Toward a Reconsideration of Disease and Contact in the Americas

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ABSTRACT. The common assumptions that serious infectious disease epidemics did not occur before prolonged European contact and that introduced European and African pathogens produced inevitable and devastating epidemics in the Americas are discussed. Evidence for pre-contact epidemics in southern Ontario Iroquoia is presented. The importance of fertility data for evaluating the demographic consequences of virgin soil epidemics is discussed and illustrated with a case study of the 1918 Spanish influenza epidemic at Norway House, Manitoba. It is argued that pre-contact infectious disease loads in specific regional sequences must be evaluated before the differential impact of post-contact pathogens can be adequately assessed. Monte Carlo simulations, mathematical modelling, the tracking of epidemic cycles, application of models developed for disease and sequences outside of the Americas and more extensive use of information contained in parish registers are suggested avenues for developing a more comprehensive understanding of disease and contact in the Americas.

SOMMAIRE. Dans cet article, on remet en question des postulats communs qui veulent qu'il n'y ait jamais eu d'épidémies de maladies contagieuses avant le contact prolongé avec des Européens et que ce sont des pathogènes européens et africains qui ont entraîné des épidémies fatales et dévastatrices aux Amériques. On y présente des preuves que la nation iroquoise du sud de l'Ontario a connu des épidémies avant le contact avec les Européens. On s'appuie sur l'épidémie de grippe espagnole qui a frappé Norway House au Manitoba en 1918 pour discuter et illustrer l'importance des données de fertilité afin d'évaluer les conséquences démographiques des épidémies qui s'abattent sur un terrain vierge. On y affirme qu'on doit évaluer les conséquences d'une maladie contagieuse avant le contact par séquences régionales précises avant de pouvoir bien évaluer l'impact différentiel des pathogènes après le contact. Afin de mieux comprendre la maladie et le contact aux Amériques, on suggère d'avoir recours à des simulations Monte-Carlo, des modèles mathématiques, des relevés des cycles épidémiques, à l'application de modèles élaborés pour les maladies et les séquences en dehors du continent américain et à une utilisation plus approfondie des registres paroissiaux.

The 500th anniversary of Columbus's journey from Spain to the West Indies has rekindled popular interest in the significance of Europe's first prolonged encounter with the Americas and with the people in it (Booth 1991; Nikiforuk 1991). One facet of the "Columbian encounter" that has preoccupied anthropologists and historians for several decades is the idea that devastating, imported European diseases precipitated an unprecedented demographic crisis in the Americas. For some, epidemiological reconstructions constitute "the key to explaining and describing Native American depopulation" (Dobyns 1984: 17); for others, "It was their germs, not these imperialists themselves, for all their brutality and callousness, that were chiefly responsible for sweeping aside the indigenes and opening the Neo-Europes to demographic takeover" (Crosby 1986: 196).

To date, most of the vigorous debate about disease and contact in the Americas has centred on the post-contact period (an exception is Verano and Ubelaker 1992). Major issues include: estimating initial and subsequent population sizes (see Ubelaker 1988 for a review); assessing the demographic impact of introduced epidemics (Meister 1976; Helm 1980; Dobyns 1983; Krech 1983; Crosby 1986); evaluating and developing appropriate methods and sources of data for deriving depopulation estimates (Dobyns 1966; Joraleman 1982; Thornton 1987; Henige 1990); and

estimating the timing and patterns of diffusion of early epidemics (Ramenofsky 1987; Decker 1988; Snow and Lanphear 1988; Dobyns 1989; Reff 1991).

Bruce Trigger (1985: 244) in *Natives and Newcomers* cautions, however, that "scholars should not succumb to the temptation of believing that in prehistoric times illness had not been prevalent or of concern to native people," stressing that "there is physical anthropological evidence of much chronic illness prior to the sixteenth century." But despite the recognition that infectious agents and pathogenic processes affected Amerindian populations well before prolonged European contact, few attempts have been made to evaluate the extent of pre-contact disease loads or to speculate on the effect these might have had on the experience of post-contact epidemics.

In fact, two common assumptions persist in visions of the relationship between European contact and disease: that serious epidemics did not occur prior to 1492 A.D., and that introduced European and African pathogens produced inevitable and virtually universal devastating epidemics. This article reviews some of the evidence for pre-contact disease in the Americas and discusses recent research initiatives in southern Ontario Iroquoia that suggest that epidemics occurred in the region prior to European contact. I then consider some of the evidence for variation in the experience of virgin soil epidemics and studies that indicate that mortality data alone are insufficient to gauge the demographic effects of epidemics. Lastly, I suggest a number of research directions that may lead to a more comprehensive understanding of disease and contact in the Americas.

Pre-contact Disease and Epidemics in the Americas

The evidence for health and disease in the Americas prior to European contact has been pieced together by physical anthropologists and paleopathologists from surviving bones, teeth and mummified tissue in archaeological context. This research has demonstrated that infectious diseases, with the capacity to erupt into epidemic form, were clearly present in the pre-contact Americas. Communicable diseases such as tuberculosis (Allison, Mendoza and Pezzia 1973; Katzenberg 1977; Buikstra and Cook 1981; Clarke et al. 1987), hookworm (Allison, Pezzia, Hasegawa and Gerszten 1974), Carrion's disease and other rickettsial diseases (Allison, Pezzia, Gerszten and Mendoza 1974), salmonellosis (Sawicki et al. 1976), amoebic dysentery, arthropod-borne viral fevers, American leishmaniasis and trypanosomiasis (Newman 1976), to name a few, appear to have been part of the American microbiological environment prior to 1492 A.D. Even supposedly harsh environments like the arctic which were once thought to have filtered out pathogens harboured by trans-Beringian migrants (Stewart 1973), accommodated an impressive catalogue of viruses, bacteria and parasites which likely infected people prior to European contact (Fortuine 1989: 45-72). Environmental mycobacteria,[1] in particular, flourish in arctic tundra conditions, especially in sphagnum moors (Clarke et al. 1987: 51).

It is not sufficient to simply identify the presence of infectious agents prior to European contact, for microorganisms will not erupt into epidemic form unless there are frequent opportunities for contact with animal reservoirs or insect vectors, or unless there is regular contact which allows them to spread from person to person. Social circumstances create the opportunities for the spread of infection and thus define the limits of a microorganism's reproductive success within a human population (Dubos 1965; Black 1990).

Recognition of the importance of the socioecological context of human disease has lead to the frequent observation that domestic animal reservoirs were lacking in the Americas, contributing to "the relatively disease-free pre-contact condition of Indians and Eskimos" (Newman 1976: 668). The potential for contracting zoonotic diseases, however, is high in any society in which people come in frequent contact with animals, animal wastes and insects (Fortuine 1989: 45-72). The impact of zoonotic disease in prehistoric societies, moreover, likely varied to a great degree, depending on local environmental features and subsistence activities.[2] Cohen (1989: 33) suggests, for instance, that tularemia, a dangerous relative of bubonic plague, may have been a serious affliction in American Indian communities whose subsistence economies involved the regular handling of game and fur-bearing animals. In a similar way, bison were a possible reservoir for *Mycobacterium bovis* in the pre-contact Americas (Buikstra 1981:13; Clarke et al. 1987:51) and infected herds may have exposed populations dependent on them to the risk of tuberculosis.

There is no doubt that small, sparsely distributed populations are unable to sustain density-dependent infectious agents (Black 1975, 1980). Microorgansims that follow a K-selection strategy,[3] however, can survive in endemic form under these demographic conditions. Examples of K-strategists include the treponematoses, mycobacteria and herpesviruses, none of which require large populations to take a constant toll of human life. They simply multiply slowly, persist in walled-off lesions, and provoke recurrent bouts of infectivity and disease (Fenner 1980). Once again, it is clear that even pre-contact gatherer-hunter communities were at risk of epidemics of infectious disease prior to the introduction of acute community infections from abroad. Given the general observation that infectious disease loads have increased through time, broadening the range of infections and increasing the opportunities for infection through increased population size and socioecological changes (Cohen 1989: 54; Armelagos et al. 1990), it must be concluded that the large number of agricultural and urbanized societies in the pre-contact Americas were fertile soil for infectious disease epidemics.

Over and above these general epidemiologic and demographic considerations, archaeological reconstructions make it quite clear that social conditions favourable for microorganisms to flourish were present in the Americas prior to European contact. Saunders, Ramsden and Herring (in press) argue, for instance, that pre-contact demographic and sanitary-

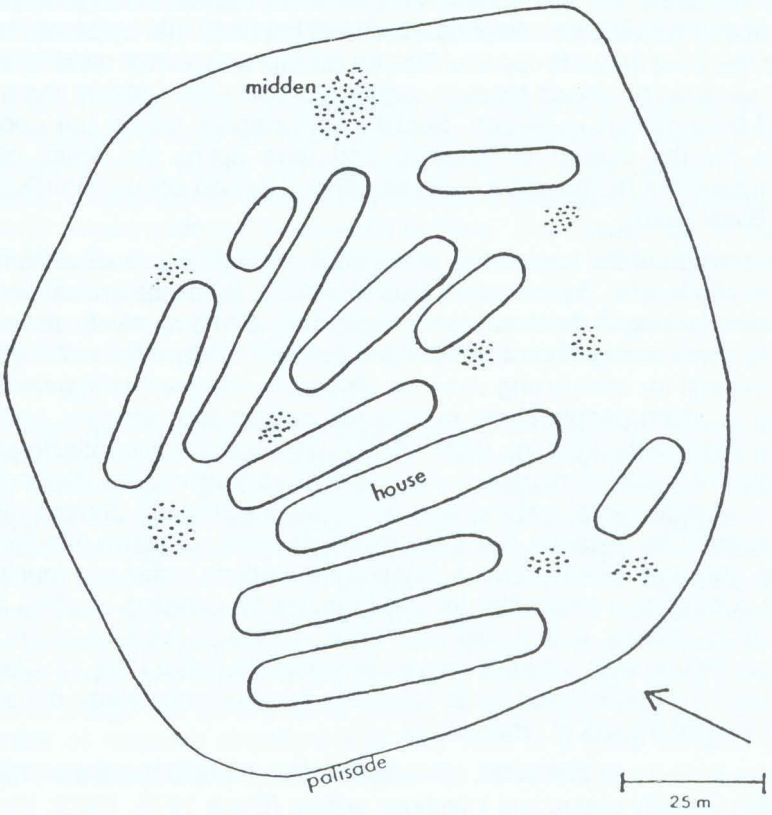

Figure 1. A "typical" pre-contact Ontario Iroquoian village as represented by the earliest Phase A of the Draper site. (From Saunders et al., 1992: 120.)

social conditions in southern Ontario Iroquoia were conducive to infectious disease epidemics. From about the eighth century to the seventeenth century, southern Ontario experienced a major demographic transformation through population increase, the emergence of larger and more numerous villages, increased village coalescence, and augmentation of the local population with migrant Iroquois from the south and southeast (Warrick 1984). The net effect of these processes was a substantial increase in village population densities and the creation of conditions necessary for person to person spread of infectious agents both within and between villages in the region.

Even the structure of Iroquoian villages was conducive to epidemic outbreaks. This is illustrated by the prehistoric Draper Site (phase A), dated to about 1450 A.D., which Finlayson (1985) conservatively estimates was occupied by 200 to 400 people who lived in eleven long-houses (Figure 1). The crowded village environment, in which the space between longhouses rarely exceeded three metres, was also pervaded with refuse dumps of

organic waste. Such sanitary-social circumstances meant that villagers not only were in close contact with each other, but also with the insects, dogs, and rodents that infested or were periodically drawn to the dumps.

Village inhabitants thus would have been at risk of contracting a variety of infections, such as encephalitis, rabies, and rickettsial diseases,[4] and living conditions would have facilitated outbreaks of endemic enteric bacterial infections like dysentery or airborne diseases such as pneumonia and tuberculosis (Saunders et al., in press). Recent work in Sarawak suggests, moreover, that longhouse living itself may be conducive to the flourishing of mycobacterial disease (Chen 1988). It is possible, therefore, that the regular relocation of Iroquoian villages every few decades or so was impelled by pest infestations and declining sanitary conditions, not just resource depletion (Fecteau et al. 1991).

There is strong evidence for pre-contact infectious disease stress and epidemics in southern Ontario Iroquoia, in keeping with the densely populated communities, complexly networked social structures, and demographically and epidemiologically dynamic populations that occupied the region at that time (for reviews see Pfeiffer 1984; Saunders et al., in press). In particular, resorptive lesions resembling tuberculosis have been identified at a number of Ontario Iroquoian sites dated from 900 to 1300 A.D. (Wright and Anderson 1969; Hartney 1981). Interestingly, these lesions begin to be detected around the time that the first longhouse villages appear in the archaeological record. If this observation represents a real increase in the bony expression of tuberculosis, then its coincidence with a change in the structure of social networks is consistent with the argument from theoretical ecology that the most important determinant of transmission patterns in person-to-person diseases is the organization of contact and interactions (McGrath 1988a: 329).

Temporal increases observed in the prevalence of tuberculosis lesions in southern Ontario Iroquoia, coupled with skeletal evidence from the Uxbridge site[5] (radiocarbon date estimated at 1490 ± 80 A.D.), lead Pfeiffer (1984: 188) to hypothesize that the disease was in the endemic, terminal phase of an epidemic cycle.[6] Taken together, the various lines of evidence, both osteological and socio-ecological, suggest that outbreaks of tuberculosis may have occurred in southern Ontario Iroquoia well before European contact. Whether this was the case for other areas in the northeast remains to be demonstrated. McGrath (1988b), for instance, argues from computer simulations of the lower Illinois River valley from middle Woodland to Mississippian times that *M. tuberculosis* could not account for tuberculosis-like lesions found in the archaeological record for Mississippian times. The application of mathematical models of specific infectious diseases to the archaeological record is clearly an important new development for understanding pre-contact health and disease (see Ramenofsky 1987).

Other presumptive evidence of declining health conditions in the region, prior to European contact, comes from studies of dental caries (Patterson

1984) and of the quality of subadult and adult cortical bone (Pfeiffer and King 1983; Pfeiffer 1986; Saunders and Melbye 1990). If these reconstructions are a reliable reflection of health in the prehistoric and protohistoric periods, then it is tempting to speculate that introduced epidemics of European acute community infections such as measles, smallpox, and whooping cough probably took a more devastating toll in southern Ontario than they did in other areas where health was less compromised, or where living conditions were less favourable for the spread of airborne disease.

Variation in the Experience of Epidemics

Beyond the effects that preexisting infectious disease loads may have had on the impact of introduced pathogens, other avenues of research continue to demonstrate that the impact of virgin soil epidemics in the Americas was not homogeneous, nor was the transmission of infectious agents from community to community inevitable. There must have been significant differences in the biosocial toll of epidemics, as is the case everywhere else in the world, simply because of the immense variety and complexity of the sociocultural and demographic fabric of aboriginal societies (McGrath 1988a; Reff 1991: 181-242). It is also important to acknowledge that microorganisms do not, in and of themselves, cause high mortality during virgin soil epidemics. Rather, high mortality is firmly embedded in the disintegration of daily life which accompanies community-wide sickness (see also Neel 1982a: 48). This is especially true for groups whose subsistence strategies do not include large stores of food or water.

Janet McGrath's work (1991: 412-14) on epidemics listed in the human relations area files suggests that there is a continuum or gradient of social responses to epidemics. The extent to which social disruption occurs, moreover, depends to a great extent on the speed at which the disease spreads, as well as on its mortality rate. The magnitude of disease and death, in turn, are influenced by social responses. Flight from an epidemic, for example, can either break the chain of transmission or spread micro-organisms to other locations. In other words, local historical, social, and demographic circumstances are critical for understanding population responses to epidemics and these are extremely diverse.

This point is illustrated by the significant mortality differences observed in Ojibwa/Cree trading-post communities in the central Canadian subarctic during the 1918 Spanish flu epidemic (Herring 1989, 1990, in press).[7] Analysis of parish and Hudson's Bay Company records revealed that Norway House, the headquarters for the Norway House Fur District, experienced a horrendous influenza mortality rate of 183 deaths per 1,000. That winter, some 18 percent of the population perished during the six-week duration of this virgin soil epidemic. This is six times higher than the estimated 3 percent of the total Canadian Indian population lost to Spanish flu (Graham-Cummings 1967: 149). In contrast, no one died or even got sick at God's Lake or Oxford House, two other posts in the Norway House district. Indeed, aboriginal communities in relatively close contact with the

central source of infection (Winnipeg, Manitoba) suffered more severely than those further removed from it (Herring, in press).

While the intensity of Spanish flu at Norway House remains to be fully explained and obviously depends on a host of interrelated factors, its key position in the fur-trade and transportation network and frequent contact with locations to the west, northwest, northeast and south, made it particularly vulnerable to imported diseases, as Ray noted for the mid-nineteenth century (Ray 1976: 156). This underlines the importance of exchange networks as routes of contagion, channelling the movement of microorganisms and patterning their dispersion across some regions (see Dobyns 1983: 12-13; Reff 1991: 119-24), while bypassing others.

The study also serves to illustrate the danger of generalizing mortality rates from small numbers of communities to derive regional epidemic mortality rates. Overestimates of the death toll can ensue if they are based on worst-case scenarios, that is, communities in close contact with European pathogens and recorders (see also Ferguson 1992). The findings also caution against assuming that post-contact virgin soil epidemics were uniformly calamitous and inescapable; clearly, this was not the case for the 1918-19 influenza pandemic.

It is noteworthy, moreover, that despite the sudden loss of about one-fifth of the adults during the epidemic, the Norway House population rebounded to its preepidemic size within five to ten years of the Spanish flu crisis (Herring 1990; Herring in press). This rapid recovery essentially stemmed from a modest postepidemic marriage boom and from the maintenance of birth rates, both of which helped to blunt the effects of influenza mortality. These results underline the well-known caveat that mortality data alone provide an insufficient basis for estimating depopulation from infectious disease epidemics. Fertility and nuptiality data — as well as information on migration and mobility — must also be examined in detail (Clark 1985; Thornton 1987; Thornton et al. 1991; Mielke and Pitkanen 1989; Sattenspiel and Powell, in press).

The importance of fertility on postepidemic depopulation is well expressed in Stannard's work (1990) on Hawaiian history. Working from information in records, station reports, and other medico-demographic data for the eighteenth and nineteenth centuries, he develops the argument that infertility and a high infant death rate prevented population recovery from mortality from introduced diseases. He further hypothesizes that post-contact declines in Amerindian populations may best be explained by a similar process: infertility and subfecundity following on the heels of the disease-malnutrition-stress-disease cycle. Stannard's research offers an excellent illustration of how historical sequences *outside* of the Americas can provide powerful analogies against which to test assumptions about disease and contact *within* the Americas.

Thornton et al. (1991) have tried to envisage the range of demographic consequences for smallpox epidemics in the Americas via an interesting

series of Monte Carlo simulations. The simulations predict changes in population size over time under different growth rate regimes, after an initial smallpox epidemic and then subsequent epidemics, ten and thirty years later. The program assumes a base population of 5,000, a 100 percent infection rate, 40 percent mortality rate (factoring in higher mortality rates for pregnant women and infants), and a return to preepidemic, age-specific fertility and mortality levels in the year after the epidemic. When post-epidemic growth rates of -.5 percent, 0 percent, 5 percent, and 1 percent were loaded into the simulation, remarkably different consequences emerged for the hypothetical populations. The most dramatic effects emerged after two epidemics, thirty years apart. A population with a growth rate of -.5 percent failed to regain its preepidemic numbers and continued to decline over a 120 year interval; the population with a growth rate of 1 percent rebounded within eighty years. While no empirical data are generated, simulations of this sort are valuable heuristic tools for charting the range of variation in population responses to epidemics, especially since population parameters other than crude mortality are built into the experiments.

Both approaches point up the pitfalls of making generalizations about postepidemic demographic responses without taking into account the substantial recuperative or debilitative potential of fertility, marital, or migration responses. But these are rarely taken into account in arriving at post-epidemic demographic estimates. Neither is population loss through the emergence of new ethnic groups, such as the Métis, figured into the numbers.

Finally, the almost exclusive emphasis on mortality has tended to overshadow other significant contributors to demographic decline (Thornton 1987: 42-59). Warfare, the nature of contact with colonial society, the extent to which the local ecology was transformed through the introduction of new plants and animals, changes in technology, trade, and lifeways, all had epidemiological repercussions (Krech 1983; Crosby 1986; Ferguson 1992). The relocation of aboriginal people to reserves with minimal resources and appalling living conditions, for example, served to catapult mortality rates in the nineteenth and twentieth centuries and to facilitate outbreaks of epidemics. As Walker noted in 1909 (cited in Bryce 1909: 282), increasing tuberculosis mortality rates represented "the whole story of the passing of the Indian from the nomadic to the settled habits of life."

New Directions

I have argued that there is good evidence for pre-contact epidemics in the Americas, that these in turn likely influenced the extent of devastation by introduced pathogens, and that the experience of post-contact epidemics must have been highly variable in aboriginal American societies. The magnitude and severity of introduced diseases undoubtedly depended on a host of local factors, including previous disease experience, proximity to and connections with sources of contagion, and local sanitary-social conditions.

This raises the question of what can be done to provide a more

comprehensive appreciation of variation in epidemiologic and demographic responses to prolonged European contact. One of the first and most obvious research initiatives involves a careful knitting together of the archaeological, physical anthropological, and ethnohistoric evidence in specific regional sequences with a view to evaluating pre-contact disease loads. What may appear superficially to be a lack of attention to the possibility of pre-contact epidemics is, in part, the natural outgrowth of the artificial structure of academic inquiry which parcels human history in the Americas into "historic" and "prehistoric" components. The tendency of researchers to specialize in one or the other periods inadvertently contributes to the impression that disease was not a serious problem before European contact and to the noticeable lack of attempts to connect pre-contact infectious disease experience with differential encounters with post-contact epidemics. Before we can understand the effects of introduced disease, we need to know more about disease loads and disease stress already operating in aboriginal communities before prolonged European and African contact. Research in this direction has already begun (Larocque 1991), but is certainly in its infancy.

Another avenue for widening the scope of our vision would be to look more closely at disease and contact sequences elsewhere, like Stannard's work (1990) in Hawaii, and interpret local sequences in the Americas in terms of these models. The tracking of epidemic cycles, advocated by Thornton et al. (1991), also offers a useful means of assessing the impact of a series of disease experiences in single populations. Recent work along these lines by Decker (1989), based on Hudson's Bay Company records, suggests that depopulation through disease in the central subarctic may not have been as extensive as has been suggested for other parts of the Americas. Monte Carlo simulations (Thornton et al. 1991) and mathematical modelling of a variety of diseases under very different demographic regimes (McGrath 1988b) will also help evaluate the relative impacts of tuberculosis, influenza, measles, whooping cough, and other infections traditionally viewed as agents of depopulation.

In addition, there is a wealth of virtually untapped information in parish records that can provide information on fertility, nuptiality and sometimes even census data necessary to truly assess the demographic consequences of virgin soil epidemics (Herring, in press). Inexpensive, powerful, and readily accessible software, such as Populate (McCaa and Brignoli 1989) can use these data to project the full range of population parameters when only birth and death information is available.

Finally, we must be careful about the "implicit narrative structure ... [the] story we tell about the peoples we study," (Bruner 1986: 139) in the course of piecing together the disease history of the Americas. The depopulation to nadir model, which is central to the disease and contact discourse, can also be interpreted as a romantic story, one that describes a golden past in which the Americas were populated by people with immune systems relatively unchallenged by infectious disease. To accept the assertion that "'germs',

not these imperialists themselves ... were chiefly responsible for sweeping aside the indigenes and opening the Neo-Europes to demographic takeover" (Crosby 1986: 196) is to ignore the massive social upheavals that ultimately underlie the efflourescence of disease in post-contact North America (Trigger 1985). We need to be aware of the stories that underlie our research as we work to create credible scientific imaginings about North American disease history.

NOTES

I am grateful to Peter Ramsden, Shelley Saunders and Colin Varley for their comments on an earlier version of this work, presented in October 1991 at the Canadian Association for Physical Anthropology annual meeting in Hamilton, Ontario. I would also like to thank William O. Autry for introducing me to Populate software. The comments of three anonymous reviewers strengthened the piece immeasurably.

1. Also known as "atypical mycobacteria," these relatives of *Mycobacterium tuberculosis* are saprophytes normally found in watery environments and can produce pulmonary lesions and disease in humans, clinically indistinguishable from that caused by *M. tuberculosis* (Clarke et al. 1987: 48).
2. See Cohen (1989: 33-36) for an extensive list of potential zoonotic infections in prehistoric band societies.
3. Also referred to as macroparasites.
4. Newman (1976: 669) notes that typhus may have been part of American pre-contact disease ecology because the Aztecs had a name for it and depicted its symptoms, as well as on the basis of its generally subclinical manifestation in the contemporary South Peruvian Sierra.
5. At the Uxbridge site, Pfeiffer (1984) identified a minimum of at least eight children and eighteeen adults with clearly distinguishable tuberculosis lesions.
6. For an excellent summary of tuberculosis epidemic wave theory, see Grigg (1958). The theoretical period of a tuberculosis epidemic wave is 300 years and epidemic waves are asymptoptic, showing a strong mortality peak at the beginning of the wave as susceptibles are eliminated from the population. Mortality gradually declines as herd immunity is acquired and as the natural cycle of the wave shifts to endemicity.
7. Parish records, cross-checked against Hudson's Bay Company post journals and other official documents for the period, make it possible to generate comparative mortality rates from the epidemic for a number of subarctic communities (Herring 1990).

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