

Mortality from Smallpox: The 1780s Epidemic in the Hudson Bay Region

Ann M. Carlos
University of Colorado at Boulder
Boulder, CO 80309
ann.carlos@colorado.edu

and

Frank D. Lewis
Queen's University
Kingston, Ontario K7L 3N6
lewisf@econ.queensu.ca

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Abstract

The smallpox epidemic that passed through the Hudson Bay region in 1780-82 is reported to have devastated the native population, causing mortality of 60 to 90 percent. We reassess this claim using three approaches. First we review case-fatality rates from smallpox in more recent, well-documented epidemics; we discuss the nature of smallpox transmission; and we review the possible impact of genetic factors and lifestyle on mortality from smallpox. Second we analyse the volume of trade in the region during the period surrounding the epidemic. Finally we estimate the native population before and after the epidemic. Prior to the 1782 our estimate is based on the carrying capacity of the region, where our focus is on moose, the large ungulate that was the basis of the natives' survival. The post-epidemic population is based on a backward projection of an early nineteenth-century population count. The three approaches lead broadly to the same conclusion. Mortality from the smallpox outbreak was modest, likely under 20 percent.

Introduction

Conjectures, assumptions and assertions surround the debate on the size of Native American populations just prior to European contact. Current estimates for North America north of the urban civilizations of central Mexico range from lows of 1,041,480 argued by Kroeber and 1,893,350 by Ubelaker to Dobyns' estimate of 18,022,006 (Ramenofsky 1987: 7; Ubelaker 1992:170). A consensus view would put the population in the range of six to twelve million. Critical to the wide variation in estimates are differences in views about the timing and impact on native peoples of the diseases brought by the first Europeans. There is little dispute about the size of the native population in the nineteenth century, when direct counts were made. The issue is whether the seemingly low numbers in these counts are a consequence of a very much larger pre-contact population that was decimated by disease, or whether native population densities, even in the pre-contact period, were low. Epidemics with mortality rates of 90 percent, as has been suggested by Doybns (1983), have far different implications than disease outbreaks that were more limited and less severe. As Shepard Kretch points out: "to decide on a sensible [pre-contact population] number, does not mean trivializing the extent of disease nor the extent of biological change introduced by Europeans. But to agree with the highest estimates assumes that diseases arrived early, spread widely and were invariably fatal ... and that diseases can actually be identified" (Kretch 1999: 85).

Native populations prior to 1500 and for the first three centuries of European contact are hard to determine largely because we have so little information. Prior to contact, the main source is the archeological evidence (Ramenofsky 1987). This evidence, drawn from excavations of particular sites, has been used to help describe many aspects of native life, including their health (Steckel et al. 2002; Steckel 2009); but, given the necessarily small samples, this sort of evidence is of limited use in determining, by extrapolation, the size of

the overall population. For the immediate post-contact period there are estimates of aboriginal populations based on the reports of European travelers and traders who observed native settlements. There is also a literature that has tried to infer native populations from the nature of their agricultural and hunting activities, and the types of flora and fauna that were available (Dobyns 1983; Ramenofsky 1987). But it is only in the last century and a half that we have direct census counts. Whatever the initial native population, it seems clear that the declines were due more to disease than war, as Owsley's (1992) study of archeological sites and skeletal remains has shown for the Northern Plains Indians.

In this paper we explore the impact of perhaps the earliest epidemic to hit natives living in the Hudson Bay drainage basin: the smallpox epidemic of 1780-82. The Hudson's Bay Company had erected its first trading posts on the bay coast in 1670s, yet nowhere in the written record of the next hundred years is there any indication of smallpox. Indeed, the traders and the natives' own oral history refer to the 1780s as the first time smallpox reached their region. This first outbreak provides an opportunity to examine the impact of smallpox on a native population that had never been exposed to the pathogen.

We begin by reviewing contemporary descriptions of the epidemic and the way Europeans and natives reported how smallpox affected the region. As we discuss most contemporary accounts speak of mortality rates ranging from 60 to 90 percent. We then study the epidemic using three quite different approaches. First, we summarize the mortality experience associated with other smallpox outbreaks, including the mortality among "virgin soil" populations, those with no previous exposure to smallpox. We include as well a discussion of the process of transmission and recent evidence on the role of genetic factors. Second, we place the epidemic in the context of the region's fur trade. Natives in the Hudson's Bay Company's hinterland were the sole trappers of beaver and other furs; and so

any serious decline in the native population would certainly have been reflected in the company's fur returns. Finally, we infer the extent of the population decline by making backward projections of the native population from the early nineteenth century to the 1780s. The resulting estimates are compared to the likely pre-epidemic population, which we base on the carrying capacity of the region, namely the population that could have been sustained by the large game, which in the region was mainly moose. Our three approaches to the epidemic all point in the same direction. We conclude that, although mortality was likely high during the smallpox outbreak, the disease had an impact much lower than has previously been asserted; the likely mortality was likely under 20 percent. Importantly, although these findings are for one episode, our results may have broad implications for the interpretation of pre-contact aboriginal population and the impact of European-borne disease.

The Smallpox Epidemic of 1780-82

The first major epidemic to affect natives trading in the Hudson Bay region appears to have been transmitted through the trading villages along the upper Missouri River by the Sioux in 1780 and 1781. This was part of a much larger series of smallpox outbreaks that began five years earlier, during the American Revolution.¹ By the fall of 1781 the disease vector had reached the Assiniboin, who were trading at Hudson House and Cumberland House, two interior collection points along the Saskatchewan River that the Hudson's Bay Company had established in the 1770s to help compete with the trade from Montreal (see Figure 1). Both collection centers traded goods for furs which were then sent on to York Factory, the company's largest trading post.

The most direct knowledge we have of the epidemic comes from the daily journals of the chief traders, which report the activities at each of these trading houses as required by the

Hudson's Bay Company management. Typically these journals described the daily routine, as in the following two entries from late November of 1781:

November 26th Monday Wind and Weather as Yesterday two Men still lame, sent five Men to the Nets, also fitted out Mr Longmoore and George Hudson and sent them away to trap Martins. 30 Sturgeon and 3 Pike [caught] yesterday.

November Friday 30th Wind E.S. E a stiff Gale, with Cloudy weather till noon... one man net Making two Hewing timber for the saw, sent others to overhaul the Nets 10 Sturgeon to day (HBC, *Cumberland House Journals*, 221).

Cumberland House began to received news of the epidemic in December. The entry, for Tuesday, December 11, reads in part:

Wind Westerly, a fresh Breeze Weather for the most part cloudy, with a low Drift... In the Evening three men and four women arrived from the southward with Furs to Trade also one family came across the lake from the Westward, the former has brought the Disagreeable news of many Indians dying, and the latter complain much for want of food. Indeed one of those that came from the southward does not seem to me to live long as she is troubled with violent pain in her back and much inclined to Vomitting, these inform me of seeing several Tents without anybody alive in them and some of the Dead not Buried (HBC, *Cumberland House Journals*, 223-24).²

Reports of smallpox and its effects now began to dominate the reports. In a letter dated December 4th, William Walker, clerk at Hudson House, had written: "small pox is rageing all round us with great violence, sparing very few that take it, we have received the News of above 9 tents of Indians within here all dead, ... as for the Stone Indians there are very few if any left alive..." Mr. Walker went on to say that the post would have problems getting in provisions "when the Indians is dying daily and them that has not taken the small pox is

frightened to look after any thing for fear of falling with others that is bad” (HBC, *Cumberland House Journals*, 225-26).

From the Cumberland and Hudson House journals it is possible to roughly track the course of the disease (see Figure 2). The journals note that as of January 2nd, 1782, four Indians from Le Pas, which was west of the disease vector, “had not heard of the disorder;” but by the 25th of the month “many sick Indians [were] arriving” from there. On February 19th, Cumberland House was told that in some tents near Le Pas, all the Indians were dead and that one-third of the Piegan Indians had died. There was a report on March 23rd that all in a group of ten tents in the Swampy River area (to the south) were dead.³

Based on the journal entries, it appears that the epidemic had largely run its course by the spring of 1782. Thus the outbreak in this area appears to have lasted five or six months, from November 1781 to March or April 1782. As of that time, smallpox did not appear to have spread further northwest towards York Factory. On March 1st, the journal notes that five men and three women arriving at Cumberland House from further north had heard nothing of the disease. Yet in following years, natives who had apparently recovered or were recovering from smallpox were reported in the vicinity of York Factory.⁴ The disease seems to have moved south east to the head of Lake Superior where it was reported among the Ojibwa in 1782.

The smallpox epidemic of 1780-82 clearly devastated and dislocated some native settlements.⁵ Here, however, we consider in detail the impact on the overall population, and in particular on those groups living in the path of the disease vectors. Hudson’s Bay Company personnel did not merely describe the event, they also tried to assess its overall impact, which they reported to the directors in London. Samuel Hearne, who had spent time at Cumberland House in its early years and later became chief trader at Fort Churchill, was

living at York Factory during the outbreak. He reported that 90 percent of the Indians in the Northern Barrens, the area to the east and north of the post, had died (Tyrell 1934: lx). This assessment was based on what native traders told him when they came to trade. York Factory's journal entry of July 2nd 1782 reported devastation among several tribes in the region: "not one in fifty of those tribes are still living" (HBC, *Post Journals: York Factory*). Four years after the epidemic, David Thompson, who provides one of the first travel narratives, journeyed from York Factory to the Rocky Mountains. Based on conversations with natives, and from discussions with company employees who had been at Cumberland or Hudson House during the epidemic, he concluded that "far more than one half had died, and from the number of tents remaining, it appeared that about three fifths had perished." Thompson goes on to assert that "more men died than women and children."⁶

The virtue of contemporary reports is that they are based on first-hand accounts of the outbreak as experienced by those living in the region. In fact it is primarily from these accounts that researchers have concluded that mortality from smallpox ranged from 60 to 90 percent. In his work on the Northern Athapaskan social organization, Shepard Kretch, citing Hearne, writes: "there is no evidence that any epidemic disease reached the Kutchin in the eighteenth century. In 1781, smallpox ravaged the Cree and Chipewyan, with losses among some Chipewyan groups estimated up to 90 percent" (Kretch 1978: 712). In *Indians and the Fur Trade*, Ray writes that "lacking any immunity ... the Indians suffered terrible losses" (Ray 1974: 105). While not being specific about actual mortality, Ray tends to agree with David Thomson's claim that mortality was one-half to three-fifth of the population. In his introduction to the *Saskatchewan Journals*, Glover wrote that "among the natives the scourge swept as murderously as the Black Death through medieval England" (Tyrell 1934: lviii). These contemporary accounts are direct evidence of the epidemic. Still, Europeans, and

indeed natives in the region, would have had only a partial picture of the epidemic. They did hear about and witness native deaths, but given the limited area where they lived and worked, they could obtain no more than a rough idea of the broader impact.

Variola and Mortality

Smallpox or variola is an infectious disease. *Variola major* is the more severe class and would have been the form that afflicted America in the eighteenth century. Yet even in the case of *variola major* there could be larger differences in mortality depending on the progress of the disease, whether haemorrhagic, flat, ordinary, or modified. Modified refers to those who have had previous exposure and so is not relevant to this discussion.

Haemorrhagic and flat are both extremely severe and nearly always fatal. As its name suggests, haemorrhagic refers to visible bleeding into the skin and flat refers to pustules that are not raised on the skin. None of the entries by company officials describe these forms. For example, the Cumberland House journal entry for December 27th notes “This morning could observe the small pox coming out very thick upon sick lads heads and thighs.” This is the classic location for smallpox. Ordinary *variola major* is therefore probably what was contracted.

The nature of the rash is itself a leading indicator of mortality. The number of lesions touching one another within a defined area is called confluence. The greater the degree of confluence the more dangerous the disease. A twentieth-century study of an unvaccinated population in rural India found case fatality rates of 62 percent for *confluent ordinary-type smallpox*, 37 percent for *semi-confluent ordinary-type smallpox*, and 9 percent for *discrete ordinary-type smallpox* (Fenner et al.1988: 22). That same study found the incidence of these three types to be 22.8, 23.9 and 42.9 percent, respectively, of the total cases. Thus the

weighted case fatality rate over the three types of smallpox was 30 percent.

We do not know which sub-type comprised the smallpox epidemic of the eighteenth century. However, given the wide variation in later smallpox fatality rates, the strain of smallpox that afflicted North America in the late eighteenth century could have had important implications for the mortality of the native (and European) populations. In the 1721 outbreak in Boston, the death rate was 15 percent among those who contracted smallpox, indicating perhaps that the sub-types of smallpox that characterized the epidemics were not the most virulent, but we do not know how many might have had prior contact with the disease (Fenn 2001: 33). As we noted, the descriptions do suggest the ordinary type rather than the much more fatal haemorrhagic or flat. At the same we have to be careful not to infer too much given that the 1780-82 outbreak in the Hudson Bay region was a “virgin soil” epidemic, which may have led to higher mortality than epidemics among previously exposed populations.

There have been a number of well-documented studies of case-fatality rates in smallpox outbreaks since 1900 (see Table 1). These rates, which apply to unvaccinated populations, or those victims of the outbreak who were not vaccinated, are in the range of about 15 to 35 percent, with rates similar, although slightly lower, for adults than children. The highest rate reported in Table 1, 35 percent, is for Madras, India over the period 1961-69, but the average over these outbreaks is closer to 20 percent. These rates have the advantage of applying to populations who were carefully studied, and are likely more reliable than rates reported in earlier epidemics. An account of a “virgin-soil” epidemic in 1795 in a village on the Japanese island of Hachijo-Jima implies a case-fatality rate of 38.3 percent (Fenner et al. 1988: 227). During the U.S. Civil War an outbreak of smallpox among a largely unvaccinated Union army led to case-fatality rates of about 35 percent - similar for

whites and Negroes; while at the time of the Franco-Prussian war, French soldiers, who were not vaccinated, had case-fatality rates of 18.7 percent. There also are reports of total mortality in some early epidemics. In 1242, the first smallpox epidemic to hit Iceland is said to have killed 30 percent of the population; and it was reported that an outbreak in 1707 resulted in similar mortality (Fenn 2001: 229; Ramenofsky 1987: 161).

Given the comparatively low mortality among European and other populations outside the Americas relative to the rates of up to 90 percent and even more reported for native groups, there is speculation that, because of the thousands of years of isolation, there was less diversity in the Native Americans' immune system antigens which rendered them less able to survive smallpox and other new infections (Fenn 2001: 26). In her classic work on this epidemic, Fenn (2001) points out, however, that this lack of diversity was more likely an issue in measles epidemics rather than smallpox; and in other work, scholars have expressed skepticism of a genetic explanation, certainly as it relates to ABO blood groups (Crosby 1976: 291-92; Fenner et al. 1988: 166). On the other hand, there has been experimental work on mousepox that finds a genetic component to resistance, and a small study of Dutch soldiers indicates that the presence of a particular HLA (human leukocyte antigen) group, Cw3, possibly implies greater susceptibility to smallpox (Fenner et al. 1988: 166). The HLA allele, Cw3, which is present among 30 percent of the Netherlands population, is common among Amerindians (Bernal et al. 1990: 1050).⁷

The documented mortality rates and case-fatality rates are much lower than the reported experience of Native Americans; nevertheless, we should heed Ramenofsky's warning that we should not simply assume Native American mortality was equal to these, generally more recent, estimates (Ramenofsky 1987: 160-62). One question is whether mortality was increased by the way the disease was treated by Native Americans, as well as

because of the harsh subarctic environment. Even in the twentieth century there was, other than early vaccination, really no effective way of treating smallpox. As Fenner et al. (1988: 64, 68) puts it: “No disease better illustrated the adage ‘Prevention is better than cure.’” In poorer countries “patients were usually better looked after at home in their village surroundings.” Of course counterproductive treatments could increase mortality. Observers noted that the sweat lodge would have increased the fever associated with the early stage of the disease, when mortality was greatest. And it has been noted by others that natives responded to their fever by jumping into cold streams which, according to one fur trader, caused “instant death;” but, in fact, cold water may have been an effective way of reducing fever. In any case, and as Fenn points out, it is not at all clear that the treatment Europeans typically received, such as bloodletting, was better than the practices of Natives Americans.

Native lifeways may also have affected mortality from smallpox. The Cree and Assiniboin were migratory hunters, and a prolonged period of forced inactivity could have been devastating not just to the adult males, but to their families as well. Smallpox, although very deadly, has symptoms that last for a relatively brief period; and although the sequelae include blindness, the more severe consequences of smallpox are rare. In fatal cases, death occurs between the 10th and 16th day of the illness; and among survivors scabs separate by the 22nd to the 27th days (Fenner et al. 1988: 22). Thus, the disease would have very seriously limited hunting and other activities for perhaps 3 to 4 weeks. The scabbing left on the soles of feet might have affected mobility for a somewhat longer period, and victims would have taken longer still to regain their full strength.

Although the impact of the disease would have largely dissipated after a month, even a month of inactivity could have serious consequences for the hunters and their families. Throughout the eighteenth century the post journals do record instances where natives arrived

at company posts, not to trade but to obtain food. There is no indication, however, that these cases increased either during or after the smallpox epidemic. In fact during what would have been the normal trading 1782 trading year, which followed the epidemic, hardly any natives came to the interior trading posts for any reason. Moreover, the few natives, who did come to the post, and reported the devastation, made clear that it was during the course of the disease that victims died.

The case-fatality rates for documented epidemics are very much lower than the overall mortality of natives that has been given in some of the historical accounts; but of course case-fatality rates apply only to those who contracted the disease. There has been considerable work done on the transmission of smallpox; and the conclusion is that smallpox is less contagious than other common infectious diseases, notably measles, influenza and whooping cough.⁸ The only route of infection of smallpox is through the respiratory tract. Depending on individual susceptibility, this would normally require direct contact with an infected person, and exposure to a relatively high concentration of the bacteria. According to the U.S. Center for Disease Control: “direct and fairly prolonged face-to-face contact is required to spread the disease from one person to another,” contact within about 6 to 7 feet for a period of a few hours. The disease does not normally spread as a result of casual or brief encounters.⁹ Indirect transmission via fomites, including clothing and blankets, is virtually impossible (Fenner et al. 1988: 194).

The epidemic swept through the Hudson Bay region in the fall and winter when natives would have been in their winter grounds, occupying wigwams or tepees. Since the living space in the tents was just over 100 square feet for perhaps nine to eleven individuals, the disease, once brought in, would likely have spread to all the occupants. On the other hand, transmission between tents or from one native group to another seems much less likely.

Of course, we know smallpox did spread to some in the region; but because of the dispersed population - a density of at most one wigwam per 500 square kilometers (see below) - many native groups appear to have been untouched by the epidemic. In summary, the evidence on case-fatality rates in other smallpox epidemics, the relative difficulty of transmission of smallpox, and the low population density in the Hudson Bay region belie the sorts of mortality rates claimed in the historical literature.

Mortality from the 1780-82 Smallpox Epidemic and the Trade Records

Given the nature of smallpox and the more recent evidence on how it has affected diverse populations, one might be sceptical of mortality rates of 60 percent, or higher, as reported in the diary and journal accounts from Hudson Bay. As an alternative to these reports of mortality in the region, we now approach the epidemic and its impact by focussing on the trade records. In the mid-1770s the Hudson's Bay Company set up two interior trading houses, which served York Factory and were designed to compete with the Montreal traders, who were an increasing presence in the region. Cumberland House and Hudson House are located in the interior several hundred kilometers from York Factory (see Figure 1). The posts quickly generated a trade comparable to the returns at York Factory from the rest of its hinterland. The trade at these the two posts is especially pertinent to our discussion of mortality since they were in the direct path of the epidemic (see Figure 2).

Neither Cumberland House nor Hudson House kept separate accounts. Although we do have their journals, these provide no more than a rough indication of the extent of their trade. Better are the accounts from the main trading post, York Factory. Its records include all the furs received; but, more important in terms of assessing the trade at its collection points, the accounts give a detailed list of the trade goods sent to Cumberland House and

Hudson House each year both before and after the smallpox outbreak. As shown in Figure 3, the volume of trade goods sent in 1777 was relatively modest at 6,060 *made beaver (mb)*, not surprising given that the two sites had just been established.¹⁰ But activity increased, reaching 11,770 *mb* in 1781, the year before the epidemic struck. Of course, central in terms of inferring the impact of smallpox on the aboriginal population is what happened after 1781.

There is no question that the epidemic year itself was devastating to the fur trade. Hardly any natives arrived at the posts, and those who did come brought few furs. The Cumberland House journal comment on the virtual disappearance of the trade and the York Factory accounts are consistent with the traders' descriptions. Realizing that little additional inventory was needed, York Factory sent just 800 *mb* in trade goods that year. There was very little trade the following year as well, although in this case the reason was political as much as environmental. The western hinterlands of Hudson Bay had entirely escaped conflict during the Seven Years' War of 1756 to 1763, but the involvement of the French in the American Revolution spilled over to the Hudson Bay region (Rich, vol. 2 1960: 84). In 1782, Comte de Lapérouse set out with a 74-gun ship and two frigates to capture the main English bay-side posts. On August 8th he took Fort Churchill and two weeks later York Factory. There was no resistance from the English. Both posts, though, were severely damaged, but not completely destroyed. Still, it was nearly a year before the company could re-establish trade at York Factory and Fort Churchill.

Despite the temporary loss of York Factory, the inland houses continued to trade from their depleted inventory.¹¹ The Cumberland House journal entry of June 20, 1783 reports that 115 bundles of furs in ten canoes were sent to York Factory. At roughly 50 *mb* per bundle, the total trade would have been close to 6,000 *mb*, an indication the trade was already

beginning to recover. But much more revealing is what happened after York Factory was again operational and resumed sending trade goods to its inland collection points. In 1784 deliveries of trade goods totalled more than 6,850 *mb*, and in 1785 their value of 9,400 *mb* was more than in any year other than 1781. Following 1785 the trade continued to increase, and in 1787 it surpassed *any* previous year. Even recognizing that some of the trade goods sent after 1783 might have been needed to replenish inventories, it seems inescapable that the natives were bringing greater numbers of furs to the post. This level of trade was taking place after an epidemic that was claimed to have decimated the population, and even more so the segment of the population, adult males, who were the main participants in the fur trade.

To highlight the change in trade before and after the epidemic, we compare the trade goods sent from York Factory to Cumberland House in 1781 and 1785 (see Table 1). In 1785, just three years after the epidemic had swept through the region and less than three years after the York Factory post had been sacked by the French, trade was already recovering. There is considerable variation by commodity, but two goods in particular give a perspective on the yearly volume of the trade, since it is unlikely these would be stored for long periods. In 1785, 448 gallons of brandy were sent to Cumberland House as compared to 675 gallons in 1781, a decline of one-third. Meanwhile the shipment of tobacco, a perhaps even more important trade good fell by only 15 percent, from 2,348 lbs to 2,007 lbs. The total trade declined by a roughly equal percentage as the trade in tobacco. Valuing the goods at the official *made beaver* prices, the total value of shipments declined by 20 percent between 1781 and 1785, from 11,769 *mb* to 9,401 *mb*. Such a reduction in trade seems totally out of line with claims that the population of native hunters during this period had fallen by 60 percent or more. Just three years later, in 1788, York Factory sent 13,856 *mb* in trade goods to Cumberland House, or nearly 20 percent *more* than the value of goods sent in 1781. One

might argue that perhaps native traders were coming to Cumberland and Hudson Houses from greater distances. We explore the issue of migration later in the paper. But especially given by the path of diffusion the disease vector, it is unlikely that people were moving into the region.

The accounts for Cumberland and Hudson Houses have the advantage of providing clear measures of the pattern of trade over the period that includes the smallpox outbreak. We can use the trading evidence to make inferences about the mortality impact of the epidemic because only native people trapped and traded animal pelts. Assume a standard harvest function:

$$H = H(E, X), \tag{1}$$

where H is the harvest, X is the population of fur-bearing animals (mainly beaver), and E is harvesting effort. By applying equation (1), observations on the size of the harvest can shed light on the extent of harvesting effort and, by extension, the number of people involved in the trade. We have derived elsewhere an effort elasticity for beaver of $2/3$.¹² From this elasticity, a sixty percent decline in effort, reflecting the lower end of reported declines in the native population, would have reduced the harvest for a given animal stock by more than 45 percent. If we allow for some growth in the animal population due to the temporarily lower harvests, the decline is somewhat smaller, 40 percent.¹³ Prior to the smallpox epidemic, the value of trade goods sent from York Factory to Cumberland House in 1781 was 11,770 *mb*. A reduction in the harvest of 40 percent would have reduced the trade to about 7,000 *mb*. This in fact approximated the value of goods sent in 1784. But in 1786 11,000 *mb* in trade goods were sent, and the value of trade goods sent in 1788 was nearly 14,000*mb*, roughly double what might have been expected if the reports of mortality had been even approximately true.

We do not mean to suggest that the number of native traders was unaffected by the smallpox epidemic; rather that the pattern of trade from an area that was in the path of the epidemic is inconsistent with extreme mortality.

Population Estimates, Carrying Capacity and the Smallpox Epidemic

The volume of trade after 1782 at Cumberland House and Hudson House appears to belie the contemporary reports that sixty percent or even ninety percent of the natives in the region were carried off by smallpox. Given that such factors as a changing fur resource base, the degree of competition from the French traders, the size of the hinterland served by the houses, and population shifts could also affect the trade, it is important and perhaps ultimately more useful to deal directly with the issue of mortality by focussing directly on the actual size of native population.

Our approach initially follows along the lines of Ray (1974: 94-116) who uses contemporary descriptions of the population of native groups. Some of the European travellers to the region not only described the native settlements they visited but often included a commentary on the number of tents. Such reports include the years preceding and following the smallpox outbreak. For the early nineteenth century we have Alexander Henry the Younger's breakdown for the Assiniboin groups, many of whom had been in the direct path of the disease. His report is very detailed and is generally accepted as the most accurate. In 1808, twenty-six years after the epidemic, Alexander Henry put the total for eleven different groups of Plains Assiniboin at 850 tents (Coues 1897: 522-23). Assuming nine persons per tent gives a population of 7,650. At eleven per tent, the number Peter Fidler assumed in the early nineteenth century and reported by Demollie and Miller (1981: 590), the population was 9,350. In 1776, Alexander Henry the Elder reported 300 tents for the Plains

Assiniboin. This is just 35 percent of his nephew's later estimate. Even had there been no epidemic this number generates implausibly high growth rates for the Assiniboin population.¹⁴ It seems clear that Alexander Henry the Elder's count was incomplete. In fact elsewhere he wrote: "The Osinipoilles [Assiniboin] have many villages composed of from one to two hundred tents each" (Henry 1969: 303). This statement suggests that the Assiniboin population in the region may have been closer to, or even above, the more detailed estimate of thirty-two years later.

Given that the eighteenth-century reports on native settlements are incomplete and vague, we approach the question of native population and the impact of the smallpox epidemic in a way that relies on knowledge that is more firmly based. We begin by taking the nineteenth-century population estimate of Henry the Younger and project it backwards to just after the epidemic. We then compare that projected value to an estimate of the pre-epidemic population, which we base on the carrying capacity of the region; namely the native population that could have been supported by the local food supply. During the subarctic winter adult males required 4,500 to 5,000 calories per day; and, although fewer calories were needed at other times, the average daily requirement was at least *four* pounds (1.8 kg) of flesh food (Rogers and Smith 1981:141). Moreover, because a high fat content was necessary, the requirement had to be met mainly from the meat of large game. The native population was therefore limited by the population of large ungulates.

The northern boreal forest that covered nearly all the fur-trading hinterland of the Hudson's Bay Company was ideal habitat for moose and this was the main food source of the natives who lived in the region. The Cumberland post journal for 1774/5 is replete with description of moose as a primary meat source. Indeed, no other large ungulate is mentioned.

For example, in September “Early in the morning an Indian man came to the tent and informed me of his having killed a moose not far off for which I payed him and sent the people with two canoes to fetch home the meat;” in December “The Indian Man who were sometime since mentioned as starving came in with some of his family brought 4 sledge load of Moose Flesh;” and in February the journal notes: “one Indian man with news that 3 tents of Indians were within 5 hours walk and some had killed 5 moose and coming with most of the meat” (HBC, *Post Journals, Cumberland House*).

The maximum density of moose and the amount of meat available from optimally exploiting the biomass thus provide us with a way of conjecturing the maximum human population density, and, by extension, the total native population in the region. Even allowing that some of the flesh requirement was met by rabbit and other small game in the winter and by fish as well during the summer, consumption of large ungulates could hardly have averaged less than 1.5 kg per day for an adult male, and 5 kg per day for a family of five (Carlos and Lewis 2010: 220-30). Over the year consumption would therefore have totalled about 1,800 kg per family.

There are a wide range of estimates of moose density in the type of boreal forest that characterized the Hudson Bay region. Recent moose surveys showed a marked increase in densities over the period 1975 to 1995 in Ontario, especially in the late 1980s after more selective harvesting and moose habitat guidelines were introduced (McKenney et al. 1998). The average for the province for the years 1990-1995 was 0.209 moose/km², with considerable spatial variation across the roughly 70 wildlife management units. Densities ranged from 0.05 to 0.79 moose/km², with some of the higher densities reported in northwest part of the province, the region that would have been part of the Hudson’s Bay Company’s fur

trading hinterland. In that region the average density was 0.298 moose/km².¹⁵ If we take this to be the density consistent with maximum sustained yield management, then density at capacity in this region would have been roughly 0.5 moose/km².¹⁶

Crête et al. (1981, 609) have estimated potential harvesting rates for moose for different kill ratios of calves, cows, and bulls. For a moose population of 1,000 at capacity, they derive a potential harvest of between 62 and 169 animals, depending on the category of animal taken; and a potential biomass harvest of between 22 thousand and 50 thousand kilograms per year. Given the more limited ability of native hunters to select the kill, it is unlikely that their harvesting could have produced the top of this range. On the other hand, natives likely could have obtained more than the lower limit, which assumes only bulls are hunted. A potential harvest of 30 to 40 thousand kilograms live weight would seem to be toward the upper end of what would have been possible. Using current dressing techniques, “lean edible tissue” of bull moose is 36.5 percent of their live weight.¹⁷ If natives obtained this proportion, the meat they might have harvested each year, from a region with a capacity of 1,000 moose, was 10,950 to 14,600 kg, which would have been sufficient to maintain between six and eight families.

The part of the Hudson Bay region that was in the path of the epidemic was one of the better habitats for moose, so we take the upper end in Ontario of moose density at capacity, 0.5 moose/km². At this density the families, who could have been supported by a standing crop of moose at 1,000 capacity, needed a hunting ground of 2,000 km². At five persons per family, the population would therefore been in the range one person per 67 to 50 square kilometers. These values are well above the high end of the population density estimate, one person per 130 km², given by Rogers and Smith (1981) for the northern Canadian shield, and

they are about twice the population density in the entire York Factory hinterland based on tent counts during mid-eighteenth century (Carlos and Lewis 2010, 72).¹⁸ On the other hand, they are close to Ubelaker (1988, 291), who takes one person per 50 km² as the likely density in all the subarctic at the time of initial European contact.

We infer the possible impact of the smallpox epidemic by backward projection of the population to 1782 using what was likely the most complete count of the native population, that made by Alexander Henry the Younger in 1808 of the Assiniboin. At that time his count of the various groups of Assiniboin totaled 850 tents, or 8,500 individuals, assuming ten persons or two families per tent. The Assiniboin population occupied a territory in the early nineteenth century of about 415,000 square kilometers (Ray 1974: 101). The implied population density is one person per 50 km², which is the upper end of the range based on the carrying capacity of the region.¹⁹ We do not know how fast the native population was growing following the epidemic; but increases in population with life expectancies typical of the time could not have been much greater than one percent per year.²⁰ At one percent annual growth from 1782, the year of the epidemic, to 1808, the Assiniboin would have numbered 6,560 in 1782. Assuming the upper end of our estimated maximum population density, one per 50 km², the population of the region just prior to the epidemic would have been 8,300, implying a loss of 20 percent due to the outbreak. If the lower density of one per 67 per km² is assumed, then the implied population, 6,200, is *less* than the population based on the backward projection.

The calculation of population based on carrying capacity is subject to a range of error at each stage, including those involving meat requirements, edible meat per animal, maximum harvest rates, and potential animal densities. Our approach, we believe, has been to err on the

side of overestimating densities, but further adjustments including making an allowance for the fact Assiniboin occupied some non-boreal forest areas, could result in higher densities than we have suggested. As it is, though, our densities are well above those suggested by Rogers and Smith, and close the value given by Uberlaker for this type of region. The overall implication of this approach based on carrying capacity is that the smallpox epidemic reduced the native population in the region by no more than 20 percent, and quite possibly less.

Interpreting the Epidemic

Our conclusion that the smallpox epidemic of 1780-82 led to relatively low mortality is contrary to nearly all that has been written, both by contemporary observers and historians. Given that we have no reliable population counts for the pre-epidemic period, such an iconoclastic result should be viewed with scepticism. At the same time, it is important to recognize that the generally accepted narrative is based on evidence that is less than firm. Statements by the English about mortality were in fact their interpretation of what the Assiniboin, Ojibwa, and other groups were telling them. For example, Samuel Hearne the chief trader at Fort Churchill thought that nine-tenths of the Indians in the hinterland of the post had died and it is this statement that has been taken as evidence in most of the current historiography. Yet given how far removed Fort Churchill was from the path of the epidemic, this impression could only have come from the statements of the natives who came to the post, many of whom were not even living in the main areas hit by the disease. Indeed, Fort Churchill was established to maintain a separation between the Chipecwan and their traditional enemies the Cree and Assiniboin. Thus traders to Fort Churchill would have little if no direct knowledge of events in the Cumberland House region. Even the entry in the York Factory journal for July 2, 1782 that among natives who had been in the La Pas area “not one

in fifty of those tribes is still living,” must be viewed with care given that it is a translation of what the Governor thought the Indians were saying.

Mortality rates of 90 or 95 percent for the overall region should probably be discounted, but other more modest estimates by contemporaries suffer from the same problem in that they are based either on incomplete death counts or on vague comparisons of pre- and post-epidemic native populations. David Thompson, who traveled widely in the region, but did not arrive until four years after the smallpox outbreak, gives 60 percent as the possible mortality. This estimate comes in part from Mitchell Omam, one of the Company’s interior traders, who accompanied Thompson in 1786. From what the natives told Omam and what he observed about the number of tents that remained he told Thompson that “it appeared about three-fifths had perished” (Glover 1962: 236). Thompson also talked about “seeing” tents in the vicinity in which the natives “were all dead.” Clearly these eye-witness accounts are testimony to a disease that killed many. Still, the lack of anything approaching firm numbers calls for scepticism about this evidence as well.

Despite the claims of contemporaries and the interpretation of historians of the smallpox epidemic of 1780-82 in the Hudson Bay region, the three approaches to smallpox mortality used in this paper lead to broadly similar conclusions. There is a long history of variola in its various forms, some more virulent than others, but in no case where numbers are reliable did case-fatality rates begin to approach the sorts of mortality claims made about the 1780-82 epidemic, and this includes rates for other “virgin soil” populations. In areas of rural India, where the population had no previous exposure to smallpox, case fatality rates for the adult population under age 40 were found to be on the order of 20 percent, and the experience of other populations where data are reliable has been similar. In fact recent work on smallpox

suggests there may be little or no genetic link, suggesting Native Americans may have been no more likely to die from the disease than unvaccinated populations with a long history of the disease. It also appears, given the difficulty of transmitting smallpox as compared to other infectious diseases, that many native groups were likely untouched by this smallpox epidemic. If observed case-fatality rates are reflective of what Native Americans might have experienced, and recognizing that the population was thinly dispersed over the subarctic and far northern plains, overall mortality could very well have been on the order of 20 percent or less.

Our second approach to mortality is to examine the trade at the Cumberland and Hudson outposts, which were in the direct path of the epidemic. The volume of furs brought to these sites was entirely dependant on the native trappers and traders. It seems inconceivable that a large decline in that population would not have been reflected in the trade returns. In 1782, the year smallpox passed through the region, the trade at these posts did indeed collapse, but this might not because of native mortality. Rather, fearing the effects of the disease, natives redirected their effort from obtaining the luxuries associated with the fur trade to concentrating on their survival. Had a decline in native population been the reason for the loss in the trade, the effects would have been long-lasting. What we find instead is that the volume of trade, as reflected in the goods sent by York Factory, exceeded within six years the peak of the trade prior to the epidemic. There is the possibility that the vacuum left by the epidemic was filled by other native groups moving into the region; but evidence on aboriginal migration gives no indication that this happened. Over the period 1780 to 1821, despite some changes within in the region, the Cree, Assiniboin, Ojibwa and Chipewyan were occupying very similar territories (Ray 1974, 99-110). Indeed it would have taken some courage for natives in areas unaffected by smallpox to move into a region that had so recently

been devastated by the disease.

Finally we infer mortality on the basis of a backward population projection and the carrying capacity of the land in the region. We have for the Assiniboin what appears to be a careful population estimate for 1808. By assuming a relatively high annual growth rate of 1 percent, we estimate that the human population in 1782, just after the epidemic passed through the region, was at least of 6,500. Even had the pre-epidemic population been close to the maximum carrying capacity of the region - one person per 50 km² or 8,300 in total for the 415,000 square kilometer region - the smallpox outbreak could have reduced the population by no more than 20 percent.

Undoubtedly some native people in the region died of smallpox; the contemporary accounts on this score are indisputable. What is in question is how widespread was smallpox in terms of the numbers contracting the disease, and how lethal the disease was to those who contracted it. We conclude that the smallpox epidemic of 1780-82 in the subarctic and far northern plains had a modest impact on the native population. Importantly, this finding could have broader implications for views about the size of aboriginal populations prior to the coming of Europeans and how contact affected those populations and native society.

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Endnotes

1. The disease appears to have begun in Boston in 1775 and had three epicenters along the east coast of the United States. The disease spread from the Gulf of Mexico northward through the central plains, reaching the southern part of the Hudson Bay Company's fur-trading hinterland in the summer of 1781. This same epidemic continued to the Columbia River basin and Puget Sound (Fenn 2001: 7).
2. The tone set in this entry is similar to that found in other entries. William Walker does not mention smallpox here but does subsequently. The women mentioned died two days later and were buried by the company men because the natives would not touch her body.
3. Arthur Ray (1974: 94-116) has used these reports to map the progress of the smallpox epidemic as shown in Figure 2.
4. In 1786, David Thompson saw a group of Indians sitting on a beach about 150 miles up river from York Factory: "to our surprise they had marks of the small pox, were weak and just recovering" (Glover 1962: 235-36).
5. The disease did not merely result in increased mortality. There was also the longer term impact of morbidity, which we touch on below. One occasional effect of morbidity was on future fertility. Ramenofsky (1987: 147).
6. This observation was based on information from Mitchell Omen who had been at the posts during 1781/2. See Glover (1952: 236).
7. It should be emphasised, though, that the connection between Cw3 and susceptibility to smallpox is tenuous at best. The impact of a diminished immune response to a virus is ambiguous in terms of clinical outcomes; moreover vaccinia has a somewhat different structure than smallpox. So it is not even clear that the immune response to smallpox and vaccinia is the same. I thank Dr. Roy Ilan, Faculty of Medicine, Queen's University for these observations.
8. <http://www.emergency.cdc.gov/agent/smallpox/overview/disease-facts.asp> (accessed 12/09/2010).
9. Ibid. The incubation period for the disease is normally 12 to 14 days but can range from 7 to 17 days. Rarely has smallpox been spread in settings where contact is fleeting such as buildings, buses, and trains. Humans are the only natural hosts of variola. Smallpox is not transmitted by insects or animals.
10. The *made beaver* (*mb*) was the unit of account used by the Hudson's Bay Company at all its trading posts. A prime beaver pelt had a price of *1mb* and all other fur and trade goods were assigned prices relative to that standard.
11. Company policy did not allow posts to maintain high inventories, indeed, the London management kept a very close watch on the level of inventories and the annual request for new trade goods. However, in this case both Cumberland and Hudson Houses would have

the inventory from the epidemic year to work from.

12. The harvest function for beaver has been derived as: $H = K E^{\frac{1}{3}} X^{\frac{1}{3}}$ (Carlos and Lewis 1993: 492).

13. A 60 percent decline in effort combined with a 30 percent increase in the beaver population gives this result (see note 12).

14. The implied population growth rate is nearly 5 percent per year.

15. This was the average density in Wildlife Management Units (WMU): 5, 6, 7A, 7B, 8, 9A, 9B, 10, 11A, 11B, 12A, 12B, 15A, 15B (McKenney et al. 1998: 1929). Densities in WMU's to the north and to the east were much lower.

16. Crête et al. (1981: 608) estimate, for different assumption about the pattern of mortality, the maximum sustained yield population for moose in southwestern Quebec as between 48 percent and 72 percent of capacity. Their intermediate values are about 0.6.

17. This is for a moose weighing 914 lbs. The field dressed weight is 72.9 percent of live weight, and the edible tissue is 50 percent of that (Marchelle and Garden-Robinson 2006: 8).

18. On the other hand Uberlaker (1988) estimates population density at the time of contact as one person per 50 km² in the entire subarctic and Dobyns (1983) has derived population estimates for North America that imply densities that are much higher still.

19. In the early nineteenth century the area occupied by the Assiniboin included in the plains, which likely supported greater population densities than the boreal forest.

20. Life expectancy at birth of these populations would certainly have been under 35 years. The required gross reproduction rate (GRR) that would have allowed an annual population growth of 1 percent was between 2.75 and 3.00 (Wrigley and Schofield 1989: 243). A GRR in this range (roughly 5.5 to 6 births per woman) is close to the upper end of what would have been possible. There could also have been morbidity impacts on fertility in the years after the epidemic.

Table 1. Smallpox Case-Fatality Rates in Unvaccinated Populations

	Period	Case-fatality rate (percent)	
		Adults ^a	All
Liverpool, England	1902-1903	24.0	27.2
Tabriz, Iran	1954-1955		13.8
Madras, India	1961-1969	35.4	35.5
West Pakistan	1966-1967	15.9	15.7
Noakhali, Bangladesh	1972-1973	20	21
India, 6 States	1974-1975	18.1	26.5

^a Ages ranges are generally from 15 to 50.

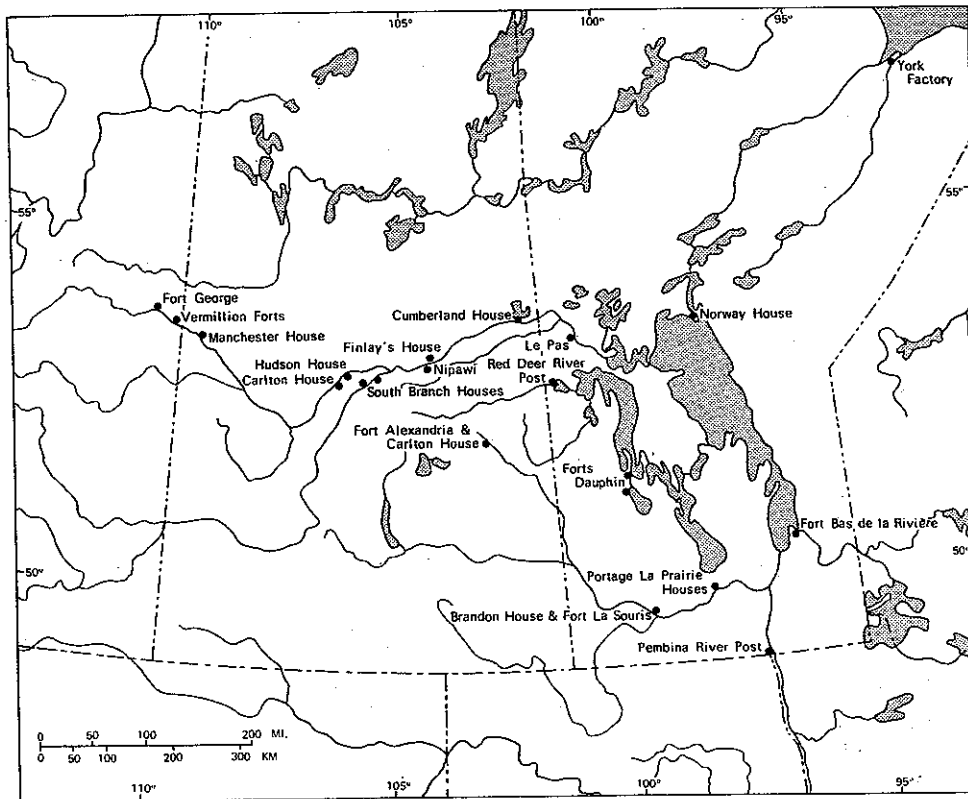
Sources: Fenner et al. (1988, 51, 53, 54, 176); Fredericksen and Motemani (1957, 854); Mack et al. (1970, 483).

Table 2. Trade Goods Sent from York Factory to Cumberland House, 1781 and 1785

Trade Good	1781	1785		1781	1785
awl blades	288	26	kettles	52	61
baize (yds)	20	71	knives	1,830	1,818
bayonets	204	126	lace (yds)	180	
beads (lbs)	39	28	looking glasses	40	48
blankets	86	43	needles	388	504
brandy (gals)	675	448	net lines	6	12
buttons	24	37	pistols		10
cloth - various (yds)	1,316	761	powder (lbs)	1,308	934
combs	84	100	powder horns	7	9
duffel (yds)	172	48	rings		516
files	66	144	rundlets	31	53
fish hooks	160		scrapers		10
flints	3,000	2,000	shirts	68	125
gartering (yds)		836	shot (lbs)	2,416	1,240
gun worms	144	288	stockings		24
guns	60	99	thimbles		24
hatchets	288	211	thread	3	6
hats	8	21	tobacco (lbs)	2,348	2,007
hawk bells	216	500	tobacco boxes	30	57
ice chissels	202	80	vermilion (lbs)	14	10
<i>Total - made beaver</i>				11,769	9,401

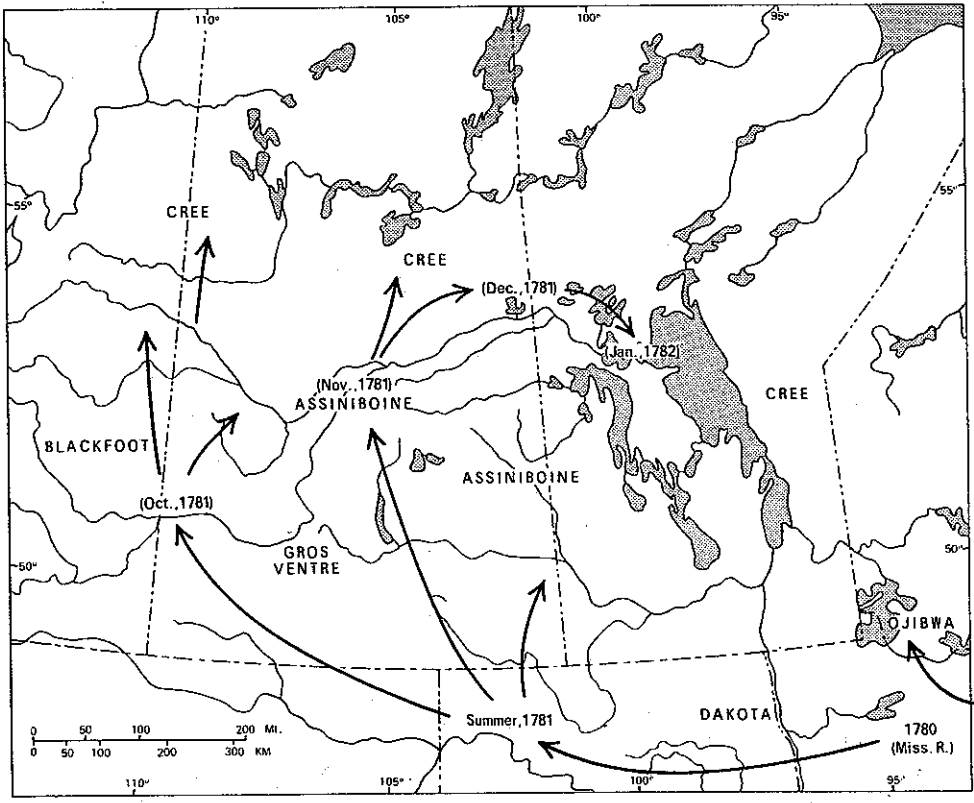
Source: HBC, Post Accounts: York Factory, 1781,1785.

Figure 1. Fur Trading Posts in the York Factory Hinterland, 1763 – 1821



Source: Ray (1974:127).

Figure 2. Principle Path of Diffusion of the Smallpox Epidemic in the Hudson Bay Region, 1780 – 1782



Source: Ray (1974:107).

Figure 3. The Value in *Made Beaver* of Trade Goods Sent from York Factory to Cumberland House, 1777 – 1789



Source: HBC, *Post Accounts: York Factory, 1777-89*.