TRACING HISTORICAL DIFFUSION PATTERNS: THE CASE OF THE 1780-82 SMALLPOX EPIDEMIC AMONG THE INDIANS OF WESTERN CANADA

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INTRODUCTION

The history of a human disease is inseparable from human history. Each civilization makes its own diseases. Some pestilences remain historical enigmas because diseases wax and wane, vary in severity from one region to another, and change their character. In order to write accurate historiographies, or to predict the future about diseases, we must have a better understanding of how diseases have acted in the past. This paper demonstrates the use of a methodology that increases the accuracy in interpretation of historic infectious disease episodes. The method, based on epidemiological principles, tackles circumstantial evidence, yet discriminates between accuracy and inaccuracy. This is critical in analyzing specific epidemic episodes (cases in excess of normal expectancy) and ascertaining how and where the disease spread, and the conditions, attitudes, and behaviour reported during that time by both Europeans and Natives. Such information is also essential in dealing with the broader issue of the cumulative impact of infectious diseases over time on interacting Native groups.

The spread, or diffusion of the 1780-82 smallpox epidemic among the Indians of the Western Interior of Canada will be used as a case in point for demonstrating the use of this method. Probably the most dreaded and lethal of the Euroamerican-introduced infectious diseases was smallpox, which was first recorded on the Plains from 1780-82, although there may have been earlier visitations of this disease. Since the beginning of the seventeenth century, smallpox had erupted with regularity in the North American colonies and Latin America. The disease was endemic (constantly present) at that time in most cities of the British Isles and Europe,[1] and consequently the Europeans had

immunity to smallpox from previous exposure. The Indians of the Western Interior of Canada, however, had no immunity, and so the epidemic spread rapidly among them with catastrophic results. They were decimated, devastated, and demoralized.

The 1780-82 smallpox epidemic on the Plains has been given less attention by scholars than other epidemics in this part of the world. The Hudson's Bay Company (HBC) had only begun to establish posts along rivers inland in 1774, so that by 1780 there were very few posts (and journals available from those posts) to record events. What is known is that this epidemic was Plains-wide in scope, and that the Indians suffered appalling ravages. According to Ray, the Plains Assiniboine were depopulated to a lesser extent than the Western Cree, even though the disease spread more rapidly through the grassland area of the more mobile Assiniboine.[2] In contrast to this epidemic, the Cree were largely spared from smallpox during a later epidemic in 1837-38, due to the vaccination efforts of the HBC men.[3]

The analyses by Dollar and Ray of the 1837-38 smallpox epidemic on the Plains are revealing for their conclusions regarding the diffusion of the disease. Epidemiological principles were integrated by Dollar into his reconstruction of the epidemic as it diffused up the Missouri River.[4] He utilized the clinical stages of infectious disease, and virulence of smallpox in various Indian groups, to deduce specifically who could have acted as the source of infection for the Mandan Indians near Fort Clark (now Bismarck, North Dakota) on the Upper Missouri River. The diffusion pattern, Dollar concluded, was a "riverine upstream" pattern caused by infectives on the steamship St. Peter's, who, although they were only weakly contagious, were the only possible sources of smallpox according to epidemiological principles. A downstream diffusion pattern was ruled out because the other Indian groups around Fort Clark were not as yet communicable, and transmission of the disease during the downstream stop of the steamship "would have presupposed a shortened incubation period, a situation requiring a virulent case of the disease as the infecting agent."[5] As the Mandan Indians were ultimately almost

annihilated, and the American Fur Company was accused of deliberately spreading the disease to them, it was imperative that a meticulous investigation of this epidemic be conducted in the interests of an accurate historiography of this region.

According to Ray, the Indians themselves "appeared to be the principle carriers" of the disease as it spread northward from the Missouri River into the Saskatchewan area during the 1837-38 smallpox epidemic.[6] He also analyzed the diffusion of other contagious diseases on the Western Plains from 1830-50, but the diffusion of measles, influenza and scarlet fever led him to a different conclusion: "in most instances the Hudson's Bay Company boat brigades served as the primary carriers of the diseases."[7] The question then remains: during the 1780-82 epidemic, was smallpox spread along the Saskatchewan River by the Indians, or by the HBC men, and how was it diffused to York Factory? A closer examination of the spread of this epidemic among the Indians will be carried out in this paper using epidemiological principles and probability theory. Information obtained on such short-term, episodic variations in populations could eventually help to explain the complex, dynamic relationships among demography and depopulation epidemics, the environment, and socio-cultural change in Indian groups.

THEORETICAL ISSUES IN THE STUDY OF EPIDEMICS

Analyzing diffusion patterns and epidemic processes of infectious diseases from the past confronts researchers with a difficult task. Archaeologists and paleopathologists may provide some clues in tracing the history of a disease, but a laboratory diagnosis of past epidemics from culturing pathogens or through immunological surveys is all but impossible. Therefore, clinical signs and symptoms as interpreted from written records, are the main sources for the diagnosis of a past infectious disease. Ascertaining the origin and subsequent spread of the disease becomes even more problematic since historical accounts in themselves are often fragmentary, and not always clinically accurate. The epidemiological literature, however, on the

prevalence, incidence, and spread of infectious diseases in populations offers guidelines for researchers engaged in this topic. The basic components of the mechanisms of this spread are the agent (or factor causing the disease), the host, and the environment (a holistic term including physical, social and cultural factors). In analyzing the modes of transmission, the focus is primarily on the dynamics of movement, and on the geographical concentration of the human host. The key element for understanding infectious diseases is this transmission cycle. It is helpful in identifying an unknown infectious disease, confirming a suspected disease, revealing the speed and direction of a disease, and assessing the virulence of the disease. When used along with other factors such as seasonality, susceptibility, and cultural characteristics, these principles could be readily applied by researchers, and would assist in unraveling a great deal of interdisciplinary information.

A basic working knowledge of the dynamic relationship between a microbe and its host can assist a researcher in identifying a disease and tracing its diffusion pattern. Micro-organisms including the viruses of smallpox and measles are host specific, and do not infect other kinds of animals. If the microbe successfully invades the host, becomes a parasite, and produces infection, then specific toxins, or poisons are produced in the host which in turn produce specific clinical signs and symptoms. How the host will react depends on the route of entry of the disease, how much of the pathogen or disease-causing organism was transmitted, and the immune mechanisms available in the host to counteract the pathogenic agent.[8] Environmental factors such as humidity and population density mediate interactions between the host and the parasite and can extensively modify that interaction. This parasite-host interplay and the resultant set of clinical stages enable a researcher to identify that particular infectious disease, and theoretically trace its spread from host to host in a population.

The clinical stages of an infectious disease such as smallpox, also called a disease spectrum, include the exposure, the

incubation and prodromal period (defined below), the acute stage, and the early and late convalescence stages (Figure 1). Exposure takes place when a pathogen from a host, that either has the disease or is carrying the disease without symptoms (or subclinically), infects a new host. The time from this infection through to the manifestation of clinical signs and symptoms is known as the incubation period. This period can be as short as a few hours, as in cholera, or can vary from seven to seventeen days as in smallpox (Figure 1). The virulence of a parasite, that is, its capacity to damage its host, and the resistance of the host, along with the mode of transmission and the site of entry of the pathogen, determine the length of the incubation period. Not all infectious diseases manifest the third stage or prodromal period, but for those historically significant diseases such as measles and smallpox that do, this stage is characterized by the first overt symptoms of the disease, which include a headache and general malaise. The acute stage of the disease begins with the onset of a rash and fever. If death ensues, it is usually during this stage. Figure 1 shows, however, that the probability of death from smallpox, and the communicability period (when the disease is either directly or indirectly transmissible) are asymptotic, or become infinitely closer to zero through time. With smallpox, there is a possibility that the virus has been able to thrive for several weeks on a contaminated object such as a blanket, even though it is only weakly communicable (or infectious), and ultimately may lead to the death of the infected individual. Generally, a person with smallpox is potentially infectious from the time of development of the earliest lesions, with maximum transmission occurring during the first two weeks.[9] Thus, communicability and the probability of death both decrease considerably through the course of convalescence, and although these probabilities are slim, they are not impossible. Finally, the last stage, or recovery period, consists of both an early and late convalescence.

The prodromal and acute stages, and subsequent convalescence, are important stages for differentiating a clinical

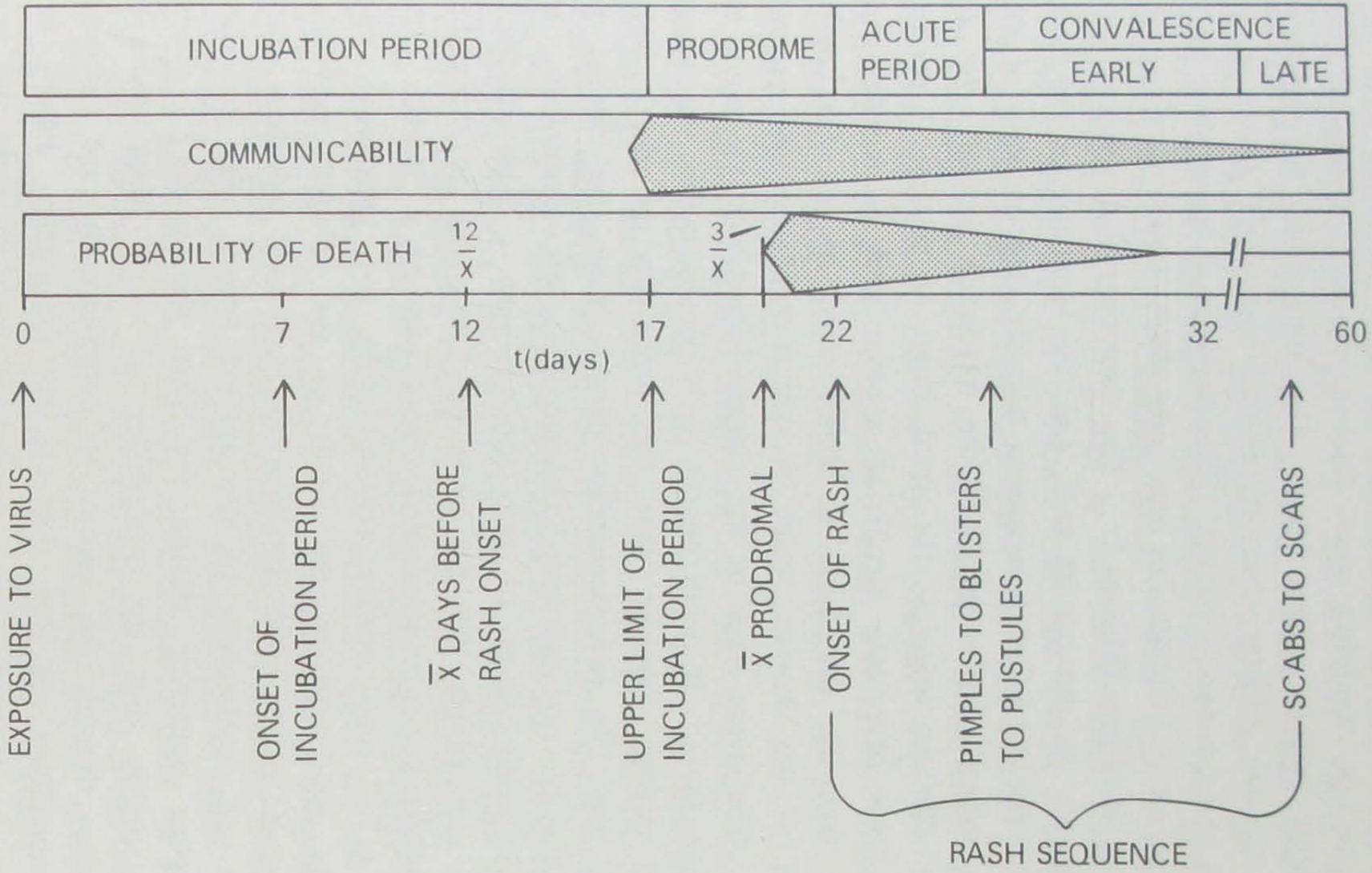

FIGURE 1 The stages of illness in the Variola Major strain of smallpox. The probability of death is greatest in the acute period (as is communicability), and decreases considerably through the convalescence stage. SOURCE: Benenson, A., Control of Communicable Diseases in Man (American Public Health Association, 1985), pp. 350-357.

diagnosis of a particular disease. Figure 1, for example, depicts the characteristic course of the classical progression of a smallpox rash from pimples to scars. The timing and type of the death during each of the stages is also significant. Side effects or any permanent damage from a disease can also help in identification. Facial scars and blindness, for instance, are suggestive of a person having had smallpox, whereas deafness is more suggestive of measles. Identification of a disease as it spreads is important, as many infectious diseases, such as measles and whooping cough, have been known to occur simultaneously in a particular population. Secondly, knowledge of the average length of each stage and at what point in the stages the disease is communicable, can assist the researcher in identifying a disease and deciding where and at what time an infected individual was exposed to the pathogen. For example, if smallpox was definitively diagnosed on a certain date, then that individual was first exposed to the disease on an average of twelve days earlier. If the individual was a traveller or explorer, it is then critical to ascertain his/her location at that earlier, significant time. The corollary is that knowing the travelling times of an individual between a known infected area and when the disease manifested itself on the host, allows one to estimate the incubation period of a disease. This deductive process is particularly useful in a micro-analysis of a relatively small region, and is vital in ascertaining the probability that a hypothesized diffusion pattern is accurate or not.

Epidemiologists have relied on Philip E. Sartwell's assertion that ". . . the dynamics of disease is closely related to incubation periods."[10] These periods vary among individuals so that, over a number of cases, a frequency distribution will show a positive skewness (or is asymmetrical) as indicated in Figure 2a. If a logarithmic transformation was applied to this skewed distribution, the transformed distribution would become essentially normal, as shown in Figure 2b. The resultant normal curve is symmetrical about the mean (which is the same as the median in a normal distribution), and half the area beneath the curve lies on either

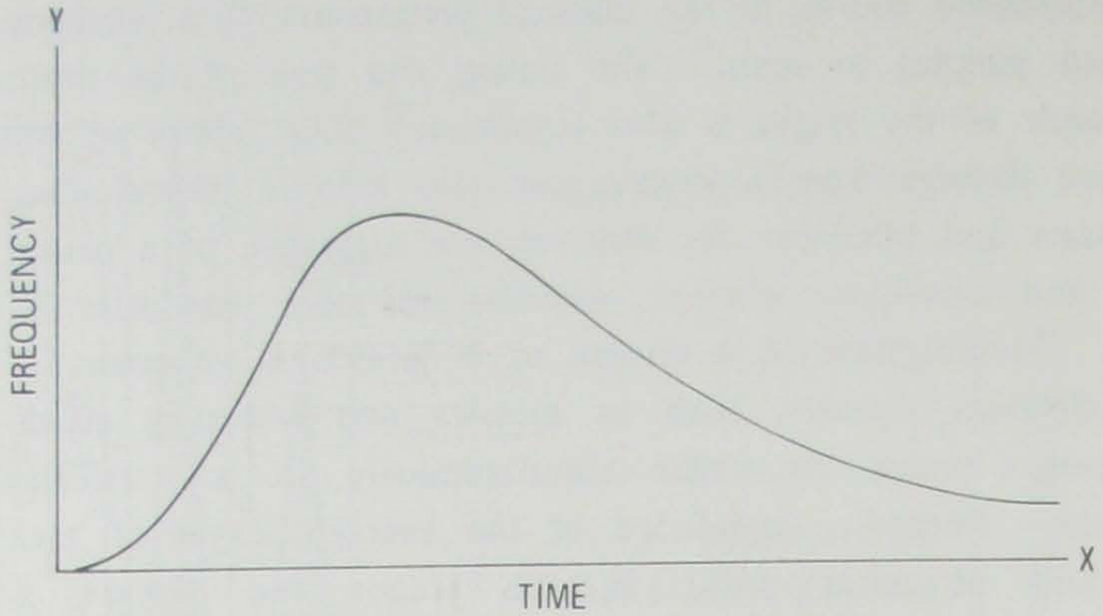

FIGURE 2a Incubation Period: Positively skewed distribution.

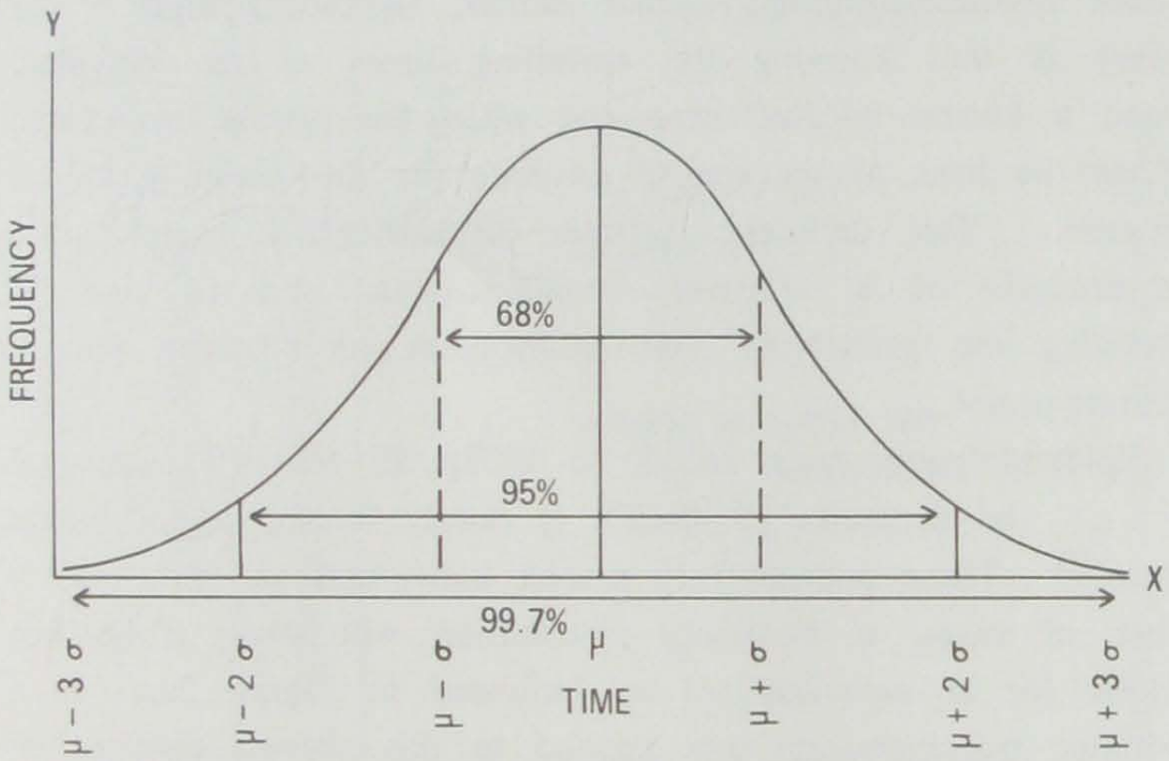

FIGURE 2b The area under a normal curve with one, two and three standard deviations.

side of the mean. The empirical value of using this approach lies in estimating certain probabilities about a variable such as an incubation period. As Figure 2b shows, approximately 68 percent of the area under a normal curve lies within one standard deviation of the mean, 95 percent of the area lies within two standard deviations, and 99.76 percent lies within three standard deviations of the mean. If these values were applied to the case of smallpox in Figure 1, a clinically verified incubation period of seven days would be the expected lower limit for the curve, the mean incubation period would be twelve days, and the upper limit would be seventeen days. The probability that a victim would experience an incubation period of seven days, or of seventeen days, (which are both three standard deviations from the mean) is, for a typical case of smallpox, therefore unlikely.

Sartwell also demonstrated that each infectious agent has a characteristic incubation period whose distribution fits a log-normal model.[11] Using this model he calculated what he called dispersion factors for numerous infectious diseases. Small dispersion factors, indicating little variation about the median, were prevalent in viral diseases such as measles and smallpox, whereas bacterial diseases tended to have larger dispersion factors.[12] This graphic curve-fitting method is a relatively easy way to calculate the ranges of the incubation period on either side of the estimated median, if the times of onset of the disease in a group of cases is known. This is not the same as the "reported date" used in some previous geographic studies. This median can help to identify the agent, since each agent has a characteristic incubation period. It can also assist in estimating the time at which the host was exposed to the agent. Finally, dispersion factors, which help to assess the probability of the time when a host will develop a disease, are also helpful in studying unusual cases that fall outside the range of the median. A fuller discussion on the uses, applications, and measurement problems of the incubation period are presented by Armenian and Lilienfeld.[13]

If the times of onset of illness are known, an epidemic curve could be constructed by plotting these times by the distribution of cases.[14] The curve helps to distinguish the origin of the disease (Figure 3). If it rises and falls rapidly, the epidemic is likely a common-source epidemic from contaminated food or water, since many persons are affected in a short period of time. The number of cases reported each day is related to a single incubation period of that disease. Conversely, if the number of cases over time is related to several incubation periods and the epidemic curve has a less distinct peak, the curve suggests a propagated or person-to-person epidemic such as smallpox.[15]

Along with the clinical stages of infectious diseases, there are additional aspects to consider in the spatial analysis of an epidemic diffusion pattern. Ecological factors including precipitation, temperature, population densities, agricultural and cultural practices, and other factors linked to geographical location, may help to determine where the disease will occur. The geographic extent of a disease might suggest a particular vector or disease carrier; for example coastal areas and inland waterways would tend to harbour water-borne diseases such as cholera and typhoid. The geographic extent of a disease might also be related to seasonal factors. In areas with marked wet and dry seasons, smallpox is worse in the dry season, in part due to climatic conditions such as low humidity, which favour the persistence of the virus outside of the host. The exception to this seasonal variation in humidity are areas with year-round rainfall, such as a coastal region.[16] In temperate climates it spreads more rapidly during the winter months. Yorke, et al. argued that "seasonal variation in transmissibility can greatly increase the minimum population size in which persistence of an epidemic is possible"[17] and it can play a major role in the size of the population required for perpetuation of a disease epidemic. The rate of population change (births, deaths, and migration), the density of population, the immunity of the population, and age structures of the population (children may be very susceptible but have little mobility and therefore may not readily be transmitters of disease)

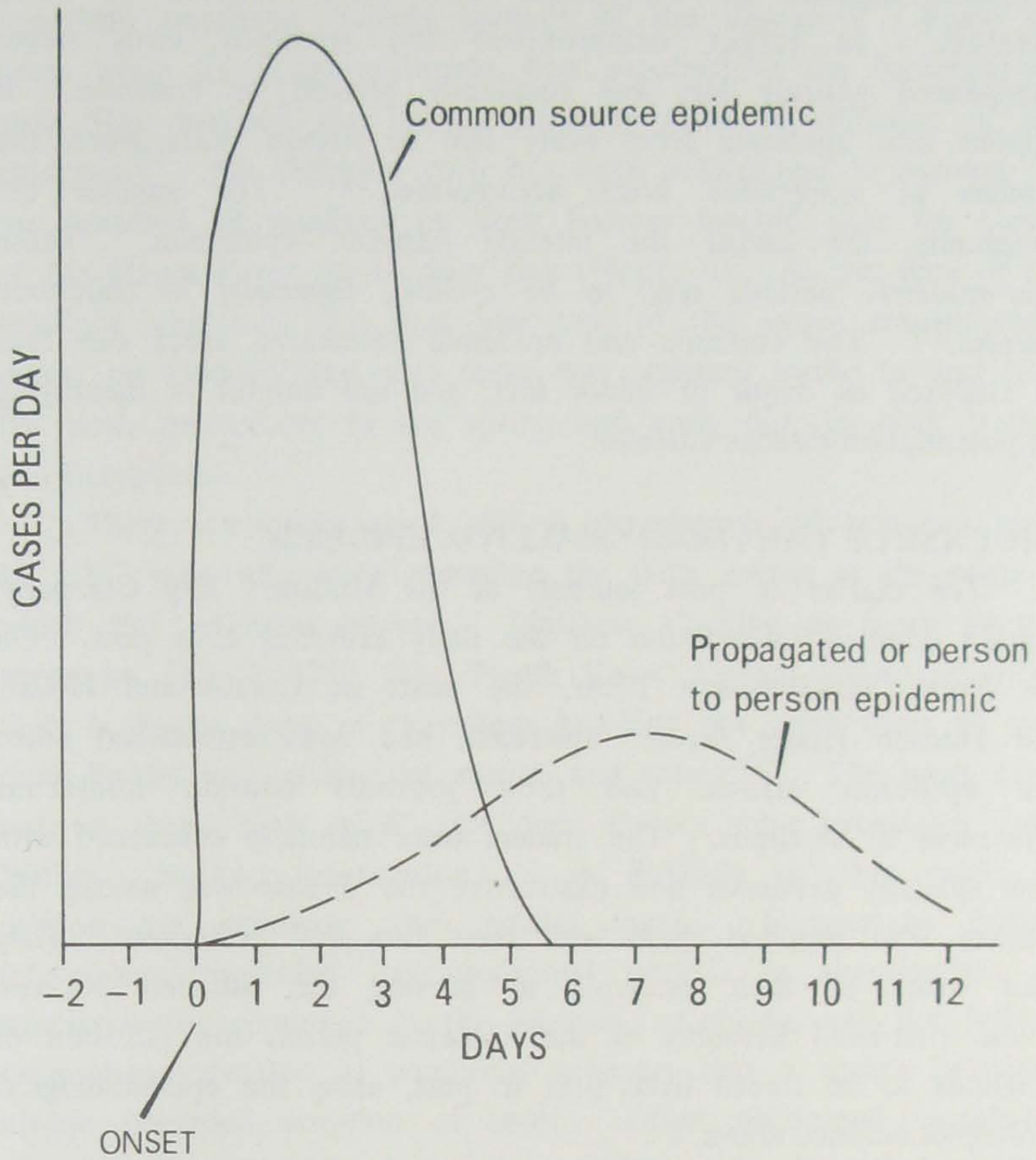

FIGURE 3 A schematic representation of the epidemic curves.

are also important considerations for perpetuation of agents in human populations.[18] Regardless of how virulent an agent is, it cannot become epidemic in a population unless there are a sufficient number of susceptible new hosts capable of being infected. In larger communities the smallpox virus never disappeared entirely but was constantly present or endemic. It erupted into epidemic form every five to fifteen years when the number of susceptible hosts accumulated.[19] The smaller the community, the longer the interval between epidemics. These inter-epidemic periods tend to be cyclical, especially in childhood diseases.[20] The endemic and epidemic delineated areas can thus be analyzed as major or minor foci, and are helpful in identifying the potential for disease diffusion.

THE CASE OF THE 1780-82 SMALLPOX EPIDEMIC

The diaries of post journals of the Hudson's Bay Company, contain detailed information on the daily activities at a post. On the North Saskatchewan River, the posts at Cumberland House, and Hudson House further upstream, had been established when the epidemic struck, and their journals contain numerous references to smallpox. The traders were naturally concerned with how spatially pervasive and destructive the disease was among the Natives who supplied them with their furs and provisions. They also wrote of their attempts at nursing the afflicted Natives. These first-hand accounts of the epidemic permit the diffusion of smallpox to be traced from post to post, using the epidemiological principles outlined above.

David Thompson wrote that the smallpox, which swept the Plains in 1780-82, was likely picked up by the Dakota Sioux from the trading villages along the Missouri River, who then spread it to the Ojibwa and Plains Indians to the north of them.[21] Tomison at Cumberland House believed from his sources that the disease originated from the Spaniards, who traded with the Snake Indians, and who subsequently spread it to the Western Plains Indians.[22] There may have been, therefore, two points of entry of smallpox into the Saskatchewan area; one originating from the Dakota Sioux

in the southeast, and a second from the more westerly Plains Indians. According to Dobyns, these small diffusion waves were part of a large diffusion wave of smallpox that originated in Mexico City in 1779, which ultimately resulted in a North American pandemic (widely spread) of the disease.[23] From the times when the local outbreaks first occurred in the Saskatchewan area, Ray mapped the broad, principle paths of diffusion of this epidemic.[24] No diffusion path has been established to account for the presence of smallpox at York Factory located near the mouth of the Hayes River on Hudson Bay (Figure 4). At the time of the smallpox epidemic, this post was one of the major transshipment points on Hudson Bay, and there was constant traffic to and from the post, particularly in the spring and early fall, by both Natives and Europeans.

There are many good clinical descriptions of smallpox given by HBC men who were recording the daily events at the posts to which this epidemic spread. Matthew Cocking at York Factory wrote on July 2, 1782, that "North River" Indian women described their husbands dying of "a violent breaking out upon them all over their bodies and within the mouth and throat."[25] The spots came out on them "very thick" and their throats were excessively sore. Without further information, it is difficult at this point to differentiate smallpox from scarlet fever. Fortunately, further information regarding the prodromal period, so characteristic of smallpox, was contained in the journals. Cocking, and the Indians themselves, revealed in numerous accounts that a severe headache always preceded eruption of spots. Other prodromal complaints that identify smallpox, included a violent pain in the back and vomiting. A description of the side effects of smallpox as it progressed through the convalescence stages is also telling. One of the Indians was "much troubled with swellings which succeed each other in different parts of his body and limbs,"[26] while yet another Indian became blind. The final definitive diagnosis of smallpox can be made with the following chronology of the disease as it progressed in a young Indian lad.

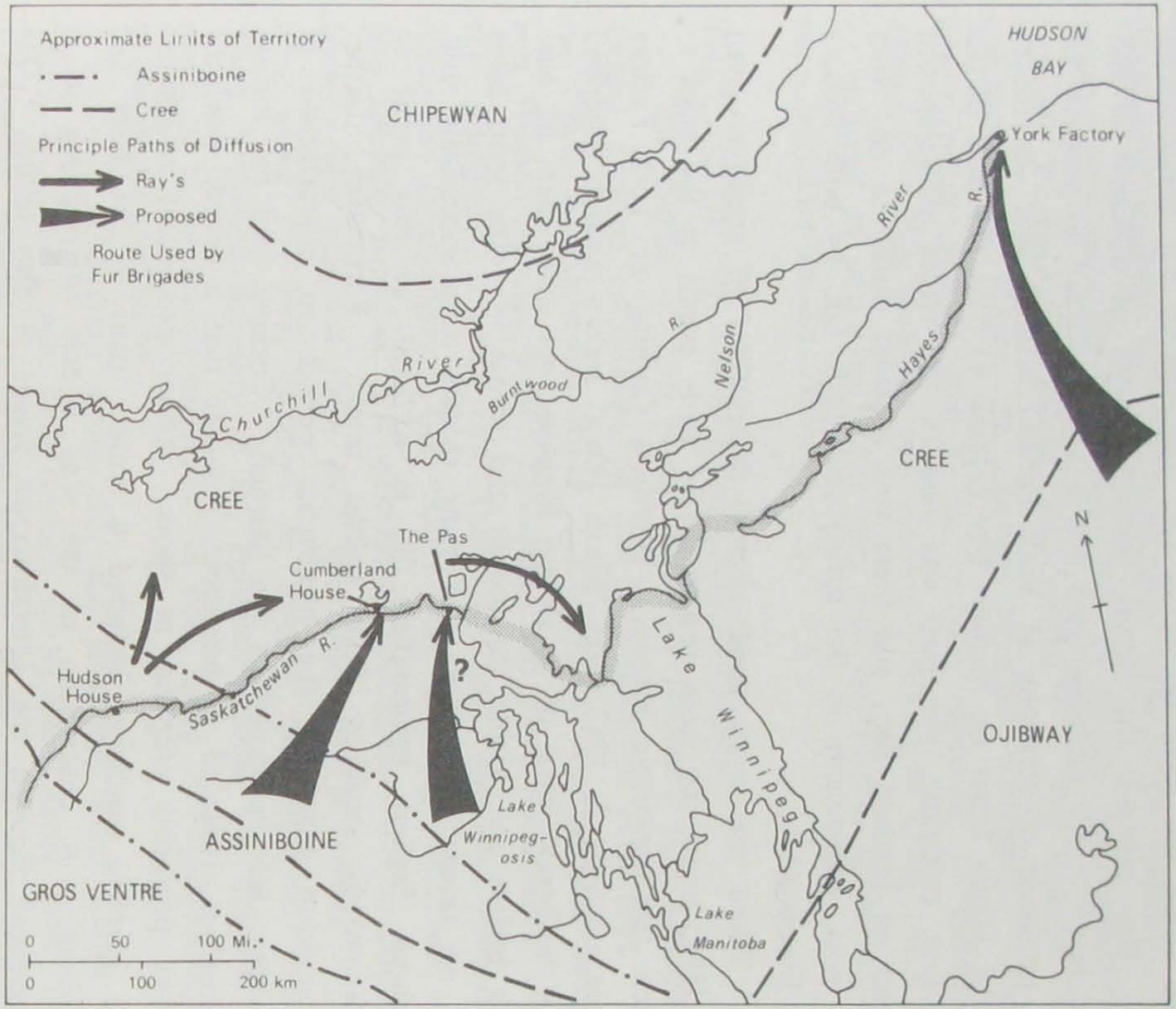

FIGURE 4 The Western Canadian Interior from Hudson House on the Saskatchewan River to York Factory on Hudson Bay, showing Ray's principal paths of diffusion, and the revised paths of diffusion based on the usage of epidemiological principles.

On December 11, 1781 several Indians arrived at Cumberland House bringing the "disagreeable news of many Indians dying of the latter complaint much for want of food."[27] One of the Indian women was very ill with "a violent pain in her back and much inclined to vomiting.[28] She died the next day, and as Tomison pointed out, it was only the fourth day of her ailment. He may have guessed then that she had smallpox, but he did not actually write about it in his journal until December 17th, when eight men and an Indian lad arrived from Hudson House further upstream (Figure 4) and reported that "smallpox was raging all around us [Hudson House] with great violence, sparing very few that take it . . ."[29] At Cumberland on December 20th, a young Indian lad and his family, who were hired by the HBC men as hunters, were sent out to hunt, but on December 24th the boy had taken ill with a "violent pain in his breast and belly," and was returned to the post on a sled on Christmas day. On the 27th, Tomison wrote: "this morning we could observe the smallpox coming out very thick upon the sick lad's head and thighs."[30] This peripheral distribution of the rash is classical of smallpox. By the 30th, the lad was very ill and "had a great stoppage of his throat." Two days later, the lad was blind (a characteristic complication of smallpox), and by January 4th he was having "deliriums." Death came mercifully the next day. From his prodromal period until his death, the lad had lived thirteen days, but Tomison said this was an exception, as this disorder was so cruel that the Indians were chiefly dying within the third and fourth day of their illness.

The unanswered question, however, is: where did the lad become infected? There are two viable possibilities here. The Indian woman who arrived at Cumberland from the "southward" on December 11th, was likely in a highly infectious stage of illness from the haemorrhagic or bleeding type of smallpox. This usually fatal variety of smallpox has an increased virulence which could shorten the average incubation period, and produce a prodromal period that is prolonged and very severe. Haemorrhages may appear and the individual may die even before the rash is seen. Headaches, backaches, and vomiting are the usual symptoms, and

death commonly ensues in four to five days.[31] The woman's clinical signs and symptoms were similar to other accounts of those with smallpox, and she died, characteristically according to Tomison, in four days. If the lad became infected from her, his incubation period would have been fourteen days, which is only slightly higher (within one standard deviation) than the average of twelve days.

The second possible infective could have been one of the traders or their Indians who arrived from Hudson House, where the disease was raging, on the 17th. The incubation period in this case would have been seven days, which is highly probable in a case of haemorrhagic smallpox, but only about three percent or highly improbable in typical varieties of smallpox. But Tomison made no mention of anyone from the Hudson House group being ill or of having had any spots or scarring, in which case even if they had, they would have been only weakly infectious. Furthermore, there was only one instance of a trader contracting smallpox in this epidemic, and he was a part Indian and would not have had immunity from previous exposure that the European-born HBC men would have had. It is more probable, then, that the lad's source of infection was the sick woman, whom he could have contacted at the post on December 11th, as all the Indians were in the habit of tenting together in a clearing near the post. From the clinical description Tomison gave of the woman's illness, it is highly probable that she had a haemorrhagic variety of smallpox, and was highly infectious, but we do not have the information to estimate the length of her incubation period.

It is puzzling, however, that the lad took much longer to die than did some of the other Indians. In assessing this lad's stages of illness, it is clear that the prodromal period (from when the lad took ill until his rash appeared), was four days in length, which is well within the normal range for a smallpox prodrome. He died nine days after his rash appeared and was obviously in an acute stage of illness. However, he was brought in on a sled by the HBC men, and cared for by them during the course of his illness. This was not the case for the vast majority of the afflicted

Indians, who would have been left to perish in their tents with little or no medical assistance from other Natives or Europeans, in one of the coldest months of the year. For this reason, the lad could have survived longer than the usual three to four days.

In summary then, it seems highly probable that this particular smallpox epidemic was a more virulent, perhaps haemorrhagic variety, which produced a very severe prodromal and acute stage, and during which death ensued in three to four days. It is less likely that the fourteen day incubation period that the lad demonstrated was typical of this type of smallpox, but his is the only case in the records to draw upon. With this epidemiological information in hand, it is now possible to trace the diffusion pattern more accurately.

Smallpox, we may conclude, was spread to the post at Cumberland from an infected Indian woman from the "Southward," who visited the post December 11, 1781, and not from the HBC traders who came from Hudson House a week later. By February of 1782, there were reports of the disease having spread to the area around The Pas, which is approximately sixty miles east of Cumberland (Figure 4). On May 21, 1782 a brigade of HBC men and four canoes of Indians left Cumberland for York Factory. On June 13th, they met two tents of Indians stricken with smallpox. On June 23rd, they met more Indians who had lost some of their friends to the disease on their way inland from York Factory. Three days later on June 26th, the brigade met two canoes of Lake Bungees who told Tomison that many of their friends had died since leaving York. Tomison arrived at York on July 2nd.

Meanwhile at York Factory, Chief Factor Matthew Cocking had a visit from sixteen canoes of Lake Bungee (Ojibwa) Indians on June 10th who came to trade, and who told him of the smallpox raging among their countrymen. North River Indians (Cree north of York Factory) who arrived June 23rd, had also been exposed to the disease, and some of them were covered with red spots, while others were recovering.[32] When Tomison arrived at York Factory on July 2nd, he informed Cocking that he had

met those Bungees who had traded at York on June 10th, and that only two canoes of Indians remained alive out of sixteen.

Figure 5 helps us to interpret this anecdotal data. Twenty days after initially coming in contact with the disease on June 13, Tomison arrived at York. If the Indians travelling with the brigade had contracted smallpox at that time, and given an average twelve day incubation period, they should have either died or had been well into the rash phase and only weakly infectious. Some of the Indians that Tomison met, however, at ten and seven days out of York had already died of smallpox on their passage down from York. Of the sixteen canoes of Lake Bungees who had arrived at York June 10th, and left on the 13th, only enough Indians remained alive to paddle two canoes when they met Tomison fourteen days upstream from York. Those Bungees had to have brought the disease to York when they arrived June 10 because somewhere between three and fourteen days later, most of them had gone through an incubation period of twelve days or less, and had died. It was highly probable then that it was the Bungee Indians who originally brought the disease to York, rather than the North River Indians or the HBC brigade from Cumberland. The day after Tomison's arrival at York on July 2nd, he wrote in the journal that those Indians who had clearly contracted the disease at an earlier time were being buried.

CONCLUSION

Using the intrinsic clinical characteristics of an infectious disease, such as smallpox, and assessing the reliability of the analysis using basic probability theory in uncertain situations, we may conclude a great deal about the 1780-82 smallpox epidemic. The inference to be drawn from this case study is that it was likely haemorrhagic smallpox (a virulent type of the variola major strain) that swept through the Western Interior of Canada in 1780-82. As Figure 4 shows, the diffusion pattern of the disease was not exclusively along the HBC river route used by the fur brigades, nor was the disease spread by the traders. It was the Indians themselves who travelled along the rivers and overland

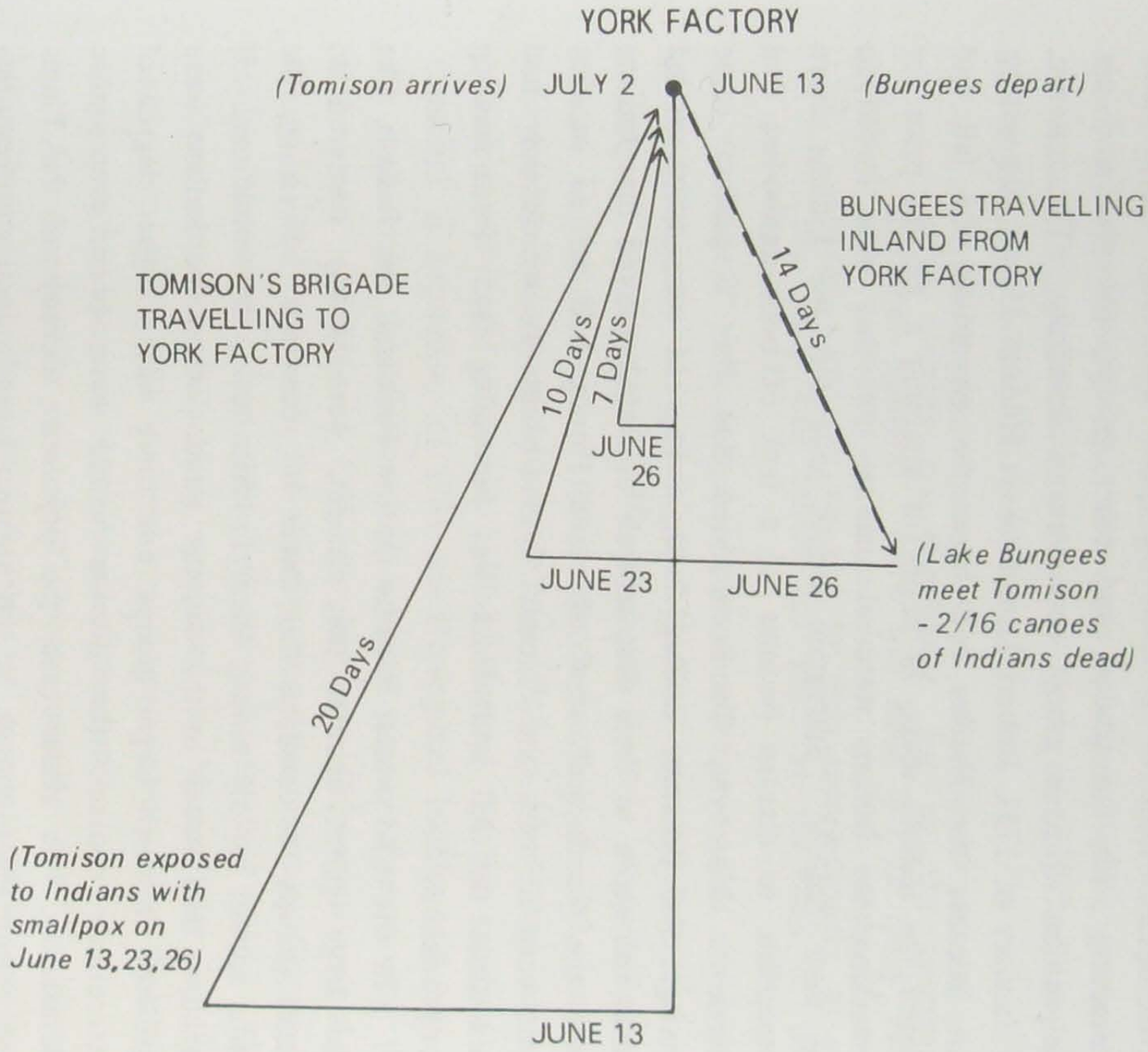

FIGURE 5 Tomison's journey to York Factory and the Lake Bungees inland journey from York Factory.

from their tenting grounds to trade, or to seek help from traders at Cumberland House and York Factory, who unwittingly spread smallpox to those posts.

The underlying processes in this epidemic were a reflection of the cultural characteristics of the Indian groups, their environment, their long and short-term travelling habits, their interaction with the traders and other groups, the size and age composition of each group, and previous immunity. For instance, by January of 1781, Robert Longmoor at Hudson House was writing that, because the Natives had burnt the ground in the fall and chased the buffalo away, in hopes of attaining a greater price for provisions, the Indians were so starving that they were forced to eat their dogs.[33] Starvation would have left the Indians more susceptible to disease because of a lack of both antibodies and calories. Moreover, Tomison added that the "Canadians came amongst the [Swampy Indians] with 4 kegs of rum, which added more and more to their destruction."[34] He also noted that Indians from the "southward" of Cumberland House, had arrived at that post very ill with the "bloody flux" (dysentery), which they had had since the fall and which was preventing them from hunting for provisions.[35]

In the cold winter months on the Parkland, the Indians, like the large game animals they sought, were widely scattered in small groups to decrease the threat of starvation. Even so, the virus spread rapidly among them in these cold, dry conditions. If smallpox had struck in the summer months when the Indians were gathered in much larger groups, and there was a higher degree of interethnic contact, the disease would have been even more devastating. In either case, the population density on the Plains was never great enough in the eighteenth and nineteenth centuries to maintain smallpox endemically. Consequently, every thirty to fifty years it reappeared to a new generation of susceptibles who lacked any immunity to the disease, in epidemic form.

The Indians did not understand the concept of contagion. As a Blackfoot Native who survived this epidemic told David Thompson: "We had no belief that one Man could give it to

another, any more than a wounded Man could give his wound to another."[36] They also thought that smallpox had eyes and could see who was afraid of it, so they gathered around the ill rather than isolate themselves. The usual remedies of the Indians failed, and the most common medical treatment of spending time in a sweat lodge and then plunging into cold water, actually led to shock and consequent death for many smallpox victims, particularly the men. When Tomison arrived at York on July 2nd, he told Cocking how the Indians had died:

> Numbers died through want of food or starved with colds, having no attendance, for us the complaint became general there was none left in many tribes able to hunt for or administer to the wants of each other; many put an end to their own existence to end the pain, and others for grief at the loss of their families. 37

Cocking at York Factory made an attempt to halt the spread of the disease by not allowing well Indians into the Fort, and not sending his Homeguard Cree to Churchill for fear of spreading the disease to the Homeguard there. In September of 1782, however, the French raided and burnt York Factory, and had already captured and burnt Churchill. When Samuel Hearne returned to Churchill in September of 1783, his Homeguard Indians told him of the devastation the disease had wreaked among them during the last winter. In May of 1784, the Northern Chipewyan arrived at Churchill and informed the traders that the famous Chipewyan leader Matonabbee and many of his followers had died. This smallpox outbreak that likely originated in Mexico City in 1779 and took until 1783 to run its course among the Northern Chipewyan, had become a North American pandemic.

As detailed as the HBC post journals are, insufficient information is contained in them to ascertain how smallpox spread to The Pas, or to Churchill. As well, the 1783 post journals for York and Churchill are non-existent due to the French raids. This illustrates one of the drawbacks that those interested in Native history often face: rarely are there adequate, detailed data with which to work. The case study in the paper has demonstrated the use of anecdotal data in tracing a

diffusion pattern, but as Armenian states: "anecdotal data from investigations are useful to estimate ranges but they cannot provide a final and specific measure of incubation periods, because of lack of precision and the assumption made in the specific situation."[38] Although this level of precision is not always possible, this method potentially yields a great deal of information: which agent caused disease and how the disease preformed in specific environments, the potential for further diffusion, which groups were contracted and an idea of mortality and morbidity within those groups, and the response to the disease by both Natives and Europeans. Knowledge of the diffusion of each epidemic wave could suggest temporal orders of introduction, and subsequent demographic or systemic change. Did the timing of disease contact vary over space, and was there differential survival? Knowledge of epidemic episodes such as the one described herein may contribute to the broader question of whether diseases had catastrophic consequences on Native populations before or after European contact. Basic epidemiological principles outlined in this paper may be useful tools for any researcher interested in these aspects of Native history.

ACKNOWLEDGEMENTS

The author would like to thank the Hudson's Bay Company for permission to consult and quote from their archives. I would also like to thank Shirlee Anne Smith, Hudson's Bay Company Archivist, and Don Freeman, Conrad Heidenreich, and John Warkentin for comments on earlier drafts of this paper.

NOTES

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[2]Arthur J. Ray, Indians in the Fur Trade: Their Role as Trappers, Hunters, and Middlemen in the Lands Southwest of Hudson Bay 1660-1870 (Toronto: University of Toronto Press, 1974), pp. 105-107.

[3]Arthur J. Ray, "Smallpox: The Epidemic of 1837-38," The Beaver, (Autumn 1975), pp. 8-13.

[4]Clyde D. Dollar, "The High Plains Smallpox Epidemic of 1837-38," The Western Historical Quarterly, (January, 1977), pp. 15-38.

[5]Ibid., p. 36.

[6]Ray, Indians in the Fur Trade, p. 188.

[7]Arthur J. Ray, "Diffusion of Diseases in the Western Interior of Canada, 1830-1850," Geographical Review, 66 (1976), p. 156.

[8]The term "agent" is commonly used to refer to both a micro-organism, or the smallest of infectious pathogens, a virus.

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[15]Judith S. Mausner and Shira Kramer, Epidemiology: An Introductory Text (W.B. Saunders Company, 1985), p. 286.

[16]Donald R. Hopkins, Princes and Peasants, Smallpox in History (Chicago: University of Chicago Press, 1983), p. 195. S. Upham in his article "Smallpox and Climate in the American Southwest," American Anthropologist, 88 (1986), p. 120, maintains that Variola virus rapidly looses infectivity at temperatures above 30 degrees celsius and humidity greater than 55 percent.

[17]James A. Yorke, Neal Nathanson, Giulio Pianigiani, John Martin, "Seasonality and the Requirements for Perpetuation and Eradication of Viruses in Populations," American Journal of Epidemiology, 109 (1979), p. 122.

[18]Ibid., p. 104. Hopkins, op. cit., pp. 234-235, also discusses the importance of density of population in the transmission of smallpox.

[19]Hopkins, op. cit., p. 8.

[20]Roy M. Anderson, "Directly Transmitted Viral and Bacterial Infections of Man," in Roy M. Anderson ed. Population Dynamics of Infectious Diseases. Theory and Applications, (Chapman and Hall, 1982), p. 1.

[21]Richard Glover, ed. David Thompson's Narrative 1784-1812, (Toronto: Champlain Society, 1962), p. 236.

[22]Provincial Archives of Manitoba, Hudson's Bay Company Archives, B.239/a/80, York Factory Journal (July 2, 1782). (Hereafter HBCA).

[23]Henry Dobyns, "Estimating Aboriginal American Population: An Appraisal of Techniques with a New Hemispheric Estimate," Current Anthropology, 7, 4 (1966) pp. 395-416.

[24]Ray, Indians in the Fur Trade, p. 107.

[25]HBCA, B.239/a/80, York Factory Journal (July 2, 1782).

[26]Ibid.

[27]HBCA, B.49/a/11, Cumberland House Journal, (December 11, 1781).

[28]Ibid.

[29]Ibid.

[30]Ibid.

[31]A. Ramachandra Rao, "Smallpox, Vaccinia, and Cowpox," in Abraham I. Braude ed., Medical Microbiology and Infectious Diseases, (New York: W.B. Saunders Company, 1981), pp. 1677-1678. Rao classifies and discusses four major clinical varieties of smallpox: haemorrhagic, flat, ordinary, and modified in his article. Basically, however, there are two main strains of smallpox; variola major, which is the strain discussed in this article, and variola minor, which did not make its appearance in North America until late in the nineteenth century, Hopkins, op. cit., p. 287.

[32]HBCA, B.239/a/80, York Factory Journal, (1782).

[33]HBCA, B.87/a/4, Hudson House Journal (October 23, 1781).

[34]HBCA, B.49/a/11, Cumberland House Journal (March, 23, 1782).

[35]HBCA, B.49/a/11, Cumberland House Journal (March 13, 1782).

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[37]HBCA, B.239/a/80, York Factory Journal (July 2, 1782).

[38]Armenian, op. cit., p. 4-5.