, 578, 589). In reality the safekeeping of the people of the Petit Nord was as much due to good fortune as it was to the HBC efforts, as the cordon sanitaire could not have blocked transmission through the Red River Settlement or from the Ojibway and Sioux living south and southwest of the Petit Nord. The disease was among the Sioux living along the upper Minnesota and at Lac Traverse, only a short distance to the south of the Settlement (Neill 1883: 454). Nevertheless, there is no evidence that it spread to the Red River Settlement at this time.

was sent from Montreal to the posts of the Lake Superior District, but most was shipped to the Northwest via Hudson and James Bays[48]. While the American Government struggled to get doctors to vaccinate the Indians in 1832 (Unrau 1989), and were unable to proceed to the Upper Missouri, the HBC already had supply lines and personnel in place to perform what amounted to a relatively simple medical procedure. Indeed, the vaccine was frequently provided to the Indians themselves, with basic instructions, in order that they might perform the procedure on the people who did not come to the posts. The HBC men also benefited from a pre-existing relationship with the Indians, while the American doctors might be met by suspicion from traders and Indians alike. Although the full extent of these efforts is unknown, it is likely that the Company vaccinated most of the people of the Petit Nord, removing the fuel for future epidemics, at least until a sufficient non-immune population had been built up (HBCA B.3/a/145: 1; B.123/e/14: 4; B.3/b/63: 27d; B.155/a/50: 20; Appendix 3). Thereafter, the traders needed only to remain vigilant for the disease at critical locations, and to administer the vaccine to prevent the spread of the disease into the region. Thus, as more and more diseases entered the Petit Nord, smallpox, the most feared *ACI,* did not.

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[48]Thus, on the 30th of May 1838, Thomas McMurray at Pic post wrote that he had "inoculated the women and children of the Post with vaccine matter received from Montreal" (HBCA B.162/a/10: 19). In addition to the vaccine sent from Montreal and that sent by ship to Hudson Bay, another supply was sent by ship to the Pacific Northwest, along with an additional supply that was to be brought from across the Rockies the following summer (HBCA A.6/25: 10).

ACIs IN THE PETIT NORD: 1821-1845

1821-1830: The Early Transition

There was only limited *ACI* activity in this region during the first few years following 1821. Apart from an outbreak of influenza among the Indians trading at Norway House in the fall of 1822, there was no sickness of any consequence until the mid-point of the decade (HBCA B.154/a/10: 36; Appendix 2). This brief respite may have been due in part to the decline of the Montreal-Sault Ste. Marie-Fort William canoe route during the post-merger period, in favour of ship-based access from London to York Factory, thereby temporarily lessening the potential for diffusion from the east. Although the full extent by which the traditional voyageur brigades from Canada introduced *ACIs* to the Northwest is not known, it was probably considerable[49]. Within a few years, however, external influences combined to more than compensate for the decline in fur trade traffic along this route and, thereafter, *ACIs* appeared within the Petit Nord with greater frequency.

The first major epidemic of the post-merger era was whooping cough, which broke out in 1825. The disease was first noted at Drummond's Island on Lake Huron during the early part of the summer and diffused from there in the fall (HBCA B.124/e/3: 2d). There, on the margins of settlement, native people gathered from far afield to receive

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[49]Writing during the next decade when *ACIs* had become more common, long time NWC and later HBC trader, Charles McKenzie expressed surprise at the later increase in diseases in the Northwest, "although the communication with Canada is less than formerly" (HBCA B.107/a/14: 7d). McKenzie was the same man who witnessed the 1806 whooping cough epidemic among the Mandan, and brought measles to Lac Seul during the 1819-20 epidemic. He was stationed at Lac Seul by both concerns on and off from about 1807 to 1854. No doubt, he was well aware of the role played by the Canadian brigades in introducing *ACIs* to the Northwest.

gifts from the British government and, inadvertently, whooping cough. This annual gathering would be a crucial factor in the subsequent introduction of *ACIs* in the Petit Nord prior to 1846.

Each summer the British government distributed what were termed 'presents' to the Indians of the Upper Great Lakes and beyond[50]. Prior to 1829, presents were given out at Drummond's Island, just to the east of Sault Ste. Marie in Lake Huron. In 1829, the British ceded the island to the Americans and the event moved for one year to nearby St. Joseph's Island (Figure 21). Between 1830 and 1835 it was held at Penetanguishene and, after that, at Manitowaning on Manitoulin Island (Major 1934: 22; Wightman 1982: 22-23). Participants arrived from far afield, and in large numbers. Until 1844, this included many Indians from American territory[51]. Typically, these people came from the Upper Great Lakes, the Upper Mississippi and even farther west (Major 1934: 22), as well as, on at least one occasion, Hudson Bay (Cabot 1850: 128). In 1842, for instance, 6,000 people journeyed to Manitoulin Island from as far away as Lake of the Woods and Red River (Strachan 1844: 10-11), while in 1837 there were 3,700 "Ottawa, Chippewas, Pottowottomies, Winnebagos, and Menominees" present (Jameson 1838: II: 275). In 1820 there were 4,000 native people collected at Drummond's Island (Garry 1900: 109).

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[50]These were not annuities, the annual payment of treaty monies, but were gifts intended to maintain the support of the natives.

[51]It was not until 1843 that the British Indian Department finally announced that they would no longer offer presents to the Indians living in the United States (United States 1843: 426), since providing guns and other aid to people who had fought against the Americans was perceived by Washington as a hostile act on the part of the British.

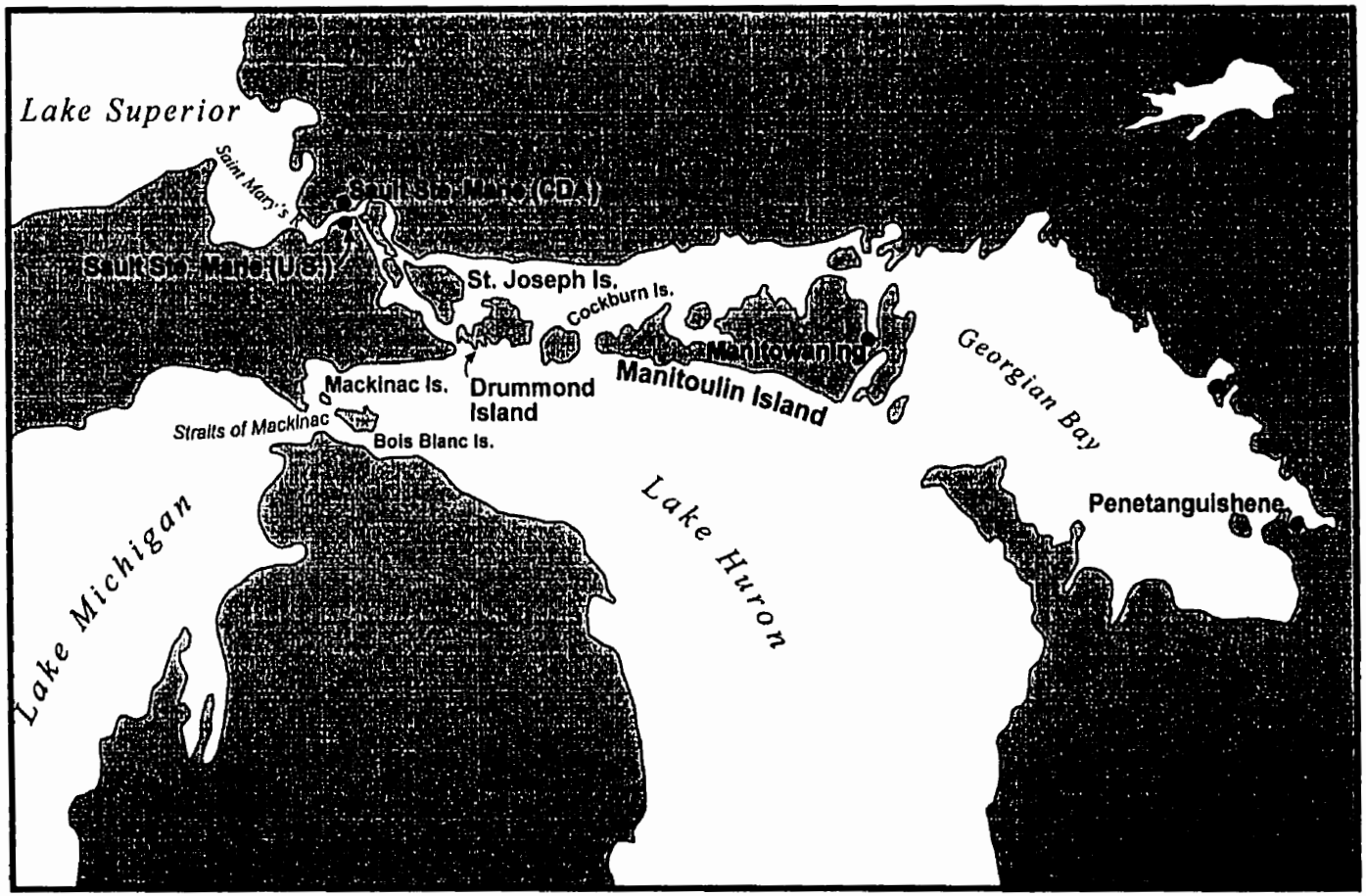

Figure 21: Locations of present-giving ceremonies in the Sault Ste. Marie Area

These gatherings were doubly threatening in terms of the diffusion of *ACIs*. First, in attracting participants from long distances, including white officials and adjuncts from the settled areas[52], the meeting both enabled the introduction of sickness to this part of Lake Huron, and presented the opportunity for epidemic diseases to spread far more widely than they might otherwise have done, as participants carried the infections back to their homes. Secondly, it led to the introduction of disease to Lake Superior by requiring the western Indians to travel through Sault Ste. Marie on their way to Lake Huron. By the end of the 1820s, Sault Ste. Marie had emerged as a seasonal focus of *ACIs*[53] (HBCA B.129/a/16: 18d).

The relationship between travel to Lake Huron, along which lay the settlement frontier, and the introduction of epidemic disease was early recognised by both the HBC traders and American government officials. For example, in 1828 the HBC post manager at Michipicoten, George Keith, dissuaded an Indian family from proceeding to Lake Huron to receive government presents, in part because in making the trip they would "expose themselves and families to some infectious disease..." (HBCA B.129/a/13: 1d). That same year, the American Indian Agent at Sault Ste. Marie, Henry Schoolcraft, advised an Indian named Red Devil (Miscomonetoes) not to travel to Drummond's Island for presents as "such visits were very injurious to them both in a moral and economical

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[52]Among those adjuncts were "petty traders", independent operators who travelled to the ceremony to trade with the Indians who gathered there (HBCA D.5/14: 138).

[53]Things seem to have changed from a decade earlier. Samuel Ashmun, junior, was a new recruit of the American Fur Company (AFC) at nearby Michilimackinac in July of 1818, and commented that "this is apparently the healthiest place in the Country in so much that it is a common saying that Mackinac is a cure for all diseases" ("With the American Fur Company": 1938: 14).

point of view [and] That they thereby neglected their hunting and gardens, contracted diseases, and never failed to indulge in the most immoderate use of strong drink" (Schoolcraft 1978: 295). The situation had worsened by the 1840s. In 1844, the HBC trader at Sault Ste. Marie, John Ballenden, wrote to Sir George Simpson regarding the annual journey, noting that the Indians of Lake Superior "Almost always on their return... carry with them the seeds of some of the maladies which are prevalent here and along Lake Huron during summer" (HBCA D.5/10: 219). In a letter to Simpson written the following year, he added that "on every occasion [they bring] with them some of the diseases more prevalent in the civilized world" (HBCA D.5/14: 138). The whooping cough epidemic of 1825,the first to penetrate the Petit Nord in the post-merger period. followed a pattern of disease diffusion that would be repeated in the Upper Great Lakes over the decades that followed.

The proceedings over for 1825, the participants at Drummond's Island dispersed, spreading whooping cough as they went. It was transmitted northward from Lake Huron to the fur trade area of both Flying Post and Matagami during the fall of 1825, by Indian people returning from the ceremony[54] (HBCA B.70/e/3: 2d; B.124/e/3: 2d; Figure 22). Thereafter, it spread among those who had not gone to Lake Huron. By the end of September this sickness had made its way to Moose Factory, and accompanied the local Indians as they departed for their winter hunts (HBCA B.135/a/128: 15; B.135/e/15: 1).

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[54]Unfortunately, there are no Lake Superior journals for this year, and so it is not known if the disease spread westward to Lake Superior with the Indians who had visited Lake Huron. Given the frequency of such diffusion to Lake Superior District from the east at this time, and Keith's and Schoolcraft's condemnation of Drummond's Island three years later, it is not unreasonable to speculate that whooping cough spread at least partly through this District in 1825.

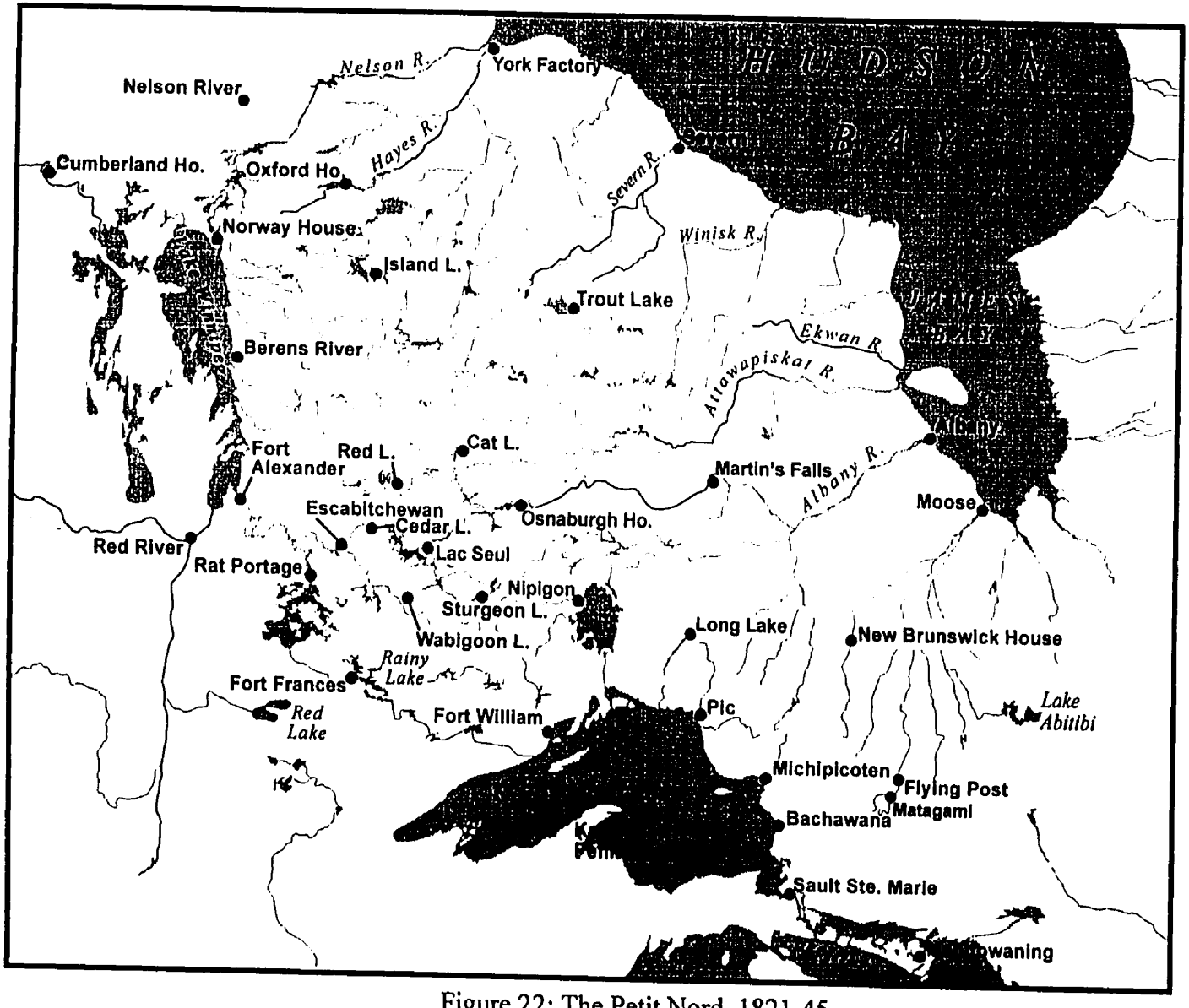

Figure 22: The Petit Nord, 1821-45

It broke out among the Indians and the mixed-blood children at Albany at the same time as it appeared at Moose. This was the first time that whooping cough had ever been identified in the Albany area. It continued to afflict the Albany natives into November, and its effects were felt into the following January (HBCA B.3/e/12: 2, 2d, 7d; B.3/a/130: 12). It was believed that the young suffered more than the aged, a pattern that is typical of the disease, which tends to be especially lethal for infants and young children in non-immunised populations (HBCA B.3/e/12: 7d; Benenson 1995: 347). It did not, however, spread far inland up the Albany River, as there is no mention of the disease at Martin's Falls.

Two years later, in 1827, whooping cough surfaced in the western part of the Petit Nord. Given its relative rarity in the Northwest at this time, it was almost certainly an extension of the 1825-26 epidemic. Here, then, was another example of the westward movement of *ACIs* from the Upper Great Lakes region to the west. Its diffusion in the west remains unclear due to a lack of evidence. Decker (1989: 102) believed that this epidemic originated in the Red River Settlement and spread northward with the brigades, but the evidence presented here indicates that this was not the case. Instead, it appeared to the north of Lake Winnipeg during the fall, before spreading to the south and west. Whooping cough broke out aboard some of the outbound boats from York Factory during the early part of the fall, spreading to Cumberland House and Îsle a la Crosse in September[55] (Decker 1989: 103). The disease spread to the men, women and children of

[55]The York Factory journal for this period stops in mid-September without mentioning the disease, and does not cover the fall of 1827. It may be that the disease did not strike these brigades at York Factory, but somewhere else to the north of Lake Winnipeg.

Oxford House, but it did not extend to the local Indian population (HBCA B.156/a/10: 9d). Once again it was especially hard on the children, some of whom were said to be bleeding from their ears and mouth, and to be choking such that they turned "black and senseless" (*Ibid.*: 10d).

Whooping cough reached the Red River Settlement in the fall of 1827, and was followed by an acute respiratory disease, most likely influenza (Decker 1989: 103; HBCA B.231/a/9: 13, 14). According to the Reverend David Jones, writing in November, "this dreadful scourge [whooping cough] was introduced here from Norway House last autumn..." (PAM CMS C.1/M.1: 323 Microfilm Reel A77, "Reverend D.T. Jones's Journal from October 15, 1826 to Oct. 15 1828"). The prior epidemic of 1819-20 probably meant that many of the adults and older children who had been in the settlement at that time were immune. Consequently, the 1827-28 epidemic was confined to the children, among whom there were several deaths, although influenza afflicted young and old alike (HBCA B.235/a/9: 13, 13d, 14). By January, the combination of whooping cough and influenza left some families with no one to care for the rest, but by the end of the month the Settlement was returning to health (*Ibid.*: 13, 14d). Whooping cough spread to Lake Manitoba, probably carried there by the Indians who visited during the Christmas holidays, and who left in haste when they learned of the epidemic (*Ibid.*: 13, 14). It did not, however, spread into the Boundary Waters to the east of Red River, and so this whooping cough epidemic was limited to the eastern and northwestern margins of

the Petit Nord[56].

Another familiar disease, measles, followed closely on the heels of whooping cough in the Petit Nord, and it was claimed that it had come from the Red River Settlement. The measles appeared first in the Lac Seul region. On April 4, 1829, Charles McKenzie noted the arrival of the Petit Francois's son and Assiniboine in his journal. He was told that "These Indians + the family to whom they belong were Sick all the fall + early part of the winter by that dreadful malady the Measles - which came from Red River" (HBCA B.107/a/7: 16). Others from the area had also been ill during the fall and early part of the winter, including those of Wabigoon Lake, to the south of Lac Seul, (*Ibid.*: 15d; 16d; Figure 22). Overall, however, this measles outbreak seems to have been limited to the southwestern part of the Petit Nord, not including the Lac la Pluie area[57]. The victims would have included only those who had been spared in 1819-20 or who had been born in the interim.

Most of the remaining outbreaks of *ACIs* in the Petit Nord during the period 1821 to 1830 consisted of ARDs (catarrhs and colds) and afflictions described as dysentery[58]

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[56]There is no obvious reason why the disease failed to spread farther from the Red River Settlement, but the late date of its appearance was probably a factor. Arriving as it did in the fall, after the HBC boats had made their rounds, it probably came too late to spread far. Those Indians from Lake Manitoba who contracted the disease during the holidays were essentially local, and the trip to the Red River was part of their seasonal rounds (see Peers 1994: 123-124). The more distant people who had visited the Settlement had already departed for their wintering grounds. Thus, by the time the disease broke out, a degree of winter isolation had already set in.

[57]Neither the Lac la Pluie nor the Osnaburgh House journals makes mention of epidemic sickness at this time.

[58]Some of these may have been extensions of influenza pandemics. Major epidemics struck the east in 1826 and 1830-32, and may have diffused into the Great Lakes area, or beyond (Heagerty 1928: I: 212-213).

(Appendix 2). These outbreaks were so frequent at this time and so dispersed that it is generally impossible to track them. However, several are suggestive of the broader role then being played by the HBC transport brigades. The 1825-27 whooping cough epidemic was one of several *ACIs* that were transported by Company crews in the Norway House-York Factory area during the 1820s. Minor sicknesses broke out at these posts at least during July of 1824, 1828, 1830 and 1831, coinciding with the arrival of the brigades from the interior[59] (HBCA B.239/a/132: 27d; B.154/a/16: 7; B.239/a/141: 72, 75, 81; B.154/a/19: 2d; B.154/a/20: 8d; B.154/a/22: 8d). This pattern of epidemic activity would be repeated on many occasions during the 1830s and 1840s, often with diseases of greater severity.

The same was true of the eastern Petit Nord, where evidence points to the brigades on several occasions. For example, an "epidemic catarrh" that struck at Moose and then Albany early in the summer of 1825 was probably brought to Hudson Bay by the canoes from Michipicoten (HBCA B.135/a/128: 6d, 7d, 9,12d; B.3/a/130: 10, 12). In 1827 epidemic colds were carried from Sault Ste. Marie to some of the Lake Superior posts by the Montreal canoes that were bound for the Northwest (HBCA B.162/a/1: 2, 2d, 3, 3d; B.129/a/12: 3 5, 10, 36; B.149/a/10: 1d, 2). The following year dysentery spread throughout much of the Lake Superior District with the HBC men, and was also carried by the brigade bound from Michipicoten to New Brunswick House in June (HBCA B.162/a/2: 4, 4d; B.149/a/11: 16, 18, 32; B.129/a/13: 2, 3d, 22). Although these disorders

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[59]By 1835 minor epidemics during the late spring or early summer, the period when the brigades were arriving, appear to have been a common occurrence at York Factory (HBCA D.4/103: 13d).

were relatively limited in their impact, nonetheless they are evidence that the post-merger HBC transport brigades were actively involved in spreading *ACIs* in the Petit Nord by the latter part of the 1820s.

1831-1840: The Transition Accelerates

The frequency of *ACIs* in the Petit Nord increased again during the 1830s, with several widespread epidemics. While cholera approached but did not enter the Petit Nord in 1832, whooping cough appeared twice in the western part of the region between 1832 and 1834[60] (Appendix 2). In the summer of 1832 Charles McKenzie at Lac Seul noted that the Indians were suffering from what he considered to be whooping cough. On August 21, 1832, he wrote that "The Indians & their Children have a very bad Hooping Cough this summer and some children fell a victim to this cough already and there is every fear that many more will be carried away by it" (HBCA B.107/a/11: 2). Ten days later the disease was universal among the people at the post (*Ibid.*: 3). Nothing further was entered regarding this outbreak, however. It seems possible that McKenzie misdiagnosed the disease or, perhaps more likely, that this was an outbreak of parapertussis, a similar disease, but of shorter duration and milder symptoms (Benenson 1995: 347-349). Two years later, McKenzie and his men were infected during a subsequent whooping cough epidemic, and it is unlikely that the two episodes were of the same disease, since whooping cough confers a lasting immunity. However, there is no

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[60]Decker (1989: 105-109) briefly addressed the 1834 whooping cough epidemic, considering the Red River Settlement to be its origin. Ray (1976: 156, fn. 56) deemed it to be a localised outbreak.

cross immunity between pertussis and parapertussis, and someone who is immune to the one can still be susceptible to the other (*Loc. Cit.*).

The path of the1834 whooping cough epidemic followed the route from the Upper Missouri to the Red River Settlement. Prince Maximilian of Wied-Neuwied spent the winter of 1833-34 at Fort Clark, located on the Upper Missouri among the Mandan. While there, he observed several epidemic diseases "which affected very many of the people; and some of the Whites, too, were severe sufferers" (Thwaites 1906: XXIII: 236-237). Two of the fatal sicknesses singled out were whooping cough, which killed many of the children, and "diarrhoea" (*Loc. Cit.*). The source of these sicknesses was not noted.

Both whooping cough and a bowel complaint appeared in the Red River Settlement during the following summer, and the former disease was especially fatal to the children[61] (PAM MG 7 D13 Microfilm reel 1 *Belleau Collection*: Bishop J.-N. Provencher to J. Signay, St. Boniface, 4 September, 1834; Same to Same, St. Boniface, 17 + 18 December, 1834; Glazebrook 1938: 160, 164, 166). There can be little doubt that both afflictions came from the Upper Missouri during the spring or early part of the summer. As in 1827, whooping cough had a prolonged stay, and it was only by December that the disease subsided (Glazebrook 1938: 148, 160). Unlike the earlier visitation, this epidemic spread from Red River to both the Boundary Waters and to the

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[61] Francis Butcher at Fort Chipewyan believed that the disease had "been making great ravages among both great and small in Red River" (Glazebrook 1938: 179). Father Provencher wrote that: "La coquelache a passé par ici cet été et a enlevé pluseurs enfants, tous ont été malades et sérieusement et longtemps."

north of Lake Winnipeg. In places it was joined by other acute infectious diseases.

Whooping cough appeared among the Indians of Lac la Pluie some time before mid-September (Decker 1989: 105). Thereafter, the disease manifested itself among some of the children and adults at the fort (HBCA B.105/a/19: 8d), and ultimately was responsible for twenty-nine deaths among the natives, including fifteen adults (HBCA B.105/e/10: 1). It seems to have been joined by influenza, as it was reported that this disease spread from Lac la Pluie to Fort William in December[62].

Influenza diffused from Lac la Pluie to Fort William on December 5, with the return of several men who had gone briefly to Lac la Pluie (HBCA B.231/a/14: 12, 14d). Thirteen days later, Donald McIntosh noted in the Fort William journal that "The most of the people + children in the Fort have been affected with a violent disease. We suppose it was brought here by the men who took Mr William Swanson to Lac la Pluie" (*Ibid.*: 15d). McIntosh referred to it as influenza and not whooping cough, and it is likely that it, too, was at Lac la Pluie, its symptoms masked by the latter disease[63]. In most cases this "fluenzy" manifested itself as a violent and painful cough accompanied in some cases by pains in the head, side and breast (*Ibid.*: 15d, 16), the last two probably the result of complications such as pneumonia (Benenson 1995: 245-246). By Christmas almost all of the HBC men at Fort William were labouring under its effects, and one had died, and the

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[62]There was also a later outbreak of colds at Lac la Pluie during the following spring (HBCA B.105/e/10: 1).

[63]Influenza may present clinically with symptoms that are similar to colds and other respiratory infections (Benenson 1995: 246), while the whooping inhalation associated with whooping cough is not always present.

disease had spread to the Indians who suffered severely[64] (HBCA B.231/a/14: 16, 16d, 17). By the end of January the men and the Indians had fully recovered (*Ibid.*: 18d). It also spread southward. In June of 1835 came word that the Spaniard's band, who hunted south of the border, had been attacked by the same affliction[65] (HBCA B.231/a/15: 2ad). While by no means extensive in its diffusion, this influenza epidemic is notable for the fact that it was perhaps the only instance during this period when an *ACI* diffused eastward in the Boundary Waters as far as Lake Superior.

Whooping cough spread to the country northward from Lac la Pluie during this epidemic, carried by the Indians. Early in 1835, McKenzie and the Lac Seul men, women and children were all ill with a "dreadful cough," brought from Lac la Pluie[66] (*Ibid.*:

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[64]Some of the Indians from Fort William visited Henry Schoolcraft at Michilimackinac in February and conversed with him on the subject of a tremendous meteor shower visible throughout much of North America on November 12, 1833. Schoolcraft wrote that:

> In consequence of the protracted mildness of the weather, Indians from Thunder Bay visited the office. They spoke of the meteoric phenomenon of November. I asked the leader of the party what he thought of it. He replied that it betokened evil to the Indian race - that sickness would visit them calamitously. (Schoolcraft 1978: 466)

This suggests that, by the mid-1830s, some of the aboriginal people of Lake Superior had great apprehensions about the possibility of devastating epidemics.

[65]A disease broke out among the people at Pic in February that was characterised by "violent spasms in the stomach & Bowels" and great pain (HBCA B.162/a/8: 18d, 19). Although there was no mention of respiratory symptoms, it may be that this was influenza or that another disease was present along the north shore of Lake Superior at this time (HBCA B.162/a/7: 19d, 26). In May, one man suffered from an eruptive disease which also caused his face to swell (*Ibid.*: 23d).

[66]In 1806 McKenzie worried about catching whooping cough while among the Mandan (Trimble 1985: 52), suggesting that he had had no prior exposure to the disease. He seems to have escaped at that time, but evidently he was not so lucky in 1843. The HBC men and Indians at Lac Seul suffered from at least two consecutive diseases that were different in nature, the latter being whooping cough. In October, McKenzie learned of an unidentified sickness among all of the Indians that caused sore eyes, sore throats and breaking out at the mouth and tongue, an affliction that had affected the people at the HBC post during the early fall, and continued thereafter among the children at the post (HBCA B.107/a/13: 11). These symptoms do not correspond to whooping cough.

14d). The same disease was carried to Sturgeon Lake around Christmas, by three Lac la Pluie Indians, and to Wabigoon Lake (Lac la Glaize), also from Lac la Pluie (*Ibid.*: 14d, 18d, 20). In this case the Lac la Pluie people may have been fleeing the effects of the epidemic, and thereby spread the disease to their northern neighbours. According to McKenzie, its effects were greatest at Lac la Pluie, but there were also mortalities at Sturgeon Lake (*Ibid.*: 18d, 20; see also B.211/a/7: 5d, 6, 7). There was some sickness among the Cat Lake Indians, located to the northwest of Osnaburgh and north of Lac Seul, during the winter or spring of 1834-35 (Figure 22). It is possible that they too had suffered from one of the diseases that struck Lac Seul (HBCA B.155/a/47: 2). However, there is no evidence that whooping cough spread beyond these areas in the southern part of the Petit Nord.

Whooping cough also passed from the Red River Settlement to the north of Lake Winnipeg, most likely with the York boats. It arrived at Norway House in August of 1834 and was widespread among the children of the post, although not exceedingly fatal (HBCA B.154/a/25: 49; Glazebrook 1938: 166). Statements made by the traders suggest that the disease was prevalent throughout this part of the country. During the fall, it spread among the Indians who traded at Norway House, Oxford House and at Nelson River, greatly affecting both fall hunts and the HBC transport (HBCA D.5/4: 98). However, it did not get to York Factory (Decker 1989: 108), or eastward into the interior of the Petit Nord. Although there appears to have been some epidemic sickness at York in October of 1834, there is no evidence that it was whooping cough (HBCA B.239/a/148: 18bd). At Severn, only an "inward complaint" afflicted the natives during

the ensuing winter (HBCA B.198/a/71: 24).

A much more destructive strain of influenza than had appeared in the post-merger period, probably of Type A influenza (Decker 1989: 112), was epidemic in the Northwest in 1835. Ray (1976: 145) concluded that it originated in the York, Island, Nelson River and Norway districts. Once again, however, it was the Red River Settlement that suffered first, although it is not known from whence the disease reached the Settlement. CMS accounts indicate that the disease was already epidemic at Red River by the second week of June, prior to the departure of the people for Norway House. On June 12 the Reverend David Jones wrote that "There is an influenza becoming very prevalent, + which I fear will prove very fatal to infants + aged invalids; it exceeds in malignity any epidemic I have witnessed in the country" (PAM CMS C.1/M.2: 143 Microfilm reel A77, "Rev. D.T. Jones' Journal, May 20/35 to July 20/36"; See also Tucker: 1858: 75). No age group was safe from the disease and in some cases entire families were incapacitated at the same time (*Ibid.*: 113 Microfilm reel A77, "Rev. W. Cockran's Journal, Aug. 14/34 to Aug 2/35"). Reverend William Cockran identified many severe symptoms which were generally consistent among the victims, and these included: sore throats, excruciating pain in the chest, weakness and pain in the limbs, violent headache and earache, discharge of pus from the ears, deafness, delirium, inflammation of the eyes, intermitting fever, severe cough and, in some, expectoration of blood (*Loc. Cit.*). The disease subsided only with the arrival of winter, and left many dead in its wake (Glazebrook 1938: 207; PAM MG2 A6: 101). By that time it had spread northward with the HBC boat brigades. Once within the HBC transport system this epidemic spread extremely widely, reaching as far

as Fort Simpson, on the Mackenzie River[67] (Ray 1976: 144).

People from Red River began reaching Norway House early in June, and the disease first manifested itself among the post's men some time prior to the 22nd (HBCA B.154/a/26: 3, 5, 6; Ray 1976: 142-143). According to Donald Ross, the trader in charge of Norway House, this severe disease arrived at the post with the return of the men who had attended the Northern Department council at Fort Garry (HBCA B.154/e/8: 2). When the explorer George Back reached the post on the 24th he found many people suffering from this disease (Back 1836: 466). In fact, influenza affected all at Norway House, including Indians, and seemingly all of the brigades that passed through to York Factory (HBCA B.154/a/26: 8, 9, 10; King 1836: II: 219-220). On July 9, word reached the Red River Settlement that the epidemic had spread throughout the country about Norway House, and that "The voyageurs were every where labouring under sickness" (PAM CMS C.1/M.2: 144 Microfilm reel A77, "Rev. D.T. Jones' Journal, May 20/35 to July 20/36"; see also B.154/e/8: 2). Ross suggested that influenza made a second, more violent, run through the post in October, and that the symptoms included "violent sickness, shivering, excessive debility... generally attended with fever" (HBCA B.154/a/26: 28, 31). The second run was probably among new people who had come to the post, as Ross told James Hargrave on July 18 that "most of the people who were here from the commencement [of the sickness], are now nearly recovered, but fresh arrivals afford new subjects for it daily" (Glazebrook 1938: 192).

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[67]McCormack (1996) identified the extremely severe effects of this influenza epidemic as it struck the aboriginal people of the Fort Chipewyan area.

The 1835 influenza epidemic spread to Oxford House as well. It was reported at nearby Knee Lake, and the Swan River and English River brigades were forced to stop near the post due to the effects of the disease among their crews (HBCA B.239/a/48: 61d, 66). In addition, some of the Oxford Indians arrived at Severn in August and, when asked why they had abandoned their post, replied that "their friends and relations had died this Summer and that they would no more return thither on that account" (HBCA B.198/a/73: 9). At about this time, Richard King, an Arctic explorer who was then at Cumberland House, learned that 13 people had died at this post (King 1836: II: 219-220).

At York Factory, outbreaks of bowel and lung complaints and other unconnected sicknesses preceded influenza (Decker 1989: 112; HBCA B.239/a/148: 40ad, 41d, 44d, 48). It appears to have arrived on June 19 with five Red River boats (HBCA B.239/a/148: 58; Ray 1976: 142-143). This influenza epidemic was very quick to spread among the men, women and children of the fort, the brigades and the Indians of the area, and it raged there until mid-August (HBCA B.239/a/148: 59d, 60d, 66, 66d). Initially, Governor George Simpson believed it to be just another spring sickness at York Factory, but by early July it was apparent that it was much worse (HBCA D.4/103: 13d). Richard King was informed that 16 people had died at York (King 1836: II: 219-220), while fourteen of the Nelson River Indians died during the epidemic (HBCA B.154/e/9: 1). There was also much sickness in the Berens River area during the fall and winter due to influenza, but few deaths (*Loc. Cit.*). However, it did not reach Severn with its northern diffusion but instead was confined to the corridors leading from Lake Winnipeg to York Factory (Ray 1976: 144; Figure 22).

The 1835 influenza epidemic also spread eastward from the Red River Settlement into the southern part of the Petit Nord. Unfortunately, there is no journal or district report for Lac la Pluie from the period following June of 1835, and so it is unknown what was happening there. However, there is evidence of the disease at Lac Seul. Charles McKenzie was absent from Lac Seul between June 11 and August 10 and it was only on his return that he learned of the presence of influenza among the Indians of the post (HBCA B.107/a/14: page before 1). During his absence all of "the natives were labouring under that cruel disease the Influenza..." including those to the westward (*Loc. Cit.*; *Ibid.*: 1). The effects of the sickness stayed with them to the fall, but none died at Lac Seul, unlike the neighbouring Indians[68], several of whom expired (*Ibid.*: page before 1). When McKenzie and his brigade returned from Albany, they too contracted influenza. Each man was forced by sickness to remain in bed for several days, but they began exhibiting improvement after three or four (*Ibid.*: 1). In some cases, the Indians were unable to harvest their wild rice owing to sickness, and the lack of food no doubt contributed to their infirmity (*Ibid.*: 2, 2d). The disease did not spread eastward to Osnaburgh and Sturgeon Lake (Figure 22).

At Lac Seul, influenza was followed in December of 1835 by an outbreak of chickenpox, an *ACI* that had never before been identified in the records of the Petit Nord. McKenzie wrote that: "about the 20th a very disagreeable disease broke out among the women + children first + afterwards some of the men caught it. It seems to be the

[68]McKenzie did not identify which neighbouring Indians he was referring to, but this statement is evidence of the varying course of an epidemic among different native groups.

Chicken-pox or something like it..." (HBCA B.107/a/14: 6). The trader described it as a mild ailment that produced small pustules from the soles of the feet to the top of the head, but no deaths, even of a baby that was born with the disease[69] (*Loc. Cit.*). It lasted only a short time, and was gone some time in January (*Ibid.*: 6d). These symptoms are consistent with chickenpox.

Chickenpox, or varicella (ICD-10 B01), is an acute, highly contagious disease caused by the *Varicella zoster* virus. It is spread through droplet emission or through contact with fluid from the blisters, and subclinical, or inapparent, infections are common. A two to three week incubation period is usually followed by a slight fever and a widespread skin eruption that may extend to the entire body. The communicable period lasts about two weeks. The morbidity rate of chickenpox is very high although it is not quite as infectious as measles (Hope Simpson 1952: 553). Expected mortality rates are low, however (Benenson 1995: 87-89).

Although a crowd disease, chickenpox is somewhat atypical. Upon the termination of the illness the individual is immune to further infection. Nevertheless, the virus may lie dormant for a period of time, up to several years, before reactivating as herpes zoster, or shingles, a less communicable disease that can nevertheless be passed on to a susceptible individual, causing chickenpox (Benenson 1995: 89; Miller and Keane 1983: 218; Ramenofsky 1987: 146-148). Potentially, an epidemic could be started by a latent carrier who develops herpes zoster, without importing the disease from an urban area. Researchers have suggested that the critical community size for endemicity of this

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[69]Congenital infections with chickenpox do occur (Benenson 1995: 89).

disease may number less than 1,000 (Black 1966: 210; Hope-Simpson 1954). Shingles is very uncommon, however, and its peak incidence is after the age of 50 (Kampmeir 1993: 1092). There is no evidence of the disease in the Petit Nord prior to this, and it does not seem to have taken hold in the region prior to 1846. Conversely, the relatively high frequency of subclinical infections means that someone who does not appear to be sick with chickenpox could also introduce the disease into a population.

The presence of chickenpox at Lac Seul is further evidence of the changing nature of *ACIs* in the Petit Nord. Aside from the possibility of infection from someone suffering from herpes zoster, the virus is not among the first rank in terms of diffusion ability (Ramenofsky 1987: 167). It appears to have been a comparative latecomer wherever it was introduced. In the Pacific Northwest, for instance, the first recorded instance of the disease occurred in 1840, long after smallpox, whooping cough and possibly measles had appeared (Boyd in press; NAC MG 19 A8, Volume 8 (Part III) — David Thompson Papers: 278, 279). It is therefore significant that McKenzie felt it necessary to ask "but what would bring such a disease to this out of the way place?,"even though he had seen epidemics of childhood diseases such as measles and whooping cough before (HBCA B.107/a/14: 6). Unfortunately, its source is not known. The Osnaburgh, Sturgeon Lake and Fort William journals make no mention of the disease. There is no journal for this period from Lac la Pluie, and so perhaps it came from the south.

In 1837 yet another influenza epidemic spread from the Red River Settlement through the Northern Department, one that was distinct from that of 1835. The pattern of diffusion was, however, remarkably similar. On June 13, about the period when the

brigades were departing for Norway House, the Reverend William Cockran noted in his journal that he had come down with influenza. He added that "We have got a species of Influenza among us again, that threatens to be fatal to the infirm + young" (PAM CMS C.1/M.2 Microfilm reel A85, "Journal of Rev. M^{r} Cockran Aug/36 to Aug/37"). The missionary was fairly clear as to its origins. The disease had "passed through the Indians of the Plains from the United States..." (*Loc. Cit.*). Whether, as is most likely, it was brought back from a trip to one of the American posts, or from another means is not known. However, it was most probably connected to an influenza pandemic that prevailed generally in Canada in 1836-37 (Heagerty 1928: I: 213). This epidemic furnishes an important clue as to why *ACIs* had become so frequent in the post-merger Red River Settlement.

There can be no doubt that this epidemic was a type that was entirely distinct from that of 1835. All were susceptible within the Red River Settlement regardless of age or prior exposure, and the 1837 epidemic proved as general as that of 1835, if not as severe (PAM CMS C.1/M.2 Microfilm reel A85, "Journal of Rev. M^{r} Cockran Aug/36 to Aug/37": June 21, July 2; PAM CMS C.1 Microfilm reel A84, Cockran to the Secretaries of the CMS, Grand Rapids, Red River Settlement, 2 August, 1837). By July, it had left Red River with the brigades bound for Norway House and York Factory.

Influenza reached Norway House early in July, and broke out almost immediately among the men, women and children (HBCA B.154/a/29: 11). It does not appear to have been as severe as the previous epidemic, as no deaths were mentioned. Subsequently, the disease spread towards York Factory. The Norway House brigade arrived at Oxford

House on July 12, suffering from influenza, and two days later the Nelson House boats arrived, also in a sickly state (HBCA B.156/a/17: 7d, 8). By the 15th, several of the Indian women and children about Oxford House were ailing, as were the HBC men (*Ibid.*: 8, 9). The word from York Factory was more melancholy, as William Sinclair arrived at Oxford House August 9 on his inward journey, and informed the Oxford House people that "numbers there being laid up with the Influenza four of which have already paid the debt of nature all Indians" (*Ibid.*: 11d). There is no York Factory journal for the period, but it would appear from this that the disease was at least moderately severe. It is unclear whether this epidemic spread eastward towards Severn. Although there was a report of sickness by some of the Indians trading at that post in May of 1838, influenza was not identified (HBCA B.198/a/77[a]: 28).

Unlike the epidemic of 1835, influenza did not spread eastward into the Boundary Waters at this time, nor did it penetrate to the Albany River system. There is no significant sickness in the Lac la Pluie journal, beyond news of the smallpox epidemic on the plains (HBCA B.105/a/20: 15d). A disease unlike influenza that could not be identified by the Lac Seul people broke out among the men in the fall of 1837 (HBCA B.107/a/16: 13). This seems to have been the same disease that was mentioned by Charles McKenzie in December as attacking the men, women and children, one that was responsible for a single death (*Ibid.*: 17d). The disease was peculiar, and the description of one of the victims included a bloated and scalded tongue, and "tumours both in + outside..." of his head (*Loc. Cit.*). As at Lac la Pluie, no suspicious sickness appeared at

Osnaburgh, Martin's Falls or Albany[70].

The diseases circulating within the Lake Superior District at this time appear to have been separate from those in the western part of the Petit Nord. Influenza again appeared at Fort William in epidemic form, breaking out during the summer of 1836, and seems to have been brought there by an Indian guide from the east (HBCA B.231/a/16: 2^b, 4d, 6). It was not very severe but two people, a woman and a child, are known to have died (*Ibid.*: 4d, 6). The following summer, beginning in June, another disease was epidemic along the southwestern tip of Lake Superior, and it may have come from the south shore. On June 5, the Spaniard and stepson arrived complaining of sickness (HBCA B.231/a/17: 1d). These men hunted south of the border, towards Fond du Lac, but traded at Fort William. By June 29, several of the women and children at the HBC post had contracted the disease, which was described as "something similar to the Meazles, but not so dangerous" (*Ibid.*: 3d). Small red spots such as those caused by measles were said to break out over the entire body, and the face became swelled (*Ibid.*: 6). Many of the children and some of the adults began to fall ill of the sickness, and most recovered in short order. However, some of the Indian men were still sick in early August (*Ibid.*: 3d, 4, 4d, 6). Reports from Grand Portage indicated that the same sickness was present among the Indians there (*Ibid.*: 5). There is no evidence that the disease spread north of the lake, although some of the Pic Indians complained of sickness in the spring of 1838 (HBCA B.162/a/10: 17).

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[70]One of the men and three of the Indians complained of sickness at Albany in February-March of 1837, after the dispatches arrived from the interior, but in all likelihood this was a minor outbreak of some ARD (HBCA B.3/a/141: 20, 21, 23d).

Although it is possible that this was another outbreak of measles, it was more likely German measles, or Rubella (ICD-10 B06). Rubella is a relatively mild viral *ACI* that can produce a rash that is very similar to that of measles (Benenson 1995: 405). It can be spread by nasal secretions through droplet spread or by direct contact with an infective person. The incubation period is lengthy, lasting between fourteen and twenty-three days, and there is usually a very good chance of recovery if the health of the patient is not already compromised. Recovery from Rubella generally yields a lasting immunity to subsequent reinfection, and so it becomes a disease of childhood in more densely populated areas (Ibid.: 407). There is no evidence for the presence of this disease in the Petit Nord prior to 1837.

There were no major epidemics between 1838 and 1840. Nevertheless, there was a good deal of sickness during these three years, and almost every post journal described what might have been an *ACI* (Appendix 2). Due to a dearth of post journals from this period, it is impossible to discern if these afflictions were part of broader epidemics. Of some significance is yet another influenza epidemic in 1839, this time afflicting the HBC people at Albany and the Indians from the surrounding country between late August and mid-October. This epidemic subsequently spread up the Albany River to Martin's Falls and beyond, towards Osnaburgh House[71] (HBCA B.3/a/145: 5, 6d, 7; B.123/a/41: 11d,

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[71]There was only one fatality reported at Albany (HBCA B.3/a/145: 6d). At Martin's Falls, George Barnston noted that:

> A severe cold, or Influenza has seized upon every one, and so violent is it for the first few days that confinement to the house becomes necessary. The children bled at the nose, the fauces [faces] are highly inflamed, and a deep hollow cough shakes the whole frame. (HBCA B.123/a/41: 11d)

15). It was preceded at Martin's Falls by a severe infection of colds that was believed to have been brought by an HBC boat from Albany (HBCA B.123/a/41: 1, 1d, 2). Also suggestive of more widespread disease was the outbreak of a cholera-like illness among the children at Norway House in March of 1840, which had been prevalent earlier on at the Red River Settlement (HBCA B.154/a/33: 31). Finally, there were outbreaks of colds and coughs among the men at York Factory in August of 1840, likely brought from the interior, and severe colds among the Indians to the west of Lac Seul in September (HBCA B.239/a/152: 39d; B.107/a/19: 3d). These, too, may have been connected.

1841-1845: The Transition Complete

The record of *ACIs* during 1841-45 is extensive, despite the fact that many of the HBC posts have not left journals from this period[72] (Appendix 2). Of those sicknesses noted during the first year, two stand out. The first epidemic of this period occurred in the Lake Superior district, and affected both natives and non-natives from Sault Ste. Marie to at least Long Lake, causing several deaths at Long Lake, Sault Ste. Marie and Bachawana (HBCA B.162/a/11: 19, 22, 28d; Figure 22). The only descriptions of the disease come from Michipicoten, where it was described as a severe cold, "joined to some other painful complaint" (HBCA B.129/a/22: 19). There, it flared up among the men late in February of 1841, only a few days after the arrival of two men from Sault Ste. Marie, and lasted into the second half of March (*Ibid.*: 19, 19d). Once again, an acute infectious disease

[72]For example, none of the Lake Nipigon, Long Lake, Fort William or Lac la Pluie journals from the period are extant. The last Pic and Michipicoten journal is for 1840-41. This makes analysis of the Lake Superior region especially difficult for this period.

had spread along the north shore of Lake Superior[73]. There were several other similar epidemics north of the lake during the next five years, perhaps every year. However, the loss of the post journals makes it impossible to identify how many and how widespread they were, except in the case of the most mortal afflictions.

The other notable sickness of 1841 occurred at York Factory, and its significance lies both in its origin and in the fact that it had never before been described in the Petit Nord. In August, mumps was brought to Hudson Bay from Great Britain by the HBC ships. On the 18th the *Prince Rupert* and *Prince Albert* arrived from Europe, the disease prevailing generally among their crews (HBCA B.239/a/154: 65d; D.5/6: 329d). Subsequently the disease spread to some of the York Factory men and to several of the natives, and it remained active at least into January of 1842 (HBCA D.5/6: 329d; B.239/a/155: 23). It was not fatal in any case, and did not spread beyond this area.

Mumps (ICD-10 B26) is a directly transmitted, acute viral disease whose sole reservoir is humans. It is spread through aerosol droplets and direct contact with infected saliva, and results in a lasting immunity. It is, thus, a typical *ACI* and was a common childhood affliction in densely populated urban areas, prior to the recent development of an effective vaccine. The incubation period is a relatively lengthy twelve to twenty-five days, commonly eighteen, and the victim is infectious from six to nine days before swelling begins in the glands to about nine days after. Prevalent symptoms include fever

[73]There had been respiratory complaints at Osnaburgh in January and later among the Albany Indians in September (HBCA B.155/a/53: 9d, 12d; B.3/a/148: 3). However, neither can be linked to the Lake Superior epidemic given the frequency with which ARDs flared up in the Petit Nord during this period.

and swelling of the salivary glands (Benenson 1995: 315-16). It is considerably less infectious than measles, however, and under endemic conditions the mumps victim therefore tends to have a much higher median age than that of the victim attacked by measles (Hope Simpson 1952: 553).

Despite being a relatively easily identified disease with a fairly lengthy infectious period, this was the first mention of mumps in the Petit Nord. In this case it was transported by the Company ships, and there is no obvious reason why a disease of childhood was prevalent among the European crews, and survived the long Atlantic voyage in this instance and not on earlier trips. Nevertheless, this disease began reaching the Northwest by land soon after, and during the next four years was present on at least two other occasions, in 1844 and 1845-46.

Whooping cough returned to the Petit Nord between 1842 and 1844, and its diffusion combined some elements of the earlier whooping cough epidemics. Once again, it broke out at the present-giving ceremony, held this time at Manitowaning on Manitoulin Island, where 6,000 native people had gathered[74] (HBCA D.5/14: 138; United States 1842: 403; Figure 22). Also circulating among the participants this year was virulent diarrhoea that proved fatal to many of the Indians on the American side (United States 1842: 403). At least whooping cough, and perhaps both diseases, spread westward

[74]At about the same time, there were reports of epidemic smallpox below Sault Ste. Marie that prompted John Swanston to vaccinate the Indians passing through Sault Ste. Marie in order to prevent that disease from diffusing to Lake Superior (HBCA D.5/7: 229d).

to Fort William, and affected the HBC people and the Indians of that area[75] (HBCA D.5/7: 372). Whooping cough arrived late in the summer, shortly after the next year's supply of goods. There, it struck the Europeans, métis and Indians with considerable force (*Loc. Cit.*). Whooping cough was also prevalent throughout the Lake Nipigon area, and caused several deaths among the Indians (HBCA D.5/8: 59).

The disease appeared in the Red River Settlement during the initial months of 1843, and was one of several epidemics that passed through Red River in that year. Overall, 1843-44 was a period of great sickness in the Red River Settlement. Whooping cough was followed by outbreaks of scarlet fever, mumps, "bilious fever," influenza and other ailments. Consequently, the Reverend William Cockran exclaimed that he had never before seen such sickness[76] (PAM CMS C.1/0 Microfilm reel A84, William Cockran to the Secretaries of the CMS, Grand Rapids, Red River, no date but marked received April 3, 1844). The Reverend John Smithurst noted in his journal for February 16 that almost all the people at his lecture were suffering from a cough, and on the 19th attendance was down considerably at his service at St. Peter's, owing "to the epidemic now in the Settlement" (PAM CMS C.1/0 Microfilm reel A96, "No. 2 Copy of Journal from Feb 1st to August 1st, 1836, John Smithurst, Indian Settlement"). Although not

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[75]A letter from John Swanston to Sir George Simpson, dated Fort William, December 20, 1842, mentioned no mortalities and only whooping cough (HBCA D.5/7: 372), but another from John Ballenden to Simpson, dated Sault Ste. Marie, July 14, 1845, stated that: "In 1842, the mortality at Fort William caused by diseases brought there from Manatowaning was very great" (HBCA D.5/14: 138).

[76]Ens (1996: 106-110) discussed what he called the crisis mortality of 1843-46 in the Red River Settlement in the context of its effects on the demography and social order of Red River. He, like others, underestimated the amount and variety of epidemic diseases circulating through this community during this period.

named by Smithurst, the symptoms of cold-like illness, accompanied by an incessant cough, sore throat, hoarseness and copious expectoration (*Ibid.*: February 19, February 20), are consistent with whooping cough[77]. The sickness persisted for several months, again characteristic of the tenacious whooping cough. However, there were only a few mortalities (*Ibid.*: March 20). Whooping cough also caused a decline in attendance at the school at Grand Rapids, St. Andrews parish, during the months of March to May, and although forty pupils fell ill, all recovered (PAM CMS C.1/0 Microfilm reel A86, Donald Gunn to William Cockran, Grand Rapids, July 6, 1843). In the end, this whooping cough epidemic seems to have made little impact on the Settlement, and was overshadowed by the scarlet fever that broke out a few months later.

Although Donald Ross at Norway House identified whooping cough as one of the diseases prevalent in the interior as of late August (HBCA D.5/8: 435), it did not spread northward or eastward from the Red River Settlement[78]. Charles McKenzie's Lac Seul journal reported no epidemic sickness among the Indians until the fall, when scarlet fever hit, and the Osnaburgh journal mentioned no widespread sickness until December, again likely scarlet fever (HBCA B.107/a/21; B.155/a/55: 11, ff.). As well, whooping cough was not one of the diseases that Nicol Finlayson at Fort Alexander identified as having struck the Lac la Pluie district in 1843 (HBCA D.5/9: 313). Neither did it appear at Norway House or in the northwestern part of the Petit Nord at this time, as it did not

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[77]Donald Gunn referred to the disease specifically as whooping cough, however.

[78]Another sickness identified by Ross was scarlet fever, and it had yet to reach Norway House as of December 21, 1843 (Glazebrook 1938: 452). It is obvious, then, that Ross was speaking for the whole of the HBC posts inland from York Factory.

spread northward with the brigades from the Red River Settlement. In all probability, the epidemic had expended much of its force by the time the men left for Norway House. Moreover, the 1834 epidemic was widespread in the Settlement and would have left many adults immune in 1843, and so incapable of supporting the disease. Thus, unlike many prior epidemics, the 1842-44 whooping cough epidemic did not diffuse through the Red River Settlement-York Factory corridor. Instead, it may have spread westward, before striking Norway House the following year. Ross reported that whooping cough broke out among the children of the post and in the nearby Methodist Indian village of Rossville towards the end of September, 1844, and was still active as of December 16[79] (HBCA D.5/12: 563d; B.154/b/1: 48d). He thought that it had come from the Saskatchewan River (*Loc. Cit.*), probably an accurate assessment, as the Reverend James Hunter found the disease prevalent among the Indians at Cumberland Station, on the Saskatchewan, early in the fall of 1844 (PAM CMS C.1/M Microfilm reel A78, "Rev^{d} J. Hunter's Journal from June 1 1844 to May 11 1845": 612, 620). There was an unidentified sickness among the Berens River Indians during the winter, perhaps whooping cough as well, as it was common for some of these people to spend their summers at Norway House, and to work on the freight boats of that place[80] (HBCA

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[79]Ross cited the earlier epidemic of the 1830s as the reason why the sickness was confined almost exclusively to the children (HBCA D.5/12: 563d).

[80]Many of the Indians around Severn House were sick late in October of 1844, but the illness was not named, and there was an outbreak of unidentified disease among the HBC men at Trout Lake in November (HBCA B.198/a/86: 10d; B.220/a/6: 24). There was no such sickness at Island Lake. However the fact that James Harrold made comments to that effect suggests that infectious disease was becoming common among the Island Lake Indians (HBCA B.93/a/7: 7d, 13). When some Indians arrived with furs in January, he wrote that "this is the hunts of 7 Indians, all those in good helth [*sic*] and there is very little Sickness amongst any of them yet" (*Ibid.*: 13).

B.154/b/1: 30, 53). This epidemic did not spread to York Factory.

The most malignant of the epidemics that passed through the Petit Nord in 1843 was scarlet fever. Scarlet fever (ICD-10 A38), or scarlatina, is one of several bacterial diseases caused by *Streptococcus pyogenes*. It is characterised by fever and a bright red skin rash that does not often appear on the face, but instead on other parts of the body such as the neck, chest, groin and elbow. Symptoms may vary widely, but there may also be nausea, vomiting, sore throat, swelling in the neck and discharge from the tonsils, among others. It is usually transmitted directly by droplet infection, and may occasionally cause widespread outbreaks due to contaminated milk or food[81]. Humans are its only reservoir and it is an acute disease. However, there can be long-term carriers. The incubation period is short, usually two to three days, and the period of communicability is lengthy, between ten and twenty-one days. It is now considered a relatively mild childhood disease, and causes a permanent immunity to the toxin that produces the rash and to the particular strain[82], and so scarlet fever rarely attacks an individual twice[83].

Although this was the first time that scarlet fever was identified in the Petit Nord, it is possible that it or other related streptococcal infections had been present before. For instance, some of the many epidemics of sore throats from the eighteenth century may have been streptococcal sore throats. Thus, an outbreak of sore throats at Albany Fort in

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[81]It can also be passed via objects handled by infected persons, but this is much rarer (Miller and Keane 1983: 1003).

[82]There are some eighty serologically distinct strains of *S. Pyogenes*, and so repeated infections with different strains is possible. Three are responsible for scarlet fever.

[83]The characteristics of this disease have been summarised from Miller and Keane (1983: 1002-

the summer of 1773 was accompanied by at least one case of erysipelas, an acute cellulitis caused by infection with Group A streptococci (HBCA B.3/a/65: 47, 47d, 48; B.3/a/66: 2d; Benenson 1995: 439). Scarlet Fever was present in epidemic form in the American colonies. as early as 1735, but was irregular in its appearance thereafter (Hardy 1993: 991-992). While it was at one time a fairly mild affliction, and is considered to be such now, a much more virulent form appeared in the 1820s and 1830s (*Loc. Cit.*). This change in virulence is reflected in the high degree of suffering experienced during the 1843 epidemic.

Scarlet fever broke out in the Red River Settlement during the summer of 1843, probably first in the lower, or northern, part, in July. It was said to have been brought by the Indians from Lake Superior[84] (HBCA D.5/9: 323, 373; PAM MG 2 C19 Bunn Papers, Thomas Bunn to Mrs. Ann Bailey, Red River, August 7, 1844; PAM MG 1 C9 #6 HBC Duncan Finlayson to HBC Governor, Chief Factors and Chief Traders, Red River Settlement, December 2, 1843). The disease soon spread throughout Red River, persisting well into the new year, and it was particularly severe in the Roman Catholic parishes (PAM CMS C.1/0 Microfilm reel A96, W. Robert Smith to Reverend William

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1003) and Benenson (1995: 438-443).

[84]A missionary, John Pitezel (1860: 76), was at the mission on the Keewenaw peninsula in November of what, judging by his memoirs, seems to have been 1843. He wrote: "In the early part of November, the scarlet fever broke out in the settlement. Several of our nearest neighbours were attacked—some were quite sick, but it did not prove fatal in any case." Pitezel includes few references to the year in which events happened, and was writing decades after the fact, and it is perhaps possible that he had mistaken the year. The American Indian Agent at Sault Ste. Marie, James Ord, wrote in his report for 1842-43 that the Indians of Ance Keewenaw had been struck by sickness during the winter (United States 1843: 429). Of perhaps 300 people wintering there, seventeen had died. Ord did not identify the disease, however.

Cockran, Middle Church School and House, August 1, 1844; Reverend John Smithurst to the Secretaries of the CMS, Indian Settlement, Red River, December 22, 1843; Ens 1996: 107). Unlike whooping cough, this sickness afflicted most of the people of the settlement, regardless of age or origin. Thus, adult Europeans contracted the disease alongside infants born in the country. It is estimated that well over 100 people in Red River died during this epidemic, most of them children (Ens 1996: 107). Apart from the age differential of the victims, Duncan Finlayson noticed a class differential in its severity, with the poorer people, who lived in crowded and unhealthy conditions and without proper diet, having much worse experience with the disease than the more affluent (HBCA D.5/9: 373).

The epidemic did not travel northward with the brigades, nor did it reach Norway House or York Factory (Ray 1976: 151). However, the Berens River Indians contracted it in the beginning of October when they travelled to Lake Winnipeg for their winter supplies (*Loc. Cit.*; HBCA D.5/9: 381). It spread quickly among them, and those who survived fled while still ill, and so no doubt carried it towards the interior of the Petit Nord (HBCA D.5/9: 381). In December Donald Ross reported that they were "dying by the half scores", and he pessimistically feared that the disease would soon be at Norway House (Glazebrook 1938: 452). Ross proposed isolating his post from the southern posts in order to prevent the northward spread of the disease (*Ibid.*: 461), and whether because of this or some other reason, the Indians and HBC people to the north of Lake Winnipeg escaped the epidemic.

It also spread east from the Red River Settlement, attacking the Indians of Fort

Alexander and Rat Portage, on Lake of the Woods, along with influenza, at least by October[85] (HBCA D.5/9: 313). Ten of the children at Fort Alexander died of scarlet fever and one man succumbed to influenza (*Loc. Cit.*). Most likely, it had reached the Winnipeg River somewhat earlier, as it was to the east at Lac Seul early in September (HBCA B.107/a/22: 5d). There, Charles McKenzie noted on the 7th that two of his men, all of the children and most of the women had scarlet fever and some of the Indians had the blotchy skin and sore throats, indicative of the disease[86] (*Loc. Cit.*). The Lac Seul Indians generally passed summers on the Winnipeg River or at Lac la Pluie, and in the summer of 1843 some were reported trading at Fort Alexander and at Fort Frances (HBCA B.107/a/21: 16d). In doing so they must have brought the disease back with them[87].

Scarlet fever, influenza, or perhaps some other disease spread through parts of the interior of the Petit Nord during the fall and winter. In December there was an unidentified illness that affected most of the people at Osnaburgh and possibly some of the Indians (HBCA B.155/a/55: 13d, 17). There was also a report from Red Lake of

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[85]In May of 1844 Nicol Finlayson noted that sickness and mortality had negatively affected the trade in his area (HBCA D.5/11: 212). The Indians had been sick at the time that he was giving out advances during the fall (HBCA D.5/9: 313).

[86]Both Nicol Finlayson at Fort Alexander and McKenzie identified native treatments as one reason for the severity of the disease (HBCA D.5/9: 313; B.107/a/22: 6).

[87]Another severe disease subsequently appeared among the Lac Seul Indians during the winter of 1843-44, one that rendered many of all ages crippled and unable to walk (*Ibid.*: 13). McKenzie commented on how the Indians of the Lac Seul area had declined in size and robustness compared to their ancestors, but not in numbers, an observation he claimed was shared by the elders (HBCA B.107/a/22: 13). If true, then it suggests that the general level of health in the Lac Seul area had declined significantly since McKenzie first came there.

sickness among the Indians during the autumn of 1843, and similar comments from an Ojibway man named the Lobster, who hunted near Escabitchewan (HBCA B.107/a/22: 12d, 15d). Nothing appeared at Martin's Falls, however, and so the 1843 scarlet fever epidemic seems to have been limited to the southwestern portion of the Petit Nord.

The whooping cough and scarlet fever epidemics of 1843 were continent spanning epidemics. Both spread westward from the Upper Great Lakes, probably coming from the eastern cities. They subsequently passed through the Red River Settlement before continuing their diffusion in the Northwest. Both later reached the Pacific Northwest. According to Boyd (In Press), scarlet fever appeared on the Columbia River early in September of 1843. He suggested that the HBC brigades had introduced it, although the timing and location of the initial outbreak indicate that it came from the Oregon Trail. The whooping cough epidemic appeared slightly later, reaching the Columbia in January of 1844. It had not spread with the overland immigrants, but instead had diffused from the east through native connections (*Loc. Cit.*), no doubt slowing its progress compared to the scarlet fever. Thus, not only was Petit Nord tied into a system of disease diffusion that brought disease from the eastern disease pools, but at least by the 1840s this system was also capable of spreading these afflictions to the westernmost shores of the continent. In fact, this system may have been in place long before, as diseases of eastern origin arrived in the Pacific Northwest as early as 1807, with the whooping cough that had been present on the Winnipeg River the previous year. More significantly, regular wintertime ARDs began to appear on the Columbia River during the 1820s and 1830s, introduced by fur traders from Red River and the Plains (Boyd 1985: 344). This new phenomenon

coincided with the emergence of ARDs as a frequent visitor to the Petit Nord, and these patterns were no doubt connected.

Ray (1976: 147-148) identified an influenza epidemic that his sources suggested broke out in the York Factory-Norway House area in 1843, and subsequently extended beyond the Churchill River, as well as to Fort Alexander. Influenza was also widespread on Lake Superior at about the same time, and also in the Upper Mississippi area[88], and it is possible that they were part of the same epidemic. On Lake Superior two other diseases accompanied influenza: mild colds and deadly dysentery. All three seem to have moved west along the north shore of Lake Superior. Influenza appeared first among the HBC men at Sault Ste. Marie, in July, and by the 17th it was beginning to exhaust itself, disappearing shortly thereafter (HBCA D.5/8: 356, 412). Subsequently it surfaced at Michipicoten with the colds, either late in July or early in August, and there the HBC men, their families, and the Indians all contracted the disease (*Ibid.*: 412, 435d, 459). It spread among the Indians along the shore of Lake Superior, and claimed five lives in the span of a single day, and seven in all[89] (*Ibid.*: 459, 514). Following in the path of influenza was dysentery, which arrived at Michipicoten some time before September 12, and lasted less than a week (*Ibid.*: 514, 546d). Only the relatively mild colds reached Pic and Fort William, and no deaths were reported at these posts (*Ibid.*: 546d). Dysentery alone appeared at Nipigon, conveyed there directly by an Indian returned from the

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[88]Influenza was said to have prevailed generally among the Sioux in the Upper Mississippi country during the summer of 1843 (United States 1843: 377).

[89]John Swanston mentioned the combination of severe coughs and diarrhoea, and whether the last was part of the dysentery epidemic or was a symptom of influenza is not known.

ceremonies at Manitowaning in August. There, the disease was extremely virulent, carrying off large numbers of children and a few adults[90] (HBCA D.5/10: 85, 197, 219; D.5/14: 138).

At about the same time that influenza was dying out at Sault Ste. Marie, it may have broken out at York Factory and Norway House (Ray 1976: 147). The Norway House journal (HBCA B.154/a/41) contains few references to sickness, but it is evident from the correspondence that there had been much disease among the people there. Towards the end of August Donald Ross wrote that "This has been a very sickly season in the interior, Hooping Cough, Influenza, Scarlet Fever and a very troublesome complaint resembling common cholera, are raging with less or more violence amongst us" (HBCA D.5/8: 435). In this, Ross was referring to all of the HBC people of the interior, and not just those at Norway House, and so it is not clear which afflictions were at his post. The sickness, which prevailed at Norway House and among the Indians at least until December, was thought by Ross to have had a serious impact on the hunts of the Indians (HBCA D.5/9: 381; D.5/10: 411). The Indians around both the Island Lake post and Oxford House also suffered severely from sickness from the late summer into the winter, and a total of twelve died[91] (HBCA B.156/a/23: 8d, 9, 11, 17, ff.; D.5/11: 1d). The disease at these posts was the same as at Norway House, and caused sore throats and a

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[90]The number of fatalities is unclear, and there were estimates of twenty-three, twenty-five, thirty-six and forty-four deaths (HBCA D.5/10: 85, 197, 219; D.5/14: 138). None of these estimates was attributed to James Anderson, the Post Master at Nipigon.

[91]At Oxford House the Indian children were especially hard it, three of whom died (HBCA D.5/9: 361d). A report from Island Lake in February stated that eight Indians, young and old, had died in the vicinity of the post (HBCA B.156/a/23: 25d).

disorder of the bowels (HBCA B.156/a/23: 13d). Both may be symptomatic of influenza.

The York Factory journal noted frequent unidentified illness among the men and a few Indians from about July 4 to late August (HBCA B.239/a/157: 44d, ff.). Perhaps significantly, two Montreal canoes arrived just prior to the start of this apparent outbreak, as did boats from Red River and Lac la Pluie (*Ibid.*: 43d, 44). By August there was a general state of infirmity among the men, including the sloopers, and work came to a standstill (HBCA D.5/8: 450). By the end of September the sickness had abated, however (HBCA D.5/7: 564d). Once again, its identity is not clear, although Ray (1976: 147) has concluded that it was influenza. Although most cases were later said to have been of bowel complaints (HBCA D.5/9: 312), comparisons were made by the HBC men to the influenza epidemics of the 1830s (HBCA D.5/8: 450d). This suggests that the symptoms were somewhat similar, and since the clinical manifestation of influenza can vary considerably between outbreaks, Ray may indeed have been correct[92].

If it was influenza at York Factory and Norway House, it is likely that it spread eastward, as a severe cold and other disorders broke out among the HBC men at Severn in January and February of 1844 (HBCA B.198/a/85: 20, 21d, 21d). By spring, reports came in that the Indians also had been sick during the winter (*Ibid.*: 24d). The fact that Post Master John Cromartie considered winter-time sickness at Severn to be very uncommon (*Ibid.*: 20, 21d) may suggest that they were accustomed to infections arriving during the summer and fall, the period when both the HBC men and Indians from the

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[92]"Mr. Gillespie... considers that the prevailing sickness has no affinity with the diseases which from 1833 to 1837 affected the Servants of this Factory" (HBCA D.5/8: 450d).

Severn area would have had contact with people from the Hayes River area.

Unlike several earlier influenza epidemics, the Red River Settlement was not the source of this disease for the Northwest, as it broke out there only after it was active in the more northern areas[93]. As noted above, influenza and scarlet fever appeared at Fort Alexander and Rat Portage during the late summer or early fall, but it is unknown if they reached Fort Frances (HBCA D.5/9: 313). Given the timing it is possible that the virus was brought from York Factory with the brigades. There is no evidence that this disease spread among the Indians living to the north of the Boundary Waters. Overall, this influenza epidemic had a relatively constrained diffusion compared to those of the 1830s.

The record of acute infectious disease from 1844 is one of manifold outbreaks in the Petit Nord, but no identifiable widespread epidemic (Appendix 2). This was more likely a reflection of the lack of journals for this period rather than it being a relatively healthy year. The situation at Lac Seul is worthy of additional comment, however. There, multiple sicknesses struck the natives and HBC men in succession during the fall and winter. It began with Charles McKenzie's return from Albany late in the summer, during which the crews of the boats fell sick of unidentified maladies on their journey to Lac Seul (HBCA B.107/a/23: 1). Nothing in either the Albany or Martin's Falls journals identifies the source of these afflictions, however. After the HBC men arrived at Lac Seul in early August, another disease broke out at the post at the beginning of the

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[93]Influenza broke out in the Red River Settlement some time after scarlet fever, which had first appeared in July (PAM CMS C.1/0 Microfilm reel A84, William Cockran to the Secretaries of the CMS, Grand Rapids, Red River, no date but marked received April 3, 1844). By that time the disease was already circulating in the Norway House and York Factory areas.

following month, spreading among the Indians and men alike (HBCA D.5/13: 117). On September 2 McKenzie commented that "almost all of the men, women, and children and Indians, have their heads tied up in Handkfs - and can scarce sleep a wink, with a most violent cough & so[re] throats" (HBCA B.107/a/23: 3). Although it is impossible to be sure, these symptoms suggest influenza. However, it is likely that other diseases were abroad as well. With all of the Indians and men ill a week later, the trader noted that "every different band of Indians bring us a fresh Cough" (*Ibid.*: 3d). Indeed, sickness lingered among the men into December, long past the time when they would be expected to have been over their initial illness[94] (HBCA D.5/13: 117d). Not only the Lac Seul people and local Indians (HBCA B.107/a/23: 7) were affected. McKenzie was informed of sickness among the Red Lake Indians and those living to the northward, as well as among many others who came to trade (*Ibid.*: 7, 8, 9d, 11d, 13d, 14). Consequently, he commented that "sick Indians coming in with nothing are no novalties here this year", and that "from whatever quarter we hear, Sickness is the Coy [company]" (*Ibid.*: 7, 8d, 9d, 10). In this case, neither winter nor the New Year brought relief, and epidemics continued among the Lac Seul Indians into the spring of 1845, as the next of wave diseases struck the area[95]. While perhaps not typical of 1844, the prevalence of sickness

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[94]On December 26 McKenzie wrote, "I may truly say that at this house, we were not a day without sickness since the arrival of the boats..." (HBCA D.5/13: 117).

[95]An undated letter from Charles McKenzie to Thomas Corcoran speaks of great sickness among the Lac Seul Indians but it is unclear if it applies to the winter of 1844-45 or 1845-46. It states that:

> There was a fatal disease raging among the Lac Seul Indians all winter nor are they free from it to this day - to which no less than 6 fell victims - all young or middle aged men - more or less in debt - as well as several women and children - there was scarcely a family free from Sickness of some kind or other during the winter. (HBCA B.3/c/1: 1ad)

at Lac Seul was indicative of the pattern of increasingly frequent *ACI* activity in the Petit Nord during this period.

1845 witnessed yet another widespread influenza epidemic in the Northwest (Ray 1976: 149). Once again it broke out initially at the Red River Settlement, late in the spring. Following an outbreak of colds in March[96], influenza was rampant in Red River by early May, characterised by "frequent coughing, much expectoration, strong fever and great debility" (PAM CMS C.1/M Microfilm reel A78, "Rev^{d} J. Smithurst's Journal April 1st to July 31st 1845": 587). It was followed by an acute "cholera", some diarrhoeal disease that was not Asiatic cholera, and a far more severe epidemic of unidentified fever or fevers, that broke out late in the summer and continued into January of 1846 (PAM CMS C.1/0 Microfilm reel A84, Cockran to the Secretaries of the CMS, Grand Rapids, Red River Settlement`; C.1/L.4 Microfilm reel A81, "Reverend William Cockran's Journal from August 1845 to June 1846": 46, 56, 61; C.1/M Microfilm reel A78, Cockran to the Secretaries of the CMS, Red River Settlement, July 30, 1845).

The boats that departed the Red River Settlement in the early part of the summer carried epidemic disease with them to Norway House (Ray 1976: 149), as they did on many previous occasions. In this case, it was not the HBC vessels but the private freight boats belonging to some of the Red River merchants that transmitted influenza (HBCA

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[96]While preaching at the Indian Church on March 21, the Reverend John Smithurst "was a good deal disturbed with people coughing..." (PAM CMS C.1/M Microfilm reel A78, "Rev^{d} J. Smithurst's Journal August 2 1844 to March 31 1845": 580). Smithurst also observed that "It is very remarkable in this country that colds are always general and perhaps 50 people will be coughing at once." Although it is tempting to conclude that the missionary mistook colds for influenza, the latter disease was identified as being prevalent in the same population nearly two months later. In any event, this is an interesting observation on the behaviour of colds in relatively isolated populations.

B.239/a/161: 47). It is not clear when the disease arrived at Norway House, although it was likely on the boats belonging to either Andrew McDermott, or those dispatched by Edward Mowat and James Sinclair. These arrived at the post in early June[97] and by July 21 the disease was "very prevalent + very severe" (HBCA B.154/a/45: 2d, 3d, 7d), its effects felt by some men as late as November (HBCA B.154/a/45: 17). In turn, it was passed to the Indians and to the crews of the freight boats that ran between Norway House and York Factory (*Ibid.*: 7d, 11). This prevented Donald Ross from providing crews for the brigades that carried goods from York Factory to Norway House (HBCA B.154/b/3: 3d). On the 30th of July, Ross told Duncan Finlayson that "the season is very unhealthy with a most severe Influenza and other complaints which prevail generally throughout the country" (HBCA B.154/b/3: 2d ; see also D.5/14: 226d), and so influenza was not the only epidemic disease spreading though the area. The disease also struck at the outpost on the Nelson River and was at least among the crews of the Oxford House freight brigade. There is no Oxford House journal for this year. However, it is likely that influenza reached there as well.

Influenza was slow to reach York Factory, appearing only in mid-July (Ray 1976: 149), and it was not the first disease to break out at that post during 1845. There had been a minor outbreak of colds and bowel complaints during March and another of colds in May (HBCA B.239/a/161: 27d, 44d; D.5/13: 376d). All had disappeared prior to the

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[97]James Hargrave noted that private traders brought the disease from Red River during the spring, rather than the summer (HBCA B.239/a/161: 47), and these were among the first crafts to reach Norway House in 1845. Ross suggested that the disease broke out at the beginning of summer, and so likely some time in June (HBCA B.154/b/1: 60d).

arrival of influenza (HBCA D.5/14: 253d). As late as July 10, none of the York Factory men had been on the sick list, but one week later Chief Factor James Hargrave noted that the crews of the four Oxford House boats who were then at his post were sick with influenza, "a disease prevalent this summer among the natives and servants in the low country" (HBCA B.239/a/161: 47). Within a few days, several of the York men were off sick with the "prevailing disease," and it was circulating among the crews of the York Factory boats (*Ibid.*: 47d, 48). By July 25, it was universal among whites and Indians alike (*Ibid.*: 48d). However, the inland brigades had already departed before the epidemic reached its height (HBCA D.5/14: 253d).

The disease seems to have spread to the Severn Indians, although not carried by the HBC Severn brigade that departed York Factory on July 26 (HBCA B.239/a/161: 48d). On August 30, John Cromartie noted that "The Indians is starving and many of them still very sick. Some of them have had a hard trial of sickness this summer but none of them has died..." (HBCA B.198/a/88: 5d). The Severn Indians had probably brought the disease back from the west, as some were known to associate with those from the York hinterland (HBCA B.198/a/86: 26). At Trout Lake the summer business was disrupted by the epidemic among the Indians, and many of the sick sought assistance at the post (HBCA D.5/16: 317d). There, the disease persisted until the onset of cold weather (*Loc. Cit.*).

Influenza and other epidemic disease also spread from Red River through the Boundary Waters and into the southern part of the Petit Nord in 1845. The Indians of the Fort Alexander area, on the Winnipeg River, contracted a very severe sickness brought

from the Red River Settlement (HBCA D.5/18: 105). Although it is not stated which disease this was, it was probably influenza, as some of the Lac Seul people contracted that disease on the Winnipeg River during the summer[98] (HBCA B.107/a/24: 1). Influenza also appears to have been at Lac la Pluie by early June (HBCA B.107/a/24: 1d). Without journals, however, it is impossible to know when the epidemic reached the western part of the Boundary Waters, and its effect on the Indian people.

More is known of events at Lac Seul. There, the pattern of sickness in 1845 was similar to that of the previous year, with what appear to have been several distinct diseases appearing over a short period of time. The first new illness of the year appeared in June, though some of the Indians remained ill of the previous year's afflictions as of April (HBCA B.107/a/23: 14). On June 1 Charles McKenzie noted that the Indians were "some + all laboring under a most violent cough + otherwise sick besides" (HBCA B.107/a/23: 17d). Most likely this was influenza. According to McKenzie, some of them had gone to the Winnipeg River earlier in the year where they had caught "this Red River disease" (HBCA B.107/a/24: 1). Another source of infection was a group of people who came with the Methodist missionary, Peter Jacobs, to Lac Seul. Jacobs, his family, a Canadian servant and six Indians arrived at Lac Seul from Lac la Pluie on June 13. The Indians and the Canadian servant were ill upon their arrival, and they were responsible for introducing influenza to the post, independent of the Lac Seul natives (*Ibid.*: 1d).

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[98]Ray's (1976: 148) map shows influenza throughout the area north and west of Lake of the Woods, and the East Winnipeg Country. Those who lived in the Escabitchewan area to the east, and summered on the Winnipeg River or went to Lake of the Woods, caught influenza early in the summer (HBCA B.107/a/24: 6d).

The Lac Seul boats left for Albany early in June and the journal was discontinued until mid-August. As in 1844, the brigade was beset by sickness on its return from Hudson Bay that made travel difficult[99] (HBCA B.107/a/24: 1; NAC MG 19 A40 1A, Thomas Corcoran to William Lane, Albany, January 1846). One half of the crews remained sick when they reached the post on August 17, and they arrived to find most of their families and the Indians all sick with violent coughs, and some already dead (HBCA B.107/a/24: 1). Influenza was not alone, however, as McKenzie discerned that "there is some other disease accompanying the cough" (*Ibid.*: 2). Similar fates befell the Indians throughout the Lac Seul District (HBCA B.107/a/24: 10d). During the summer, fall and winter, people from every quarter brought news of their affliction (*Ibid.*: 4d, 5, 6d, 10d; 20). Altogether, there were as many as a hundred sick at the post at this time, leading McKenzie to exclaim that "This house is more a Hospital than a kitchen" (*Ibid.*: 1). To his dismay, sickness was to linger at the post for over a year[100].

McKenzie noted an interesting dynamic at work between the HBC post and the

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[99]Once again there is nothing in the Albany or Martin's Falls journals to suggest where the sickness came from that afflicted McKenzie's brigades. In fact, the health of the men and Indians about these posts was very good at the time that the Lac Seul boats went through (HBCA B.239/a/151; B.123/a/48; NAC MG 19 A 40 1A, Thomas Corcoran to William Lane, Albany, January 1846).

[100]Influenza first appeared at Lac Seul during June of 1845 and lasted until the fall. According to McKenzie some form of sickness remained at the post until June of 1846 (HBCA B.107/a/24: 25). However, another influenza epidemic broke out in June of 1846, followed by measles and dysentery. There was probably not a period of more than a month or two between the spring of 1844 and the spring of 1847 when the Lac Seul Indians and HBC men were completely free of epidemic disease.

Indians of the Lac Seul area[101]. As they fell ill, the Indians of the district headed for his post in order to receive assistance from the HBC men (HBCA B.107/a/24: 3, 10d). This was a standard reaction to epidemic disease, and the Company considered providing shelter and medical and food aid as a part of doing business (HBCA E.18/3: 52). In this instance, however, the arrivals brought other diseases with them that, added to those already at the Lac Seul post, made it more dangerous than the place from whence they had come[102] (HBCA B.107/a/24: 5, 6d, 10d). Very quickly during the summer of 1845, the post gained a reputation for enhanced sickness and death, and the Indians then began to leave in large numbers for the ricing areas, and a chance to free themselves from the sickness (HBCA B.107/a/24: 3). The majority who were unable to proceed on their own were carried to their canoes "as from a Pesthouse" and none reached their destination, becoming too sick to proceed along the way (HBCA B.3/c/1: Letter 8, Charles McKenzie to Thomas Corcoran, Lac Seul, August 30, 1845). In this case at least, the post had become the antithesis of the safe haven that it was intended to be[103].

There were two distinct, but brief, outbreaks of sickness at Osnaburgh House, and

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[101]Another reaction that was observed in at least one instance was avoidance. A prominent Indian man named Okimas chose to leave his family at Red Lake rather than bring them to the post, and to avoid summertime contact with others. Thus, in August McKenzie wrote that: "Okimas came in with his old mother from the Red Lake quarter where he past [*sic*] the summer, and left his family there being so much afraid of sickness that he did not join any Indians during Sum[r] having lost 2 children last year" (HBCA B.107a/24: 4d). It is not known how many others followed Okimas' lead, but dropping out of the trade and isolating oneself may have become a common response by some people during periods of frequent epidemics.

[102]McKenzie noted that sickness was the rule throughout the district, but "no where so fatal as this place because as soon as they fell sick they congregated here from all quarters each bringing a different branch of the disorder that it joined him in one Body -which formed nothing short of a real pest..." (HBCA B.107/a/10d).

[103]One of the HBC men and three of the Indians died at the post between the 16th and the 30th of August, four others were at the point of death, and an unknown number away from the post had succumbed (HBCA B.3/c/1: Letter 8, Charles McKenzie to Thomas Corcoran, Lac Seul, September 5, 1845).

neither seems to have spread to the Osnaburgh Indians. The first appeared in mid-August of 1845, and placed most of the HBC people on the sick list (HBCA B.155/a/57: 5d). This was only days after Charles McKenzie's brigade passed by and, despite George McPherson's suggestion that all were "well and safe" on these boats, it is probable that the Lac Seul men had passed their sickness to the Osnaburgh men (*Ibid.*: 4d). The second outbreak, again unidentified, occurred during the month of December, and nearly all of the people at the post were ill (*Ibid.*: 12, 13). Most likely this was influenza from Lac Seul. Nothing was ever stated regarding the health of the Indians who traded at that post during this period. It is impossible to discern how far down the Albany River these sicknesses spread, if at all, but once again the Martin's Falls and Albany areas remained free of epidemic disease while they raged around the headwaters of the river.

There is no record of influenza in the eastern part of the Petit Nord in 1845. However, other diseases were abroad in the Lake Superior District. There was a brief outbreak of severe colds at Michipicoten that affected the HBC people early in the fall. It had been brought from Sault Ste. Marie by some of the Indians on their return from the British present-giving ceremony at Manitoulin Island (HBCA D.5/15: 84). More deadly was an epidemic of measles that spread from Lake Huron to Fort William during the latter half of the year, as part of a pandemic, accompanied by mumps (HBCA D.5/14: 190d; 299). In this case, most of its penetration into the Petit Nord took place in 1846, and it will be examined in the following chapter.

SUMMARY

The period 1821-1845 saw a more or less constant increase in the number, type and range of *ACIs* striking the Petit Nord. Not only were there many more epidemics and outbreaks in the region than before, but several new diseases such as mumps and chickenpox first appeared during these years. No disease was more prominent than influenza, however, which arrived frequently, at times almost yearly, and devastated the people of the Petit Nord on several occasions. For many of the region's people there was little respite between wave after wave of often devastating epidemic disease, particularly following the late 1820s. The situation worsened during the 1840s, as epidemics began to overlap on a regular basis, and in some places three or more afflictions appeared in a single year. However, one established disease that failed to penetrate the region was smallpox, prevented from doing so by the beneficial diffusion of the newly demonstrated prophylaxis of vaccination.

This increase in *ACIs* marked an epidemic transition for the Petit Nord, from relative isolation to inclusion within the disease frontier of formerly distant, urban disease pools. This was the direct result of changes occurring outside of the region. Most notable were the movement of large numbers of non-natives from the east into the interior of the continent, including the Upper Great Lakes, and improvements to the speed of transportation provided by the construction of canals and the widespread use of the steamboat. Together, they enabled many more diseases to spread into the interior than had ever been the case. These changes mirrored those that had resulted in the introduction of a myriad of *ACIs* among the aboriginal people of the east from the early

seventeenth century onward. With the movement of large numbers of settler families into the interior of the continent, there emerged a new means by which the diseases of the east could be conveyed towards the heart of the continent, one that overshadowed the older mechanisms of fur trader travel and aboriginal contact.

During this period, two broad patterns of diffusion into the Petit Nord emerged. Many of the diseases that struck the region entered it from the southeast, passing directly from the Lake Huron area into the Lake Superior district. These tended to be relatively constrained and seldom spread far beyond the north shore of Lake Superior. Many other sicknesses entered the region from the southwest, passing through the Red River Settlement, which played a critical role in introducing, hosting and redistributing epidemic disease throughout the Northwest. No doubt, the American trading posts established on the Upper Missouri served as a source of disease for the Settlement, due to their connections with St. Louis. Nevertheless, it is also apparent that these two broad systems of diffusion were connected through linkages that ran to the south of the Boundary Waters. On several occasions, *ACIs* that were rampant south of the Upper Great Lakes subsequently surfaced in Red River, thereafter spreading to the Petit Nord.

Within the Petit Nord, the HBC transport brigades became the main mechanism for the long-distance transport of epidemic disease. This was a major departure from earlier periods when the brigades rarely participated in the diffusion of *ACIs*. As the Company altered its hiring practices during the post-merger period, from employing mainly Europeans to using métis and Indian men born in the Northwest, the overall level of immunity to the crowd diseases among its crews declined. Moreover, the brigades

serving the western part of the Petit Nord were strongly tied to the Red River Settlement, both through the nature of the transport network and the fact that the Settlement supplied much of their labour. Consequently, the diseases of this community often spread widely in the region that these western brigades travelled. These epidemics were repeatedly extended to the Lac Seul region by the communication of the native people with those of the Boundary Waters. In the east, fur trade transport connections with Sault Ste. Marie and Lake Huron, and the movements of the aboriginal people, tended to distribute *ACIs* throughout the Lake Superior district.

Overall, the documentary record in the Petit Nord during the period 1821-45 shows that *ACIs* became increasingly common in the region as time passed. However, an examination of the journals of several posts within the area shows that not all parts of the region experienced a similar rise in activity, and hence there was significant divergence in the fate of the people of the Petit Nord during this period[104] (Appendix 4). Just as there were spatial variations in the impact of every epidemic that struck the Petit Nord, there were variations in the long-term pattern of epidemic activity for different populations within the region that saw some people overwhelmed by repeated episodes of severe sickness, and others attacked by only infrequent and mild complaints.

As Ray (1976) concluded, there was a great deal of epidemic activity at York

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[104]The posts chosen for Appendix 4 were those with the most complete span of journals over this period. The disease references shown are those from the journals only, since some posts such as York, Norway House and Albany had a far greater volume of additional records, particularly correspondence, than did Martin's Falls or Osnaburgh, making direct comparison somewhat unfair. Unfortunately, none of the Lake Superior posts had a sufficient range of journals to warrant inclusion. There can be no doubt that the journal record, like almost any primary record, was biased in favour of severe and, especially, incapacitating diseases. Thus, minor complaints were far less likely to be commented upon in the journals than diseases such as whooping cough, influenza and measles.

Factory and Norway House, introduced by the HBC's Northern Department transport network and its connections with the Red River Settlement. Many of these epidemics spread widely in the Northwest through that network. Oddly, there was significantly less disease activity recorded at Severn, despite the fact that this post was outfitted from nearby York Factory and there were strong connections between the native people from the Severn hinterland and those of the Hayes River. Instead, the HBC men, their families and the aboriginal people of the Severn area appear to have remained isolated from the sicknesses that were carried along the Red River-York Factory corridor with some regularity.

The frequency and types of *ACIs* appearing at Albany during 1821-45 were much the same as they had been during the period 1784-1818. ARDs, generally referred to as colds, were present on many occasions, but there is no evidence that any of the more widespread, and serious, epidemics reached the Lowlands in this area, with the exception of whooping cough in 1827. Once again this contrasts greatly with York Factory and is likely the result of the more limited transport connections to the south at Albany[105].

Even more remarkable is the rarity of *ACIs* at Martin's Falls. It does not appear that the Southern Department's Albany River brigade transported diseases into the interior with anywhere near the regularity that the Northern Department boats carried them between the Red River Settlement and Hudson Bay. No doubt, this was due to the small number of men who manned this brigade as well as the large gap between the

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[105]Comparative analysis with Moose, a post with far more substantial fur trade connections than Albany, would be of considerable benefit in order to explain the pattern of epidemic activity at the latter post.

Albany River posts[106]. Nor did the epidemics that periodically struck near the headwaters of the Albany river, or those that flared up in the Lake Superior area, make their way to the people living around Martin's Falls through aboriginal movements. Instead, these people enjoyed relative freedom from the devastating infections that struck those living to the south and west within the Petit Nord. The failure of these diseases to reach the Martin's Falls Indians through aboriginal connections can be attributed to their relative isolation. Despite complaints of trespass in the mid-1830s by the Martin's Falls people against the Osnaburgh, Long Lake and Nipigon Indians (Bishop 1970: 5; HBCA B.123/a/38: 13, 14, 24; see also B.123/a/48: 8), there appears to have been little contact between these groups. By 1839, the people who traded at this post were mostly from the Sucker clan of the Ojibway, who had pushed their way northward into Cree territory (HBCA B.123/e/14: 3d). Their relationship with other Indians in the Petit Nord was complex, and they maintained ties to the Severn and Albany Cree, intermarrying with them, but regarded their Ojibway relatives to the south and west with fear and suspicion (Ibid.: 4). In particular, they did not cross the highland between Lake Nipigon and the Albany River drainage basin when hunting (Ibid.: 4d). When Anglican Bishop David Anderson passed down the Albany River in 1852, he suggested that the Indians whom he met at Martin's Falls were very isolated from others (Anderson 1967: 202).

Finally, the situation at Lac Seul and at Osnaburgh clearly shows that there were other important factors in the diffusion of *ACIs* besides fur trade transport. These posts

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[106]It also seems probable that many of the men would have recovered from their afflictions before leaving for Albany. As well, many of the more severe epidemics at Lac Seul broke out after the brigade had departed.

lay at the farthest reaches of the Southern Department of the HBC but experienced far more epidemic activity than did Martin's Falls. In fact, there were more outbreaks recorded at Lac Seul than at Albany. Unlike at York and Norway House, where the HBC brigades were most frequently responsible for introducing *ACIs*, at Lac Seul and Osnaburgh these diseases were almost always transported across departmental boundaries by aboriginal people. Often it was the more western of the Lac Seul Indians, those who spent their summers in the Boundary Waters, who carried diseases back towards Lac Seul[107], where they were sometimes passed on to the Osnaburgh Indians. At other times it was the Lac la Pluie Indians who carried disease into the Lac Seul area. Thus, in contrast to Martin's Falls where isolation from the southern people protected them from *ACIs*, at Lac Seul frequent contacts with other Indians led to frequent epidemics among the aboriginal people. As at York and Norway House, it was communication with the Red River Settlement, either directly or indirectly, that proved the most significant factor in the increased frequency of *ACIs* at Lac Seul during the first half of the nineteenth century.

While many of the years between 1821 and 1845 saw more than one epidemic *ACI* in the Petit Nord, particularly during the 1840s, things had yet to reach their lowest point. In 1846 all of these patterns and circumstances combined, and the result was a year without precedent in the history of epidemic disease in the region. Thus, the many

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[107]Travel between Lac Seul and the Boundary Waters by the HBC men was a relatively rare occurrence during this period, but was a part of the seasonal rounds of the Lac Seul Indians who often visited the Boundary Waters. Thus, Charles McKenzie noted: "... most of our Indians go early in the Spring where Sturgeon is found. Either Lac la Pluie, Lake of the Woods or River Winnipic where they pass the summer, and... generally Return in the Autumn..." (HBCA B.3/c/1: 1a).

epidemics that passed through the Petit Nord in that year were reflective of the general trends that developed during this period. This transition was well underway by the mid-1840s, culminating in the fateful year 1846 that followed.

CHAPTER 11 — THE EPIDEMICS OF 1846

The year 1846 witnessed more epidemic activity within the Petit Nord than had ever been the case in the history of the region. This was manifest in terms of the range of diseases and the numbers of people affected, if not in terms of the mortalities that occurred. This was the product of the increasing epidemic activity in the region in the 1830s and early 1840s, and this in turn reflected growing connections with the urban disease pools of the east. For most of the people living in the Petit Nord, the year 1846 represented the nadir of their health fortunes to that time. During that year, three major disease episodes struck the region, leading to considerable sickness and death, particularly where they overlapped in time and space. Other diseases of variable virulence also appeared, but were more localised. At the same time, some parts of the region escaped all of these contagions entirely. Among the diseases present during that year, the most widespread were influenza, measles and dysentery.

INFLUENZA

Influenza, the first of the epidemics of 1846, was first noted in the Red River Settlement in January (Ross 1957: 362). The CMS missionary, John Smithurst, described its effects in his journal in February. On the 22nd he preached at his colony churches and found

> The congregations were very small and there was so much coughing that very little of what I said could be heard. Within the last few days nearly the whole of the Indians have fallen sick with a disease similar to the influenza. It is attended with a constant coughing and expectoration.

(PAM CMS C.1/M.4: 96 Microfilm reel A78, "Reverend J. Smithurst's Journal from Aug 1st, 1845 to March 31st, 1846")

Peter Garrioch attended one of Smithurst's services that day and noted in his journal "Weather very cold and a great deal of coughing in church" (PAM MG 2 C38 *Peter Garrioch Journal*: 68). The following day Smithurst found that "On going to the School this morning I found not ¼ of the children present and so much coughing that nothing could be heard" (PAM CMS C.1/M.4: 96 Microfilm reel A78, "Reverend J. Smithurst's Journal from Aug 1st, 1845 to March 31st, 1846"). The next Sunday he preached at the Grand Rapids (St. Andrew's Parish) where he found "The Influenza is raging here as well as at the Indian settlement" (*Loc. Cit.*). On the 1st of March another CMS minister in the Settlement, William Cockran, wrote that "The influenza has made its appearance among us and many are dangerously ill" (PAM CMS C.1/M.4: 62 Microfilm reel A78, "Reverend William Cockran's Journal from August 1845 to June 1846"). By the end of March it would seem to have passed from Red River leaving in its wake little or no mortality (Ross 1957: 362). In the end, this affliction at Red River was more of a nuisance than a plague.

The source of this disease is not mentioned in any of the records and remains unknown. Perhaps it had come from Pembina where the American trader Norman Kittson had a post frequented by smugglers from Red River. Kittson obtained his supplies from St. Paul, Minnesota and this would have provided an excellent route for the virus to pass to the Red River. The influenza epidemic of 1845, for instance, had spread from the upper Red River northwards (Ray 1976: 148, 149). What is certain, however, is that the disease spread far afield from this community, first to the Boundary Waters and from there to the northern end of Lake Winnipeg and beyond (Figure 23).

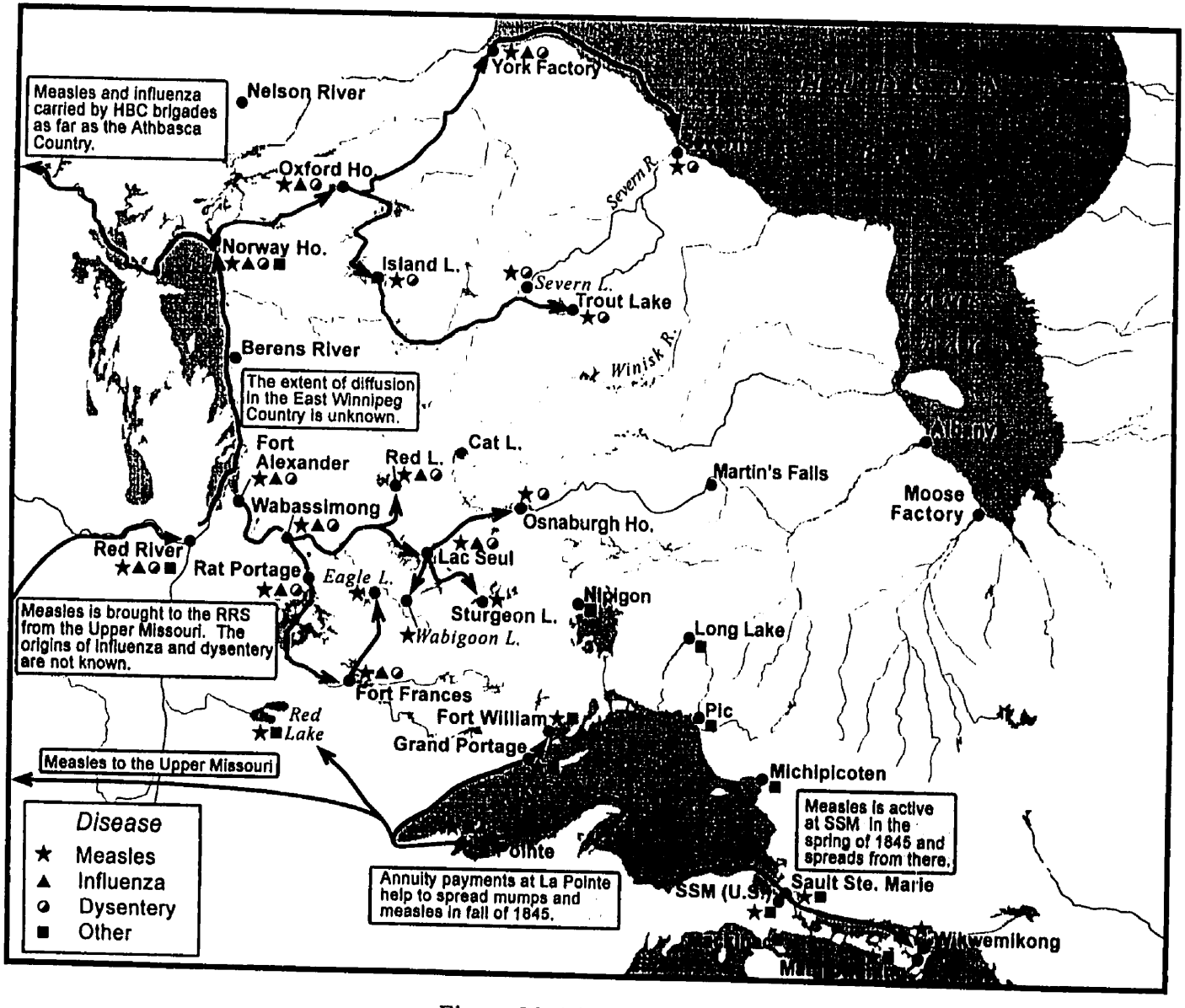

Figure 23: The 1846 epidemics

Although there are no journals for the HBC posts at Fort Alexander, Rat Portage and Lac la Pluie for this period, there is evidence of influenza among the Indians of this region. Charles McKenzie, the long time trader stationed at nearby Lac Seul, learned on March 25 that "the people of that place [Rat Portage] and quarter are all laid up with the Influenza of a malignant nature which of course will soon find its way here by the intercourse of Indians of that quarter with our own" (HBCA B.107/a/24: 19). On May 19 he learned by a letter from Lac la Pluie that "they suffer in that quarter much from Influenza" (*Ibid.*: 23d). The disease did not halt its diffusion there, and McKenzie's fears proved well-founded only days later. As he had predicted, influenza spread to his own fort as, on June 1, he noted in the journal "Our men are entirely laid up with that Confounded Influenza which the Indians brought from the River Winipic" (HBCA B.107/a/24: 25). For McKenzie, this was another devastating blow to the trade at Lac Seul, and to his Indians, and his comment concerning the health of his district illustrates his frustration at a seemingly endless string of sickness. He lamented that: "Surely never was there a more sickly year than the present. Sickness overtook us in the Albany River last Summer–and I do not believe that this fort was a month without sickness since" (*Loc. Cit.*). McKenzie did not know, however, that worse lay in the near future for, just as he was preparing to depart for Albany Fort, two other diseases, far deadlier than any that he had seen in the previous few years, were making their way towards Lac Seul.

Six days later McKenzie and his men departed for Albany Fort with the two boats, most of them, including himself, suffering from influenza. It would be with great

difficulty that they would get to Albany.

> Some of the young Indian voyageurs were Spitting Blood among other Corrupt Matter—those who were well yesterday are the worse to day—this cough did not allow myself an hour of comfortable sleep last night-and they tell me that I am only at the beginning-nevertheless-God willing! we must make a start to morrow morning—and surely never a more miserable crew faced the Albany River with so valuable a cargo. (*Ibid.*: 25d)

As they passed Osnaburgh House on the 11th, post master George McPherson penned a letter to Thomas Corcoran at Albany stating that

> M^{r} M^{c}Kenzie of Lac Seul arrived here in the 11th + went off again yesterday in company with our boat + returns, c. M^{r} M^{c}Kenzie and his men arrived here all sick, the most miserable brigade that I ever witnessed, and I do not know how they will reach Albany if they Continue in that state of Health— they obliged to leave two men here not able to go further, say Tom Tait + an Indian and taking two fresh Indians in their places. (HBCA B.155/a/57: 20)

Despite the movement of the infected brigade down the Albany the disease had reached the eastern extent of its diffusion at Lac Seul. Although two men were left at Osnaburgh, there is no evidence that influenza spread to any there. The two quickly recovered and so did the "miserable brigade," before it reached its destination (*Ibid.*: 4). At the same time, however, influenza spread northward from the Boundary Waters to Norway House and westward from there. By that time the disease's diffusion had merged with that of the next major epidemic of 1846, measles. The diffusion to Norway House will be dealt with in the context of that measles epidemic.

MEASLES

The second of the three sicknesses to strike the Petit Nord in 1846 was measles, an infrequent visitor to the region. According to Charles McKenzie there was an outbreak of measles in the spring of 1829 to the southwest of Lac Seul, near Wabigoon Lake, but it appears to have affected only a very few who had not contracted the disease during the 1819-20 epidemic (HBCA B.107/a/7: 15d, 16, 16d, 18). For many other groups in the Petit Nord, their previous experience with the disease had been in 1819-20, just over twenty-five years earlier, and the first major epidemic of the nineteenth century. Still others had had no recorded exposure. Between the two major epidemics a generation had been born and reached adulthood, a generation which lacked acquired immunity to this disease. These people, and those who had been spared earlier, provided ample susceptibles to fuel another epidemic and once again the sickness contributed to significant loss of life for the region's people. This time, moreover, it travelled farther into the area than in 1819-20. Indeed, it is probable that, as late as 1846, some aboriginal groups were being struck by this disease for the first time in their history; they were true virgin soil populations even as late as the mid-point of the nineteenth century.

Origin

As it had in 1819, measles came to the Canadian Northwest from the plains, funnelled through the Red River Settlement. This was only the immediate source, however, and its path may be traced back eastward, to the Great Lakes, and presumably beyond that to eastern North America. The few who have considered this epidemic have

pointed to the American Plains as a source of the virus (Galois 1996: 31), or specifically to the settlers travelling along the Oregon Trail (Taylor 1977: 60; 1982: 60, 63, 65). Neither, however, was the source of the infection at Red River. Evidence from the country to the east of the Red River points instead to an origin centred on Sault Ste. Marie, the gateway to Lake Superior. This was typical of many of the diseases that were reaching the interior of the continent at this time.

Measles first appeared at Sault Ste. Marie in the spring of 1845 according to the United States Indian Agent James Ord (United States 1845: 503). By mid-July it was prevalent among the people on both the Canadian and American sides of the river, and had proved fatal in some cases[1] (HBCA D.5/14: 138). This disease remained active there through July and August and towards the end of September, and by that time "several lives [had] been lost" (HBCA D.5/14: 298d; O'Meara 1847: 35). As late as October 6 measles was still rampant among the Indians of nearby Garden River (*Ibid.*: 37).

It was exactly this situation, widespread infectious disease present at Sault Ste. Marie late in the summer, that had long worried the HBC traders of Lake Superior. They

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[1]It is not clear how the disease arrived at the Sault. However, by 1845 connections with the settled regions of the east were such that *ACIs* could easily diffuse to the eastern extremity of Lake Superior by any number of means during the open season of navigation. Moreover, nearby American communities such as Mackinac and Detroit were even more strongly tied to the urban east, and to that region's disease pools. By this period travel times could be extremely brief. For instance, in the spring of 1847 Frederick Graham voyaged from Boston to Mackinac in less than two weeks, travelling by train from Boston to Buffalo and from thence to Mackinac by steamboat (Nute 1950: 40-41). In the annual report of the Commissioner of Indian Affairs for 1845, the Reverend Peter Dougherty explained that attendance was down at the Indian school at Grand Traverse Bay, on the east shore of Lake Michigan, "owing to the fact, that the school has been almost broken up by the introduction of the measles in the place. A family that wintered up the lake returned here this spring [1845], bringing the disease with them. It has spread and is spreading throughout the village" (United States 1845: 576). Depending on what Dougherty meant by "up the lake" he may be identifying a southern focus for the disease, or confirming the presence of the disease towards Mackinac and Sault Ste. Marie.

knew, and dreaded, that the Indians who traded furs at their posts would be heading east to participate in the British government's annual distribution of presents, held in 1845 on Manitoulin Island, and that the Indians would be heading into the very heart of an epidemic at Sault Ste. Marie. To make matters far worse, at the time of these proceedings, when thousands of Indians collected at the village of Manitowaning on Manitoulin Island from all over the Upper Great Lakes, the disease was spreading through not only that community but also the Roman Catholic village of Wikwemikong and the nearby mainland (O'Meara 1847: 34; Figure 23). In 1845, according to Paul Kane (Garvin 1925: 10) who travelled to Manitoulin Island from Toronto, this included Indians "from all parts of the shores of Lake Huron, Nipissing, and Superior, as well as from all the neighbouring islands." Indeed, it is likely that these visiting Indians were responsible for carrying measles to Manitoulin in the first place for, according to Frederick O'Meara, the Anglican missionary stationed at Manitowaning, the disease had been brought by "some of the strange Indians" (O'Meara 1847: 34). Although it is not known, it may be that the disease also diffused among the 2,600 Indians on the US side who were gathered at Mackinac from all over the territory south of the border (Garvin 1925: 18).

In July the Lake Superior Indians began to make their way to Manitowaning. On July 14 Chief Trader John Ballenden wrote to Governor Simpson informing him that he had met some Indian people heading eastward and had attempted to keep them from continuing on to Sault Ste. Marie. He wrote that:

> On my way to Michipicoton this season, I met a party of Indians from Fort William, who were on there [*sic*] way to Manatowaning to receive a portion of the presents distributed there annually by the British Government among the Indians. Previous to my departure from Sault de

> S^te^ Marie, the measels [*sic*] were prevalent among the Inhabitants on both side of the river, + in some cases had proved fatal. Fearing that these Indians might be infected by that disease, + on the return introduce it among the natives in the Interior, I urged them to return to Fort William, representing to them the risk they incurred, + the consequences to themselves and families should they be infected with the malady, but they persisted in proceeding on their Journey. (HBCA D.5/14: 138)

Prior experience may have given Ballenden the ability to predict what would happen, but he was nonetheless impotent to prevent it. Once again, the Indians of Lake Superior headed east to partake of the British Government's largesse and, regrettably, to expose themselves and their families to the diseases of civilisation.

The ceremonies over, late in August Ballenden was visited at Sault Ste. Marie by the Indians returning to Lake Superior. As he had predicted, "the Lake Superior Indians who went to Manatowaning have caught the infection, and one of their Children died at Missassague[2]. They passed here about a week ago, several of the children still unwell" (HBCA D.5/14: 299). Once again Ballenden tried desperately to prevent the spread of the epidemic to the Indians of Lake Superior, this time recommending that these people remain near Sault Ste. Marie, rather than continue to their home territories (*Ibid.*: 299-299d). Although he was unable to persuade them to stop, they nonetheless failed to reach their destination. They were still at Sault Ste. Marie as of September 9, barely surviving with the aid of the HBC (HBCA D.5/15: 658). This group had departed Sault Ste. Marie attempting to continue to Fort William, but the illness of most of their group had forced them to return. By that date, four had perished, an old woman, two boys and a child, and others were so low as to raise doubts about their recovery (*Loc. Cit.*). Even before they

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[2]Mississagi, on the north shore of Lake Huron just to the west of Manitoulin Island.

reached Sault Ste. Marie they had begun to suffer from that common sequel of measles, bronchopneumonia, and Ballenden's assistance of food, medicine and shelter seemed not enough (*Ibid.*: 658-658d). Ironically, though, by virtue of the severity of the disease, in this instance measles was prevented from diffusing along the north shore of Lake Superior.

As did Ballenden, the HBC men stationed farther west and north of Lake Superior worried about the possibility that such a terrible disease might spread among their Indians. Thus, at Michipicoten John Swanston wrote in July that:

> The measles which were said to have made their appearance at the Sault S[t] Marys in the early part of the present month, have not I am happy to state, got this far, and I trust through the merciful interposition of a kind providence that we shall be permitted to escape this dreadful scourge, as it would make sad havoc amongst the natives should it unfortunately get amongst them. (HBCA D.5/14: 190d)

The disease had still not progressed as far as Michipicoten by September 16, although another "eastern" disease had. Swanston informed Simpson that he, his family and all of the servants had been plagued by a severe cold, "brought hither from St Mary's, by some of the Indians on their return from the presents" (HBCA D.5/15: 84). He also noted that measles had not arrived and the chances of it diffusing there were decreasing as the season progressed, as communication with Sault Ste. Marie was drawing to a close[3] (*Loc. Cit.*). At Nipigon, James Anderson expressed similar concerns on December 26, although

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[3]He wrote:
With all our misfortunes we have every reason to be thankful that we have so far escaped that scourge the measles, and as our communication with the Sault is now nearly brought to a close, for this season, I hope that we may be so fortunate as to escape them altogether, for it would be a most dreadful and fatal disease, if it was to get amongst the poor Indians.

the health there was excellent among whites and Indians alike (*Ibid.*: 612d). In the end, the delays caused by the severity of the disease were enough, and none of the Indians between Sault Ste. Marie and Lake Nipigon was exposed to measles at this time.

Instead, the virus spread south of Lake Superior, accompanied by mumps, which had also been at Sault Ste. Marie during the spring and summer (United States 1845: 503; Figure 23). The latter disease was also epidemic in 1845-46 and crossed into the Petit Nord, although most of its diffusion occurred in American territory. Mumps was not a frequent visitor to the Canadian Northwest prior to the 1840s, as the only earlier direct references occurred in the fall of 1841 at York Factory (HBCA D.5/6: 329d), and at Isle à la Crosse and the Red River Settlement in 1844 (Ray 1976: 156: fn. 56; PAM CMS C.1/0 Microfilm reel A84, Reverend William Cockan to the Secretaries of the Church Missionary Society, Grand Rapids (Red River), No Date [Marked Received 3 April, 1844; Postmarked Lachine, 19 March, 1844]).

At the HBC's post of Lac D'Orignal (or Moose Lake), an outpost of Fort William near Grand Portage on the Pigeon River route, 1846 started with an outbreak of mumps. This disease had been brought from La Pointe, in U.S. territory and south of Lake Superior, by Indians who had travelled there to receive annuities from the Americans[4] (HBCA D.5/16: 115, 135). Once again the large, temporary gatherings of Indian people

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[4]Fort William Post Manager John Mackenzie informed Simpson that:

> I had a visit from old [Antoine] Collin from Lac D'Orignal about Christmas who informed me that 9 families of his Indians had last Fall caught a disease commonly called I believe the Mumps which rendered them incapable of doing any thing + that three of them were at the point of death when he left. This disease was brought into the district by a set of worthless fellows who were in the habit of going every year to Lapointe for a

created by government policy had inadvertently enabled the diffusion of infectious disease. La Pointe was the location of the payments of annuities to those bands who signed the 1842 treaty, and this included those living in the Upper Mississippi and along the south shore of Lake Superior (Wentworth 1892: 12-13). These payments were, then, a potent mechanism of infection and would have fostered westward diffusion from the eastern settlements.

Mumps persisted in the Grand Portage area throughout the winter of 1846 (HBCA D.5/17: 214a). By February, it had progressed as far north as Lake Nipigon as James Anderson informed Sir George Simpson that:

> The Indians and the people of the Establishment enjoyed excellent health till the month of February, when two hands were attacked with the Mumps, some of whom are still unwell, fortunately with one exception, they are indifferent hunters. (HBCA D.5/17: 246)

Apparently, it had also continued its westward diffusion as it was epidemic at Red Lake, Minnesota, during the winter of 1845-46 (Minnesota Historical Society [MHS], American Board of Commissioners for Foreign Missions Papers Correspondence [A.B.C.F.M.], May 1844 to May 1848, MSS: No. 29, BA10.A5126, Box 6: 8-9, F. Ayer to D. Greene, Red Lake, March 10, 1846). Finally, there was also an outbreak of the same disease at Fort Union on the Upper Missouri in the winter of 1847-48, witnessed by John Palliser (Palliser 1856: 84). This last, however, was two years after Red Lake and there are no known intermediary outbreaks, and so the two may not have been related, but instead were symptomatic of the growing frequency of acute infectious disease west of the Great

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share of the presents distributed annually by the American Government. (HBCA D.5/16: 115)

Lakes.

Measles also moved westward, travelling south of Lake Superior, and its spread also seems to have benefited from the gathering at La Pointe. According to James Anderson at Lake Nipigon, both measles "and a kind of putrid sore throat" were prevalent at Fort William by Christmas (HBCA D.5/15: 612d). There is no evidence that the disease diffused north and eastward along the Lake Superior coast at this time. Instead, it continued spreading to the west. During the winter it was rampant at Red Lake and few escaped its ravages (MHS, A.B.C.F.M: MSS No.29, F. Ayer to D. Greene, Red Lake, March 10, 1846, *Op. Cit.*). Between fifteen and twenty were thought to have died there at this time, about half of them very old people (*Loc. Cit.*). This suggests that the Ojibway of Red Lake (Minnesota) had not been exposed to measles in 1820 unlike the nearby Sandy Lake and St. Croix Ojibway, who had suffered severely (Warren 1984: 335).

This pattern of diffusion involving the Red Lake Ojibway and Lake Superior, and the presence of the disease west of Lake Superior, is not unexpected. In 1842-43 the American Missionary Frederick Ayer was stationed at Red Lake and noted the patterns of movement of these people. He wrote that:

> When the rivers open in the Spring, the men generally leave, and descend the Red River to the Colony... They are absent about 20 or 25 days, The principal object of this visit to the Colony, is to traffick sugar with the half-breeds and others, for which they receive clothing and goods. Again in the first part of June, a considerable number start out to hunt buffalo in the plains to the West. A few also visit La Pointe and the Sault Ste Marie during the summer. With these exceptions, they spend the spring and summer at Red Lake. The men only go to the Colony and on their hunts in summer. And in their winter hunts, the men do not usually take their families with them. ... As a general thing the women and children remain here both summer and winter. (Quoted in Moodie and Kaye 1986: 180)

Ayer also stated that their number included 100 warriors and 500-600 women and children (*Loc. Cit.*). Thus, the Red Lake people were an excellent vehicle for the westward diffusion of the virus, especially since they probably had been present at La Pointe, which was the location for distributing the government annuities that year[5]. Winter counts kept by the Lower Yanktonai Sioux (Howard 1976: 51) confirm the presence of the disease in the Red River-Upper Mississippi area at about this time, although some counts, and at least one U.S. government report, suggest that it may have been active in the area in 1844-45, as well[6].

Thereafter the disease continued its westward diffusion before heading north. It is evident, for instance, that measles was present on the Upper Missouri during 1846. Winter counts belonging to both the Mandan, and the Hidatsa who lived with them (Howard 1960a: 32; Beckwith 1969: 32), identify measles or a measles-like disease for

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[5] As we have seen, mumps was spreading from La Pointe and since the Red Lake people also had mumps it seems likely that they, too, had been to Lake Superior.

[6] In the Annual Report of the U.S. Commissioner of Indian Affairs for 1845, an undated extract from the report for the mission school at Lac Qui Parle adds some confusion to the question of timing. According to Thomas Williamson, "The measles and dysentery caused a great mortality among the Dakota last fall and winter. Out of a population of 400 souls who reside here some part of the year more than 30 have died within a year" (United States 1845: 564-565). Lac Qui Parle is on the Minnesota River near Lac Travers. It seems likely that an unconnected outbreak of measles was active at Lac Qui Parle a year earlier than at Red Lake. Less likely is that the disease had started in the west, worked its way east and then returned to the west. Measles was said to be among some of the families travelling along the Platte River Trails, during the spring or summer of 1845 (Mattes 1988: 64; Chambers 1975: 11). Alternatively, it may be that something was incorrect about Williamson's timing or that it was written later than other reports contained within the Commissioner's Report. Several of the Teton counts that identify epidemic disease at this time indicate that the mortality level was low or that none died (Howard 1960b: 377), an interesting comment considering the devastation wrought by the 1819-20 measles epidemic. None indicates that the mortality rate was high. The body of winter counts suggest that many of the Teton had been exposed to this earlier measles epidemic, while the Yanktonai were not [Compare the John K. Bear count (Lower Yanktonai — Howard 1976: 44, 51) with, for instance, the High Hawk count (Brule Teton — Curtis 1970: 172, 175)]. Perhaps prior exposure some 27 years earlier helped to lessen the number of fatalities among the Teton.

this period[7]. Several Teton winter counts, although not all, describe epidemic disease that may be interpreted as measles in about 1846[8] (Cohen 1942c: 29; Curtis 1960: 175; Howard 1960b: 377-378; Mallery 1972: 322). The disease also appeared among the Arikara, living in villages located near the Mandan and Hidatsa, some time in 1846. A first hand account of the effects of the epidemic, if not of the epidemic itself, has been left by an American Indian agent who visited these villages after the epidemic had subsided. On May 23, 1846 the AFC's *General Brooke* left St. Louis, the first steamship to make the journey to the Upper Missouri country that year. Among its passengers was the Indian agent for the Upper Missouri, Thomas P. Moore (Sunder 1965: 86). After a leisurely journey, Moore finally reached Fort Clark in July,

> contiguous to which reside the entire Arickaree nation. They had been recently visited by the measles; and, in consequence of their ignorance of the disease, and its appropriate remedies, it destroyed a large number of them[9]. (United States 1846: 78)

Although recently broken out, the disease had evidently had sufficient time to spread throughout the Arikara nation, which numbered about 600 prior to the epidemic (Sunder 1965: 83, fn. 12). Thus, what remained of all three of the village tribes of the Upper

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[7]The Butterfly count specified smallpox for 1845-46 (Howard 1960a: 32) while the Foolish Woman count noted chicken pox for 1846 (Beckwith 1969: 312). At this time the Mandan and Hidatsa were living just to the north of Like-a-Fishhook, which was located on the same site as Fort Berthold, built in 1845.

[8]Significantly, a few do not, suggesting that the epidemic was not universal among the Dakota. See, for instance, the Lone Dog count (Mallery 1972: 282). John K. Bear's Lower Yanktonai count also notes the disease (Howard 1976: 51). Battiste Good's mnemonic phrase was "Broke-out-on-faces-had-sore-throats-and-camped-under-the-bluff-winter" and "Also-had-a-bellyache" (Mallery 1972: 322). The latter may be referring to the effects of dysentery.

[9]Meaning the Arikara. Report of Thomas P. Moore Indian Agent for the Upper Missouri Agency, Sept. 21, 1846.

Missouri had contracted measles in 1846. Interestingly enough, several of the Arikara attributed the disease to the whites[10] (United States 1846: 78).

Once on the plains measles spread widely, infecting many of the more northerly groups. Indeed, one settler stated that in 1847 "Measles was general that year on the Plains[11]," (Quoted in Boyd 1994a: 12). Just how far the epidemic spread is not known, but it must have rivalled the most far-flung epidemics preceding it, including the smallpox epidemic of 1779-83. According to Ray (1976: 152) this epidemic touched most of the area lying between Lake Winnipeg and the Rockies, eventually reaching as far north as Great Slave Lake through the HBC's boat brigades. Moreover, the epidemic also crossed over the Rocky Mountains to California and the Pacific Northwest, thereafter spreading northward through much of present-day British Columbia through a combination of white settlers, HBC transport and Indian trade networks, and also into Alaska (Boyd 1990, 1994a; Galois 1996). Eventually, the disease was carried across the Pacific to Hawaii aboard an American warship (Boyd 1994a: 47, fn. 85). Influenza

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[10]In the Pacific Northwest the introduction of the disease was also blamed on whites by the Indians of the Columbia River valley and around Fort Simpson. In the heavily edited published version of Paul Kane's journal the infamous Whitman massacre was said to have been triggered by the effects of this measles epidemic (Garvin 1925: 222-225). However, MacLaren (1989: 16) demonstrated that this information, which was not in the draft journal, was simply added later by whoever was editing the published journal. See also Schlissel (1982: 75, fn. 31) and Boyd (1994a). The disease was active among a great many of the Indians living near the juncture of the Powder and Snake Rivers, in eastern Montana, in the winter of 1847-48 (Ebbert 1918: 264). Many were said to have died due to the use of the cold plunge for treatment (*Loc. Cit.*). In 1848, measles was carried by the Mormon immigrants into the Great Salt Lake region, perhaps part of the same general epidemic (Stoffle, Jones and Dobyns 1995:183).

[11]Farther down the Missouri River from the Mandan, Hidatsa and Arikara, at Council Bluffs, Subagent R.B. Mitchell wrote in his annual report, dated September 11 1846, that the Ojibway, Ottawa and Potawatomi of that subagency, living about the Missouri River, had suffered much from unnamed and varied diseases, and that they had lost an estimated ten percent of their population (United States 1846: 88). One of these may have been measles.

generally accompanied or preceded measles throughout the Northwest, and may have reached the Pacific coast with that disease. Galois (1996: 34; 40) noted the presence of influenza at several places in British Columbia during the westernmost extension of the measles epidemic. Earlier, however, measles also diffused northward to the Red River Settlement from the Missouri, and from there into the Petit Nord.

The Red River Settlement

Measles arrived at the Red River Settlement in May of 1846 (Ross 1957: 362). Its path may be pieced together from statements made by three men occupying significant positions in the colony. According to the Anglican missionary William Cockran, the disease had "marched towards the west, from the United States, into the plains and the vagrant hunters brought it to the Settlement" (PAM CMS C.1/M.4: 68, Microfilm reel A78, "Reverend William Cockran's Journal from August 1845 to June 1846," May 24, 1846). There is nothing unreasonable in Cockran's statement, and it appears to fit the portrait of the disease's diffusion presented above, that is, that it spread westward from Sault Ste. Marie along the south shore of Lake Superior to Minnesota, and subsequently found its way to the Upper Missouri and then to the Red River Settlement. There can be little doubt that these hunters had brought the disease back from an excursion to one of the American trading posts on the Upper Missouri, or through contact with one of the American tribes, since Father J.-N. Provencher, the Roman Catholic bishop at St. Boniface and someone well acquainted with the goings on in the Settlement, wrote that

measles had come from the Missouri[12] (PAM MG 7 D13 Microfilm reel 1 *Belleau Collection*: Bishop J.-N. Provencher to J. Signay, St. Boniface, 16 June, 1846.). Finally, Chief Factor Alexander Christie of the HBC wrote to Governor George Simpson from Fort Garry on May 25 and informed him that:

> We are all here much in the usual way, with the exception that from the continual intercourse between the Settlement and the Assiniboins during the last Winter, the Measles were brought amongst us, and are now going over the Settlement. (HBCA D.5/17: 287)

This disease, then, had been brought to the Red River Settlement from the Missouri River by some of the Assiniboine, either directly or indirectly (Figure 23). Once again, the Red River Settlement acted as the entry point or conduit for epidemic disease into the Canadian Northwest.

The Assiniboine, who by 1846 were living much farther west of the Red River than they had in La Vérendrye's time, had long since begun trading with the American traders, primarily at Fort Union at the confluence of the Missouri and Yellowstone Rivers (Ray 1988: 212). Despite the fact that the Americans had only been trading on the Upper Missouri since the beginning of the nineteenth century, this trade was not greatly different from that which these people had engaged in for centuries. Even before the Americans settled these posts, the Indians, métis and white traders annually went south to trade with the Mandan, a pattern witnessed as early as 1738 by La Vérendrye, and one which may have dated to the seventeenth century (Ray 1988: 89). When the Americans opened posts

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[12] The full text is:

Je n'ai point vu M. Belcourt depuis l'ariveé des lettres. Ses sauvages sont malades ainsi que presque tout le monde dans le pays. La rougeole, **venue de Missouri** [Emphasis mine], est la maldie [*sic*] du temps; des sauvages en s'ont morts.

on the Missouri River, these too were frequented by Indians living north of the international border. One unintended consequence of these trips was, unfortunately, the diffusion of disease from the American plains into the Canadian Northwest. In 1837-38 this trading connection proved costly as smallpox caught at the Fort reduced the Assiniboine numbers greatly (Ray 1988: 187; see also Ray 1975), just as smallpox in 1781 and measles in 1819 were contracted by the Canadian tribes while raiding or trading with the village tribes of the Upper Missouri. In 1846 the pattern was repeated and measles was carried to the Red River Settlement.

Once within the Settlement, measles spread throughout with considerable rapidity. Within parishes this rapid diffusion was likely abetted by the churches that would have facilitated widespread diffusion among parishioners during the services. On May 24 Reverend William Cockran noted in his journal that the disease had already made significant inroads among the congregation of the Anglican church in St. John's parish. That day he:

> Rode to the Upper Church [St. John's] and performed the divine service. A very thin Congregation. Whole families are confined to their houses and bed by measles. This disease having never visited the settlement before, it finds numerous victims. ... Many persons have suffered severely from it, but thank God it has not yet ended fatally in any case[13]. (PAM CMS C.1/M.4: 68 Microfilm reel A78, "Reverend William Cockran's Journal

[13]Cockran's statement regarding the lack of prior exposure to measles in "the settlement" is either erroneous or refers only to the "Indian Settlement" of St. Peter's since the disease had raged in the Red River Settlement in 1819-20, admittedly long before he arrived at Red River (See Hackett 1991), and perhaps again in 1828 (See Chapter 9). Ens (1989: 186-187; 1996: 107) used this statement in support of his conclusion that this was a virgin soil epidemic. In his view, since the Red River Settlement population had never before been exposed to the disease, mortality should have been extreme and among all ages. Ens's (*Loc. Cit.*) contention that mortality from this measles epidemic and its "complications" was very high is incorrect, given the testimony of observers. Instead, it was dysentery that was responsible for most of the deaths.

from August 1845 to June 1846").

The sickness had yet to reach lower down the Red River to St. Paul's, since the same day Cockran "Read prayers and preached in the Middle Church in the afternoon, [and had] a good Congregation [,] the epidemic has not yet become general in this part of the Settlement (*Loc. Cit.*)." By June 7 measles had travelled down the Red to St. Paul's Parish for on that day Cockran noted in his journal that he had "Read prayers and preached at the Middle Church. The Congregation slender owing to the measles being now prevalent in this quarter (*Loc. Cit.*)." On June 4 Reverend John Smithurst first made note of the disease at the Indian school at St. Peter's when an employee of the school, a Mrs. Cook, came to work while exhibiting symptoms (*Ibid.*: 107). Ten days later it had spread widely among the people of that parish, and by June 16 nearly half were afflicted with measles[14] (*Ibid.*: 108, 109). Meanwhile, the same sickness had diffused throughout the Roman Catholic parishes along the Assiniboine as, on the 16th of June, Bishop Provencher wrote to Bishop Bourget of Montreal, explaining that the Indians of Father Belcourt's Baie St. Paul community had the measles, as did almost everybody in the region[15] (PAM MG 7 D13 Microfilm reel 1 *Belleau Collection*: Bishop J.-N. Provencher

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[14]On June 4th he wrote "On going to the school this morning to proceed with the clothing of the children I was surprised to see Mrs. Cook with the measles. I at once sent her home sorry she had been among the children." On June 14th he "Attended school. Very few present. Many are now sick with the measles. The disease is making its way with great rapidity among the Indians at present however in a mild form". Finally, on June 16th he wrote: "Nearly half the Indians being under the measles I have to supply them medicines and other little things necessary. This is by far the heaviest visitation our little community has ever experienced." (PAM CMS C.1/M.4: 107-109 Microfilm reel A78, "Reverend J. Smithurst's Journal from April 1st to July 1st 1846")

[15]The text reads: "Sir George [Simpson] n'avait pas encore vu M. Belcourt qui est retenue chez lui par la maladie des sauvages; la rougeole a jeté tout la monde sur la grabât; il m'a dit qu'il desirait le voir." See also Provencher's letter to Bishop Signay, quoted above.

to Bishop Ignace Bourget, St. Boniface, 16 June, 1846). Diffusion was rapid and the epidemic involved all of Red River. If indeed this disease had been introduced to the Red River Settlement through the western parishes, as seems probable, it had moved rapidly eastward along the Assiniboine and then northwards down the Red, spanning the entire regional community in a matter of a few weeks.

DYSENTERY

Shortly after measles arrived in the Red River Settlement a far more mortal affliction, the third of 1846, joined it in attacking the now weakened population. In June dysentery appeared first among the people of the White Horse Plain, also called St. Francois-Xavier, "and soon spread with fearful rapidity and fatal effect among the whites" (Ross 1957: 363). Although the disease entered through White Horse Plain there is no other evidence suggesting its origin[16]. Eventually it would spread as far north in the Red River Settlement as the Indian Settlement at St. Peter's, according to the Anglican missionary John Smithurst, where it raged until near the end of September (PAM CMS C.1/M.4: 101, 197 Microfilm reel A78, "Reverend J. Smithurst's Report for the Year Ending Aug 1st, 1846"; Reverend John Smithurst to the Secretaries of the CMS, Red River Settlement, 18 November, 1846). As did measles, dysentery escaped the confines of Red River, although in some cases masked by the more obvious symptoms of the measles.

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[16]There was a very fatal epidemic of dysentery in the Pacific Northwest during the second half of 1844, brought by ship from somewhere in the Pacific (possibly Polynesia; Boyd In Press), and it is possible that these were part of the same epidemic. Boyd suggested that this was shigellosis.

By most accounts this dysentery epidemic was as mortal a visitation as had struck the Northwest since the smallpox of 1779-83. In severity it rivalled earlier smallpox or measles epidemics, and some observers compared it to the devastating cholera outbreaks in the east and in Europe. Although sharing the same name, this was behaviour unlike the dysentery that broke out periodically among some individuals in the Northwest and which was more of a minor inconvenience[17]. In fact, the term dysentery, also called bloody flux, is commonly used for several disorders with markedly different causative agents and characteristics, and which may vary greatly in severity (Miller and Keane 1983: 347). However, all are marked by a similar set of symptoms involving watery, often bloody, stools, fever, stomach cramps and strain when passing faeces (*Loc. Cit.*). In this case, given the severity of the outbreak, the rapid and wide spread of the disease and the lengthy duration of patient confinement, it was possibly bacilliary dysentery. Bacilliary dysentery, or shigellosis (ICD-10 A03), is a self-limiting, potentially lethal disease with known case fatality rates as high as twenty percent under hospital conditions (Benenson 1995: 421). The infectious agent, *Shigella* bacteria, is transmitted through faecal-oral contact, either directly or indirectly, such as by contaminated food, water or milk, and thrives under poor sanitary conditions and where personal hygiene is inadequate (*Ibid.*: 422), conditions undoubtedly to be found throughout the Northwest. It is communicable for up to about four weeks, and has an incubation period of between twelve hours and one week, depending on serotype, and illness may last between four and seven days on

[17]Dysentery, in both chronic and epidemic forms, had been a significant health problem in the eastern colonies since at least the first decade of the seventeenth century (Duffy 1953: 202-203; Earle 1979).

average (*Loc. Cit.*). Major symptoms include diarrhoea, bloody stools, fever, nausea, vomiting, cramps, tenesmus[18] and even convulsions (*Ibid.*: 421). Unlike some other forms of dysentery, shigellosis is a significant epidemic threat and may result in many deaths.

By mid-June, having spread throughout the communities along the Assiniboine, and begun their attack on those along the Red, both measles and dysentery were carried out of Red River and onto the plains during the annual spring bison hunt. This was especially devastating for the Ojibway and métis of Baie St. Paul and White Horse Plain who had the great misfortune to suffer from both during this communal event (Sunder 1965: 101-102). The hunt, an integral part of the colony's resource economy, was an ideal mechanism for the spread of both measles and dysentery and served to ensure as complete a diffusion as possible among many of the mixed blood and Indian inhabitants of Red River.

There were two distinct groups who pursued the bison herds during the spring, those of Red River and those of the White Horse Plain (Hind 1971: 179), and there is evidence of disease among both parties. Each of these started from a different location, took a different route and generally hunted in a different region (*Loc. Cit.*.; Hargrave 1970: 40). The Red River people started from the Settlement and took the road to Pembina, and hunted in a southerly direction (Hargrave 1970: 40). Those from White Horse Plain headed southwest from the Assiniboine River (*Loc. Cit.*). The noted painter Paul Kane accompanied the Red River people on their hunt in June of 1846 and made

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[18]"Ineffectual and painful straining at stool or in urinating" (Miller and Keane 1983: 1095).

mention of the death of his guide due to measles (MacLaren 1989: 28; Garvin 1925: 62-66). Beyond this he made no other note of sickness among the bison hunters in either his draft or heavily edited published journals. Most likely, Kane simply failed to comment on the diseases as he would later fail to mention the diseases at Norway House as they raged around him, as it is apparent that both measles and dysentery had made significant inroads throughout Red River by the time the bison hunters departed for the hunt.

There is better evidence for disease among the people on the other hunt[19]. This evidence stems from the correspondence of the Roman Catholic priest George-Antoine Belcourt who accompanied the people of White Horse Plain and Baie St. Paul on the spring hunt. Early in June Father Belcourt, whose mission was at Baie St. Paul on the Assiniboine, was prevented from accompanying Father Pierre Aubert to the mission at Wabassimong on the Winnipeg River, by the outbreak of dysentery among his guides (Belcourt 1847: 70). Soon thereafter the disease became general among the people on the Assiniboine River and, when the time for departure on the bison hunt came, they left with it in their midst (*Loc. Cit.*). Father Belcourt, touched by their plight, quickly obtained permission to accompany the hunt onto the plains and left his station on June 22. He carried with him "a good supply of medicine" which, nevertheless, was quickly exhausted (Belcourt 1849: 314). While on the hunt he was employed almost constantly ministering to the sick and dying, of whom three or four died each day. In a single day, he related, he had buried eight people (Belcourt 1847: 70-71). By the 5th of July, when he wrote to his

[19]Both measles and dysentery were active among the people of Father Belcourt's parish, although he mentioned only dysentery in his letter. See Belcourt (1847: 70) and the letter from Bishop Provencher to Bishop Bourget, dated St. Boniface, 16 June, 1846 (PAM MG 7 D13 Microfilm reel 1 *Belleau Collection*).

superior Father J.-N. Provencher, twenty-five people had already died (Provencher 1913: 258). As a result, he and six of the hunters resolved to go to Fort Berthold, near the villages of the Mandan and Hidatsa on the upper Missouri, in order to obtain medicine (Sunder 1965: 101; Belcourt 1847: 71). There he was well received by both the Indians and the man in temporary charge of the post, a M. Bruguier, who provided him with medicine for the sick (Belcourt 1847: 75). On July 29 he left Fort Berthold for the encampment of the bison hunters, and thereafter returned to Red River. By August 2 he was at the forks (*Loc. Cit.).*

The combined effects of dysentery and measles proved devastating for all in the Red River Settlement and beyond. It is significant that Sir George Simpson, who had first arrived in the Canadian Northwest on the heels of the 1819-20 measles epidemic, stated that the sickness in 1846 "led to a greater mortality than at any former period within my recollection" (HBCA A.12/3: 275). The statement is even more astounding considering the devastation of the 1837-38 smallpox epidemic. Overall, however, it would seem that dysentery accounted for the bulk of the mortality during the summer of 1846 and was far more feared than measles, which proved more of a secondary nuisance. In fact, several reliable reports suggest that there were few fatalities owing to measles at this time and that the vast majority of mortality was due to the unrelated but concurrent

dysentery epidemic[20]. This may be illustrated in the case of Partridge Crop, on Lake Manitoba, where the CMS missionary Abraham Cowley noted the effects of first measles and then dysentery. On July 11 Cowley wrote:

> My home correspondence will occupy me a long time I fear, as the measles are rapidly spreading among our people and I attend them there being no one here so well acquainted with such diseases as myself. The present appearances are all favourable though some are suffering acutely. ... I fear lest the Indians who are still here should follow their own conceits rather than my directions and thus unnecessarily expose themselves. Some have left the place lately for I am told the following reason, viz., that their medicines might act the better and that they may be under no restraint in beating the drum &c. nearly all the School children are under it but are doing well. (PAM CMS C.1/M.4: 148-149 Microfilm reel A78, "Reverend A. Cowley's Journal from August 1st to July 28th, 1846")

Nothing in Cowley's writings suggests that measles alone had led to any deaths. On December 17 Cowley followed up with information about the ensuing epidemic of dysentery which, conversely, proved far more fatal.

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[20] See, for example, Ross (1957: 362-363); (PAM CMS C.1/M.4: 101, 113 Microfilm reel A78, "Reverend J. Smithurst's Report for the Year ending Aug 1st, 1846"; "Reverend J. Smithurst's Journal from April 1st to July 1st 1846"); (PAM CMS C.1/0 Microfilm reel A96, Cowley to Reverend Davies, 21 July, 1847); and (PAM CMS C.1/M.4: 273 Microfilm reel A79, "Reverend A. Cowley's Journal from July 25th 1846 to July 21st, 1847"). After the epidemic had largely run its course in the Settlement, Reverend John Smithurst informed his CMS superiors that:

> Notwithstanding the alarming extent to which the measles prevailed the fatal cases among the Indians have been comparatively few, and those few for the most part have been occasioned by mismanagement or by the interference of their own Medicine men. (PAM CMS C.1/M.4: 101 Microfilm reel A78, "Reverend J. Smithurst's Report for the Year ending Aug 1st, 1846")

Nevertheless, some mortality was attributed solely to the measles epidemic within the Red River Settlement. Edwin Denig (Ewers 1961: 115) stated that "The halfbreed settlement on Red River suffered severely at the same time [as the Cree] from the same distemper [measles]," and identified the well known sequel of measles, pneumonia, as a major cause of death for those who succumbed during this epidemic. In the Pacific Northwest it is apparent that in those areas where dysentery followed measles, mortality was far greater than where measles alone appeared, and that some observers blamed most of the deaths on the "bloody flux", or dysentery, rather than the relatively mild measles (see Boyd 1994a: 20-21, 34, 40).

> When I last wrote the Measles were raging amongst us, few, if any who heretofore had not had them escaped. They were followed by the dysentery which also prevailed as an epidemic. This last carried off as far as I am at present informed some of the Indians left here while suffering under it and I do not know if any more have died since we last heard from them since the death of Hewaytinnoos eight Indians of whom three were adults, one a young woman and the other four children. (*Ibid.*: 205 Microfilm reel A78, Reverend Abraham Cowley to the Secretaries of the CMS, Partridge Crop, December 17, 1846)

Alexander Ross, in his history of *The Red River Settlement*, provided a sobering vision of the impact of dysentery at this time. He wrote that:

> In no country, either of Europe or America, in modern times—not under the severest visitation of cholera—has there been so great a mortality as in Red River on the present occasion. Not a smiling face in a summer's day. Hardly anything to be seen but the dead on their way to their last home; nothing to be heard but the tolling of bells, and nothing talked of but the sick, the dying, and the dead. In other more populous places such things might be more common and less horrifying, but in a country hitherto so healthy, and a population so scant, it was a new and awful sight. From the 18th of June to the 2nd of August, the deaths averaged seven a day, or 321 in all; being one out of every sixteen of our population. Of these one-sixth were Indians, two-thirds half-breeds, and the remainder whites. On one occasion thirteen burials were proceeding at once. Many houses were closed altogether; not one of the family, old or young, being left in them. (Ross 1957: 363)

Although there is an element of hyperbole in Ross's statement, it is also clear that many fell victim to these illnesses and, perhaps, that they had a profound impact on the psychological state of the Red River people. Governor Simpson drew a similar comparison in a letter to the Governor and Committee in London written on August 20, stating that: "at Red River the mortality principally among the children, was for about six weeks greater, for the extent of the population, than that occasioned by cholera in any part of Europe or America during the years 1831 and 1832" (HBCA A.12/3: 275d). This was a severe blow to the people of the Red River Settlement.

Other evidence confirms the tragic consequences of these diseases. On the 28th of July Simpson estimated that there had been 300 deaths in the Settlement out of a population of roughly 6,000, during a one-month period (HBCA A.12/3: 242). This amounted to the loss of about five per cent of the total population in a span of about four weeks, and so comparisons with cholera were perhaps not unreasonable. As well, Chief Factor Christie informed Simpson that there had been eight deaths on July 27 alone, and another five the next day[21] (HBCA D.5/18: 89). The Reverend Cowley wrote the Reverend F.W. Rice from his mission at Partridge Crop on July 20, 1847 echoing some aspects of Ross' statement. After describing the wellbeing of his own family, Cowley wrote of the people of the area:

> When I think of my family circle unbroken while so many around us have been called upon to endure the most distressing bereavements, e.g. the father of a numerous family the only survivor of which I think there were several instances in the R.R. Settlement. I also know of a whole encampment of Indians cut down by it (the dysentery) I think myself highly favoured & praise God for his goodness. Health has long since been established but as every where we have sickness & death warning us of hereafter[22]. (NAC MG 19 E9 F.W. Rice Fonds, Rev. Abraham Cowley to F.W. Rice, Partridge Crop, July 20, 1847)

The disastrous consequences were felt throughout the Settlement. In his annual report for 1845-46, dated August 1, 1846, CMS missionary John Smithurst described the

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[21]Simpson informed the Governor and Committee that in the vicinity of Upper Fort Garry alone there had been eight deaths on July 29, six on July 30 and five on July 31 (HBCA A.12/3: 322d).

[22]Ironically, Cowley and his mission were partly responsible for bringing measles to the Partridge Crop settlement from Red River. In June of 1846 he had been briefly at the Red River Settlement before departing on his return for Partridge Crop about the middle of the month (PAM CMS C.1/M.4: 146-148 Microfilm reel A78, "Reverend A. Cowley's Journal from Aug 1st to July 28th, 1846"). While on board the boat taking the flour to the mission two of the men fell ill of the measles. The ship reached the mission on June 29 and by July 11 the disease was widespread throughout the region (*Ibid.*: 148-149).

mortality in the Protestant parishes, stating that:

> The number of deaths in the Upper Church and Rapids District have been great beyond whatever before occurred in the same space of time. The deaths in the Rapids district last week amounted to eleven out of a population of about six hundred. The deaths of the Indian Settlement within the last ten days amount to nine out of a population of about five hundred. (PAM CMS C.1/M.4: 104 Microfilm reel A78 "Rev. J. Smithurst's Report for the Year Ending, Aug 1ST 1846")

By the end of September dysentery was on the decline in his parishes and Smithurst could then tally the fatalities, which amounted to a total of 33 at St. Peter's[23]. Sickness had an equally terrifying effect in the Roman Catholic parishes. On July 9 Smithurst learned of the deaths at White Horse Plain from the Roman Catholic priests, Fathers Taché and Laflèche, mortality which, although the priests attributed to the effects of measles, Smithurst correctly deduced was the result of dysentery[24]. Thus, he wrote in his journal that:

> The Priests informed me that the mortality among the Catholic population during the prevalence of measles has been very great. At White Horse

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[23]Smithurst informed his superiors that:

> Since I last wrote to you on the 1st of August I have had to pass through one of the most trying and anxious seasons that has ever yet fallen to my lot. You will recollect that I then mentioned the prevalence of a very fatal disease, which was making great ravages throughout the whole settlement. During the last 12 days of July there were eleven funerals at the Indian Church, and eighteen in the month of August, making a total of twenty nine in six weeks, out of a population of about 450. (PAM CMS C.1/M.4: 196 Microfilm reel A78, Reverend J. Smithurst to the Secretaries of the CMS, Red River Settlement, 18, November, 1846)

There were also four more funerals in September for a total of 33 during this epidemic (*Ibid.*: 197). However, the epidemic was slackening by the beginning of that month (PAM CMS C.1/M.4: 242 Microfilm reel A79 "Account of Proceedings Aug 1 to Nov 1 1846"). Smithurst was to lose twenty of his students at the Sunday school, alone, during the epidemic (PAM CMS C.1/M.4: 264 Microfilm reel A79, "Rev J. Smithurst's Report for the Year ending Aug 1847").

[24]Father Belcourt, who was at Baie St. Paul until June 22, identified dysentery as the sickness which was devastating the métis, although it seems likely that both diseases were present then and thereafter among the families that ventured onto the plains for the hunt (Belcourt 1847: 70-71).

> Plain out of a population of about 400 there have been about 70 deaths amounting to above 1/6 of the whole. It appears to me, however, from their description, as if the measles were followed by another disease distinct from it and far more dangerous. It is a sort of dysentery or bloody flux[25]. (PAM CMS C.1/M.4: 113 Microfilm reel A78 "Reverend J. Smithurst's Journal from April 1st to July 1st, 1846")

Father Provencher, the sole Roman Catholic missionary remaining in Red River after the departures of Fathers Belcourt, Aubert, Taché and Laflèche, wrote that sickness was killing many in the three parishes of St. Paul, White Horse Plain and St. Boniface, and that he had had to bury as many as nine in one day, mostly children (Provencher 1913 258-59). As noted, dysentery was also making great havoc among those on the bison hunt with Father Belcourt throughout the months of June and July. Thus, throughout the Red River Settlement the summer of 1846 was one of sickness and heightened mortality. Indicative of the severity of this extraordinary period of epidemic disease was a request by Simpson to Smithurst late in July "that the prayer appointed by the Church in times of sickness should be read[26]," (PAM CMS C.1/M.4: 115 Microfilm reel A78, "Reverend J.

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[25]Almost certainly, there would be more dead than this among those who lived at White Horse Plain since the bison hunters and their families had departed early in June and were still succumbing to these sicknesses even after the two priests had left Smithurst on their way to Isle a la Crosse (See Belcourt 1847).

[26]The response of the secretaries of the CMS was, as expected, an equally spiritual one and the two show that Euro-Canadian attitudes towards epidemic disease in the middle of the nineteenth century were in some ways similar to those of the aboriginal people of the continent.

> We are grieved to hear of the succession of severe diseases with which it has pleased God, during the last year, to visit various portions of Prince Ruperts Land; + we heartily sympathize with the Missionaries in the trials which they were called to endure in consequence of the awful calamity around. We would, however, acknowledge with gratitude the preservation of the health of the Missionaries themselves when so many on the right hand and on the left were cut off. (PAM CMS C.1/L.1: 7-8 Microfilm reel A76, The Secretaries of the CMS to Reverends James and Smithurst, Montreal, March 27, 1847).

Smithurst's Journal from April 1st to July 1st 1846").

The two sicknesses subsided almost as suddenly as they had arrived. They had entered the Settlement in late May and early June, and by early August it would seem that the tide was turning on the epidemic, perhaps other than in Smithurst's parishes. According to Adam Thom at Lower Fort Garry on the 7th,

> Within these last ten days the dysentery seems to have taken a favorable turn. The new cases are less virulent as well as less numerous; and perhaps there is also something in this, the people have been so thoroughly frightened that they resort to remedies at an earlier stage of the maladie [*sic*]. (HBCA D.5/18: 113d-114)

Thom's conclusion was echoed by several others who saw an end to what had become a summer of misery[27]. There was, however, another sickness at Red River later in the year. On December 30 Dr. John Bunn informed Governor George Simpson that "The health of the settlement is still far from good a troublesome fever causes much distress and some casualties" (HBCA D.5/18: 539d). By then, however, both measles and dysentery had long since been carried from Red River and had begun a long journey of diffusion. As was the case so often before, in the summer of 1846 these diseases would not be contained within this region.

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[27]A similar opinion was expressed by Adam Beatty and, on August 8, by Alexander Christie (HBCA D.5/18: 95, 119), although on the 1st Christie had informed Simpson to the contrary, stating that:

> "I am concerned to say the prevailing malady continues, with much about the usual daily deaths, our shop man at the Upper fort is no better, and another two of the men are taken ill this morning, we are hopeful that as the season advances health will be returned. (*Ibid.*: 93d)

See also Simpson to Governor and Committee, York Factory, 23 August, 1846 (HBCA A.12/3: 322d).

DIFFUSION FROM THE RED RIVER SETTLEMENT

In June of 1846 the Red River Settlement again played a major role in the diffusion of epidemic disease throughout much of the Canadian Northwest, for both measles and dysentery spread far afield, largely in a hierarchical pattern reflecting the HBC's extensive transportation network. As during several earlier epidemics it was this community's transport linkages to the entire Canadian Northwest via the HBC's boat brigades that enabled these afflictions to spread considerable distances in a short period of time. Once again acute infectious disease spread from Red River into the Petit Nord by two different routes: along the Red River-York Factory corridor and by way of the Winnipeg River.

Although there were instances where other mechanisms were responsible for transporting the diseases out of Red River during the summer of 1846[28], it was mainly the York Boat brigades, quick and operating within a rigid schedule over long distances, that were responsible for spreading sickness to those living north, west and northeast of Lake Winnipeg and the Red River. Later in the summer, the Reverend Abraham Cowley learned from the Swan River brigade that "the Measles have been carried in every direction by the Voyageurs who have suffered severely from it and that many Indians and others have died from it" (PAM CMS C.1/M.4: 274 Microfilm reel A79, "Rev. A. Cowley's Journal from July 25th 1846 to July 21st 1847"). The timing of the initial outbreak could hardly have been worse for, just as these diseases were becoming

[28]For instance, as noted above, the boat delivering flour to the Anglican mission at Partridge Crop carried measles to the people living in its vicinity, while the same disease was carried to Lac Seul by some women who had gone to visit relatives (PAM CMS C.1/M.4: 146-148 Microfilm reel A78 "Reverend A. Cowley's Journal from August 1st, 1845 to July 28, 1846; HBCA B.107/a/25: 1).

entrenched in the colony, the HBC's York Boats were departing from Lower Fort Garry bound for Norway House, on the north end of Lake Winnipeg. These boats were the backbone of the Company's internal supply line at this time and brigades manned by Indian and mixed-blood tripmen served between Red River and Norway House, Red River and York Factory, and Norway House and Methy Portage in the Athabasca country (Morton 1973: 82). Norway House was a central hub in this transport network and played a key role in the widespread diffusion of the 1846 measles epidemic in the Canadian Northwest (Ray 1976: 151-153). Red River also played an important role in this network, supplying most of the mixed-blood labour and, in this instance, providing the initial infection that eventually would spread throughout much of the Canadian Northwest, including into the Petit Nord[29]. Thus, some of the men on board the first boats that departed Red River at the end of May were infected with measles (Ray 1976: 151).

As early as May 25 Chief Factor Alexander Christie had expressed concern about infection aboard the brigades, not for the safety of those who lay in their path, but for the delays that such sickness would cause the Company's summer transport. It was his task to arrange the crews and departures of the brigades from the Red River Settlement, and the measles threatened to hinder his plans. As such, he wrote to Sir George Simpson expressing these views, stating that: "I am most apprehensive of the malady breaking out amongst the trip men, thereby retarding the summer transport very materially - because when the men are exposed to <u>wet</u> in the Voyage, the danger will be infinitely greater"

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[29]In his pioneering study of the diffusion of epidemic disease in the period 1830-50 Ray (1976) delineated the broader patterns of diffusion of this epidemic in the Northern Department of the HBC which included the westernmost part of the Petit Nord.

(HBCA D.5/17: 287). By this period the HBC's boat based transport scheme was so complex and demanded such precision that any long delays could have meant a significant setback, to the point where distant regions such as the Athabasca might not get their supplies during the assigned season, and their furs might sit at the posts for an additional year, tying up the Company's profits. In 1846 this was aggravated by the need to transport several hundred of the 6th Royal Regiment of Foot from York Factory to the Settlement towards the close of the transport season. They had come by ship from Britain and it was impossible for the Factory to support such a large number of people during the winter, over and above the men stationed there[30]. Failure to transport them prior to freeze up would have had disastrous consequences. Consequently, Christie made the decision to dispatch the first brigades from Lower Fort Garry "without delay" (*Loc. Cit.*).

Norway House

The first boats left Red River shortly after Christie wrote this letter, too early for dysentery, which only entered the Settlement in June, but still harbouring the measles virus among their crews. On June 1 Reverend Abraham Cowley met the first brigade on Lake Winnipeg, just to the south of Berens River. That day he "Passed several boats on their way from Red River to York Factory and learned from them that the Measles were in the Settlement, indeed members of the Crews were suffering from it" (PAM CMS C.1/M.4: 144 Microfilm reel A78, "Rev. A. Cowley's Journal from August 1st 1845 to

[30]The troops, under Lieutenant-Colonel John Crofton, arrived at York Factory in August and started for the Red River Settlement on the 28th. The total party, including wives and children, amounted to 369 people (Ingersoll 1945: 15).

July 28, 1846"). By June 7 they were at Norway House (Ray 1976: 151; Figure 23). There, the interim manager[31] wrote in the journal that: "Early this morning 4 Boats of M^{r} Mowet's [*sic*] + 4 of Tho^{s} Sinclairs arrived with cargoes for York Factory + as they had the measles among the crew, I sent them off after taking fresh bills of lading - M^{r} Campbell + family were passengers in Sinclairs Boat + remained here" (HBCA B.154/a/46: 3-3d). The disease had arrived at the north end of Lake Winnipeg.

Here was one of the few examples of the HBC failing to follow a preventative policy with respect to the health of the Indians of the Northwest, a policy it had followed at almost every step during the course of its history. This divergence began with Christie's decision to dispatch the York Brigades from the Red River Settlement without delay, a move that was perhaps necessary in his mind given the disastrous consequences failure to proceed would bring. It continued with the decision to pass on the boats at Norway House without taking appropriate precautions. This time, no concern was shown for the possibility of spreading disease among the native people. Instead of being isolated from the local population, or quarantined on their journey to York Factory, precautions similar to those which had been taken by the HBC during earlier epidemics[32], the diseased boats were simply sent on. In advancing on their journey, these and subsequent brigades spread disease from Norway House to York Factory and west along the

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[31]Ray (1976: 151) believed that this was Chief Factor Donald Ross, and that the decision to send the brigade on was his. However, Ross was then at Lower Fort Garry at the Northern Department Council, along with Governor Simpson and most of the other Chief Factors, and returned to Norway House only on June 21 (HBCA B.154/a/46: 5). Most probably this was instead Weymys Simpson, the nephew of Sir George Simpson's wife (and cousin) Frances. Weymys Simpson was ordered to supervise Norway House while Ross was away, according to James Hargrave writing in March of 1846 (HBCA D.5/16: 317d).

[32]Such as in 1782 at York Factory and in 1820 on the Albany River. See Chapter 7.

Saskatchewan River and beyond, as far as the Athabasca country. Thus, this simple decision was to have dire consequences for many of the people living to the north, northeast and west of Lake Winnipeg.

Ten days later, on June 17, the first local cases of measles appeared at Norway House as the journal recorded "several of the new hands are laid up with the Measles" (HBCA B.154/a/46: 4d). As Ray (1976: 152) has noted, this timing is reasonably consistent with infection by the boats from Red River. By the 19th there had been at least one death among the people at Norway House and the disease had become widespread among the men (*Ibid.*: 5). A week later Donald Ross, who had just resumed control of the post, could foresee the disastrous effects the disease would have on the returns for the year to come, and its impact on the natives[33]. On July 3 the Indians living near the post were "with hardly an exception, under the measles..." and he was finding it impossible to find able bodied men to man the brigades (HBCA B.154/b/3: 17d).

By early July other sicknesses had joined measles at Norway House, and all around were suffering. On July 9 Ross wrote to Simpson stating that:

> Since then [he had written to Simpson a few days earlier] I am sorry to say the sickness has become even more prevalent than before, Influenza and severe bowel complaints have become universal as well as the measles, and the poor Indians are in a truly pitiable condition - many of the Company's Servants are also quite incapable of doing their duty.—by the last account their evil had not yet extended much to the low country, but

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[33]The Returns of this Department for the last Outfit have turned out remarkably well but I am sorry to say that the measles have made their appearance amongst us this spring and are likely to derange our summer business very materially and do much injury among the natives. (HBCA B.154/b/3: 17)

Donald Ross to John McKenzie (Fort William), Norway House, 27 June 1846.

> there is no question that at a later period of the season, they will extend over the whole of the land. (HBCA D.5/18: 30a)

The presence of dysentery too was perhaps inevitable. Although the first boats departed Red River too early for this disease, subsequent vessels were dispatched from Lower Fort Garry after this epidemic had broken out[34]. All were directed to Norway House. The presence of influenza, however, requires some explanation, since it had died out in Red River long before the brigades began to depart for Norway House.

First described at Norway House on July 3 as a very bad cold, this disease was thereafter identified as influenza (HBCA B.154/a/46: 8, 8d), a disease that Ross would contract later in the month (HBCA D.5/18: 66d). Ray (1976: 149) suggested that sickness diagnosed as influenza in 1846 "may have been complications resulting from measles." However, other evidence contradicts this conclusion. Certainly the fact that Ross claimed to have influenza, and had not had measles, suggests that this malady did not result from the complications following measles[35]. Moreover, as we have seen, influenza preceded both measles and dysentery at the Red River Settlement and in the Boundary Waters, and so was present in 1846. Indeed, when evidence from a broader region is taken into account it becomes apparent that there was a widespread epidemic of influenza during 1846. In all probability the malady arrived with the Lac la Pluie brigade,

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[34]Although dysentery was first mentioned directly in the Norway House journal only on August 4 (HBCA B.154/a/46), it is evident that it had appeared far earlier in this region.

[35]William Sinclair at Churchill reported that a sickness that was not measles and which affected the lungs preceded measles at that post (HBCA D.5/18: 131d). It is possible that this was bronchopneumonia arising from a bout of influenza. Measles arrived at Churchill by way of two packateers returned from York Factory on July 16, and by August 2 many of the HBC men and the Indians were suffering from the disease (HBCA B.42/a/183: 53, 54).

which reached Norway House on June 10 (HBCA B.154/a/46: 4) and thereafter spread among the post's men. By July 16 there were "8 men laid up with the Influenza," the same day the Athabasca brigades departed for home (*Ibid.*: 8d). Subsequently, this disease was carried by the boats along with measles to Isle a la Crosse in the English River department (Ray 1976: 153). By the fall of 1846, it had appeared at Forts Chipewyan and Resolution, again almost certainly carried by the brigades (cf. *Ibid.*: 154).

Finally, a fourth disease struck the Norway House region after the others had largely run their course. On December 27 Ross wrote to Simpson informing him that:

> The Measles and Dysentery seem to have passed away, but I am grieved to say a new pestilence has lately appeared in this quarter apparently of a still more deadly nature. I cannot say what the disease really is, but it seems to possess the chief characteristics of a malignant putrid fever very infectious, and with this melancholy particularity, that no one has yet recovered from its attacks, all those who have caught the disease have either died or are still hopelessly lingering under it. (HBCA B.154/b/3: 23)

It is not certain if this unidentified disease spread from Norway House. On January 25, 1847, word came from the Moose Lake region to the west of Lake Winnipeg of continued mortality among the Indians there. Two Indians arrived from that area and, as Ross wrote, "They report very unfavourably of the poor Indians among whom much sickness and deaths still prevail and consequently but little doing in the way of fur hunting" (HBCA B.154/a/46: 16-26d). However, there is also some evidence of dysentery in the region west of Lake Winnipeg about this time (PAM CMS C.1/M.4: 310, 311 Microfilm reel A79, "Rev. J. Hunter's Journal from the 1st of August, 1846 to the 17th of May, 1847"), and it cannot be ascertained whether this represented the continuation of the earlier epidemic or was part of this unidentified outbreak at Norway House.

Initially, only the Norway House men and the Indians and métis manning the brigades were sick with measles, but the disease must have spread quickly to the Indian people living near the post, likely through the medium of returned trip men infected with the virus or simply through contact with the post. On June 28 Ross lamented that "the native population are with very few exceptions laid prostrate with the measles" (HBCA D.5/17: 414). On July 20 there were reports of deaths among the Indians at the nearby village of Rossville, which was run by Methodist missionaries, and which was the home of many of those who manned the freight brigades between Norway House and York Factory[36] (HBCA B.154/a/46: 9). These deaths were not attributed specifically to measles so it may be that dysentery also played a role. Early in August other reports started coming in of deaths among the Nelson River Indians who were generally ill by this time (*Ibid.*: 11, 11d). There the sickness would become "rather severe" before it ended (HBCA B.154/b/3: 27). About the end of August the "Wood Indians," Ross's term for those who did not live in the Rossville settlement (HBCA D.5/17: 277d), began to arrive for debt. Unfortunately, many of the people living near the post still suffered from measles and dysentery, and it is likely that these were passed on to any "Wood Indians" who had not already been infected (HBCA B.154/a/46: 13d).

Estimates of casualties among the Norway House Indians are unavailable. However, according to Chief Factor W. McTavish of York Factory they were "very great"

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[36]Rossville was located about three miles away from Norway House and was founded in 1840 by the Reverend James Evans (Jacobs 1853: 30). By 1844 there were thirty houses, a chapel, a schoolhouse and a workshop. There was also a garden which in 1845 produced nearly 1,000 bushels of potatoes (*Ibid.*: 31). In 1846 there were between 300 and 400 people living there, according to Simpson (HBCA A.12/3: 320).

as of December of 1846[37] (HBCA D.5/18: 430). Donald Ross believed the sicknesses were equally as mortal at his Norway House post as they were at Red River (HBCA D.5/18: 66). Given the rapid sequence of mortal epidemic diseases that struck the people living in the vicinity of this post, at a time of year when they were preparing for the winter, such human devastation was perhaps inevitable. Indicative of the state of despair then prevailing at Norway House, in July Ross felt it necessary to distribute alcohol among the Indians to improve their outlook[38].

While these diseases afflicted most of the people living in this area, perhaps the hardest hit were those who manned the brigades to York Factory. During the period between the passing of the first infected boats and the last boats of the season, which left Norway House on October 3, there are frequent references to sickness and death among the men manning the brigades between Norway House and York Factory[39]. Typical of the experience of the crews was that of the Oxford House freight boats. On July 14 Laurence Robertson recorded in the Oxford journal that

> The 4 Frët [freight] Boats of this place arrived from Y F. On the voyage the [they] got Sick with the Measles and has brought up the Boats with only 3 of the men able to work. —and at present almost all the crew is sick. The Guide is at the point of Death. (HBCA B.156/a/25: 5d-6)

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[37]"Among the Indians the mortality has been very great particularly at this place and Norway House." Chief Factor W. McTavish to George Simpson, York Factory, 1 December, 1846.

[38](HBCA B.154/b/3: 18d) Donald Ross to Alexander Christie, Norway House, 28 July 1846 The state of depression which at present so universally prevails among our voyaging and other Indians is such that I have considered it absolutely necessary to retain a cask of Spirits out of the quantity now passing on to the Settlement, for the purpose of being used in small quantities among the Indians of this place and Oxford House, chiefly as a medicine....

[39]See, for example, HBCA B.154/a/46: 3, 4d, 10d; HBCA B.156/a/25: 4, 14, 6.

The men of the Norway House freight brigade fared no better, and on the 25th Robertson observed "The 4 boats from Norway House in charge of w^{m} Ballantyne Guide arrived from YF after a trip of great suffering from the Sickness of the Crews.—one Indian from Berens River and 2 Indians from this place has died on the passage" (*Ibid.*: 7). The impact of the epidemics on the crews was perhaps best summed up by Chief Factor Hargrave in a letter to Simpson.

> It is with deep pain I have to report that the sickness which I then [his previous letter of July 12] noticed as prevailing here among our indian Freight Brigades, has proved more fatal to the poor natives than our worst apprehensions had then anticipated. The Norway House, Oxford House and York Brigades, have been swept by it for the season, almost all the Outfits for the Northern Districts next year (which are absolutely required to be rendered at Norway House this Autumn) are still in our stores; likewise the whole of the Oxford House and part of the Norway House Outfits for the current season. Four of these Boats, (the last of them from the Interior) reached us on the 6th Ultim^{o}, the crews of which were generally affected with Measles, which malady, in a few days, was communicated both to the Indians around us and to the many of our native servants in the Factory. The latter have now nearly recovered without loss of life, but these boats are still on the beach, many of the crews are dead, and such as remain are either in the last stages of disease or are so enfeebled as to be totally incapable of even the slightest labour this season. (HBCA D.5/18: 180)

In these cases the crews generally had to cease travel and attempt to weather the sickness where the men fell ill or, in some cases, died. At the very least, they were forced to bring up the boats severely undermanned. Such was the case with a brigade that arrived at Norway House on August 4 on its way through[40] (HBCA B.154/a/46: 10d), and those operating to Oxford and Norway Houses (HBCA B.156/a/25: 5d, 6d, 7). Since the

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[40]"the crews of this Brigade suffered severely from the Measles and Dysentery, five of them having been cut off on the voyage."

brigades were manned exclusively by males, and the conditions while travelling no doubt led to heightened mortalities, Donald Ross could comment that "the lamentable mortality among the Indians last summer has left a vast number of widows and orphans unprovided for..." (HBCA B.154/b/3: 21).

For the Company these sicknesses among the men of the brigades during the summer of 1846 "materially deranged" the summer transport, and left cargo for the following outfit in a state of disarray (HBCA D.5/18: 428, 430). As crews fell ill and lost members, and were thereby forced to delay or cut off their journeys, the HBC was unable to fulfil its transportation needs. These losses were to hinder the transport the following year as well, as the labour force had been depleted and there was a much smaller population of able-bodied boatmen from which to choose[41]. Even among the survivors, the suffering which had come to be associated by the Indians with employment on the brigades made it difficult for some posts to hire men[42] (HBCA B.220/a/10: 18d; B.198/a/90: 31).

Finally, there appears to have been a subtle but fundamental change in the way some of the Norway House Indians mourned the loss of the dead during this epidemic, one noted by Ross. Consistent within the fur trade records from a very widespread area

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[41]Ross felt it necessary to inform Nelson River outpost manager John Isbister not to draw upon any of the Norway House Indians, telling him: "at all events you must have nothing to do with any of the voyaging Indians of this place, their number is already greatly reduced by deaths and Sickness, and none of the Beren's River Indians are coming here at all next Summer" (HBCA B.154/b/3: 27).

[42]Similar problems were experienced elsewhere at about this time. For instance, in June of 1845 Charles McKenzie also found it difficult to find Indians willing to accompany his Lac Seul boats to Albany Fort during the epidemic of that year. He found that "They are also loathe to leave their families during the Summer, particularly at this season, as they are some + all laboring under a most violent cough + otherwise sick besides" (HBCA B.107/a/23: 17d).

which included the Petit Nord, was a set of very specific customs which dictated how the living behaved following the loss of a close relative. At least as far back as 1737, when La Vérendrye's son witnessed the Cree of the Saskatchewan River throwing away their furs and those of their deceased relatives "according to their custom" (Burpee 1927: 258-259), and probably much earlier, the Indians of the Northwest had adhered to either one or both of two actions while mourning. During such times they would either discard all of their furs and, in many cases all belongings beyond what they needed for survival, or they would refrain from trapping furs for an extended period of time, or both. Between 1737 and 1846 the records are replete with trader's comments describing just such practices[43]. Elsewhere in 1846, Indian behaviour appears to have been consistent with these beliefs, including at Oxford House (HBCA B.156/a/25: 36d), Fort Frances (HBCA D.5/18: 232; D.5/19: 129d), and on the Winnipeg River (HBCA B.107/a/25: 4).

Such was not the case at Norway House. On September 19 Ross wrote to

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[43]At Albany Fort in 1767, Lieutenant Earchekeshick, an Uplander, informed Humphrey Marten that he mourned the loss of a son by throwing away all his goods, which left him in poverty (HBCA B.3/a/60: 31). Three years later, in July of 1770, Marten was told of a similar act by an Indian who had failed to come to the fort (HBCA B.3/a/62: 34d). See also HBCA B.3/a/74: 19 in which, in 1778, Thomas Hutchins verifies that it is a common custom to throw away everything of value when mourning. In January of 1829 John McIntosh at Lake Nipigon wrote to George Keith reminding him that "you know that an Indian who loses a child or near relation resigns himself to melancholy + gloom for a season, and neglects hunting" (HBCA B.149/a/11: 32). Charles McKenzie at Lac Seul had his own theory for the temporary cessation of hunting. When an Indian failed to pay his debt in May of 1842 due to the loss of a child he noted in his journal that: "and of course - in such cases the trader must 'by right' loose [*sic*] his debt - Should an Indian lose a child (or even a relation) and strive to pay his debt for the rest of that season - all the other Indians would accuse him of being voide of Natural Affection" (HBCA B.107/a/20: 13d). McKenzie also noted that, among the northern Indians, as compared to those of the prairies and the southern Indians, "Here as well as there, [while mourning] they throw away all their property" (HBCA B.107/a/21: 12). Writing in 1807 about the Ojibway, Aeneas McDonell noted that: "They often on the death of a relation give or throw away their clothes and necessaries etc for fear of burthening their deceased relation" (NAC MG 19, C4 Volume 53: 5). See also Bishop (1974a: 210, 225 fn. 61, 249) and Lytwyn (1993: 375, fn. 3). Excellent descriptions are available for the Chipewyan, as collected by Esau (1986: 95-98), which included relocation to lands that were scarce in fur bearer but rich in game animals.

Simpson and informed him that:

> the numerous deaths among the hunters would of itself occasion a serious falling off and amongst Indians the event extends much farther as most of those who survive generally lose all heart and energy and for months on end do little more than moan and mourn helplessly for their lost friends and relatives their little property is either recklessly destroyed or given away thus rendering themselves a great measure unable to hunt even after their fit of despondency may have passed away. I noticed this season however, what I never perceived before, a marked degree of callousness and indifference among the Indians, to the loss of even their nearest and dearest relations and even death itself seemed to have become so familiar to them as to have lost much of its usual terror. (HBCA D.5/18: 466-466d)

Evidently this abandonment of a longstanding custom was seen not just at Norway House, but was in evidence at several of the other Northern Department posts[44]. The following July, Simpson wrote to London informing the Governor and Committee of the fears that he and the traders had had that the epidemic would severely hurt the returns. He informed London of the:

> great mortality that took place last summer among the natives which we were apprehensive would have distracted the survivors (as is usual in cases of mourning) from giving attention to the chase. The discontinuation of this usage, however, reflects much credit on the management of the gentlemen in charge of districts and posts, to whose influence with the natives may, in a great degree, be ascribed the abandonment of many of their old and useless customs. (HBCA A.12/3: 444d)

For the traders this uncustomary behaviour was welcome, for such customs always cut

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[44]At Oxford House, during the epidemic, Laurence Robertson noted similar strange behaviour, recording that he "met 3 Indians coming for a supply of ammunition, stating that if they had such they were going off, so as to shun the light of their Friends Death- and that of their own. I gave none but encouraged those in health to try + bring their Friends in reach of the Fort" (HBCA B.156/a/25: 10d). In October of 1846, Charles McKenzie noted that "the [Lac Seul] Indians are going off as many as can having many at the point of death - Sons leaving their Fathers and Mothers - brothers leaving brothers - careless whether they can ever see them again (HBCA B.107/a/25: 6d).

into their trade, especially following widespread mortality during the epidemics[45]. For the Indians of Norway House, it signalled a small but significant departure from past behaviour, behaviour that appears to have been mandated by strong prohibitions. In turn, this suggests that something had been altered in their relationship with the dead. Ross's observations should not be dismissed without further consideration of the broader effects of repeated epidemics on the Indians of the Norway House area. It may be that changes such as these, although they may appear minor of themselves, actually hint at more fundamental transformations to belief structures.

Oxford House

The diseased brigades were first noted at Oxford House, the next post located on the Hayes River route to York Factory, on June 21 (HBCA B.156/a/25: 3, 4), and by the 7th of July, the Indians of this area were infected with measles (Figure 23). On that date post master Laurence Robertson noted in the journal that "The Indian Peter was brought here from the Island Dead with the Measles, owing to the Indians being constant with the Brigades in their way as they pass" (HBCA B.156/a/25: 5). It would appear from this that the virus had spread directly from the men of the boats to the local Indians. By July 16

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[45]The switch to the ready barter system of trading, which did away with advances for the Indians, eliminated some of the losses that the traders would normally take in an epidemic year. Charles McKenzie explained in 1835 that:

> I am fond of this system, but I must say that it is a most cold calculating system - + not at all times to the [mind?] + disposition of the natives - but a very blessed system to the Traders in a year of sickness + death as this last winter was in this and the surrounding country" (HBCA B.107/a/13: 21-21d).

If the natives destroyed their furs or refrained from hunting for an extended period of time, the traders would lose no advances, but only what they stood to trade for that year.

Robertson could not find a healthy native to send to Norway House with a message, and on July 21 he lamented that "This sickness is prevalent all around the Fort, Consequently a great demand for Store Provisions" (*Ibid.*: 7). Two days later it was apparent that all of the Indians residing near Oxford House were in a bad way with the measles (*Loc. Cit.*).

Unlike at Norway House, where the men became sick very shortly after the arrival of the first boats from the Red River Settlement, measles was not a problem for the Oxford men until much later, except for those who had served on the brigades. On July 22 Robertson himself fell ill and was confined to bed for several days, while at the same time the men were "very low with this Sickness" (*Ibid.*: 7). This was six weeks after the first infected brigades had passed by. By mid-October dysentery was also common at the post and among the Indians[46] (HBCA B.156/a/25: 16d). The Anglican Reverend, Robert James, wrote of the disease at Knee Lake near Oxford House after having passed through in late September of 1846[47]. In doing so he noted both the severity of its effects and the fatalistic reaction of the Indians. There he witnessed many Indians in a miserable condition all belonging to Oxford House, "A fatal dysentery had left but fraction of their number alive. If they have the least ailment they lie down, regardless of all efforts for recovery, and sorrowfully await their death believing that they are so fated" (PAM CMS C.1/M.4: 202 Microfilm reel A78, Reverend R. James the Reverend R. Venn, Red River, 11 January, 1847). In all likelihood, the loss of hope would have contributed greatly to

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[46]On October 13 Robertson wrote: "Magnus Harper confined in the House.—with bowl complaints which is very prevalent here at present particularly amaung the children."

[47]The Reverend and Mrs. James passed Oxford House on September 19 (HBCA B.156/a/25: 13d).

the suffering experienced by these people.

During this epidemic Robertson identified other factors contributing to heightened mortality among some of the Oxford House Indians. Perhaps most significantly, he perceived a marked disparity in outcome between those who conformed to more traditional ways and those who had closer ties to the fur traders, the former dying at a greater rate. On July 29 he learned that the Indians encamped at nearby Trout Fall[48] were refusing his aid and advice, despite their great suffering (HBCA B.156/a/25: 7d). During the course of the next month he made several comments in the journal regarding the fate of these people, particularly in contrast to the fate of those who camped near the fort, and who readily accepted both relief and advice. On August 25 he recorded:

> The sickness amaung the natives of this place has been and is very heavy, but those in the neighbourhood of the Fort, being well cloathed and having tents, they have recovered, very few Deaths occurring:- But those from the Out Post who camps at the Trout Falls, being almost destitute of cloathing + tents - and Sticks close by their own ways of "Conjuring" has and is suffering and many dying. Every possible help has been and is offerded [*sic*] them, but advice or medicine except their own they refuse. I again visited them to day + find a great many very lo,- and 5 men 7 wives & 5 + children has Died. (HBCA B.156/a/15: 10d)

Here the trader identified two key factors that exacerbated the health problems of the people at Trout Fall. The first was the lack of clothing and shelter available to these people, and the shortfall of these necessaries would have complicated the course of their sickness by exposing the victims to the elements. In fact, given the increasing rarity of

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[48]Trout Fall was a portage of some sixty yards length near Oxford House on the way to York Factory (Jacobs 1853: 23). See Franklin (1823: 37) for a sketch of the Fall made by Robert Hood.

animals that could supply fur and leather at this time throughout much of the Northwest, these effects were most likely widespread.

The second factor, one that has long been implicated in the massive mortality of Indian people during post contact epidemics, was the use of traditional, but inappropriate, treatments (e.g. Taylor 1977: 58; Schoolcraft 1978: 312; Dobyns 1983: 16; Trimble 1989: 46). When Robertson suggested that the Trout Fall people were dying in part due to reliance on "their own ways of "Conjuring,"" he was observing the same deadly relationship that many others had witnessed before him. The two most widespread treatments among the aboriginal people of the Americas were the sweat lodge and exposure to cold water, or some variation, and both were often fatal under these conditions. Boyd, in his study of this same measles epidemic in the Pacific Northwest, identified these treatments as major reasons for extreme fatalities among certain groups[49] (Boyd 1994a: 41). For those at Trout Fall in 1846, treatment revolved around attempts to relieve the terrible fever by cooling the body. Thus, on August 28, Robertson:

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[49]Boyd wrote that:

> A major, if not the most significant, factor in accounting for different mortality rates appears to have been variations in treatment of the disease. In areas where Indians persisted in exposing themselves to cold water (either through drinking or bathing), or where (as in the Columbia River drainage) Natives treated the disease by placing infected individuals in sweat lodges, mortality appears to have been higher.

Saukamappee told David Thompson, with reference to the 1779-83 smallpox epidemic, "We did not suffer so much as those that were near the river, into which they rushed and died. We had only a little brook, and about one third of us died, but in some of the other camps there were tents in which every one died" (Tyrrell 1916: 337). Compare this with the comments by the Anglican Missionary John Smithurst stating that the few casualties due to measles in his parishes in the Red River Settlement were due to either mismanagement or interference by medicine men (PAM CMS C.1/M.4: 101 Microfilm reel A78, "Reverend J. Smithurst's Report for the Year ending Aug 1st, 1846").

> went down to the Trout Fall ... on arriving there I find 3 men + several children dead.- and all the others very low.- about the time the Measles is broke out on any one they then resort to the horrid custom of Cooling the Boy [meaning body?] by covering their naked Body up in wet moss - or lay exposed to the rain or if able [plaing?] in the water nothing can persuade them against such and this is what makes their Sufferings & Death. (HBCA B.156/a/25: 11)

Finally, during the following January Robertson observed that the epidemic had had a deleterious effect on the Indians' quest for provisions, even after the sickness had passed, since it had disrupted their normal seasonal pattern of activity. He wrote that the Indians were "Suffering for a want of food, being kept from their usual fishing + hunting lands owing to the lateness of their getting winter supplies, last autumn and the effects of the past Summer Sickness is greatly felt by the Indians all over. These are great drawbacks to the fur hunting" (*Ibid.*: 27). Thus, the hardship engendered by epidemic disease did not necessarily end with the termination of the epidemic.

Robertson's observations suggest that many had, temporarily at least, come to rely on the HBC post for clothing, medical assistance, food and shelter during this epidemic, a common pattern of behaviour whenever epidemic disease struck the Indians of the Canadian Northwest during the fur trade era. Indeed, according to Governor George Simpson in 1836, the HBC posts spent considerable effort "...administering to the sick and infirm, every Trading Establishment being in fact an Indian Hospital, where those who are unable to follow the Chase during the Winter months, are fed, clothed, and maintained throughout that inclement season with the most tender solicitude..." (HBCA E.18/3: 11). This was another facet of the Company's traditional paternalistic role in times of want, a role that it was to continue to fill into the late nineteenth century (Ray

1984; 1988; 1990). While this dependency on the traders was a short-term measure, it also may be that a more permanent reliance upon the trading posts was developing among some Indian people as a result of these epidemics.

Krech (1984: xvi) identified the need to consider epidemic disease when examining native adaptations to the fur trade, including dependency. While his suggestion is an important one with regard to the process by which a specific group may become more dependent on the traders' goods, a different process was identified at Oxford House, one that derived from differential mortality rates during such epidemics. Given Robertson's observations, it is quite possible that survival of these epidemics favoured the more "tractable" during the fur trade period, or at least those willing to adopt non-traditional practices. In this instance, and probably in many others, refusal to accept assistance from the traders and reliance on traditional treatments was a negative factor for survival. This was precisely the relationship that Boyd identified in the Pacific Northwest during this epidemic, where those who clung to traditional practices died in significantly greater numbers than those who took the simple medical advice and assistance of the whites (Boyd 1994a: 41). The resultant differential in the rates of mortality would have decreased the numbers and relative strength of the traditional factions with respect to those who were more acculturated. A similar outcome was experienced where opposition to smallpox vaccination was championed by traditionalists. Roman Catholic priests in the Pacific Northwest, for instance, observed a considerable differential in mortality during the epidemic of 1837-38 between Christian Indians who submitted to vaccination, and thus escaped the disease, and more traditional groups who refused, the latter

succumbing in the hundreds (Chittenden and Richardson 1905: 1235, quoted in Stearn and Stearn 1945: 64). This remarkable disparity, according to Father Adrian Hoeken, "had the effect of increasing the influence of the missionaries" (*Loc. Cit.*). Similarly, the AFC trader on the Upper Missouri, Edwin Denig, noted of the same epidemic on the Plains, despite the HBC's long-standing vaccination efforts, "Still many young children died who had not been vaccinated, with some few grown persons whose superstitions would not admit of its being applied" (Ewers 1961: 115).

A related outcome was that those native people who tended to remain in the vicinity of the posts throughout the year also had a better chance of survival, as the traders could provide medical aid or subsistence when needed. The Company sought to provide the sick with aid and comfort in all possible cases (HBCA E.18/8: 174, 92, 93). However, those who were out of reach had to make do on their own. When illness was widespread among such groups, with few healthy individuals to nurse the rest, mortality would be greater. The assistance afforded by the fur traders did not have to be complex to be effective. Shelter, food and clothing could have reduced their mortality significantly. An example of this is provided by the fur trader and northern explorer Thomas Simpson. Speaking of an influenza epidemic near Fort Chipewyan in the winter of 1836-37, he noted that that disease

> carried off nearly two hundred of the distant Chipewyans. I say *distant,* because all who were within reach of the establishments were sent for and carried thither, where every care was taken of them; warm clothing and lodgings were provided, medicines administered; the Traders and servants fed them, parting with their own slender stocks of luxuries for their nourishment. (Simpson 1843: 67-68)

It is not inconceivable, then, that in a period when severe epidemic disease had become an almost annual event, as it had in the 1840s, that trading posts increasingly came to be seen as refuges by some native people.

York Factory

The same boats that brought measles and dysentery to Norway and Oxford Houses carried these diseases to York Factory, infecting both the Indians and the HBC men early in July[50] (Figure 23). On July 12 James Hargrave informed George Simpson that "...The only difficulty I foresee arise from sickness now almost universal among our Indians both around this place and in the Brigades" (HBCA D.5/18: 36). The same day, in a different letter to Simpson, Hargrave painted a telling picture of an epidemic well advanced among the Indians of the York area.

> The routine here moves slowly ... but more from the despondency and fear that have crept into the minds of both men and indians.—The House and the Factory and the indian tents -are filled with the sick,—two natives were buried at the old Factory yesterday and the previous day —more of them are not expected to live,—and the disease is found to be far more lingering and fatal than at first it was expected to be.—The Whites are not [illegible] affected @ yet but both the indians and half caste [word?] are at this time widely under its influence—(more than half of them being presently more or less indisposed) and I fear that scarcely any of them will ultimately escape being attacked. (*Ibid.*: 38-38d)

By August 5 Hargrave would write that: "The greatest distress and suffering presently

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[50]Although there is no regular journal extant for York Factory for 1846, in its place there is a medical journal with entries beginning on August 26 which contains references to both measles and dysentery among the men of the fort (HBCA B.239/a/166). According to James Hargrave four of the Norway and Oxford House boats "reached us on the 6th Ultimº [July 6], the crews of which were generally affected with Measles, which malady, in a few days, was communicated both to the Indians around us and to the many of our native servants in the Factory" (HBCA D.5/18 130).

prevail among the natives from measles and other diseases. The loss of life has been considerable and the survivors are greatly reduced in strength and energy" (*Ibid.*: 107). Francis Ermatinger wrote his brother Edward from York Factory on the 8th, and noted that "the poor natives around us have been dying by wholesale" (McDonald 1980: 271). Two days later, Hargrave noted that the mortality during the previous month had been severe.

> The distress and mortality among the natives throughout the past month have been very heavy. The number of deaths around this Factory, up to this date, reaches 31; but this applies only to those in our immediate vicinity and buried by the Company Servants: many no doubt have sunk elsewhere who were beyond reach of our assistance; while, as I have said, many more of our most valuable indians are in so low a state that their lives are also despaired of[51]. (HBCA D.5/18: 130d)

At one point Hargrave's own children suffered from measles although, unlike many of the Indians, they recovered[52] (Macleod 1947: 221-222). Overall it is reasonable to conclude that the Lowland Cree of the York Factory region were struck with considerable severity during this epidemic, although it is not known how many died. The Indian population about York Factory had declined considerably some years before, as many Cree had abandoned the Lowlands for interior fishing stations or the settlement of Rossville near Norway House (HBCA D.5/11: 4). Thus, the thirty-one whom Hargrave's men buried

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[51]Hargrave identified "inflammation of the heart and of the chest generally," which followed as a sequel to the original disease, as the most frequent cause of death (Ray 1976: 154). The Reverend R. James was at York late in the month and noted that there had been as many as six deaths in a single day (PAM CMS C.1/M.4 : 185 Microfilm reel A78, Reverend R. James to Reverend H. Venn, York Factory, 27 August, 1846).

[52]On August 28 Letitia Hargrave wrote to Mrs. Dugald Mactavish that "Doey Dame [Their son Joseph James, born 1 April, 1841] & Tash [Their daughter Letitia Lochart, born 24 October, 1844] have had measles, which raged all over the country, & been fatal among the Indians, old young & middle aged."

may have comprised a significant portion of the local aboriginal population.

Severn and Trout Lake

From York Factory other HBC boats carried measles eastward into the Hudson Bay Lowlands to the Severn River region (Figure 23). The diffusion process began on June 19 when Severn trader John Cromartie departed the post for York with two Indians in a canoe. Initially, the men travelled towards the interior, and away from Hudson Bay, employing the Beaverdam- Shamattawa River route to the Hayes which they paddled down to the coast (HBCA B.198/a/90: 4-4d). En route they met up with William McKay and his men from the Trout Lake outpost and proceeded as a group[53]. On June 29, on the Steel River part of the Hayes River route, they observed other boats bound for York "with most of their Crews all sick" (*Ibid.*: 5). This was an inauspicious portent of their own fate in the near future. The brigade soon arrived at York Factory in good health, but quickly fell victim to measles (HBCA D.5/18: 131d, 428). Then, according to Chief Factor James Hargrave: "In the course of a few days however they were also added to the sum of invalids at this place. Two of their best hands sank under disease [i.e. died] and it was not until the 31st Ult^{o} [July] that I was enabled to get them off again, many of the crew being still in a weak and sickly condition[54]," (*Loc.Cit.*).

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[53]This route was probably an ancient one, but had only just come into use by the Trout Lake men. In April of 1844, Chief Factor James Hargrave suggested that, in future, the returns of Trout Lake be brought to York Factory by canoe, meeting up with the Severn brigade about June 15 "(each bringing the prime Otters of their respective trades in small canoes) at the mouth of the stream which falls into Severn River + through which a communication is found with the Shamatawa and this Factory [Hayes] River" (HBCA D.5/11: 2d).

[54]Hargrave's statement may indicate that the men contracted dysentery first, since the incubation

Although almost all of the boats' crews were still ill with measles and perhaps dysentery, and some had died, the Severn and Trout men departed for their posts together, their return journey being along the coast to Severn and from thence inland to Trout Lake (*Ibid.*: 428). According to Cromartie, they left York Factory with only two able hands in each boat, and along the way four fell dead (HBCA B.198/a/90: 4d). Others had to be left with relatives along the Hudson Bay coast, in turn infecting them. McKay's account of the journey corresponds to Cromartie's and provides greater detail.

> This morrining [*sic*] [19 September] I arrived with the Boat + outfit of this plase [Trout Lake] after a long and harresting [*sic*] voyage, from york factory to this. I left the above place upon the 1st Augst and ever since being on the voyage—I left york with three boats, two for Severin [*sic*], one for this, when we started all the Crews were all sick the Company's Servants excepted, all the others bein hired Indians for the voyage, not one of the latter that was not sick when we started the Boats were obliged to be loded by the people of the Factory and we started off with only two in each Boat that could work. Having bad weather along the Cost [coast] we were 19 days along the cost, in that time 4 of the Indians died, and we were obliged to leave the most of them with their friends at Goose River in passing. The cargo of the three Boats were all weate, and would not effect it to be othere ways, with such weather we had, and all along the Cost only two to three all the way in each Boat to work them[55]. (HBCA B.220/a/9: 5d)

The men were still sick when they reached Severn on August 19 and were responsible for bringing the disease among the people of this area, for when they arrived they were met

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period of measles is generally ten days, with a normal range between seven to eighteen (Benenson 1995: 294). It is unlikely that these men would have been delayed at York for that length of time, unless they were sick. More likely, had it been measles alone they would have been infected and departed for their posts before any symptoms became apparent. Hargrave suggested instead that they fell ill after only a few days. If the dysentery then epidemic was a form of Bacilliary dysentery (Shigellosis), then the incubation period may have been as little as twelve hours, although generally one to three days (*Ibid.*: 422).

[55]Not surprisingly the surviving tripmen declined hiring on for the next year's journey to York, both for Severn and for Trout Lake (HBCA D.5/19: 420d; B.198/a/90: 31, 31d; B.220/a/10: 18d). One man deserted from the brigade during the trip (HBCA B.220/a/8: 35).

by Indians who were waiting to trade (HBCA B.198/a/90: 5-5d).

By August 28 there were two sick Indians at Severn House (*Ibid.*: 6d). On the second of September sickness had become far more widespread. Cromartie noted "...I am afraid that the Indians will not be able to hunt as most of them is sick and no appearance of them getting getting [*sic*] better as yet" (*Ibid.*: 7). A day later he received word of the death of the eighth hunter belonging to the post (*Ibid.*: 7d). Critically, the sickness disrupted the important fall goose hunt, ironically during a year when there were plenty to be had (*Ibid.*: 9, 10, 11), and reports of starvation were received throughout the fall and winter. Part of the problem was that this affliction prevented the Indians from walking (*Ibid.*: 9, 28d), and thus circulating through their resource areas. Whereas normally they would have moved often during the winter in search of food, this year they were forced by the sickness to remain in a single location, which limited their opportunities to obtain sustenance. Although neither mortality figures nor the extent of the epidemic in the Severn House area can be pinpointed, it is evident that this epidemic spread east of the Severn River, although probably not as far as the Winisk, and that there were significant casualties among the Lowland Cree population of this region[56] (HBCA B.198/a/90: 19, 25, 18d). Perhaps indicative of the degree of disruption engendered during this epidemic, the trader wrote on March 28: "Serene + mild for this time of the year but no Indians being sumething very Strange this season above all others they Cannot be all Dead sume

[56]On March 8 Cromartie learned of the death of several of his best Indians the previous fall (HBCA B.198/a/90: 25). On April 15 the Winisk River Indians finally arrived and, though they complained of starvation and poor hunting, made no comment regarding sickness, suggesting that they had been spared during this epidemic (*Ibid.*: 28d).

of them must be in the land of the living yet" (*Ibid.*: 27).

Although McKay's men continued ill after they departed Severn, it is not likely that they were the means by which disease reached Trout Lake (HBCA B.220/a/9: 5d). After a journey of about 49 days (HBCA B.220/a/8: 35) it is highly unlikely that they remained infective with either measles or dysentery. Instead, measles was transmitted directly to the Trout Lake Indians by some of their friends from Oxford Lake[57] (HBCA D.5/19: 420). Once again, there is little in the way of evidence in McKay's journal to establish the epidemic's extent since few details are provided regarding the origins of the Indians he was trading with. The only concrete information was relayed by an Indian called the Gull's Nose, who informed the trader that those hunting towards Severn Lake, to the westward, had contracted the disease during the fall and by early winter were doing poorly (B.220/a/9: 14). Curiously, a large party of Sandy Lake Indians, living about midway on the road between Trout Lake and Oxford House, arrived on June 19, 1847 not having been in during the winter, and made no mention of disease among themselves (HBCA B.220/a/10: 20). If indeed they had escaped all sickness it was undoubtedly a stroke of great luck. Conversely, it was reported that some of the Indians wintering at Island Lake, between Oxford House and Sandy Lake, and on the same track, had been confined with sickness for the most part of the winter (HBCA B.156/a/25: 37).

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[57]William McTavish wrote to Sir George Simpson from York Factory on March 20, 1847, explaining that:

> M[r] M[c]kay [Trout Lake] had last autumn arrived at his post with the outfit on the 19[th] Sept[r], the Indians attached to it having been equipped and sent off to their hunting grounds at an early date, but as measles had been communicated to several families of them by some Oxford Indians whom they had been visiting, it is feared their exertions will have been very materially crippled and of course the Returns suffer in proportion.

Overall, the mortality at Trout Lake, while perhaps not exceptional when compared to other places, was nonetheless significant. In summing up the outfit year on May 31, McKay wrote that "The returns would have been better had the Indians been all in good health.— But a great number of them were under the measels the first part of the winter. 14 Deaths of men, women + children ammong the whole of the natives belonging to this post, which has been greatly against the returns..." (HBCA B.220/a/9: 24). When epidemics were occurring at relatively frequent intervals, even small population losses could accumulate and play a significant role in shaping the lives of Indian people.

The Boundary Waters

About the same time that measles and dysentery were being carried northwards from the Red River Settlement through Lake Winnipeg during the summer of 1846, these diseases were also making their way to the Boundary Waters (Figure 23). It is impossible to state the exact timing of this diffusion, however. Despite the presence of several missionary stations and posts in the Boundary Waters, there is, unfortunately, very little that survives in the way of primary accounts for this period[58]. Perhaps most unfortunately, none of the HBC journals for these posts is extant. Instead, available evidence is limited to a small amount of correspondence and reports from the

[58]This included at least: a Roman Catholic mission at Wabassimong on the Winnipeg River, under Father Aubert, a Methodist mission at Fort Frances, under Peter Jacobs, an American post at Warroad and HBC posts at Fort Alexander (George Setter), Rat Portage (Donald McKenzie), Rainy Lake (Chief Factor Nicol Finlayson) and Fort William (John McKenzie) (PAM MG 7 D2 *L'Esperance* 1: 135; HBCA B.239/k/2: 388; HBCA B.154/b/3: 17). The HBC's Lac la Pluie Department included one officer (Finlayson), four clerks and 25 servants (HBCA B.239/l/17).

neighbouring post of Lac Seul. Nevertheless, it is clear from this evidence that by July both measles and dysentery had followed influenza into this region.

Although the HBC brigades were key factors in the diffusion of measles and dysentery from Red River to the northern portions of the Petit Nord, they played no such role in the spread of these diseases to the southern part. Both measles and dysentery appeared early on the Winnipeg River, only a few weeks after they first broke out in the Red River Settlement, and before the brigades returned to the Lac la Pluie Department. Thus, Charles McKenzie at Lac Seul wrote on August 13 that "many deaths occurred among our Indians who pass the Summer on the River Winipic whe[illegible] the Measles was raging **since last spring** [emphasis mine] accompanied by a Bloody Flux and Billious Complaints which made a havoc among the Natives" (HBCA B.107/a/25: 1). The first Lac la Pluie brigade, consisting of three boats under the charge of C. Roussain, reached Norway House on its trip to York Factory on June 9, and did not pass the same post on its return trip until July 12 (HBCA B.154/a/46: 3d, 8). For an unknown reason there was no earlier brigade dispatched directly from Red River to Lac la Pluie[59]. Consequently, the brigades heading from Norway House could not have been the first to import the sicknesses to the Winnipeg River since they would not have arrived until mid to late July at the earliest. By then both diseases were already present in the Boundary Waters.

Instead, these diseases were more likely carried by individuals, either native or

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[59]On May 25 Chief Factor Sinclair at Lower Fort Garry informed Governor Simpson that: unforeseen circumstances have occurred to prevent our entering upon an early transport from here to Lac La Pluie, the Boats shall therefore be dispatched for York factory without delay, and may be expected to return from there about the 20th July.... (HBCA D.5/17: 287)

non-native, from Red River to the Boundary Waters. There was considerable traffic between this area and the Red River Settlement and people would have passed and repassed throughout the open season, as the Winnipeg River was part of the main route to and from Red River. One possibility may be the party of the Roman Catholic missionary, Pierre Aubert, who left Red River for Wabassimong on June 20, just as the diseases were gaining strength in the Colony (PAM MG 7 D2 *L'Esperance* 1: 135). There is some evidence that the Indians of the Winnipeg River placed the blame for the epidemic at the feet of the Roman Catholic mission, which had to be abandoned the following year over threats to its personnel[60]. This may have been because the mission had a role in the arrival of the sicknesses, since the Methodist mission at Lac la Pluie was not similarly threatened.

In all likelihood these diseases had been brought first to the Winnipeg River, perhaps in late June, and then spread eastward from there. The timing of the sickness is significant. The people living around the Winnipeg River gathered together in larger numbers during the late spring to celebrate the rites of the Midewiwin, and such social gatherings served as ideal vehicles for the diffusion of infectious disease. In just such a manner the ceremonies of the Grand Medicine Society had facilitated the diffusion of dysentery among the participants at Partridge Crop to the west of Lake Winnipeg, (PAM CMS C.1/M.4: 277-78 Microfilm reel A79, "Reverend A. Cowley's Journal from July 25th, 1846 to July 21st, 1847"). In 1846 people began to gather in June on the Winnipeg

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[60]PAM MG 7 D13 Microfilm reel 1 *Belleau Collection*, Bishop J.-N. Provencher to Bishop Turgeon, St. Boniface, 4 December, 1847.

River for this ceremony. Paul Kane was at Fort Frances on June 4 and arrived at Fort Alexander seven days later[61] (MacLaren 1989: 24). While at the latter place he attended a medicine ceremony where he was met by "grate swarems of Indanes" (*Loc. Cit.*). Although Kane wrote nothing of sickness among these people, it may be that he failed to observe or comment on it, or that he departed before the diseases became widespread.

Once introduced, however, the twin scourges quickly spread through the native people who spent their summers on the Winnipeg River. On August 3 Chief Factor Nicol Finlayson, who was on his downward journey from York Factory to Lac la Pluie, wrote to Governor Simpson from Fort Alexander stating that "sickness has made such a ravage among the natives this summer, and the last which has broken out in Red River and the vicinity, which has more the appearance of a pestilence than an epidemic..." (HBCA D.5/18: 105). Finlayson's letter indicates that the people of the Winnipeg River, who had been hard hit the previous year by the "Red River Disease", were again suffering greatly under sickness brought from Red River[62]. On August 27, Charles McKenzie at Lac Seul was visited by an Indian, Quisance's third son, who had come with additional news from the Roman Catholic mission at Wabassimong on the Winnipeg River. "Our Indians in that quarter are sick and dying and cannot make Rice altho' Rice is plenty on that River - The accounts these bring us of the Starve of the Measles + Dysentery are dreadful"

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[61]On June 13 1852 the Methodist missionary Peter Jacobs visited the site of the ceremony, at a rapids near Fort Alexander, and found some 100 Indians gathered in preparation for what he called the "Manito Feasts" (Jacobs 1853: 18).

[62]Due to the sickness among the Lac la Pluie district Indians Finlayson found it difficult to engage men for the district brigade and the additional transport brigades for Norway House (HBCA D.5/18: 105).

(HBCA B.107/a/25: 2d). The inability to obtain provisions and the resultant starvation in the face of widespread and severe illness were common complaints whenever aboriginal populations were exposed to acute infectious diseases with which they had little or no experience[63]. In this case starvation was playing a major role in the death of these people.

Sometime during the summer these diseases spread eastward to Lac la Pluie, arriving at least as early as by August 17 (HBCA B.107/a/25: 2; Figure 23). Again, the exact timing is difficult to establish. There is some evidence in a letter from the Methodist missionary, Peter Jacobs. Jacobs was stationed at Fort Frances and had been to the Red River Settlement earlier in the year. According to his letter, he had left Red River after the measles had broken out and had been afraid that his men would contract the disease. Despite this fear, he and the men arrived safely at the fort after a journey of twelve days (HBCA D.5/18: 19d). The letter was written on July 7, and Jacob's only comment concerning the Indians was to say that "this present year is going to be a great year for wild rice at this place, and that the Indians of this District are preparing for a great rice harvest" (*Ibid.*: 19-20). This suggests that the disease had yet to reach the Indians of Lac la Pluie by this date and, consequently, that diffusion occurred later here than on the Winnipeg River.

Nevertheless the arrival of measles and dysentery could not have been much later,

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[63]Early in September Quisance arrived with his sons after summering on the Winnipeg River. According the McKenzie "They brot but 1 Bag rice only [,] the death of children + sickness discouraged them in rice making" (HBCA B.107/a/25: 4). However, it seems unlikely that this family had declined gathering provisions as part of mourning. Instead, they were probably incapacitated at the time of the harvest, just as the Winnipeg River and Lac la Pluie Indians had been.

as epidemic diseases also prevented these people from harvesting what would have been a bumper crop in a healthy year. Early in the fall Finlayson, now at Fort Frances, wrote that "Rice had been most abundant every where upon this quarter, but unfortunately, measles and other epidemics laid up the natives at the time of the harvest...[64]" (HBCA D.5/18: 232). In most years wild rice was harvested in late August or early September (Waisberg 1984: 127). Generally, the Indians began to gather together in larger numbers in preparation for the wild rice harvest after having been dispersed through the summer (*Loc. Cit.*). Conditions would therefore have been conducive to widespread diffusion in this region. Perhaps in this case the rice harvest attracted some from the westward who had come into contact with the diseases, and then spread among the Lac la Pluie people. Certainly measles was present well before August 17, as on that date Lac Seul trader Charles McKenzie noted the presence of measles among the Eagle Lake Indians who had contracted it while summering at Lac la Pluie (HBCA B.107/a/25: 2).

As everywhere, at Lac la Pluie the Indians suffered greatly and the mortalities were significant. Nicol Finlayson explained to Simpson on January 26, 1847 that "My prospects of trade are not flattering; many of my hunters are gone to <u>follow</u> <u>the</u> <u>chase</u> with their Fathers, and all the survivors have lost so many of their children and relations, that but little can be expected from them this season..." (HBCA D.5/19: 129d). In an earlier letter Finlayson told Simpson that the Indians were mourning many of their friends and relatives who had succumbed (HBCA D.5/18: 232). When the returns for 1846-47 outfit were in, the Lac la Pluie district had experienced a reduction of about £300 over the

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[64]Nicol Finlayson to Sir George Simpson, Fort Frances, 19 September, 1846.

previous year (HBCA A.12/3: 447d). A lack of documentary evidence precludes any further analysis in this area.

Lac Seul

Both measles and dysentery spread north and east from the Boundary Waters into the interior of the Petit Nord, to Lac Seul and beyond during the summer of 1846 (Figure 23). The initial penetration of the two into this area occurred while Lac Seul trader McKenzie was away at Albany Fort, and the journal was discontinued at the time of his departure. Nevertheless, sufficient evidence exists to piece together the course of the epidemic in this region. As noted above, when the Lac Seul brigade left the post on June 8 the men, and indeed the Indians of the area, were suffering from influenza. When the boats returned on August 13 they arrived to find measles already rampant on "Bonny Point" as:

> most of the Lac Seul Indians are here on this point waiting the arrival of the craft [i.e. the Lac Seul boat from Albany] and labouring under the Measles which was brought here from Red River early in July by the men's wives who went there to see their friends in May last[65]. Every soul within this fort has had the disease but they are all now convalescence [*sic*] of a death appearance, but no deaths took place so far on the point, except one young woman a few days ago.... (HBCA B.107/a/25: 1)

The mild nature of this sickness at this point was in direct contrast to the situation on the Winnipeg River and suggests that dysentery had yet to reach Lac Seul. Throughout the

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[65]These women had departed Lac Seul on May 19 as McKenzie wrote: "No less than 3 of our Ladies went off for Red River to visit their friends in that quarter- + the two young lads who passed the winter here-two only of these are to be back this summer-the other being the widow of the late Samuel Rat, is to remain with her relations" (HBCA B.107/a/24: 23d).

remainder of August, however, reports came in of death and sickness due to dysentery in the Lac Seul region. By the end of the month, dysentery had also broken out at the post among the men and their families, as infected Indians arrived at the post for goods and medical assistance[66] (*Ibid.*: 3d).

By September 15 conditions at Lac Seul post had taken a drastic turn for the worse and all were in great misery. Between the 15th and the 24th there is a gap in the journal as McKenzie himself fell victim to dysentery and was incapacitated. Upon his recovery he explained this omission:

> I have been attacked by the most violent Flux, or Dysentery, that ever attacked man (short of destroying life) on the 15th Instant, and this is the first time I was able to take the pen - what I suffered, and still suffer, I will not attempt to describe - my case was light to that of those who died and are dying about me - no less than 5 died on this point during my sickness Bloody Flux and Bilious Complaint[67].
>
> Robert Gill is laid up with Measles of a very oppressive nature accompanied with a violent cough and sore throat - George Moor and his large family are lying side by side - without a single exception - or one able to assist the other to a drink of water.
>
> There cannot be less than from 70 to 80 Souls on this point and not 20 of these are able to go about to get a fish or a Rabbit for the sick- how they exist I cannot say. (*Ibid.*: 5)

Another man, William Sabiston, fell ill of the measles on September 29, leaving McKenzie with only three men capable of leaving their beds, and on October 4 Gill died

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[66]"The dysentery was an after affair but which followed at the heels of the Measles and which made a greater havoc..." (HBCA B.3/b/73: 12d, McKenzie to Corcoran, Lac Seul, 20 December, 1846).

[67]McKenzie was confined to the house for a total of twelve days at this time during which, he noted, there were seven fatalities among the Indians at the post. Of the HBC people and their families, only McKenzie's wife and Patrick Adhemar escaped dysentery during this epidemic. Two died, being Robert Gill and William Sabiston (HBCA B.3/b/73: 12-12d, McKenzie to Corcoran, Lac Seul, 20 December, 1846). McKenzie did not contract measles but did suffer from dysentery, and so this is definitive proof that this dysentery epidemic was an independent sickness, and not a symptom of measles.

after fourteen days' illness, "from Measles, and an accumulation disorders - Bloody Flux - Billious, Cough, and Sore Throat[68]," (HBCA B.107/a/25: 29, 6). The effects of so many ill with dysentery at the post were revolting, and were certainly conducive to all manner of disease transmission. Early in October McKenzie commented that: " there has been a pestilential infection on this point from first to last and now— no one can out about the place without his stomach being turned and his foot being sunk in human excrement—!" (HBCA B.107/a/25: 6d).

Although traditionally the HBC post had served as hospital and commissary during epidemics, in this instance neither McKenzie nor his men could offer assistance to anyone due to their own sickness. Indeed, they found it difficult to provide for themselves. By the 27th, however, some sustenance was available as the trader sent three men and women to dig potatoes, and these were quickly joined by most of the sick Indians who, because of their disabilities, had to crawl up the hill to the potato field (HBCA B.107/a/25: 5, 6d). If harvesting wild rice, fishing and trapping rabbits had proved too difficult for those suffering from these diseases (*Ibid.*: 2), it would seem that digging potatoes was within their meagre abilities. In turn, this may suggest that those native groups in the region who had adopted agriculture had a better chance of survival if an epidemic struck when crops were edible, than those who relied on the chase.

While those who had previously contracted measles would have had immunity, it is apparent that there were many in the Lac Seul area who had either been born since the

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[68]Although Gill was listed as a mixed-blood apprentice in 1838 (HBCA B.107/a/16: 24), he was employed by the HBC at least as early as 1827 (HBCA B.155/a/39), and so was born before the 1819-20 epidemic. Since he contracted measles in 1846, he must have escaped the disease in 1819-20.

last epidemic, or had escaped without being infected, including several of McKenzie's men. Unlike the case in 1819-20, the employees of the post-merger HBC, many of them of mixed descent and having lived their lives in the Northwest, had no immunity to measles, while dysentery was a scourge to which all were vulnerable. As well, despite the virulence and wide spread of the 1819-20 epidemic in this area, and the smaller, more localised outbreak of 1828-29, there were many aboriginal people who were susceptible in 1846, making for widespread suffering. Moreover, the concurrent or subsequent dysentery that, in most cases took the greater toll, made matters far worse, particularly for those who contracted both. As such, even immunity to the former disease was no guarantee of health. The fate of one Indian, Nigonice, was perhaps representative of the long term prospects of the Lac Seul Indians during the first half of the nineteenth century. He was one of the few survivors of the 1819-20 measles epidemic in the region (HBCA B.107/a/2: 14; B.107/a/25: 9). On September 23, 1822, McKenzie wrote that: "Neconise took Debts with a good deal of reluctance, he having contracted a large one the year that dreadful disease past [*sic*] in this quarter which almost carried him to his grave." In 1846 he was, of course, immune to this disease, but nevertheless succumbed to dysentery. On October 15 he had helped wash Robert Gill's body in preparation for internment. By the 3rd of November he was dead (HBCA B.107/a/25: 9). Undoubtedly, a similar fate was shared by many others who were granted only a temporary stay of execution during one of the many epidemics in the southern part of the Petit Nord. Although they might survive one epidemic there were always other sicknesses to follow which might claim them.

These diseases spread widely in the Lac Seul area, even to more remote areas. In this case, isolation from the post proved no safeguard from sickness[69]. On August 17 it was reported that the Indians of Eagle Lake, on the Wabigoon River system to the southwest of Lac Seul, were "in the heat of the Measles" which they had brought from Lac la Pluie (HBCA B.107/a/25: 2; Figure 23). Five days later McKenzie learned that the Indians to the westward of his post were suffering from both measles and dysentery. By that time two of these hunters had died, as well as an old woman and "many children" (*Ibid.*: 2d). Their sickness had yet to peak, however, and McKenzie pessimistically predicted that "more will die of the Bloody Flux than of Measles" (*Loc. Cit.*). On August 31 McKenzie was visited by John Moose and his family, returning from nearby Rice Lake[70], and was told that "The Measles and Dysentery reign there as well as here. John reports several deaths to the southward - chiefly that of children[71]," (HBCA B.107/a/25: 3). The deaths referred to here may have included those of Eagle Lake and perhaps Lac la Glaize, or present Wabigoon Lake. Rice Lake was a major ricing area attracting Indians from Lac Seul, Osnaburgh, and elsewhere (*Ibid.*: 2d, 3), and thus was a significant location for widespread dissemination of disease. Throughout the fall and early winter,

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[69]According to McKenzie, the diseases spread throughout, "scattered as the Indians were over the country, the Measles found them out the deep forest was no safety." (HBCA B.3/a/73: 12d). Although the Indians fled in panic upon hearing of the disease, in many cases it was too late, and they had already been infected.

[70]There are many ricing lakes in the area, and it is not known which McKenzie referred to as Rice Lake. One clue is that in an earlier journal he stated that Rice Lake was Gull Lake (HBCA B.107/a/9: 5d). Gull Rock Lake lies along the route to Red Lake, a short distance to the northwest of Lac Seul, and this may be McKenzie's Rice Lake. In 1836 a Lac Seul trading party had left for Rice Lake on the 11th of September and returned only three days later (HBCA B.107/a/15: 3d).

[71]Moose, an Osnaburgh Indian, died on the afternoon of September 26 (HBCA B.155/a/587d).

reports of sickness and mortality continued to arrive at the Lac Seul post. On November 15 McKenzie, inundated with bad news, wrote, "From whatever quarter we hear, we are sure of fresh reports of deaths!" (HBCA B.107/a/25: 9). Indeed, by December his information suggested to him that sickness reigned not only at Lac Seul, "but over all the country on this side Lake Winnipeg" (HBCA B.3/b/73: 12d).

Although the Indians of nearby Sturgeon Lake, to the southwest of Lac Seul (Figure 23), may have escaped the measles in 1819-20, those who resided there did not in 1846[72]. Still, the disease was late in spreading there, as initial reports were favourable. On August 29 seven of the young Loons arrived from that lake, apparently in good health (HBCA B.107/a/25: 3). A little over a week later, two more Sturgeon Lake Indians arrived at Lac Seul and McKenzie commented that "these so far escaped the Measles" (*Ibid.*: 4). On September 26th, nine more came in, and McKenzie's entry noted "all these had the Measles, and 3 deaths occurred among them (*Ibid.*: 5). Finally, on November 14 the trader received a report of several additional deaths at Sturgeon Lake since they had come to the post, chiefly among the children" (*Ibid.*: 9).

Osnaburgh

These diseases were somewhat delayed in their arrival at Osnaburgh. However, by September 26, measles had become so widespread there that post master George

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[72]There had been changes in the occupants of Sturgeon Lake since the earlier epidemic. For instance, in the early 1830s the Sturgeon Lake Indians had begun encroaching on the Lac la Pluie Indians' grounds, being in turn pushed out of their own hunting grounds by the Nipigon Indians who were moving westward into Sturgeon Lake (HBCA B.107/a/5d).

McPherson could count only two men still healthy (HBCA B.155/a/58: 7d). By the 3rd of the following month, the men were still ill with measles and dysentery had made its appearance, though by the 12th they were recovering (*Ibid.*: 8). Unlike at Lac Seul, none of the Osnaburgh men succumbed (HBCA D.5/19: 128). Almost certainly, the diseases were carried to the post from Rice Lake, to the west of Lac Seul. Many of the Osnaburgh Indians had been there in search of rice earlier in August, or when both diseases had been rampant (HBCA B.107/a/25: 2, 26, 31). Indeed, some of these people had brought measles to Lac Seul towards the end of August (*Ibid.*: 3d) and some never made it back to their home territories (HBCA B.3/b/73: 12). Despite this, McPherson was slow to learn of the extent of the mortality among his hunters[73]. On January 2 he could only write to his superior Thomas Corcoran at Albany that "The returns of present outfit has a poor appearance at present, the Measles + Bloody Flux that came among the Indians in the Fall of the year did a great deal of injury... I have seen only two Indians since the Winter set in, as far as the rest of them, I do not know whether they are alive or not" (HBCA B.155/a/58). As the month progressed, however, he began to understand the extent of the devastation and it became apparent that more than just a few had died (HBCA D.5/19: 128; B.107/a/25: 12). All told, it is difficult to judge just how severe the sicknesses' impact was at Osnaburgh, since McPherson provided little detail in his journal and correspondence in this regard, but it was certainly enough for him to anticipate a poor year for returns.

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[73]Charles McKenzie learned on November 14 of the death of many of the Osnaburgh Indians, and thus before McPherson was aware of the loss (HBCA B.107/a/25: 9).

In discussing the 1846 measles and dysentery epidemic, Charles Bishop estimated that the number of fatalities among Indians in the Lac Seul-Osnaburgh area was perhaps as many as forty (Bishop 1974a: 162). In doing so, he essentially disregarded the impact of the epidemic, at the same time ignoring the dysentery component of the epidemic entirely, despite the comments by Charles McKenzie, one of his key sources. Even so, forty fatalities would have been a significant number, given that at Osnaburgh House the total population was only 279 in 1830, and 181 in 1858, and at Lac Seul in 1838 there were 339 (*Ibid.*: 157, 160). Given the higher figures, for instance, and Bishop's mortality estimate, almost 6 per cent of the total population would have perished in the space of a few months. Given the lower estimate it would have been about a 7.5 per cent mortality rate. At the Red River Settlement, where similar rates were experienced during the summer, comparisons were made to cholera and other devastating epidemics. This was by no means a minor episode.

Moreover, it is likely that the impact was even greater than described by Bishop. As we have seen, during the ensuing years since the earlier population estimates, in 1830, this area was buffeted by repeated disease episodes that probably had a hand in lowering the population, an effect that Bishop (1974a: 157) discounted. Furthermore, Bishop's estimate of mortalities seems low. In December of 1846 McKenzie stated that he knew of at least thirty-six fatalities among the Lac Seul Indians alone[74] (HBCA B.3/b/73: 12d). However, it is highly unlikely that he was aware of all the deaths among the Lac Seul

[74]"I cannot say how many deaths among the natives at this date. I knew of 6 men but of women and children the number cannot be short of 30 souls, in short I scarcely know a family who has not lost a member, some from two to three and this Fort is now surrounded with Widows and orphans."

Indians at this point in time since many would not be in until the spring, when he would have a better idea of the final toll. At Osnaburgh, in January McPherson noted the death of eighteen Indians in two families, as well as several others[75]. The total for these estimates alone is greater than fifty-four. Thus, it seems probable that the death rate due to these diseases amounted to more than just six or seven and a half percent.

Charles McKenzie's comments suggest that epidemic disease spread throughout the country lying to the east of Lake Winnipeg. However, it is impossible to verify the situation to the north and west of Lac Seul and Osnaburgh, at least as far as the Trout Lake region, where the disease came from the north. It is not known, for instance, if either measles or dysentery diffused into the East Winnipeg Country, although at least one Berens River Indian succumbed while employed on the brigades (HBCA B.156/a/25: 7). There were boats from the Berens River outpost arriving at Norway House during the epidemic period on two occasions, June 13 and August 7 (HBCA B.154/a/46: 4; B.154/a/46: 11). Moreover, there were free traders from the Red River Settlement among these Indians during the winter of 1845-46, a pattern likely repeated the following year (HBCA D.5/17: 277d). Likewise, Indians from both the Red Lake (Ontario) and Osnaburgh areas had been known to spend time on that river (HBCA B.107/a/24: 23; B.3/c/1: 1ad). Certainly there was considerable opportunity for diffusion to the eastern side of Lake Winnipeg. There is simply no evidence to corroborate McKenzie's

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[75]On January 23, 1847 he wrote that "Two Cranes arrived they paid their debts and trade a little they are to start tomorrow, they report that six of their wives + children have died of the Measels" and on the 28th "Kanandouray + two of his brothers arrived they bring very little. They report that 12 of that family have died, viz. Whiskey, Jacob, Snake, 2 women + 7 children all of the measles." (HBCA B.155/a/58: 12d).

conclusion. Similarly, according to McKenzie, at least seven of the Indians from Rat Portage on Lake of the Woods wintered with the Cat and Crow's Nest Lake Indians to the north of Lac Seul and not too distant from Osnaburgh (HBC B.3/b/73: 29d). It is probable that one or both of the sicknesses reached this area, although there is no specific reference to these Indians in either of the two posts' journals. Perhaps the only evidence of epidemic activity farther into the interior of the Petit Nord lies with the sickness and death of several of the Cranes during the winter (HBCA B.155/a/58: 12d, 21). Although these Indians traded at Osnaburgh during January and June of 1847, they generally wintered to the north, at Round Lake[76] (Rogers and Black Rogers 1982). Indeed, in August of 1846 it was reported that the best Osnaburgh Indians were trading at Trout Lake including, probably, the more northern groups such as the Cranes (HBCA B.3/b/73: 11ad). Unfortunately, with the concentration of HBC posts in the southern and northern parts of the Petit Nord, and without any records from Berens River outpost, the fate of much of the heart of the region cannot be documented, save for the comment by McKenzie that sickness reigned "over all the country on this [i.e. east] side Lake Winnipeg" (HBCA B.3/b/73: 12d).

Martin's Falls and Albany

The fate of the lower parts of the Albany is much clearer. Although both measles

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[76]It may also be that another group wintered to the north and contracted measles. On January 28, 1847 it was reported at Osnaburgh that an Indian named Jacob had died of measles, along with several others in his winter group (HBCA B.155/a/58: 12d; see note above). On October 1, 1846 an Indian named Jacob arrived at the Trout Lake post to receive debt (HBCA B.220/a/8: 36d).

and dysentery had penetrated into the Albany River region, their downstream progress was minimal, probably not much farther east than Osnaburgh House (Figure 23). Certainly they did not diffuse as far as Martin's Falls, the next HBC post downstream from Osnaburgh, despite assertions to the contrary by Charles Bishop (Bishop 1974a: 161). In January of 1847 Thomas Corcoran, Chief at Albany, received news of the devastation at Lac Seul and Osnaburgh, as well as of affairs at Martin's Falls, via the winter mail packet. In his journal entry for January 28 he wrote:

> at Lac Seul + Osnaburgh they have been severely scourged during the last summer + autumn with measles, dysentery + bilious fever, which proved fatal to many of the Indians of both places + to two of our most youthful + active servants - Robert Gill + W^{m} Sabiston at Lac Seul. The evils that proved so disastrous at the above two posts have not reached Martins Falls where everything goes on as favourably as could be expected[77]. (HBCA B.3/a/152: 20d)

Even at this late date, when both diseases had begun to slacken at Lac Seul and Osnaburgh, there was no sign of either farther down the Albany River, nor would they ever reach this post. If the Martin's Falls journals for the period 1846-49 are a reasonable indication of the health status of the people and Indians of this post, it was remarkably healthy during this period (HBCA B.123/a/50-52). The same is true of Albany, where Corcoran could only comment on the sickness from a distance (See HBCA B.3/a/152-153). Likewise, Moose was relatively free from any significant infectious disease at this time (HBCA B.135/a/151). This pattern is remarkably similar to that of the epidemic in 1819-20, although in 1846 the disease was able to spread slightly farther east, to

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[77]Similar comments were made by Corcoran in a letter to George Simpson dated the following day. See HBCA D.5/19: 128; B.3/b/73: 16.

Osnaburgh and Sturgeon Lake.

Lake Superior

Although measles struck Fort William on its westward course, late in 1845, and an unknown number of Lake Superior Indians were infected while at Manitoulin Island or Sault Ste. Marie the same year, it would appear that none of the three major epidemic diseases of 1846 reached the people living north of Lake Superior (Figure 23). There was a good reason why they were spared these diseases; by 1846 the Indians of Lake Superior had, for the most part[78], settled into a pattern of movement that revolved around the shore of Lake Superior and journeys only a short distance into the interior. Trips to Manitoulin Island and Sault Ste. Marie and lake fishing, as well as visits to American settlements and HBC posts, all kept the Lake Superior Indians around the lake during the critical summer of 1846, when the danger of contracting these diseases was greatest. Come the fall, they

[78]Significant exceptions to this pattern of movement included encroachments by the Indians of Lake Nipigon on Sturgeon Lake (for example, HBCA B.107/a/25: 15d), and on the Martin's Falls and Albany River area by the Long Lake Indians (for example, HBCA B.3/a/151: 7; B.3/a/152: 6d; Bishop 1974a: 216). However, in 1846 many of the Lake Superior Indians remained about the lake working for the mining companies rather than going to Sturgeon Lake (HBCA B.107/a/25: 4; B.134/c/61: 201d-202), while all available evidence suggests that none of the diseases reached the lower part of the Albany River, and hence the Long Lake Indians were in no danger of contracting a disease while on their raids of the Albany River Indians' lands. As well, in 1846 most of the Sturgeon Lake Indians traded at Nipigon rather than at Lac Seul, when they heard of the cheapness of the goods at Lake Nipigon and that there would be no debts given at Lac Seul (B.3/c/1: Charles McKenzie to Thomas Corcoran, Lac Seul, 5 September, 1846). Thus there was a break in communications between the Sturgeon Lake and Lake Superior Indians, on the one hand, and those who were affected during this epidemic and lived to the north and westward. Also reinforcing this pattern of movements was the decline of furbearers and game animals in the Lake Superior region. Long before 1846, both beaver and other big game had begun to disappear from the region to the north of Lake Superior. As a result, some of the Indians had begun to remain for extended periods of time on the shores of the lake, where fish could be readily found at certain seasons. For many, the tendency had become to spend a longer part of the year along the shore, rather than heading for the interior and trapping, where subsistence might be far more precarious. This had the effect of lessening the chance of contact between the Indians of Lake Superior and those of the interior.

dispersed into smaller, more isolated, winter hunting parties with even less contact.

In 1846 this general pattern of movement was reinforced by the arrival on the scene of mining companies attempting to survey promising locations on the north shore[79]. During the early to mid 1840s there was a substantial rise in the number of non-natives travelling to Lake Superior, fuelled by the growing interest in its mineral wealth. Following a treaty in 1842 that opened up the south shore of the lake (United States 1845: 498), hundreds of American miners entered the region (HBCA D.5/8: 347d, 435d). By 1846, 500 men were employed by 24 companies along the U.S. shore, and the Americans had at least five schooners and a steamboat in operation on Lake Superior to transport men as far as the mines at La Pointe (Logan 1847: 10; HBCA A.12/3: 135d). That same year, miners began to appear on the Canadian side in substantial numbers[80] (HBCA A.12/3: 368d; PAM CMS C.1/0, Microfilm reel A84, Reverend William Cockran to the Reverend R. Davis, Toronto, 11 August, 1846). The mining companies employed many of the Indians of the area as guides and canoemen (HBCA A.12/3: 369d). In September of 1846, for example, Charles McKenzie at Lac Seul learned that most of the Lake Nipigon Indians "passed the Summer with the Miners on Lake Superior..." (HBCA B.107/a/25:4). Having little communication with the Indians to the west and northwest of Lake Superior, where the sicknesses reigned during the summer and winter, there was

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[79]These miners came on the heels of white fishermen. Commercial fishing probably became a significant attraction to American and Canadian interests in the late 1830s. However, it gained momentum in the 1840s and William Nourse told James Hargrave in May of 1840 that the Americans were flooding Lake Superior for its fisheries (Glazebrook 1938: 312). Still, in terms of the numbers of people sent to Lake Superior from the east, mining would have easily eclipsed fishing.

[80]Six years later there were 8,000 non-natives engaged in mining on Lake Superior, and another 1,000 in commercial endeavours (Jacobs 1853: 10).

almost no chance of contracting these three deadly diseases.

This does not mean that the Lake Superior people were free from acute infectious disease in 1846, however. Instead, they were exposed to several other infectious diseases that were comparatively localised and, with one exception, lacked the severity of the measles, influenza and dysentery faced farther west. No doubt the miners played a role in these outbreaks, both by introducing disease and by encouraging the Lake Superior Indians to remain along the shore year-round, where the danger of exposure was much greater[81]. As has already been noted, mumps spread from Grand Portage to the Fort William area and on to Lake Nipigon[82]. At the latter post trader James Anderson also described a severe but unidentified affliction suffered by two women. This was characterised by delirium and vomiting of blood. Despite this, the returns for the outfit ending in June were excellent (*Loc. Cit.*) and there is no mention of any deaths in either post's correspondence. Later, in the fall of 1846, a considerable number of Lake Nipigon Indians fell seriously ill while visiting the miners of the Montreal Mining Company at Point Gourgon on Lake Superior, and had to be attended by the company's representative

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[81]Governor Simpson worried that Indians from as far away as the upper part of the Albany River would seek employment with the mining companies, and thus counselled Thomas Corcoran of Albany that:

> Should these reports have reached the Indians of the upper parts of the [Albany] River it would be proper to let them know that such demand for labor was merely temporary while the operations were going forward, but now that the shores of the lake are thoroughly examined in reference to mining purposes it is not possible that the Indians will be furthered [*sic*] employed in that way, and instead of benefiting by a visit to Lake Superior, they are more likely to injure themselves and their families by harsh treatment on the part of the miners who feel no interest in their welfare, **while the whole race would in due time be destroyed by the diseases contracted in their intercourse with strangers** [Emphasis mine]. (HBCA B.3/b/73: 17-17d)

[82]There was also an unidentified sickness among the Indians of Fort William in the summer of 1847 (HBCA D.5/20: 12).

(HBCA D.5/19: 318). Again, there is no indication that anyone perished.

Farther east, at Long Lake, there was a widespread but unidentified sickness during the summer and ensuing autumn of 1846. This was far more virulent. John Swanston wrote to Simpson from Michipicoten in February of 1847 explaining that:

> Sickness was very prevalent at Long Lake last Autumn amongst the Indians, and proved fatal to 17 individuals, man woman and children shortly after having received their supplies for the winter, and M[r] Laronde was apprehensive that he would hear of some other deaths, as there were some others very low late in the autumn. (HBCA D.5/19: 161d)

Three more deaths were reported at the Pic (*Loc. Cit.*). At Michipicoten, Swanston and his men suffered from a very severe ARD, accompanied by a cough[83], early in the summer of 1846 and by the fall they had come down with dysentery (HBCA D.5/18: 28d, 223d). The latter was not the same devastating disease that swept through the people farther to the west and resulted in countless deaths. Instead, this was probably an outbreak of some less severe affliction since Swanston stated only that he and his men "suffered somewhat". Clearly this disease lacked the virulence and epidemic nature of the bacilliary dysentery that was then striking Lac Seul and Osnaburgh. Had it been the same type, his reaction undoubtedly would have been far different. Once again, there is no indication that either the colds or dysentery resulted in any fatalities. In 1846 the people living along the northern coast of Lake Superior suffered from infectious disease,

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[83]This may have been influenza. John Swanston wrote to Governor Simpson, dated Michipicoten, 9 July, 1846:

> I am sorry to acquaint you that nearly the whole of the inhabitants of this Establishment are suffering severely from Colds, the cough attending which is most distressing and found by all more severe than the Influenza we had in '43 - I have become so weak from its effects, that for a few days could scarcely manage to go about and altho far from being well, am improving a little. (HBCA D.5/18: 28d)

in some cases fatally, but they appear to have escaped the worst of what was extant at this time[84].

SUMMARY

In 1846 several *ACIs* of widely varying severity struck the people of the Petit Nord. Those living in the western part were ravaged by sequential, in places concurrent, epidemics of influenza, measles and dysentery, as well as other localised and unidentified diseases. Those in the southeastern quarter suffered from several different afflictions that appear to have spread from the east, but none was as widespread as the eastern epidemics. Only the people living on the lower stretches of the Albany River seem to have escaped *ACIs* entirely. The multitude of sicknesses that struck these people in 1846 was symptomatic of the epidemic transition that had occurred during the previous era, and the mechanisms and routes by which these epidemics diffused had long been established as part of the region's system of diffusion. Although this was probably the worst year to date for epidemic disease in the Petit Nord, it was not greatly dissimilar to those that preceded it during the 1840s.

Measles followed the traditional route from Lake Huron to the Red River Settlement by way of the country to the south of Lake Superior and the Boundary Waters, and the Upper Missouri. This was essentially the same pattern as the 1819-20 epidemic. All three of the major epidemics of 1846 appeared in the Settlement during the spring

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[84]Including smallpox, which was then ravaging the Indians living to the south of the lake (United States 1846: 33; United States 1847: 91, 92; Harstad 1963: 216, 220).

before spreading to the north and east with the HBC brigades and through native movements. Typically, these diseases failed to penetrate far into the region, but caused great mortality where they struck. Conversely, those diseases that struck the Lake Superior region were more localised and were less mortal, with the exception of the outbreak at Long Lake. While the source of these afflictions is not known, there is a strong possibility that some were introduced by the growing influx of non-natives in the Lake Superior area, and through native and fur trader contacts with Sault Ste. Marie and Lake Huron.

CHAPTER 12 — CONCLUSIONS

This study has examined the epidemic history of the vast fur trading region known as the Petit Nord during the period 1670-1846. In doing so, it has answered a call for detailed epidemiological studies of aboriginal peoples in specific regions and at different times, not only within Canada, but within the New World as a whole. As a result, it has documented the epidemics and outbreaks that struck this region during that period. In addition to delineating the internal ways by which *ACIs* spread within the Petit Nord, it illuminates the larger patterns of disease diffusion that carried them into the region. This dual approach has resulted in new insights into the timing and distribution of these diseases, including the means by which they reached the interior of the continent and the limitations to their diffusion. These insights in turn bear upon the broader patterns of disease diffusion in the New World and in North America in particular.

A major finding of the study is that diverse changes both within and beyond the Petit Nord shaped the type and number of *ACIs* that struck the region. For many reasons, the region's disease load was altered during the study period in ways that led to more frequent outbreaks and epidemics and a wider variety of afflictions. These culminated in an epidemic transition within the region by the middle of the nineteenth century. Within the overall pattern, however, there were temporal and local exceptions among the far flung people of the Petit Nord, and thus disparities in the types and frequencies of diseases at the local level. In general, the increased frequency and variety of *ACIs*

reflected changes in the patterns of movement of people and innovations in transport technology. Together these changes brought the Petit Nord closer to external urban disease pools. In the process, the region slowly went from epidemic isolation to progressive inclusion within the disease frontier of formerly distant pools. By 1846, the Petit Nord had become a "near" region after having been "peripheral" throughout most of its history. In effect, it was part of the global process of disease homogenisation outlined by William McNeill (1976) in his study of the emergence and history of crowd diseases.

Historically, these changes began in the Petit Nord with the arrival of the HBC in James Bay in 1668. This event set the stage for much of the *ACI* activity that followed, as it also led to the expansion of the French fur trade into the region. Thereafter, diseases were periodically carried into the region by the annual brigades from Montreal and, in lesser degree, by the HBC's ship-based transport to Hudson Bay. Despite these new mechanisms of diffusion, the first major epidemics to reach the margins of the Petit Nord, the smallpox epidemics of 1669-70 and 1737-38, did so by spreading through aboriginal connections after having come to the eastern colonies from Europe. This was a pattern that can be documented in eastern Canada back to the 1630s, although it is not known if these earlier epidemics reached as far into the interior as the Petit Nord. However, the existence of this mode of diffusion among peoples connected to Sault Ste. Marie in 1669-70 lends support to the possibility that epidemics had reached the Petit Nord in the protohistoric period. Indians of the interior often visited the Canadian communities on

the St. Lawrence or traded for European goods through Indian middlemen such as the Montagnais, Algonquin and Huron, at least as early as the arrival of Old World diseases in the east.

For the Petit Nord, the period between 1739 and 1780 saw an increase in the number of *ACIs* and the first appearance of measles. Several factors contributed to this trend. Of considerable importance was the emergence of the HBC ships as a factor in introducing *ACIs*, mainly ARDs. This corresponded to a rise in the number of respiratory disorders affecting the region, most probably including influenza. These appear to have been most common among the HBC personnel and the Cree of the Hudson Bay Lowlands. There was also an increase in disease in the interior due to the French expansion during this period. It was also at this time that the horse arrived on the Northern Plains as a trading commodity, introduced through an aboriginal trading network that centred on Mesoamerica. While this undoubtedly increased the risk of diseases spreading from the southern part of the continent, it also intensified connections between the peoples of the Northern Plains and the native peoples living to the east, who acted as a bridge for eastern epidemics. Finally, some of the temporary changes in the arrival of epidemic disease in the region can be attributed to the disruption of the fur trade caused by the Seven Years War and its immediate aftermath. There were stretches lasting several years during this period when no *ACIs* appeared in either the eastern or the western part of the Petit Nord.

In identifying these earlier epidemics, this study has challenged a common belief in the secondary literature that the 1779-83 smallpox epidemic was the first significant or at least the first recorded epidemic in western Canada (Kehoe and Kehoe 1966: 434; Young 1979: 197; 1991: 35; Thistle 1986: 62, 63; Decker 1989b: 59). An extreme example of this belief was expressed by James G. E. Smith (1980: 811), who stated that "the Hudson's Bay Company archives do not provide evidence of epidemic disease [among the Cree and Chipewyan west of Hudson Bay] until the winter of 1781-82". Instead, it is necessary to look much earlier than 1781 for an epidemic baseline for the Canadian Northwest, and especially in the Petit Nord. Moreover, given the frequency with which *ACIs* struck the people of the Petit Nord prior to the merger of the HBC and the NWC, Young's (1991: 97) characterisation of the "Early contact" period (1670-1821) as one of excellent health among the Indians of the central Subarctic needs to be reassessed.

While the arrival of the horse on the Northern Plains increased the potential for the spread of diseases from far to the south, this did not translate into a significant increase in epidemic activity in the Petit Nord. Only once in its history did a major epidemic reach the region from Mesoamerica. Probably the most significant epidemic in the history of the Northwest, the 1779-83 smallpox epidemic was carried northward in stages from Mexico by aboriginal interaction, first through trading ventures and then through warfare. It is noteworthy that only smallpox spread through the Interior Plains

after the arrival of the horse on the Northern Plains, a development that overcame fundamental barriers to the diffusion of this disease from the south (Ramenofsky 1987: 167; Jenness 1989: 130). Without the advantages of the speed and range provided by the horse, earlier contacts between the Mesoamerican people and those of the Northwest would have been slow and too limited to foster the spread of smallpox, let alone other *ACIs*, over this great distance.

The 1779-83 epidemic entered the Petit Nord along two main transportation corridors. From the Saskatchewan River the disease moved along the Hayes and Nelson Rivers to York Factory, and from thence to the Severn hinterland. After reaching Severn it spread in the Hudson Bay Lowlands towards Albany Fort. About the same time, the disease also spread from the Red River region throughout the Boundary Waters to Lake Superior, and into the interior towards the Albany River. However, this sickness was variable in its effects. Mortalities among a few of the bands of this area were confined to the young, indicative of the band's prior exposure to smallpox. While it diffused widely within the region, its spread was not complete. Contrary to what Lytwyn claimed, it did not travel far along the Albany, but reached only to its headwaters. Nor did it strike at Lake Nipigon or the eastern part of Lake Superior. However, for many groups in the Petit Nord for which this was a virgin soil epidemic, the effects of smallpox epidemic were devastating.

Overall, *ACIs* appeared in the Petit Nord on a more frequent basis between 1783 and 1818, including in the interior of the region. This corresponded to a period of major fur trade expansion, both by the Canadians and by the HBC. However, there was also a decline in disease activity between 1810 and 1818. There were also intraregional ebbs and flows in the incidence of *ACIs* in the Petit Nord during this period, this time more the result of changing fur trade hinterlands than of external forces.

In the long run, the most important development of this era was the emergence of endemic disease pools in the larger American cities of the east. This put in place an essential component for epidemic transition in the region, as it meant that crowd diseases were now constantly available in eastern North America to diffuse into the interior of the continent. Initially, the new urban disease pools had little impact on the Petit Nord, and there was no sudden or momentous increase in the number of epidemics in the region following the emergence of these pools. Prior to 1819, only the 1788-89 smallpox epidemic can be traced to the movement of the American settlers into the interior, which was just beginning at this time. Because of the limited numbers involved in this westward migration during its initial phase, and the fact that this human flow was directed well to the south of the Upper Great Lakes, it was several decades before this migration and the expansion of the American settlement frontier had a significant epidemiological impact on the people of the Petit Nord. Events in 1819-20 signalled the beginning of this new era of increased epidemic activity. At this time the region was hit

by its first true compound epidemic. Both whooping cough and measles from the Upper Great Lakes area spread widely within the region. These two diseases overlapped in the Boundary Waters, and a third epidemic disease, influenza, diffused through the people living in the eastern part of the region. The simultaneous presence of three major epidemics was unprecedented in the region's history. Mortalities among some groups were extreme, rivalling those of the 1779-83 smallpox epidemic. Thereafter, a bewildering array of *ACIs* began to appear in the Petit Nord in rapid-fire succession, offering little respite for many of the native people of the region.

The Petit Nord underwent a remarkable transition following 1820, as it moved closer to the eastern pools (epidemiologically speaking) than it had ever been before. This coincided with the movement of large numbers of non-natives, and especially families of settlers, into the interior of North America from the east, many of them into the Upper Great Lakes area. As improvements in transportation technology enabled them to travel more quickly and in larger groups, *ACIs* began to penetrate farther, faster and more frequently. Here was the complement to the earlier development of the eastern disease pools, and together they unleashed a flood of epidemic disease on the Petit Nord. Only partly offsetting this epidemic transition was the disappearance of smallpox from the Petit Nord until much later in the nineteenth century, despite its frequent appearance in surrounding areas. Nonetheless, this period saw a wider array of diseases strike the region than ever before, and they were far quicker to return.

Although is it clear that the Petit Nord had undergone an epidemic transition by 1846 and was no longer a peripheral region, it is also clear that its relationship with the urban disease pools was not precisely the same as those of other near regions or populations as postulated by McNeill. Here, the true crowd diseases such as whooping cough and measles did not return often enough to become afflictions of childhood as, for instance, smallpox did in the smaller towns of the near populations of Britain during the eighteenth century, or as measles did in Iceland during the second half of the twentieth. In large part this was due to the absence of a regular movement of children and other susceptibles between the settlement frontier and the Petit Nord. Instead, *ACIs* reached the region either with the few adult non-natives who journeyed into the region or with aboriginal people who ventured beyond its limits. In both cases, there was little possibility for frequent reintroduction of crowd diseases since, once exposed, they would have acquired immunity that prevented contracting the disease a second time. Moreover, the low population density found throughout the Petit Nord during the study period prevented such afflictions from flaring up into epidemics affecting larger areas when they were introduced, except when a sufficient susceptible population had been built up. This might require several decades of births. As long as these conditions prevailed in the Petit Nord, they would not become childhood diseases in the region. Nevertheless, this should not be taken as evidence that an epidemic transition had yet to occur. Although not present here as frequently as among near populations who are in more intense and closer communications with endemic pools, these crowd diseases reappeared far more often

than had been the case throughout most of the study period. Moreover, the frequent, in some cases overlapping, epidemics of ARDs in the region are evidence that, by the end of the study period, the Petit Nord was well within the disease frontier of the urban east.

In addition to the movement of non-natives into the interior, other factors also favoured the spread of acute infectious disease to, and within, the Petit Nord following 1821. The establishment of several American trading posts on the Upper Missouri helped to bring sickness from the settlement frontier. The growth of the Red River Settlement during the post-merger period served to introduce *ACIs* into the Northwest, to prolong their stay by hosting them temporarily and, finally, to disseminate them throughout western Canada. At the same time, changes in the ethnic composition of the HBC brigades ensured that they assisted in the spread of epidemic diseases, particularly through the western and southeastern portions of the Petit Nord.

All of these factors had converged by 1846, leading to the worst epidemic year in the Petit Nord to that date. Major epidemics of influenza, dysentery and measles were joined by many other afflictions, and very few people in the region escaped from these disorders. All three of the main afflictions came through the Red River Settlement, with measles arriving from the Upper Great Lakes via the American trading posts of the Upper Missouri. Once hosted within Red River, the three diseases spread rapidly and widely with the HBC brigades, striking the western half of the region with great severity. They did not reach the Lake Superior area, where several other afflictions of variable severity

appeared instead. Moreover, no significant sickness reached the lower part of the Albany River during this year. Consequently, the marked frequency of *ACIs* in the Petit Nord in 1846 masked considerable variation in the fates of its inhabitants depending on the part of the region in which they lived.

The limited diffusion of the epidemics of 1846 illustrates a significant and consistent characteristic of the epidemics that struck the Petit Nord. None spread to the limits of the region. Whether due to buffer zones, relative distances from major corridors of travel, low population densities, limited contact between groups, the season of introduction or varying degrees of immunity, there were always some people who escaped a devastating visitation while those living nearby succumbed. In many cases, large parts of the region were disease-free while others were swept by disease. Moreover, there also could be significant variation in the effects of such diseases on those groups who were afflicted. Thus, rather than being a shared or common influence on the people of the Petit Nord, *ACIs* had a highly variable impact on the human landscape. They should not be seen as a unifying force, then, but instead as an agent of diversity. Given this diversity of experience, it is highly unlikely that protohistoric epidemics swept throughout the entire Petit Nord, let alone throughout the continent as a whole. This is similar to the findings of Harris (1994: 615) for the Pacific Northwest and of Newson (1992: 108-109).

The highly variable epidemic experiences of the people of the Petit Nord reinforces the need to reconstruct epidemic histories on a local scale in order to understand the effects of the introduction of exogenous diseases in virgin soil populations (e.g. Taylor 1989: 28; Harris 1994: 615; Waldram et al. 1995: 49-51; Crosby 1992). Indeed, under certain circumstances it may be necessary to consider the impact of these diseases on a family or band level, rather than at a regional or tribal scale. In turn, the inevitable disparities in their impact on different populations are important topics in their own right. Diverse secondary consequences such as shifts in balances of power, increased dependency by some, altered marriage and medical practices and even divergent spiritual beliefs could all come as a direct result of variations in epidemic impact, either in the short term or over a succession of epidemics (e.g. Boyd 1985: 525-531; Trimble 1985: 293, 296-299; Peers 1994: 20-21; Taylor 1977; Krech 1984a: xvi; 1984b; Hurlich 1983).

One important secondary consequence in the Petit Nord was the movement of people following some major epidemics. It has long been recognised, and has been well documented, that especially severe virgin soil epidemics were often followed by migrations of aboriginal people (e.g. Wissler 1936: 36; Boyd 1985: 525-526; Dobyns 1983: 302-306; Taylor 1977: 64-66; 1982: 90-94; Ramenofsky 1987: 104; Trimble 1985: 293). These movements occurred as differential mortality created population vacuums, making former boundaries more permeable, or as surviving groups sought to maintain minimum viable sizes. Although Rogers (1963: 66) doubted that the epidemics that

struck the Cree and Ojibway of northwestern Ontario "affected the settlement patterns in any significant manner," both dynamics were in evidence in the Petit Nord during the study period. Population movements of varying extent followed the epidemics of 1737-38, 1779-83 and 1819-20. With further research it is likely that evidence of other epidemic-induced movements will emerge.

The epidemic record of the Petit Nord suggests that the sequence of *ACI* introduction was not entirely random, but instead reflected specific attributes of each disease as well as the changing circumstances surrounding the region's communication with the external disease pools. Certain diseases appeared early and often, and others rarely and not until towards the end of the study period. This is consistent with Ramenofsky's (1987: 167) concept of disease-specific *diffusion potential*, referred to in Chapter 2. In this case, however, the epidemic record of the Petit Nord supports the basic concept but does not conform precisely to Ramenofsky's rankings. As expected, smallpox first struck the region during the early part of the contact period, appearing on several occasions prior to 1800. Later epidemics were prevented by vaccinations, but it is likely that smallpox would have been a regular visitor to the Petit Nord during the nineteenth century had it not been prevented by artificial means. Measles arrived later, first breaking out in 1751 and then again in 1819-20, 1828 and finally in 1845-46. Chickenpox (1835), mumps (1841) and scarlet fever (1843) were among the last to arrive, reflecting their more limited diffusion potential. These latter diseases arrived only during

the period when large numbers of settler families began to move into the areas immediately to the south and southeast of the Petit Nord. As such, the relative order of appearance of these diseases tends to support Ramenofsky's classification of diffusion potential.

Running contrary to the predicted order of arrival were the acute respiratory diseases (ARDs) and whooping cough. Although colds and influenza have comparatively brief infectious periods, placing them in the third class of diffusion potential according to Ramenofsky, they arrived early on in the Petit Nord. Scattered colds broke out at Albany as early as 1715-16, with a larger outbreak in 1719, well before the first epidemics of measles (second class) and whooping cough (first class). By the 1750s, outbreaks or epidemics of colds or "great colds," some of them influenza, were becoming increasingly common among both the Europeans and natives around the HBC's Bayside posts. By the third decade of the nineteenth century influenza had emerged as the *ACI* appearing most frequently in the Petit Nord (excluding other ARDs). Conversely, whooping cough appeared far later in the epidemic record than would be predicted based on its infectious period. It first broke out in 1806, long after measles and the ARDs, but returned at least once a decade following the 1819-20 epidemic. Given these departures from the predicted order of first introduction, it seems likely that the diffusion potential of a given *ACI* may, in part, be determined by disease-related characteristics besides the infectious period.

Ramenofsky (1987: 170) noted that the singular ability of the smallpox virus to survive for prolonged periods on fomites enhances its diffusion potential. Several other possible disease-related factors affecting diffusion potential can also be identified. For instance, Trimble (1989: 42-43) listed pathogenicity/virulence, infectivity/transmission, invasiveness and stability of the disease agent as core factors affecting the spread of a specific disease in a given population. While diseases with higher rates of infectivity may be more successful at prolonging a chain of infection than less infective diseases, highly pathogenic afflictions may actually hinder diffusion by preventing travel. This was especially important when humans provided the power for travel, such as walking or canoeing, but became far less so as mechanised forms of travel were introduced, such as the steamboat or the train. Moreover, afflictions with longer incubation periods will have greater opportunity to travel longer distances from the initial point of infection before the onset of the disease (relocation diffusion), and thus to find new susceptibles (Burnet and White 1972: 122-123; Cook and Lovell 1992b: 235. e.g. Trimble 1985: 79; Boyd 1985: 44; Crosby 1976: 297). Of crucial importance in the context of the introduction of *ACIs* to more isolated regions such as the Petit Nord is the immunological nature of the disease. If most of the contact between the isolated population and the disease pools is through the movement of adult individuals who were born and raised in the proximity of a large urban area, then there would be limited opportunities for crowd diseases to spread to the region. Conversely, diseases that do not provoke a lasting immunity, including the ARDs that flooded the Petit Nord after the 1820s, would have a much greater opportunity to

arrive through these connections, since all would be susceptible. Possible factors influencing a disease's diffusion potential therefore extend far beyond the infectious period.

The concept of diffusion potential is nonetheless a useful tool for historical studies of exogenous disease introduction. A carefully considered system of classification could help shed light on the diffusion of *ACIs* into areas for which there is a limited historical record. It is clear, however, that Ramenofsky's rankings require fine-tuning through comparison with other situations, and it is equally clear that a simple ranking scheme based on a single disease characteristic would prove inaccurate in many contexts.

Finally, this dissertation has documented some of the broader patterns of diffusion that carried *ACIs* to the Petit Nord between 1670 and 1846. What is perhaps most striking is the apparent infrequency of diffusion from Mesoamerica through the interior Plains, even after many Plains groups had adopted the horse, as well as the consistency with which they spread from the east. No doubt this was partly a function of the region's distance from the disease pools, with far more afflictions reaching the Petit Nord from the relatively nearby eastern pools. However, orientation and intensity of communications also appear to have been factors, as several of the epidemics spread from the east to the Pacific Northwest, a distance that easily rivalled that from Mesoamerica to the Petit Nord. This may mean that the connections running from the east to the west were better suited

to transporting disease than those running from the south to the north. Still, much more work needs to be done in order to clarify these broader patterns of diffusion. If this was indeed the case, then it would suggest that there was little chance that hemispheric protohistoric pandemics from Mesoamerica spread directly to the Northern Plains during the sixteenth century, a period for which the barriers to diffusion were far greater than during the historical period.

APPENDIX 1: EPIDEMIC REFERENCES IN THE PETIT NORD: 1784-1818

	NATURE OF DISEASE	SOURCE
1784	Universal colds, sore throats at Albany Fort in December and following August (1785).	B.3/a/84: 15-15d, 53d
1785	Smallpox said to be raging again inland from Gloucester House in July.; Suspicious sickness among Indians at Gloucester House in October.	B.78/a/12: 21d; B.78/a/13: 6
1786	A great sickness west of Gloucester House.	B.78/a/16: 28, 29+
1787	Epidemic catarrhs and sore throats at Albany Fort in January to July.	B.3/a/88: 16d, 17, 23+
1789	Suspicious sickness inland from Severn (Winter 1789-90).	B.198/a/39: 34
	Some sickness, perhaps significant, at Gloucester House in late winter/spring 1789.	B.78/a/18: February, 20d
	Outbreak at Henley House and at Gloucester House in spring.	B.86/a/43: 16, 45, 47d
1790	Suspicious sickness at Severn (Winter 1790-91).	B.198/a/40: 33d, 35
	Sore throats, colds, etc., men and Indians at Henley House (Fall-Spring 1790-91).	B.86/a/45: 3, 8, 9+
1792	Outbreak of colds at Albany Fort in July.	B.3/a/93a: 56, 56d
1793	Great death at Lake of the Woods and Lac la Pluie in summer.	B.166/a/1: 2d
1794	A plantation of sickly Indians at Severn in December.	B.198/a/46: 18
	Epidemic sickness(es) at Albany Fort fall 1794-spring 1795.	B.3/a/96: 5, 6d+
1795	Suspicious mortality at Lac la Pluie (Winter 1795-96)	B.105/a/3: 32
	Suspicious sickness at Severn in August, possibly from York Factory	B.198/a/47: 5
1796	Epidemic sickness at Moose.	B.198/a/49: 44d
	Colds at Henley House from Albany Fort in September.	B.86/a/52: 4d
	Epidemic colds at Albany Fort from sloop September-November. Dysentery and colds from Albany Fort to Martins Falls.	B.3/a/97: 33d, 34, 34d; B.3/a/98: 1, 1d, 2+; B.3/b/34: 5d-6, 8d, 22; B.3/a/100: 1, 4d
	Epidemic sickness at Albany Fort, Martin Falls and Moose Fort.	B.123/a/3: 13, 14d, 18d, 33
1797	Epidemic colds and possibly another, worse, disorder at Lac la Pluie (April 1797).	B.105/a/4: 20
	Suspicious mortality at Escabitchewan in May; Epidemic(s) at Escabitchewan in Summer and Fall.	B.64/a/2: 14d; B.64/a/3: 3, 7d, 8, 16

	NATURE OF DISEASE	SOURCE
1798	Epidemic sickness among Indians northwestward from Albany Fort in April.	B.3/a/101: 18d
	Epidemic sickness at Sandy Lake in October-November.	B.192/a/1: 4d+
1799	Epidemic colds at Albany Fort in winter 1799-1800.	B.3/a/103: 13d, 21d
1801	Epidemic at Albany Fort in July, possibly from Martin Falls.	B.3/a/104: 11, 20d, 21+
1802	Smallpox at Lake of the Woods.	Tanner 1987: Map 32
1803	Epidemic sickness at Martin Fall in July.	B.123/a/8: 32, 33
	Illness of short duration at Osnaburgh in March.	B.155/a/17: 16
1804	Unidentified sickness on Rainy River	Tanner 1987: Map 32
	Epidemic sickness at Albany Fort in spring-summer.	B.3/a/106: 13, 20+
1805	Epidemic colds at Albany Fort in September, possibly from schooner.	B.3/a/108: 3d
1806	Epidemic colds at Albany Fort in September.	B.3/a/109: 2
	outbreak at Portage de l'Isle; possibly whooping cough.	Nelson Papers
1807	Epidemic sickness at Osnaburgh Fall-winter.	B.123/a/12: 7
	Epidemic colds at Albany Fort in August; sickness at Albany Fort in Winter 1807-08	B.3/a/109: 18; B.3/a/111: 5
1808	Suspicious sickness, colds, at Albany Fort in July, September.	B.123/a/13: 13; B.3/a/111: 12, 18
	Colds at Sandy Narrows from Osnaburgh in August.	B.193/a/3: 2d, 3, 3d
1815	Suspicious sickness at Osnaburgh in winter-spring.	B.155/a/27: 13+
1817	Outbreak of dysentery at Michipicoten in September.	B.129/a/8: 4d
1818	Possible sickness at Gloucester House in spring	B.78/a/26: 13d
	Inflammatory sore throats at York Factory August to September.	B.239/a/126: 8

APPENDIX 2: EPIDEMIC REFERENCES IN THE PETIT NORD: 1821-1845

Part 1: 1821-1831

	NATURE OF DISEASE	SOURCE
1822	Influenza at Norway House and among Indians in late summer and fall.	B.154/a/10: 8, 17, 36
1823	Possible sickness at Jack Lake, to the southwest of Island Lake	B.93/a/4: 23
	Outbreak of colds and coughs at Escabitchewan in December.	B.64/a/10: 9
	Outbreak of colds and dysentery at Albany in August.	B.3/a/128: 6d
1824	Sickness among the inland men at York Factory in July	B.239/a/132: 27d
	Outbreak of colds at Albany in January.	B.3/a/128: 13d
1825	Throat sickness at Osnaburgh (winter 1825-26)	B.155/a/37: 16
	Whooping cough around Flying Post and Mattagami, brought from Drummond Island, Lake Huron.	B.70/e/3: 2d; B.124/e/3: 2d
	Epidemic colds at Moose in summer, probably from Michipicoten, whooping cough in September and into winter.	B.135/a/128: 7d, 8d, 9, 12d, 15, 30; B.135/e/15: 1
	News of epidemic colds at Moose followed by whooping cough at Albany.	B.3/a/130: 7, 10, 12, 20; B.3/c/54: 6d; B.3/e/12: 2, 2d
1826	Outbreak of severe colds at Lac la Pluie in October.	B.105/a/12: 5d
1827	Quinsy at Cat Lake (north of Lac Seul).	B.155/a/38: 24d
	Whooping cough Oxford House.	B.156/a/10: 9d, 10d, 11, 12d
	Epidemic colds at Pic from Sault Ste. Marie (by Montreal Canoes).	B.162/a/1: 2, 2d, 3d
	Epidemic colds and coughs at Albany in June-September.	B.3/a/132: 2, 2d, 3, 8, 8d
	Unidentified sickness at Lake Nipigon in June-July.	B.149/a/10: 1d, 2
	Epidemic colds in June, later on brigades to Long Portage. Possibly a second disease later in the year.	B.129/a/12: 3, 5, 10, 36

	Influenza in the Red River Settlement in early part of 1827. Whooping cough in fall-winter at Red River Settlement and Lake Manitoba, followed by epidemic influenza in January of 1828.	B.235/a/9: 13, 14; PAM CMS C.1/M.1 [Reel A77] P. 228 Rev. Jones to the Secretaries; P. 319, 323, 327 Jones "Journal" Oct. 15, 1826-Oct. 25, 1827
1828	Epidemic dysentery at Pic, Lake Nipigon throughout Lake Superior District.	B.162/a/2: 4, 4d; B.149/a/11: 16, 18, 32
	Dysentery at Michipicoten and at Lake Nipigon. Typhus at Bachewana Bay.	B.129/a/13: 1d, 23d, 21d
	Epidemic typhus at Sault Ste. Marie (fall-winter of 1828-29).	B.194/a/4: 9, 15
	A great many sick Indians at Trout Lake (Severn), probably winter 1827-28.	B.220/a/5: 24d
	Brief outbreak of unidentified disease at Norway House in July.	B.154/a/16: 7
	Measles at Lac Seul fall and winter of 1828-29 brought from Red River Settlement.	B.107/a/7: 16, 16d, 18
1829	Possible Sickness at Cat Lake (winter 1829-30).	B.155/a/41: 13
	Violent colds (from Michipicoten) at Pic.	B.162/a/3: 13, 14, 15
	Epidemic colds at Michipicoten in June.	B.129/a/14: 1
	Epidemic sickness at Red River Settlement and Lake Manitoba, possibly colds.	B.235/a/12: 7, 7d, 8d
	Suspicious sickness and mortality Lac Seul area in fall of 1829.	B.107/a/8: 3, 4, 7d, 14d
1830	Dysentery at Oxford House.	B.156/a/12: 41
	Dysentery at York Factory in January, epidemic colds in July-Aug.	B.239/a/141: 37, 38, 40, 72, 75, 81
	Outbreak of sickness among brigade coming to Norway House from York Factory in July, probably colds.	B.154/a/19: 2d
	Epidemic colds among Indians at Albany in June.	B.3/a/135: 4
1831	Epidemic colds at Norway House in July.	B.154/a/20: 8d

Part 2: 1832-1840

	NATURE OF DISEASE	SOURCE
1832	Suspicious sickness at Merry's House.	B.156/a/13: 2d
	Epidemic colds at Norway House in July.	B.154/a/22: 8d
	Bowel disease at Pic.	B.162/a/6: 8, 9d, 11, 18
	Sickness among some of at Fort Alexander Indians.	B.4/a/7: 2d, 9d
	Suspicious deaths at Nipigon.	B.231/a/12: 15
	Epidemic colds at Michipicoten from Sault Ste. Marie in February.	B.129/a/16: 18d; B.129/a/17: 3d, 6d
	Cholera in Canada, US, at Mackinac and quarantine at Sault Ste. Marie.	B.105/a/17: 3d
	Whooping cough at Lac Seul in August.	B.107/a/11: 23
1833	Bowel complaints at Pic.	B.162/a/7: 6d, 7
	Minor illness at Lake Nipigon.	B.149/a/16: 1d, 2d
	Epidemic colds at Fort William.	B.231/a/13: 9d
	Outbreak of colds at Michipicoten in March. Another in the summer. Latter affects Indians at Montreal and Agaywam Rivers.	B.129/a/17: 15; B.129/a/18: 5d, 6d, 7
	Sickness among the Michipicoten Indians during the fall and part of the winter.	B.129/e/9: 4d
	Epidemic "colds" and dysentery at Norway House Jan-May.	B.154/a/22: 28, 28d, 36, 38
	Suspicious sickness and mortality among Indians inland from Severn during winter (1832-33)	B.198/a/70: 14
	Influenza at Martin Fall brought from Albany in July.	B.123/a/32: 4, 5
1834	Whooping cough at Norway House, Nelson River, Oxford House.	Simpson Refs
	The Severn natives suffer from some "inward complaint" in December.	B.198/a/71: 14d
	Outbreak of dysentery at Norway House in February-April, news of sickness at York Factory in April, whooping cough at Norway House August-December.	B.154/a/24: 64, 76, 79-80; B.15/a/25: 49
	Epidemic whooping cough (Nov.-Feb.) followed by outbreak of colds at Lac la Pluie. No mention of influenza — see Fort William.	B.105/a/19: 8d, 12d, 16d, 17d
	Whooping cough at Lac la Pluie in fall, accompanied by the cold.	B.105/e/10: 1
	Whooping cough followed by bowel complaints in Red River Settlement.	Glazebrook 1938: 148, 160, 164

	Whooping cough killed many children in the Red River Settlement during the summer. Whooping cough and another sickness of the head, stomach and eyes during summer in Red River Settlement.	PAM CMS MG 7 D13 Belleau Collection [Reel 1] Provencher to Signay, 4 Sept 1834; Provencher to Signay, 17 + 18 Dec., 1834
	Whooping cough raging at the Rapids and Indian Settlement, and fatal to children.	PAM CMS (PAM CMS C.1/M.1 [Reel A77] P. 85 Cockran "Journal" Aug. 14, 1834-Aug. 2, 1835
	Epidemic colds at Pic in February.	B.162/a/7: 19d
	Epidemic colds at Albany in June.	B.3/a/138: 18d, 19
	An epidemic sickness, possibly Influenza, at Fort William said to be from Lac la Pluie (December-January 1834-35).	B.231/a/14: 15d, 15d, 16, 16d, 17, passim; B.117/a/9: 10
	Strange epidemic disease at Lac Seul, and a very bad cough (same as at Lac la Pluie) undoubtedly whooping cough.	B.107/a/13: 11, 12d, 14d, 18d, 20, 21
1835	Possible Sickness at Cat Lake (winter-spring 1834-35).	B.155/a/47: 2
	Epidemic sickness of swelling in head and throat at Oxford House.	B.156/a/16: 30d, 32d
	Possible outbreak at Sturgeon Lake (from Lac Seul in February?).	B.211/a/7: 5, 5d, 6, 7
	Continuation of influenza from previous year reported at Fort William in June (but Spaniard's band so may have spread to Grand Portage), a different influenza in September at same, and an outbreak of fluxes at same in January of 1836.	B.231/a/15: 2ad, 7d, 16
	Sickness among crew of brigade arriving at Lake Nipigon in August.	B.149/a/18: 5
	Influenza at Lac Seul in summer to fall and followed by chicken pox in winter	B.107/a/14: (page before 1), 1, 22d, 6
	Reference to an outbreak of water on the brain among children at York Factory in previous spring (1834), epidemic dysentery among the men of York Factory in March -June, same as occurred previous year (1834). Severe colds and sore throats in June-July among men, Indians and the crews of all of the boats, i.e. influenza.	B.239/a/148: 18bd, 37 40ad, 41d, 44d, 47, 47d, passim
	Epidemic influenza at York Factory and among brigades in early part of the summer, also at Red River Settlement during summer and fall, winter.	PAM MG 2 A6: 100, 101
		B.154/a/26: 6, 7, 8,

	Epidemic influenza at Norway House in June-October.	9, 10, 28, 31
	Dysentery among natives at Severn in June-Aug, fear of complaint prevailing at York Factory (influenza?), suspicious deaths at Oxford House.	B.198/a/73: 9
	Suspicious deaths at Fort Alexander.	B.105/a/19: 17d
	Sickness of several people at Fort Alexander, mainly children, due to inflammation of the brain. See York Factory.	B.105/e/10: 1
	Influenza in Red River Settlement in summer, later several deaths at same due to "suffusion of the brain". See York Factory and Fort Alexander.	Glazebrook 1938: 207
	Colds brought from interior to Albany in February by packeteers.	B.3/a/139: 17, 17d
	News of sickness among Pic Indians winter of 1835-36.	B.117/a/10: 7d
1836	Suspicious "swelling" sickness at Michipicoten, possibly at Bachewana.	B.194/a/9: 7d
	Influenza at Fort William in summer.	B.231/a/16: 4d
	Widespread sickness at Lake Nipigon in summer.	B.149/a/19: 24, 27
	February coughs accompanied by headache among Lac Seul men.	B.107/a/14: 7d
1837	Smallpox general on plains.	Simpson References
	Influenza at Oxford House and York Factory, smallpox on plains.	B.156/a/17 — 7d, 8, 9, 11d, 13, 34d
	Outbreak of influenza at Norway House in early summer.	B.154/a/29: 11
	Influenza in Red River Settlement early in June had just come from US to Red River via the plains Indians.	PAM CMS C.1/0 [Reel A85] Cockran Journal 1836-37
	Influenza in the Red River Settlement.	PAM CMS C.1 [Reel A84] Cockran to the CMS Aug 2, 1837
	Measles-like sickness at Fort William and Grand Portage summer.	B.231/a/17: 3d4, 4d, 5, 6, 6d
	Epidemic dysentery at Lake Nipigon.	B.149/a/19: 24; B.149/a/20: 6a, 10
	Colds brought from interior to Albany by packeteers in Feb.	B.3/a/141: 21d, 23d
	Fort Pelly boats had been suffering influenza on return journey from York Factory.	B.159/a/17: 2

	Strange disease epidemic at Lac Seul in fall and winter. Breaking out at the mouth and nose, tongue bloated and scalded. Men and Indians.	B.107/a/16: 13,17d
1838	Prevalence of headaches and pain in shoulders at Oxford House.	B.156/a/18: 2d, 3d, passim, 31
	Possible sickness at Pic.	B.162/a/10: 17, 19
	Severe bowel complaints in February at Norway House.	B.154/a/29: 58, 67, 71, 73
	Outbreak of St. Anthony's Fire at Michipicoten.	B.129/a/19: 3, 4d
	Dysentery in August at Martin Fall.	B.123/a/39: 6d, 22, 22d, 23, 31, 33d; B.123/e/14: 4
1839	Outbreak of colds at York Factory.	B.239/a/151: 40
	Reports of widespread sickness among the Lac Seul Indians in February.	B.107/a/17: 12
	Outbreak of colds at Michipicoten in May, several men complain of stomach problems in December.	B.129/a/19: 20; 19
	Epidemic colds at Martin Fall in June. Epidemic influenza in October-December, possibly from Albany.	B.123/a/41: 1, 1d, 2, 3d, 11d, 15
	Epidemic sickness at Albany in August-September.	B.3/a/145: 1, 5, 5d, 6d, 7
1840	Outbreak of colds and coughs at York Factory.	B.239/a/152: 39-39d
	Severe colds among Indians to the westward of Lac Seul in September.	B.107/a/19: 3d
	Outbreak of cholera-like disease at Norway House in March among the children, probably from Red River Settlement.	B.154/a/33: 31
	Unknown sickness among Indians of Pays Plat (Lake Superior) winter of 1839-40.	B.129/a/22: 1

Part 3: 1841-1845

	NATURE OF DISEASE	SOURCE
1841	Mumps at York Factory.	Simpson References
	Frequent sickness among men at York Factory in Autumn, but not identified as mumps, an Indian with mumps in January of 1842.	B.239/a/155: 23
	Respiratory complaints at Osnaburgh (from Lac Seul) in January.	B.155/a/53: 9d, 12d
	Sickness on Saskatchewan Boats (at Oxford House).	B.156/a/20: 6
	Severe epidemic sickness at Michipicoten, Bachewana, Sault Ste. Marie, Long Lake and probably Pic.	B.162/a/11: 19, 22, 28d
	February-March men unwell with bad colds at Michipicoten.	B.129/a/22: 19, 19d
	Outbreak of colds among Indians at Albany in August.	B.3/a/148: 3
1842	Smallpox at Sault Ste. Marie. Colds and fever at Michipicoten. Whooping cough at Fort William	Simpson References
	Suspicious sickness and mortality near Severn in fall and winter of 1842-43.	B.198/a/84: 14, 20, 20d, 32
1843	Whooping cough at Lake Nipigon and Norway House. Influenza at Sault Ste. Marie, Michipicoten, York Factory, Norway House, Rat Portage and Fort Alexander. Dysentery at Michipicoten, York Factory, Lake Nipigon and Norway House. Scarlet fever at Norway House, Fort Alexander, Rat Portage, Fort Frances, Red River Settlement, Berens River and Nelson River. Unidentified at Island Lake and Oxford House.	Simpson References
	Epidemic influenza at Sault Ste. Marie and Lake Huron in July, August at Michipicoten.	B.134/c/56: 58, 180
	Epidemic in Red River Settlement, cold and cough with much expectoration sore throat, constant inclination to cough. Possibly concurrent whooping cough.	PAM CMS [Reel A96] Journal of Rev. Smithurst — Feb- March 1843

	Attendance at the school had been down in March-May due to whooping cough. Same to same, Aug 1, 1844. In August 1843 another decrease due to scarlet fever. Cowley to the CMS Secretaries Dec. 28, 1843 scarlatina in Red River Settlement likely to become general.	PAM CMS C.1/0 [Reel A86] — Letter Donald Gunn to Cockran Grand Rapids, 6 July 1843
	Scarlet fever in Red River Settlement.	PAM MG 2 C19 *Bunn Papers* Bunn to Bayley; PAM MG1 C9 #6 Finlayson to HBC Govs, Chief Factors Dec. 2, 1843; Glazebrook 1938: 452.
	Many sick men at York Factory in February.	B.239/a/157: 22d, 34d, passim; B.239/a/159: 3d, 4, passim
	Unidentified at sickness Osnaburgh (winter 1843-44).	B.155/a/55: 13, 13d, 14, 17
	Unidentified sickness at Oxford House and Island Lake (probably Influenza or whooping cough) during fall and winter 1843-44).	B.156/a/23: 11, 13d, 25d, 33d, 34d
	Reports of sickness among the Trout Lake Indians in June.	B.198/a/85: 16
	Strange mortality at Lac Seul, but no "running sickness" early in the year. Followed by reports of smallpox at Lake Nipigon early in summer and then scarlet fever in the fall.	B.107/a/21: 9, 9d, 11d, 16d; B.107/a/22: 1d, 5d, 6, 12d
1844	Whooping cough Norway House. Unidentified sickness at Lac Seul	Simpson References
	Whooping cough at Norway House in September, poss. From the Saskatchewan River. Some sickness at Berens River winter of 1844-45 (perhaps whooping cough?).	B.154/b/1: 48d, 53
	Epidemic sickness at Severn and among the Indians January-February, October	B.198/a/85: 20, 21d, 24d; B.198/a/86: 3
	Outbreak among men at Trout Lake November.	B.220/a/6: 24

	A crippling disease at Lac Seul in early part of year. McKenzie and brigade suffered sickness on way back to Lac Seul from Albany in summer. Men and Indians there to meet them were already sick with colds and coughs	B.107/a/22: 13; B.107/a/23: 1, 3, 6d, 7, 8d, 9d, 10d, 11d, 14
	August a "bilious fever" epidemic breaks out in the Red River Settlement. October 1844 a typhus-like disease in the Red River Settlement, a "local cholera" in November. Whooping cough at the Pas in September.	PAM CMS C.1/M [Reel A78] p. 531 Cockran to the Secretaries, 30 July 1845: 531. p. 541, 542, 543 Cockran journal Aug 1844-July 1845. P.612, 620 Hunter's Journal 1844-45.
1845	Measles at Sault Ste. Marie.	Simpson References
	Two sicknesses, August and December at Osnaburgh.	B.155/a/57: 5d, 12, 13
	Epidemic colds (May-June) followed by epidemic influenza at York Factory and environs (July-August).	B.239/a/161: 36, 37, 44d, 45d, 47, 47d, 48, 48d, 49, 51d, 54
	Epidemic influenza at Norway House summer.	B.154/b/3: 2d, 3d
	Epidemic influenza at Norway House in July among brigade crews.	B.154/b/1: 60d; B.154/a/45: 7d, 7d, 11, 17
	Epidemic influenza at Red River Settlement in spring, a type of cholera in summer. Influenza May of 1845. March 21 people coughing en masse at Indian Church. Influenza had been in the upper part of the settlement for some time, as of May 1.	PAM CMS C.1/M [Reel A78] p. 531 Cockran to the Secretaries, 30 July 1845: 531; P.555, 557 Cockran Journal 1844-45; P.580 Smithurst Journal 1844-45; P.587 Smithurst Journal April-July 1845

Fever-swollen throat in Red River Settlement Aug 1845-Jan. 1846	PAM CMS C.1/L.1 (Reel A 81) Cockran Journal 1845-46
Much sickness among Severn Indians during summer of 1845.	B.198/a/88: 5d
Many epidemics in Lac Seul vicinity some from west, some disease brought by McKenzie from Albany. Coughs, dysentery, peeling skin, sore bones etc. See D.5/18: 105 for evidence of disease at Fort Alexander in summer of 1845. A fatal disease at Lac Seul undated, great sickness during summer 1845	B.105/a/24: 1, 1d, 2, 3, 4d, 5, 6d, 8, 10d; B.3/c/1: McKenzie to Corcoran Lac Seul undated [but either 1844 or 1845]; same to same, Lac Seul 30 Aug. 1845

APPENDIX 3: DISTRIBUTION OF SMALLPOX VACCINE IN 1838

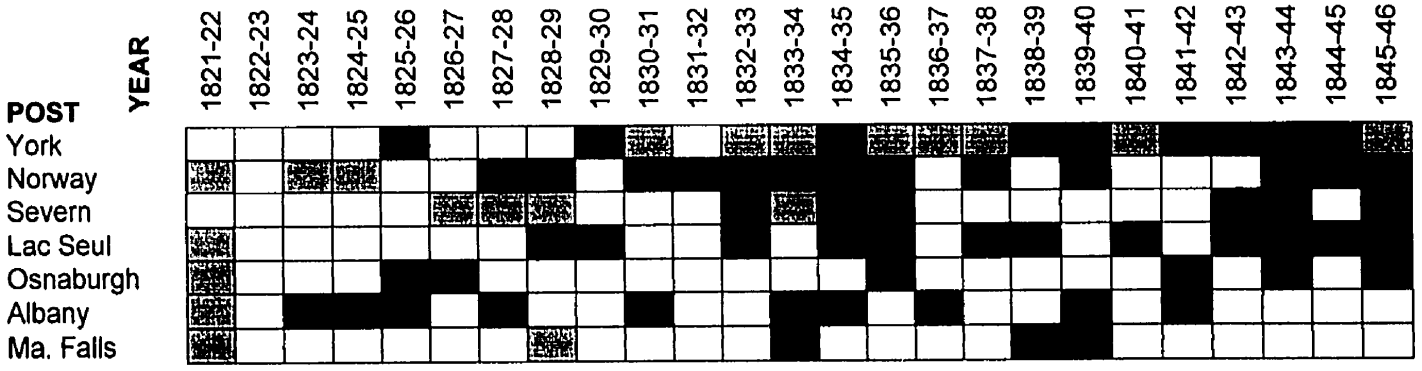
DISTRICT	# OF JARS	DISTRICT	# OF JARS
Athabasca Lake	6	Fort Frances	5
Mackenzie River	8	Norway House	5
English River	5	Oxford House	3
Edmonton House	8	Arctic Expedition	2
Carlton House	3	Churchill [Fort Prince of Wales]	2
Cumberland House	3	Severn	1
Swan River	6	York Factory	5
Red River	4	D^{r} Bunn Red River Settlement	2
Fort Alexander	2		
		Total	59 Jars

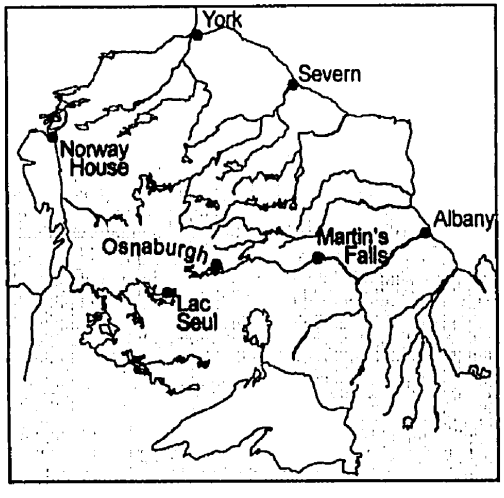
(Source: HBCA B.239/a/151: 16)

On May 29, 1838, shipments of smallpox vaccine were sent from London to York and Moose Factories on board the HBC ships *Prince Rupert* and *Prince of Wales* for distribution throughout the HBC's territories (HBCA C.4/1: 20-21). In the Southern Department the late arrival of the *Prince of Wales* threw some confusion into the distribution of the vaccine supply, and the necessary equipment was not sent to the inland posts until the following January (HBCA B.3/b/63: 2; 3d, 4, 4d, 13d). Nevertheless, the traders at Albany, Osnaburgh House, Lac Seul, and Martin's Falls all received the material and were able to complete their vaccinations (HBCA B.3/a/145: 1; HBCA B.123/e/14: 4; HBCA B.3/b/63: 27d; HBCA B.155/a/50: 20). Shipment inland proceeded more regularly in the Northern Department, under the direction of Chief Factor James Hargrave of York Factory. On December 8, 1838, the winter packet was

closed in preparation for its departure into the interior two days later. In the journal Hargrave carefully noted the destination of each jar of vaccine. This table shows the destinations of the vaccine that Hargrave sent inland. Note the provision of vaccine for the Arctic expedition and for Dr. John Bunn of the Red River Settlement.

Appendix 3 — Distribution of Smallpox Vaccine





APPENDIX 4: SIGNIFICANT DISEASE OUTBREAKS RECORDED AT SELECTED POSTS, 1821-46

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These documents are housed in the *Provincial Archives of Manitoba*, in Winnipeg. The following refers only to those documents that have been cited in this dissertation.

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HBCA A.1/172	Minutes of Subcommittee 1872
A.6/2	London Correspondence Book Outwards – HBC Official 1688-96
A.6/25	London Correspondence Book Outwards – HBC Official 1838-42
A.10/2	London Inward Correspondence – General 1817-1835
A.11/114	London Inward Correspondence from HBC Posts, York Factory 1716-56
A.11/44	London Inward Correspondence from HBC Posts, Moose Factory 1774-1784
A.12/3	London Correspondence Inward from Governors of HBC Territories – Sir George Simpson 1846-47

SECTION B: RECORDS OF NORTH AMERICAN POSTS

Post Journals

B.3/a/	Albany Fort
B.4/a/	Fort Alexander
B.16/a/	Berens River
B.22/a/	Brandon House
B.23/a/	Brunswick House
B.27/a/	Carlton House
B.39/a/	Fort Chipewyan
B.42/a/	Churchill Fort
B.49/a/	Cumberland House
B.51/a/	Fort Dauphin
B.59/a/	Eastmain
B.64/a/	Escabitchewan
B.78/a/	Gloucester House
B.86/a/	Henley House
B.93/a/	Island Lake

B.105/a/ Lac la Pluie
B.107/a/ Lac Seul
B.117/a/ Long Lake
B.123/a/ Martin's Falls
B.129/a/ Michipicoten
B.135/a/ Moose
B.145/a/ New Brunswick House
B.149/a/ Nipigon House
B.154/a/ Norway House
B.155/a/ Osnaburgh House
B.156/a/ Oxford House
B.162/a/ Pic
B.166/a/ Portage de l'Isle
B.186/a/ Rupert House
B.192/a/ Sandy Lake
B.193/a/ Sandy Narrows
B.198/a/ Severn
B.211/a/ Sturgeon Lake (Albany River)
B.220/a/ Trout Lake (Severn)
B.231/a/ Fort William (Lake Superior)
B.235/a/ Winnipeg
B.239/a/ York Factory

Correspondence

B.3/b/ Albany Correspondence Books
B.3/c/ Albany Correspondence Inward
B.68/b/ Flamborough House Correspondence Books
B.123/b/ Martin's Falls Correspondence Books
B.134/c/ Montreal Correspondence Inward
B.135/b/ Moose Correspondence Books
B.154/b/ Norway House Correspondence Books

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B.3/e/ Albany
B.16/e/ Berens River

B.51/e/	Fort Dauphin
B.60/e/	Edmonton
B.64/e/	Escabitchewan
B.70/e/	Flying Post
B.93/e/	Island Lake
B.105/e/	Lac la Pluie
B.107/e/	Lac Seul
B.123/e/	Martin's Falls
B.124/e/	Matawagamingue (Mattagami)
B.129/e/	Michipicoten
B.135/e/	Moose
B.145/e/	New Brunswick House (No. 1)
B.154/e/	Norway House
B.198/e/	Severn
B.239/e/	York Factory

Miscellaneous

B.239/k/	York Factory Minutes of Council
B.239/l/	York Factory District Statement

SECTION C: SHIPS' RECORDS

C.1/104X	Ships' Logs — Seahorse (II) 1771
C.1/1047	Ships' Logs — Seahorse (III) 1782
C.4/1	Book of Ships' Movements 1719-1929

SECTION D: RECORDS OF THE GOVERNOR OF RUPERTSLAND AND COMMISSIONERS

D.1/2	Governor William Williams Correspondence Book Outwards 1819-20
D.4/74	Governor George Simpson Correspondence Book Outwards (General) 1853-54
D.4/103	Governor George Simpson Official Reports to the Governor and Committee in London 1836
D.5/4-20	Governor George Simpson Correspondence Inward

SECTION E: PRIVATE MANUSCRIPTS

E.3/2	Peter Fidler Journal of Exploration and Survey 1789-1804
E.3/3	Peter Fidler's Journal of Exploration and Survey 1794-1808
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E.18/8	"Parliamentary Select Committee on H.B.C. – Miscellaneous Papers" 1857 and undated
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Citations of **HBCA Files** refer to Search Files compiled on a variety of subjects by the HBCA archivists. These files include information collected on a specific subject and may be consulted at the HBCA. **Post Histories** have been created for many of the Company's posts and forts and these are an excellent source of basic information about the posts.

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MG 10 F4	Minnesota Historical Society
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MG 19 A40	William Douglas Lane Papers
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