European infectious disease and depopulation of the Wendat-Tionontate (Huron-Petun)

Gary Warrick

Abstract

The timing and impact of depopulation of the Wendat-Tionontate (Huron-Petun) was examined using historical, epidemiological, archaeological, and bioarchaeological data. Historical and epidemiological research indicates that the documented 1634–40 epidemics that devastated Wendat-Tionontate villages had their origins in the large numbers of European children who emigrated in the 1630s to the New World colonies from the disease-ridden cities of England, Netherlands, and France. There is no evidence in Wendat-Tionontate archaeological settlement remains or large burial populations for any significant outbreak of European disease prior to AD 1634.

Keywords

Iroquoian; epidemic disease; Huron-Petun; seventeenth-century Ontario; depopulation.

Introduction

In AD 1492, the population of North America (i.e. north of the urban civilizations of Central Mexico) was somewhere between 1.89 million (Ubelaker 1992) and 18 million (Dobyns 1983). By 1890, only 546,000 Native North Americans were alive (Ubelaker 1992). Most historians and archaeologists agree that the catastrophic depopulation of Native America was caused by epidemics of European disease but there is disagreement about when this depopulation occurred. Some scholars believe that much of Native America experienced sixteenth-century pandemics of European disease far in advance of face-to-face contact with Europeans and support high pre-European population estimates (Cook 1973; Dobyns 1966, 1983; Crosby 1976, 1986; Ramenofsky 1987; Upham 1992). Others believe that Native American depopulation occurred as a result of local disease epidemics, after first direct contact with Europeans (Milner 1992; Reff 1991; Snow 1995, 1996; Snow and Lanphear 1988; Snow and Starna 1989; Ubelaker 1992). In the

Routledge Taylor & Francis Group

World Archaeology Vol. 35(2): 258–275 *Epidemic and Infectious Disease*

ISSN 0043-8243 print/1470-1375 online
DOI: 10.1080/0043824032000111416

absence of sixteenth-century pandemics, historical first-contact population estimates for Native America are deemed to be an accurate reflection of pre-contact population, resulting in numbers that are one tenth of those cited by proponents of pandemics. Unfortunately, surprisingly few investigators (cf. Ramenofsky 1987; Snow 1995, 1996) have actually used archaeological data to trace the demographic history of a Native group to measure the timing of depopulation and pre-contact population numbers.

The debate over the population of the New World in 1492 is not confined to academia – Native Americans are keenly interested in knowing whether the first Europeans to contact their ancestors found them in an unaltered pre-contact state or as the surviving, disease-scarred remnants of a formerly populous and complex society (Krech 1999: 83–4; Sioui 1992). At present, population estimates for Native America in 1492 are unacceptably contentious. In order to achieve reliable estimates of Native population at the regional or hemispheric level, censuses of individual Native American groups or tribes must be compiled. The best method for estimating Native American population involves meshing data from historical accounts, epidemiology, archaeology, and bioarchaeology. The use of multiple data sets permits one to trace a particular Native population from pre-contact to late historic times and to assess the impact of European contact and disease. This paper examines the timing and demographic impact of the first European disease epidemics to affect the Wendat-Tionontate (Huron-Petun) who occupied southern Ontario, Canada, and demonstrates why archaeological data are crucial to the writing of Native American population history.

The case of the Wendat-Tionontate

The Wendat-Tionontate or Huron-Petun are one of the best-known Native groups in all of North America and provide an ideal case study for examining the timing and impact of European disease epidemics for several reasons. First of all, seventeenth-century documents, written by French visitors to the Wendat-Tionontate country (Samuel de Champlain in the winter of 1615–16 (Biggar 1922–36), Gabriel Sagard in the winter of 1623–4 (Wrong 1939), and various Jesuit priests from 1634 to 1650 (Thwaites 1896–1901)), provide descriptions of daily life, census data and eyewitness accounts of epidemic disease. Second, there is abundant archaeological data for the Wendat-Tionontate, including the partial and complete excavation of dozens of village sites occupied from 1000 to 1650, allowing one to make inferences about population change from settlement size and number (Warrick 1990). Lastly, several large burial populations have been exhumed and analysed, permitting one to trace changes in pathology and vital rates from 1300 to 1650 (Pfeiffer and Fairgrieve 1994).

In the early seventeenth century, the Wendat-Tionontate were one of several confederacies of Iroquoian speakers in north-eastern North America (Fig. 1). Northern Iroquoians occupied the mixed deciduous forests of the St Lawrence Valley-Lower Great Lakes region and numbered around 100,000, distributed in thirty distinct nations (Snow 1992, 1996; Warrick 2000). Prior to 1634, when the first recorded epidemics of European disease struck the Wendat, the combined Wendat-Tionontate population totalled 30,000–35,000, according to seventeenth-century accounts (Biggar 1922–36, 3: 122; Thwaites 1896–1901,

6: 59; 7: 225; 8: 115; 10: 313; Wrong 1939: 91). The Wendat consisted of four nations (Heidenreich 1971: 84–6; Trigger 1976: 30; 1990: 19–20) who occupied eighteen villages. The Tionontate, divided into two nations, occupied seven villages (Biggar 1922–36, 3: 95–101, 122; 4: 278–84, 302; Garrad and Heidenreich 1978; Wrong 1939: 91). The typical village was 1.8 ha in size and contained thirty to forty longhouses surrounded by a double-row palisade (Warrick 1990: 391). Longhouses averaged 20 metres in length (Dodd 1984: 414), sheltering thirty to forty people (Warrick 1990). Fireplaces for cooking and heating were arranged along the centre of a longhouse and each was shared by two nuclear families (Biggar 1922–36, 3: 122–4; Thwaites 1896–1901, 8: 105–7; Wrong 1939: 93–5). The Wendat diet consisted of at least 50 per cent maize, beans, and squash, 25 per cent fish, and 25 per cent wild game (primarily white-tailed deer) and fleshy fruit (Heidenreich 1971: 163–4; Katzenberg 1993). The French remarked on the robust health of the Wendat (Trigger 1990: 13).

Historical population and disease epidemics among the Wendat-Tionontate

The first estimates of Wendat-Tionontate population are 32,000 by Champlain in 1615 (Biggar 1922–36, 3: 122) and 30,000–40,000 by Sagard in 1623–4 (Wrong 1939: 91). Prior to the summer of 1634, the French Jesuits who had lived among the Wendat consistently estimated their population at 30,000 persons and the number of their villages at twenty (Thwaites 1896–1901, 6: 59; 7: 225; 8: 115; 10: 313). There are no recorded estimates of Tionontate population for the early 1630s, but in 1639 they are said to have occupied nine villages (Thwaites 1896–1901, 19: 127). In the spring of 1639 and over the winter of 1639–40, the Jesuits took a house-by-house census of the Wendat-Tionontate:

> we have had means to take the census not only of the villages, large and small, but also of the cabins, the fires, and even very nearly of the persons in all the country, – there being no other way to preach the Gospel in these regions than at each family's hearth, whereof we tried to omit not one. In these five missions there are thirty-two hamlets and straggling villages, which comprise in all about seven hundred cabins, about two thousand fires, and about twelve thousand persons.
>
> (Thwaites 1896–1901, 19: 127)

The total of 12,000 applied to both the Wendat and their neighbours the Tionontate. This census documents the impact of the 1639–40 smallpox epidemic – it counted hearths before the epidemic struck and survivors after the epidemic passed. Two thousand fires should translate into 20,000 people, assuming two families per hearth and five people per family. Instead, only 12,000 people are reported. This implies the loss of 8,000 people over the winter of 1639–40, from smallpox, the last in a series of devastating diseases that visited the Wendat-Tionontate between 1634 and 1640.

The first recorded disease epidemic among the Wendat-Tionontate occurred in the late summer of 1634. A 'sort of measles and an oppression of the stomach' (Thwaites 1896–1901, 7: 221), accompanied by high fever, rash, vision impairment in some cases, and ending in diarrhoea, spread throughout the western Wendat villages and lasted over the winter (Thwaites 1896–1901, 7: 221; 8: 87–9). Mortality rates were approximately 20 per

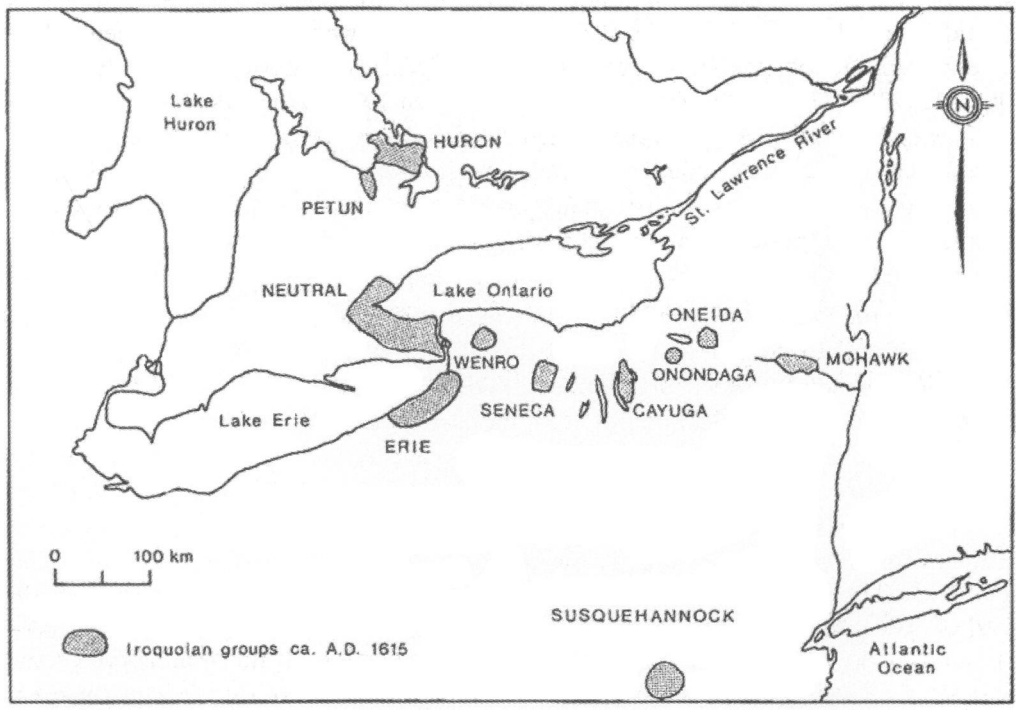

Figure 1 Distribution of Iroquoian groups in north-eastern North America c. AD 1615.

cent (Trigger 1976: 851). If the 1634 epidemic was measles (*Variola minor*) (Dobyns 1983: 17, 322; Trigger 1976: 500–1), mortality rates in a non-immune population are characteristically 10–20 per cent (Ramenofsky 1987: 148). Assuming that only the western half of the Wendat population and some Tionontate were infected (Thwaites 1896–1901, 10: 77), the Wendat-Tionontate may have suffered a 10 per cent depopulation – a loss of about 2,500 people. It is possible that this measles epidemic can be attributed to the arrival of twenty-five French children at Quebec in June of 1634 (Trudel 1973: 184–5). It is generally agreed that Wendat traders were afflicted with measles upon contacting the sick and dying Algonkin and Innu at Trois Rivieres, and possibly Quebec, in early July of 1634 (Trigger 1976: 500–1).

A 'pestilence, of unknown origin' (Thwaites 1896–1901, 11: 13) hit the Wendat in early September of 1636 and persisted until the spring of 1637 (Thwaites 1896–1901, 13: 115, 163–5). This epidemic has been diagnosed as influenza, judging from its symptoms (i.e. bouts of very high fever and cramps) and that it affected both the French and Wendat (Thwaites 1896–1901, 13: 95–101; Trigger 1976: 526–7). However, influenza is communicable for no more than three days (Benenson 1975) and probably would not have survived an Atlantic crossing on board a ship with fewer than 100 passengers. Furthermore, the sickness lasted at least two weeks among the few French residents of Wendat villages, and persisted throughout the winter and flared again in the early spring among the Wendat themselves (Trigger 1976: 526–7), uncharacteristic behaviour for a flu. A more likely diagnosis is strep. infection (e.g. tonsillitis) complicated by bacterial pneumonia, which often produces high fever and stomach cramps due to swollen lymph glands and can result

in 25 per cent mortality in a population untreated with antibiotics (Ramenofsky 1987: 150–2). Strep. infection can have a communicable period of weeks or months, depending on the strain (Benenson 1975), and is especially contagious in crowded dwellings where contact is intimate (e.g. sharing of drinking and eating utensils). It may have been transmitted by the Nipissing, who probably contracted the disease from new French immigrants at Trois Rivieres (Trigger 1976: 521; Trudel 1973: 185). The western Wendat (Attignawantan Nation) lost 500 people, half of these in the large Ossossane village (Trigger 1976: 528). Mortality would have averaged 5–10 per cent (Benenson 1975), reducing Wendat-Tionontate population by another 1300–2500 people. The Nipissing who wintered in the Wendat country lost seventy persons during this epidemic, approximately 10 per cent of their population (Thwaites 1896–1901, 14: 37).

Another epidemic of an unidentified childhood disease (no French contracted it) struck the Wendat in the summer of 1637 and lasted until the autumn of the same year (Trigger 1976: 528). The disease killed its victims quickly, sometimes within two days of onset (Thwaites 1896–1901, 15: 69) but no symptoms are explicitly mentioned. Outbreak of the disease occurred before the Wendat trading season and it probably entered Wendat-Tionontate villages via the Susquehannock, who had suffered an unspecified epidemic in February of 1637 (Trigger 1976: 528). The ultimate origins of the infection were probably from the English colony in Virginia, a fast-growing colony with numerous children, whose population exceeded 4,800 in 1625 and 8,000 in 1640 (Delage 1993: 244). If the illness was scarlet fever (rash form of *Streptococcus pyogenes*), as suggested by Dobyns (1983: 322) and Snow (1992) and supported by the lack of remarks about symptoms by the Jesuits, perhaps implying similar ones to the preceding epidemic of the 1636–7 winter (Trigger 1976: 528), the mortality rate is reportedly higher for this epidemic than the previous one (Trigger 1976: 528), suggesting at least 10 per cent mortality overall, leaving only 23,000 Wendat-Tionontate alive in late 1637.

Smallpox ravaged the already decimated Wendat and Tionontate from the early fall of 1639 until the spring of 1640 (Dobyns 1983: 322; Trigger 1976: 588–9). In 1633, smallpox was transmitted to the natives of coastal New England, reaching as far inland as the Mohawk by December of 1634 (Snow 1992; Snow and Starna 1989). In 1638, a British ship docked at Boston carrying smallpox (Cook 1973). This introduction was the one that inevitably found its way to the Wendat-Tionontate via a group of Kichesipirini returning from Abenaki country (Thwaites 1896–1901, 16: 101; Trigger 1976: 588). Smallpox is directly communicable for two weeks but can live in a dried state in scabs and on clothing for longer periods (Benenson 1975; Ramenofsky 1987: 146). Victims typically die within five to seven days of first symptoms from severe fever and toxaemia (Benenson 1975). The consequences for the Wendat-Tionontate were devastating – a 40–60 per cent mortality rate for most villages has been conservatively estimated, based on documented outbreaks of smallpox in other 'virgin-soil' situations (Heidenreich 1971: 97–8; Ramenofsky 1987: 146–9; Snow 1992). Thus, based on disease mortality rates, the post-smallpox Wendat-Tionontate population would have been somewhere in the vicinity of 10,000–12,000 people, precisely the number documented by Jerome Lalemant in the 1639–40 census (Thwaites 1896–1901, 17: 223; 19: 127).

For the Wendat-Tionontate, the epidemics of European disease ended in the spring of 1640, leaving an estimated 12,000 scarred and battered survivors. In a period of six years,

the Wendat-Tionontate were reduced from 30,000 to 12,000 – a depopulation rate of 60 per cent. Catastrophic depopulation of the Wendat-Tionontate evoked this reaction from Jerome Lalemant in 1642:

> where eight years ago one could see eighty or a hundred cabins, barely five or six can now be seen; a Captain, who then had eight hundred warriors under his command, now has not more than thirty or forty; instead of fleets of three or four hundred canoes, we see now but twenty or thirty.
>
> (Thwaites 1896–1901, 23: 109)

European disease in the Northeast before 1634

Before examining the archaeological record and burial populations of the Wendat-Tionontate for evidence of European disease and depopulation prior to the historically documented epidemics of 1634–40, it is important to investigate the potential for pre-1634 transmission of disease to the Wendat-Tionontate. It is possible that infectious disease reached Iroquoian peoples as early as 1524, as a result of the voyage of Verrazzano, who had direct contact with Native North Americans along the Atlantic coast from North Carolina to Maine (Morison 1971: 289–309). However, Dean Snow has argued (1980: 32–3; Snow and Lanphear 1988) that the average 42–day duration of trans-Atlantic crossings in the sixteenth and early seventeenth century and relatively small crew sizes acted to prevent the transmission of most European crowd contagions to North America. The communicable periods (i.e. incubation period plus period of illness (Burnet and White 1972: 124–5)) of most European diseases are less than 14 days (Benenson 1975; Ramenofsky 1987), except for smallpox (9–21 days and on clothing for months), plague (in fleas for months), whooping cough (14–28 days), strep. infections (months), typhus (28 days including human body louse life cycle), and bacterial pneumonia (months) (Benenson 1975; Ramenofsky 1987). The trans-Atlantic sea voyage acted like a quarantine period for European sailors (Snow 1980: 32–3). Nevertheless, there are several references in sixteenth- and seventeenth-century documents to disease outbreaks among Natives in north-eastern North America.

The first recorded epidemic of European disease in the Northeast occurred in 1535. While overwintering near the village of Stadacona (near present-day Quebec City), Jacques Cartier (Biggar 1924: 204) observed in December of 1535 that over fifty Stadaconans (about 10 per cent of the village population (Trigger 1985: 237)) died from an unknown disease. The mortality rate, epidemic behaviour, and timing of the disease outbreak suggest a European contagion (Trigger 1976: 193–4). Influenza and cold virus have been proposed (Snow and Lanphear 1988; Trigger 1981) but the epidemic was probably bacterial pneumonia. People predisposed to pneumonia (staph. or strep. strains) as a primary infection are those who have chronic lung disease or previous respiratory infection and who live in cold, damp, and crowded dwellings (Benenson 1975; Ramenofsky 1987: 150). Skeletal pathology indicates that tuberculosis was endemic among fifteenth- and sixteenth-century Wendat-Tionontate (Hartney 1981; Pfeiffer 1984) and also present in fifteenth-century St Lawrence Iroquoians (Hartney 1981).

Furthermore, life in an Iroquoian longhouse during the winter was cold and crowded – at night occupants slept close to the central fires, huddled together for warmth (Thwaites 1896–1901, 17: 13; Wrong 1939: 93–4). Coughing, droplet spread, and inhalation are the main routes of infection. The communicable period for bacterial pneumonia varies but the disease can survive in a person's upper respiratory tract for months (Benenson 1975). Primary infections of pneumonia, left untreated by antibiotics, result in 20–25 per cent mortality through high fever and respiratory distress (Ramenofsky 1987: 152).

Archaeology and oral history both indicate that St Lawrence Iroquoians abandoned the St Lawrence Valley by 1580, as a result of warfare among themselves and with other Iroquoian nations (Bradley 1987: 84–7; Engelbrecht 1995; Heidenreich 1990; Jamieson 1990; Kuhn et al. 1993; Pendergast 1993; Ramsden 1990; Sioui 1992: 83–6; Snow 1996; Trigger 1985). The presence of distinctive St Lawrence Iroquoian pottery in Wendat, Onondaga, and Mohawk sites between 1530 and 1580 indicates that at least 800 St Lawrence Iroquoians resettled among the Wendat (Saunders et al. 1992: 120), 600 joined the Onondaga (Bradley 1987), and perhaps a few hundred lived with the Oneida and Mohawk (Snow 1996). The possibility that St Lawrence Iroquoians were depopulated by European disease (Sioui 1992: 41–2), causing regional destabilization, cannot be ruled out.

The Wendat's first contact with the French occurred at Quebec in 1609 (Biggar 1922–36, 2: 67–71). Between 1610 and 1615, approximately 200 Wendat and Algonkians traded with the French on the St Lawrence River. In the spring and early summer of 1611, some of the Ottawa Valley Algonkians were prevented from coming to trade because 'many had died of fever' (Biggar 1922–36, 2: 207). A fever causing relatively high mortality was probably of European origin, perhaps bacterial pneumonia, and perhaps was transmitted to Algonkians via their Innu allies who frequented the Quebec trading post each winter. There is no historical evidence for its spread to the Wendat-Tionontate.

In the winter of 1615–16, Champlain and fourteen men overwintered in the Wendat country (Heidenreich 1971: 238). Although contact was intimate (Biggar 1922–36, 3: 47), there was no apparent disease transmission. French traders occasionally overwintered in the Wendat-Tionontate country (five or six traders in 1622–3; fourteen in 1623–4; ten in 1624–5; eight to ten in 1626–7; twenty-one in 1628–9) with apparently no ill effects on the indigenous population (Trigger 1976: 367–73).

From 1616 to 1622, an epidemic of viral hepatitis swept entire communities away (90 per cent mortality) in southern New England, but did not spread more than 60 kilometres inland from the Atlantic coast (Cook 1973; Snow 1992; Spiess and Spiess 1987), leaving Iroquoians unscathed. Gabriel Sagard recorded that the Weskarini (interior group of Algonkin, north of the Ottawa River) suffered relatively high mortality because of disease and hunger during the winter of 1623–4 (Wrong 1939: 263). It is not known if the disease was a European one. Finally, in 1633, an outbreak of smallpox at an English trading post on the Connecticut River spread quickly to the Mohawk and other Five Nations villages (Snow 1992), presumably through Mahican traders who had lucrative commercial relations with the Dutch and English in both the Connecticut and Hudson River valleys (Brasser 1978). Smallpox did not reach the Wendat-Tionontate in 1633, probably because of hostile relations between the Wendat and Five Nations confederacies.

Archaeology and Wendat-Tionontate depopulation

Ann Ramenofsky (1987) made the first serious attempt to use archaeological settlement data to estimate the sixteenth- and seventeenth-century population for an Iroquoian group, with the explicit goal of determining the timing of depopulation for the Five Nations Iroquois as a result of European disease. Relative estimates of population from roofed area of village sites produced ambiguous results due to biased site samples (only twenty-six sites representing three centuries) and imprecise time periods. In an attempt to improve upon Ramenofsky's (1987) research, two independent archaeological projects were launched in the 1980s to estimate fifteenth- to seventeenth-century population for the Mohawk (Snow 1992, 1995, 1996; Snow and Lanphear 1988; Snow and Starna 1989) and the Wendat-Tionontate (Warrick 1990). Using estimates of 12–20 m^2 of village area per person, Dean Snow (1995) calculated absolute population figures and found that Mohawk population increased steadily from AD 1400 to 1635 (1070–7740 people) but experienced a 63 per cent decrease between 1635 and 1640. When did depopulation occur for the Wendat-Tionontate?

The archaeological record for the Wendat-Tionontate offers several advantages for estimating past population from settlement remains. First, over a century of archaeological investigation has located 460 village sites in south-central Ontario, approximately 61 per cent of all village sites that ever existed and over 80 per cent of all sixteenth- and seventeenth-century sites (Warrick 1990: 157–9: see Table 1). Second, Wendat-Tionontate village sites were occupied for ten to fifty years (Warrick 1988), with minimal reoccupation of the same site. Furthermore, excavation has uncovered partial or complete plans for fifty village sites (Warrick 2000), permitting one to calculate density of hearths (and people) per unit area of site. Lastly, many Wendat-Tionontate sites can be dated to ten-to-thirty-year periods (based on radiocarbon dates and pottery decoration and glass bead chronologies (e.g. Kenyon and Kenyon 1983; Fitzgerald 1990)), corresponding to individual village occupations (Warrick 1988).

Gary Warrick (1990) traced change in Wendat-Tionontate population in absolute numbers from AD 900 to 1650. The methodology used archaeological settlement data from twenty-five partially or completely excavated village sites, 460 known village sites and seventeenth-century descriptions that two families shared each central hearth in a longhouse (Thwaites 1896–1901, 15: 153; Wrong 1939: 94). Central hearths are the most useful indicator of population in Iroquoian village sites. Based on excavated village plans, hearth densities in Wendat-Tionontate settlements were found to be relatively constant over several centuries, averaging fifty hearths per ha of village area. Palaeodemographic analyses of ossuary data from Fairty, Uxbridge, Kleinburg, and Ossossane provide average family sizes of ten to eleven people per central hearth for the Wendat-Tionontate. Applied to archaeological hearth density, this translates into 500–50 people per ha of village area. This matches Snow's (1995, 1996) standard population density figure of 20 m^2 of village space per person for the Mohawk (except between 1550 and 1625 when hearth density rose to seventy hearths per ha among the Wendat-Tionontate (Warrick 1990: 232–5) and 12 square metres of village space per person for the Mohawk (Snow 1995, 1996)). Hearth density was then multiplied by site area and number of people per hearth for 460 confirmed and dated Wendat-Tionontate village sites, representing 700 years of

occupation. The time periods in Table 1 are derived from radiocarbon dates, pottery seriation, and European trade item chronologies (Warrick 1990: 170–90). Adjustments were made to the village totals for each time period to accommodate site durations that were less than the length of the time periods, to avoid double counting people. Coeval village numbers in Table 1 represent the maximum number of villages that were simultaneously occupied in each time period. The cumulative size of these villages was used to calculate the total hearth number (using hearth density per ha of village area), which in turn was multiplied by the period-specific family size.

Table 1 Archaeological estimates of Wendat-Tionontate population

Time period	*No. of confirmed village sites*	*No. of historic villages*	*No. of missing village sites*	*Total no. of coeval village sites*	*Total no. of hearths*	*Population*
AD1647	21	n/a	0	21	1920	11520
AD 1633	35	29[a]	0	35	2940	29400
AD 1623	25	25[b]	0	25	3150	31500
AD 1615	19	25[c]	6	25	2940	29400
AD 1580–1609	25	n/a	6	31	3370	33700
AD 1550–1580	27	n/a	1	28	3020	30200
AD1500–1550	76	n/a	0	37	3330	33300
AD 1450–1500	97	n/a	0	35	2780	27800
AD 1420–1450	45	n/a	0	32	2590	27200

Source: data from Warrick (1990)
Notes
[a] Thwaites (1896–1901, 6: 59; 7: 225; 8: 115; 10: 313; 19: 127).
[b] Wrong (1939: 91).
[c] Biggar 1922–36, 3: 95–101, 122; 4: 278–84, 302).

Population estimates derived from hearth counts were plotted over time for the Wendat-Tionontate (Fig. 2). The resulting graph displays a rapid population increase during the fourteenth century from 11,000 to 29,000 persons, representing a growth rate of 1.07 per cent per annum (Warrick 1990: 353), a levelling off and then cessation in growth by 1475, and then a dramatic decrease in the 1630s (Fig. 2). The important part of the curve, the sixteenth century, shows stability with no significant decrease (except for fluctuations around a mean of 30,000 Wendat-Tionontate from 1500 to 1623 (see Table 1)). This can be explained by a simple 10 per cent error margin in archaeological census taking, not depopulation from a '1520s pandemic' (Dobyns 1983). A similar result has been obtained for the Mohawk. In fact, the Mohawk population was *increasing* throughout the sixteenth century (Snow 1995, 1996). Thus, for at least two relatively dense populations of the Northeast (particularly vulnerable to depopulation from contagious disease as a result of compact longhouse and village life), there is no evidence for early sixteenth-century pandemics and consequent depopulation.

Using archaeology to estimate the depopulation curve for the Wendat-Tionontate is a little more difficult. First of all, there was a high frequency of village abandonments, relocations, and amalgamations between 1635 and 1640. Many villages were abandoned after 1639 because they were no longer demographically or politically viable communities.

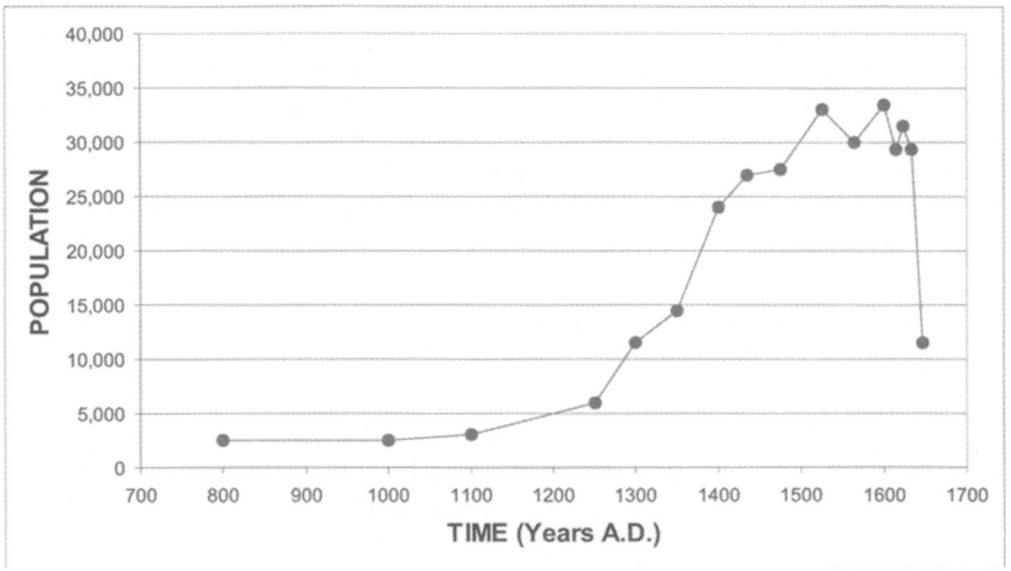

Figure 2 Population growth curve for the Wendat-Tionontate AD 800–1650.

> The remnants of the Wendat found themselves living in villages that were too large for them. Many longhouses were empty or almost empty, since up to half of their inhabitants were dead. In the summer of 1640 this resulted in a decision to relocate the town of Ossossane, although the existing settlement was only five years old (21: 159). The extra labour involved in founding a new, albeit smaller, town so soon after the last move must have been a very heavy burden to the people of Ossossane. It may be assumed that similar, premature moves were made in other parts of the Wendat country.
>
> (Trigger 1976: 602)

Second, the number of people per hearth was no longer ten (i.e. two nuclear families): based on palaeodemographic data from Ossossane and the 1639–40 Jesuit census (i.e. documenting 2,000 hearths and 12,000 people), post-1640 hearth population was only six people (Warrick 1990: 306–9). Using the 1639–40 Jesuit lists of village names and number for the Wendat-Tionontate, matching archaeological sites to villages on the 1640s Jesuit map *Corographie du Pays des Wendats*, and multiplying hearth counts by six people per hearth yields a 1647 Wendat population of 8,600 persons and a Tionontate population of only 2,900 people, for a combined Wendat-Tionontate total of 11,500 (Warrick 1990: 404). One example of the difficulty in providing archaeological estimates of post-epidemic population from settlement remains involves the Tahontaenrat village of Scanonaenrat, occupied between 1635 and 1649. Identified in 1996 with the 5.6 ha Ellery site (Archaeological Services Inc. 1993), Scanonaenrat would have contained over 2,500 people estimated from site size. However, Scanonaenrat must have lost over half of its population during the epidemics of 1634–41. If the Tahontaenrat continued to live in their pre-epidemic village, which it appears that they did, then archaeologically the depopulation would be invisible, unless upon complete excavation the village site revealed an obvious contraction in size (i.e. abandonment of a section of the village, shortened longhouses and a rearranged palisade configuration).

In summary, archaeological estimates of Wendat-Tionontate population indicate stability for 1475–1633 and a 60 per cent depopulation between 1634 and 1640. There is no evidence of sixteenth-century disease epidemics or depopulation.

Bioarchaeology of the Wendat-Tionontate

Skeletal populations provide a unique data set for tracking temporal change in disease and mortality rates, which can identify the introduction of European disease. Beginning in the early fourteenth century, the Wendat-Tionontate began to bury the their dead in communal ossuaries, often containing the disarticulated remains of hundreds of individuals, typically representing the dead which had accumulated over the ten-to-thirty-year lifespan of a village. In 1636, a French Jesuit, Jean de Brebeuf, witnessed and recorded one of these burial episodes near the Wendat village of Ossossane, which he called 'Feast of the Dead' (Thwaites 1896–1901, 10: 275–303). In 1947–8, this ossuary pit was identified and excavated (Kidd 1953; Fitzgerald 1990: 222–6). (It should be noted that, in the summer of 1999, the Wendat people repatriated the bones and grave goods of the Ossossane ossuary and reburied them with appropriate ceremony in the original pit. Other ossuaries are being considered for repatriation.) Subsequent excavation of other Wendat-Tionontate ossuaries has amassed large skeletal populations, dating from 1300–1640. Three ossuaries characterize fifteenth- (pre-Columbian), sixteenth- (indirect European contact), and seventeenth-century (direct European contact) Wendat-Tionontate population: Uxbridge (1460–90), Kleinburg (1580–1600), and Ossossane (1624–36) (Pfeiffer and Fairgrieve 1994). Is there evidence in any of these populations for European disease and depopulation?

Skeletal analyses of the Uxbridge population (dating 1460–90 and containing 457 individuals (Pfeiffer and Fairgrieve 1994)) have revealed a rather unhealthy picture of late fifteenth-century Wendat life: chronic protein-calorie malnutrition, a very high incidence of tuberculosis (at least 4 per cent skeletal involvement (Pfeiffer 1986)), 21 per cent dental caries and a number of deaths and injuries caused by interpersonal violence (i.e. tribal warfare) (Pfeiffer 1984, 1986; Pfeiffer and King 1983; Pfeiffer et al. 1986). Despite a high morbidity, however, the Uxbridge population had a lower juvenile mortality (70 per cent survival to 15 years of age) and a slightly higher adult life expectancy (life expectancy at birth of 25 years) than the fourteenth-century Fairty population (Warrick 1990: 306). Palaeodemography indicates a stable population ($r = 0.0$) (Jackes 1986; Warrick 1990: 306), confirming archaeological estimates of population stability for the Wendat-Tionontate by 1475 (Warrick 1990).

Bioarchaeological analysis of the Kleinburg ossuary (dating 1580–1600 (Kenyon and Kenyon 1983) and containing 561 individuals (Pfeiffer and Fairgrieve 1994)) reveals a life expectancy at birth of 25 years, a low juvenile mortality rate (70 per cent survival to 15 years), but a low fertility rate (Jackes 1986; Pfeiffer 1983). An extremely high rate of dental caries (41 per cent (Patterson 1984)) is substantiated by palaeodietary analyses suggesting a diet composed of at least 50 per cent maize (Katzenberg and Schwarz 1986). Palaeopathology of Kleinburg indicates a lower incidence of tuberculosis, lower rate of bone infection and higher rate of growth arrest lines than either the Fairty or Uxbridge

populations but otherwise the relative health of the Kleinburg people was comparable to that of earlier Wendat-Tionontate (Pfeiffer and Fairgrieve 1994; Pfeiffer and King 1983). There is absolutely no evidence for European epidemics in the Kleinburg mortality rates (Sullivan 1997). Palaeodemography indicates a stable population ($r = -0.002$) (Jackes 1986; Warrick 1990: 306), confirmed by archaeological population estimates (Warrick 1990).

The mortality profile of the Ossossane ossuary (dating to 1636 and containing 419 individuals (Pfeiffer and Fairgrieve 1994)), indicating elevated adolescent deaths (40 per cent survival to 15 years), suggests impact from an epidemic disease (Jackes 1986; Saunders and Melbye 1990; Sullivan 1997). Infants (0–2 years) and the old (>40 years) are selectively killed by acute crowd infections, although adolescents and young adults (15–30 years) can experience high mortality rates from smallpox, measles, mumps, and chickenpox because of over-reactive immune responses (Burnet and White 1972: 97–9). Ontario Iroquoian skeletal populations that were buried during the 1630s epidemics, at Ossossane and in the Neutral cemetery at Grimsby, reveal extremely high juvenile mortality rates for populations that we know were not growing, based on archaeological evidence (Jackes 1986). Palaeopathological data suggest a population in comparable health to that of Fairty, Uxbridge, and Kleinburg (Pfeiffer and Fairgrieve 1994). Based on the historical identification of this ossuary, the dead would include victims of the 1634 measles epidemic. It is interesting that no signs of European disease were identified in the bones of the Ossossane people. Infectious diseases of European origin tend not to leave signatures in skeletal populations – victims die before bony lesions have a chance to form, and, for survivors, lesions produced by European diseases, such as smallpox and measles, if produced at all, tend to be non-specific (Ortner 1992). One possible exception is the discovery of lesions attributed to smallpox in the bones of an adult male buried in a Neutral Iroquoian cemetery, in Grimsby, southern Ontario, dating 1640–50 (Jackes 1983). It is conceivable that this man was infected with smallpox as early as 1633–4 (Jackes 1983: 80). Glass bead chronology, in association with inferred season of death and distribution of multiple graves at the Grimbsy cemetery, suggests that European epidemics did not affect the Neutral until the mid-1630s (Fitzgerald 1990: 226–39).

In summary, palaeodemography and palaeopathology of Wendat-Tionontate skeletal populations between 1400 and 1650 document consistently moderate rates of caries, malnutrition, and endemic disease (e.g. tuberculosis), but no evidence of European disease prior to the first recorded epidemic of 1634.

Discussion: Iroquoians and European disease in the sixteenth and seventeenth century

Why were the Wendat-Tionontate and other Iroquoian groups living in the interior of the Northeast not infected with European disease prior to 1633? Factors that prevented the pandemic spread of European disease across sixteenth- and early seventeenth-century North America include long trans-Atlantic voyages, lack of European children in the first colonies of the Northeast, infrequent land-based contacts with Natives prior to colonization, low population densities of Native societies in northeastern North America, and physical and socio-political buffer zones between Native groups (preventing localized

disease outbreak from becoming epidemic or pandemic) (Carlson et al. 1992; Milner 1992; Snow 1992; Snow and Lanphear 1988; Thornton et al. 1992).

In the 1630s, shiploads of European colonists began arriving along the Atlantic seaboard. Infected children of these first colonies are believed to have been responsible for initiating a continuing series of disease epidemics among interior Native groups of the Northeast (Snow and Lanphear 1988). In seventeenth-century Europe, most acute crowd infections were childhood illnesses. Typically, most children would have been exposed to measles, smallpox, whooping cough, and other contagions by 5 years of age (Burnet and White 1972: 95). In 1629, there were only 117 residents of New France and about 500 in New England and 300 in New Netherland (Delage 1993: 243, 258; Trudel 1973: 165). Between 1630 and 1640, 700 colonists settled in New Netherland and 13,400 in New England (Delage 1993: 243). In contrast, New France added only 120 colonists in the 1630s (Delage 1993: 243). This suggests that most of the European diseases that inflicted such devastation on the Wendat-Tionontate probably originated from newly arrived Dutch or English children after 1630.

Early seventeenth-century France, England, and the Netherlands were unhealthy places to live, especially in the towns and cities. France's population was stagnant (Grigg 1980: 55–7), suffering famine in 1630 and disease epidemics in 1625, 1637, and 1638 (Delage 1993: 257–8). The rise of urbanization in Western Europe in the early 1600s (10 per cent of France's population, 15 per cent of England's, and 50 per cent of Holland's were in towns of over 5–10,000 people (Grigg 1980: 95, 110, 156)) elevated the rates of epidemic disease and mortality (Wrigley 1969: 96–7), as reflected in a decline in both life expectancy at birth and total fertility rate from 1600 to 1650 (Livi-Bacci 1992: 84). Between 1612 and 1664, plague attacked the citizens of Amsterdam on nine separate occasions (Grigg 1980: 160) and attacked Paris on five occasions (Delage 1993: 258). Unfortunately for seventeenth-century Native America, the cities and towns of Western Europe produced the bulk of North American colonists. In fact, overseas emigrants from south-eastern England in the early 1630s were predominantly (up to 80 per cent) urban artisans and their families (Grigg 1980: 98). Similarly, 40–60 per cent of French emigrants in the 1630s came from urban areas (Charbonneau and Robert 1987). The generally poor state of health of Europe's town and city dwellers in the early 1600s, the dramatic rise in colonization of North America between 1625 and 1640, and the predominantly urban origin of the first colonists of New France, New England, and New Netherland explain why the Wendat-Tionontate and their neighbours were decimated by European disease after and not before 1630.

The first encounters of Native Americans and European disease most often took the form of locally severe outbreaks. In north-eastern North America, Native population densities were relatively low and most Native nations lived in clusters of compact villages separated from other nations by hundreds of square kilometres of forest, i.e. deer hunting territories. Although all Wendat communities were within one or two days' walk of one another, the Wendat-Tionontate homeland was 21–8 days by canoe from Montreal and more than 40 days overland during the winter (i.e. 600 kilometres). A network of trails connected the Wendat-Tionontate to the Neutral (4-to-5-day walk or 120 kilometres) and beyond to their Susquehannock allies (at least 30 days overland or 800 kilometres). The Wendat-Tionontate were hostile with most of the Five Nations Iroquois. In turn, the Five

Nations fought with the Algonkian groups to the north and east (Mahican) and with the Susquenhannock to the south (Heidenreich 1990; Trigger 1985). This essentially isolated the Five Nations from European contact in the sixteenth century and interrupted the human chain of contact that could have carried European disease inland from the Atlantic Coast.

The demographic impact of European epidemics on the Wendat-Tionontate, causing close to 60 per cent depopulation, is congruent with other 'virgin-soil epidemics' (Crosby 1976). Aboriginal groups along the Northeast coast of North America were decimated in the early 1600s by European diseases (Carlson et al. 1992; Cook 1973; Snow 1980: 32–5; Snow and Lanphear 1988). Depopulation rates for New England Natives of the early seventeenth century range from 67 to 95 per cent (Snow and Lanphear 1988: 24). Smallpox was the most virulent, with 50–90 per cent mortality rates being recorded for virgin-soil epidemics of this disease (Johnston 1987: 20). The high residential density of Iroquoian villages and communal longhouse life would have hastened the spread of disease and death from secondary infections (e.g. pneumonia) (Burnet and White 1972: 16–17; Crosby 1976: 293–7). The deep spiritual concern for sick relatives, longhouse living conditions, and lack of quarantine are highlighted in an account of Wendat behaviour during the 1639–40 smallpox epidemic by Jesuit Jerome Lalemant:

> for the Wendats – no matter what plague or contagion they may have – live in the midst of their sick, in the same indifference, and community of all things, as if they were in perfect health. In fact, in a few days, almost all those in the cabin of the deceased found themselves infected; then the evil spread from house to house, from village to village, and finally became scattered throughout the country.
>
> (Thwaites 1896–1901, 19: 89)

Conclusion

In conclusion, it is possible to identify the timing and demographic impact of the introduction of European disease on a Native American group, using historical, epidemiological, archaeological, and bioarchaeological data. Archaeological estimates of pre-Columbian Wendat-Tionontate population appear to match estimates provided in the early seventeenth-century documents – 30,000 people. Furthermore, there is no archaeological evidence for depopulation of the Wendat-Tionontate prior to the recorded disease epidemics of the 1630s. It is interesting that Dean Snow (1995) made a similar finding for the Mohawk. If the Wendat-Tionontate and Mohawk cases are representative of the Native population of north-eastern North America, we must conclude that there was no sixteenth-century pandemic of European disease in the Northeast. In fact, contrary to the claims of Henry Dobyns (1983) and his supporters (Crosby 1986; Upham 1992), the only region in North America which contains any archaeological evidence for significant depopulation in the sixteenth century is the south-eastern US (Ramenofsky 1987; Smith 1987). Thus, for most regions of North America, the first European observations of Native American population numbers are probably an accurate reflection of pre-contact numbers. This implies that the Native North American population in 1492 was probably

around two million, a figure derived from European first-contact population estimates (Ubelaker 1992).

Wilfrid Laurier University, Brantford, Ontario

References

Archaeological Services Inc. 1993. An archaeological assessment of proposed pit application (Varcoe Pit No. 2) part of Lot 72, Concession 2, WPR, Township of Flos, Simcoe County, Ontario. Unpublished report on file Ontario Ministry of Culture, Toronto.

Benenson, A. S. (ed.). 1975. *Control of Communicable Diseases in Man*. Washington, DC: American Public Health Association.

Biggar, H. P. 1922–36. *The Works of Samuel de Champlain*, 6 vols. Toronto: The Champlain Society.

Biggar, H. P. 1924. *The Voyages of Jacques Cartier: Published from the Originals with Translations, Notes and Appendices*, Publications of the Public Archives of Canada 11. Ottawa.

Bradley, J. 1987. *Evolution of the Onondaga Iroquois: Accommodating Change, 1500–1655*. Syracuse, NY: Syracuse University Press.

Brasser, T. J. 1978. Mahican. In *Handbook of North American Indians* (ed. W. G. Sturtevant), Vol. 15, *Northeast* (ed. B. G. Trigger). Washington, DC: Smithsonian Institution, pp. 198–212.

Burnet, M. and White, D. O. 1972. *Natural History of Infectious Disease*. Cambridge: Cambridge University Press.

Carlson, C. C., Armelagos, G. J. and Magennis, A. L. 1992. Impact of disease on the precontact and early historic populations of New England and the Maritimes. In *Disease and Demography in the Americas* (eds J. W. Verano and D. H. Ubelaker). Washington, DC: Smithsonian Institution Press, pp. 141–54.

Charbonneau, H. and Robert, N. 1987. The French origins of the Canadian population, 1608–1759 (Plate 45). In *Historical Atlas of Canada*, Vol. 1 (ed. R. C. Harris). Toronto: University of Toronto Press.

Cook, S. F. 1973. The significance of disease in the extinction of the New England Indians. *Human Biology*, 45: 485–508.

Crosby, A. W. 1976. Virgin soil epidemics as a factor in the aboriginal depopulation in America. *William and Mary Quarterly*, 33: 289–99.

Crosby, A. W. 1986. *Ecological Imperialism: The Biological Expansion of Europe, 900–1900*. Cambridge: Cambridge University Press.

Delage, D. 1993. *Bitter Feast: Amerindians and Europeans in Northeastern North America, 1600–64* (trans. J. Brierly). Vancouver: UBC Press.

Dobyns, H. F. 1966. Estimating aboriginal American population: an appraisal of techniques with a new hemispheric estimate. *Current Anthropology*, 7: 395–444.

Dobyns, H. F. 1983. *Their Number Become Thinned: Native American Population Dynamics in Eastern North America*. Knoxville: University of Tennessee Press.

Dodd, C. F. 1984. *Ontario Iroquois Tradition Longhouses*, Archaeological Survey of Canada Paper, No. 124, National Museum of Man Mercury Series, pp. 181–437.

Engelbrecht, W. 1995. The case of the disappearing Iroquoians: early contact period superpower politics. *Northeast Anthropology*, 50: 35–59.

Fitzgerald, W. R. 1990. Chronology to culture process: Lower Great Lakes archaeology, 1500–1650. Doctoral dissertation. Department of Anthropology, McGill University, Montreal.

Heidenreich, C. E. 1971. *Huronia: A History and Geography of the Huron Indians, 1600–1650*. Toronto: McClelland & Stewart.

Garrad, C. and Heidenreich, C. E. 1978. Khionontateronon (Petun). In *Handbook of North American Indians* (ed. W. G. Sturtevant), Vol. 15, *Northeast* (ed. B. G. Trigger). Washington, DC: Smithsonian Institution, pp. 394–7.

Grigg, D. 1980. *Population Growth and Agrarian Change: An Historical Perspective*. Cambridge: Cambridge University Press.

Hartney, P. C. 1981. Tuberculous lesions in a prehistoric population sample from southern Ontario. In *Prehistoric Tuberculosis in the Americas* (ed. J. E. Buikstra). Northwestern University Archaeological Program Scientific Papers 5. Evanston, IL: Northwestern University, pp. 141–60.

Heidenreich, C. E. 1990. History of the St Lawrence-Great Lakes area to A.D. 1650. In *The Archaeology of Southern Ontario to A.D. 1650* (eds C. J. Ellis and N. Ferris). Occasional Publications of the London Chapter, Ontario Archaeological Society, No. 5, pp. 475–92.

Jackes, M. 1983. Osteological evidence for smallpox: a possible case from seventeenth century Ontario. *American Journal of Physical Anthropology*, 60: 75–81.

Jackes, M. 1986. The mortality of Ontario archaeological populations. *Canadian Journal of Anthropology*, 5(2): 33–48.

Jamieson, J. B. 1990. The archaeology of the St Lawrence Iroquoians. In *The Archaeology of Southern Ontario to A.D. 1650,* (eds C. J. Ellis and N. Ferris). Occasional Publications of the London Chapter, Ontario Archaeological Society, No. 5. London: Ontario Archaeological Society, pp. 385–404.

Johnston, S. 1987. Epidemics: the forgotten factor in seventeenth century Native warfare in the St Lawrence region. In *Native People, Native Lands* (ed. B. Cox) Ottawa: Carleton University Press, pp. 14–31.

Katzenberg, M. A. 1993. Applications of elemental and isotopic analysis to problems in Ontario prehistory. In *Investigations of Ancient Human Tissue (*ed. M. K. Sandford). New York: Gordon & Breach, pp. 335–60.

Katzenberg, M. A. and Schwarz, H. P. 1986. Paleonutrition in Southern Ontario: evidence from strontium and stable isotopes. *Canadian Review of Physical Anthropology,* 5(2): 15–21.

Kenyon, I. and Kenyon, T. 1983. Comments on 17th century glass beads from Ontario. In *Proceedings of the 1982 Glass Trade Bead Conference* (ed. C. F. Hayes, III). Rochester: Rochester Museum and Science Center, pp. 59–74.

Kidd, K. E. 1953. The excavation and identification of a Huron ossuary. *American Antiquity*, 18: 359–79.

Kuhn, R. D., Funk, R. E. and Pendergast, J. F. 1993. The evidence for a Saint Lawrence Iroquoian presence on sixteenth-century Mohawk sites. *Man in the Northeast*, 45: 77–86.

Krech, S. 1999. *The Ecological Indian: Myth and History*. New York: Norton.

Livi-Bacci, M. 1992. *A Concise History of World Population* (trans. C. Ipsen). Cambridge, MA: Blackwell.

Milner, G. R. 1992. Disease and sociopolitical systems in late prehistoric Illinois. In *Disease and Demography in the America* (eds J. W. Verano and D. H. Ubelaker). Washington, DC: Smithsonian Institution, pp. 103–16.

Morison, S. E. 1971. *The European Discovery of America: The Northern Voyages, A.D. 500–1600*. New York: Oxford University Press.

Ortner, D. J. 1992. Skeletal palaeopathology: probabilities, possibilities, and impossibilities. In *Disease and Demography in the America* (eds J. W. Verano and D. H. Ubelaker). Washington, DC: Smithsonian Institution, pp. 5–14.

Patterson, D. K., Jr. 1984. *A Diachronic Study of Dental Palaeopathology and Attritional Status of Prehistoric Ontario Pre-Iroquois and Iroquois Populations.* National Museum of Man, Mercury Series, Archaeological Survey of Canada Paper 122. Ottawa.

Pendergast, J. F. 1993. More on when and why the St Lawrence Iroquoians disappeared. In *Essays in St Lawrence Iroquoian Archaeology* (eds J. F. Pendergast and C. Chapdelaine). Occasional Papers in Northeastern Archaeology, No. 8. Copetown: Copetown Press, pp. 9–47.

Pfeiffer, S. 1984. Paleopathology in an Iroquoian ossuary, with special reference to tuberculosis. *American Journal of Physical Anthropology*, 65: 181–9.

Pfeiffer, S. 1986. Morbidity and mortality in the Uxbridge ossuary. *Canadian Journal of Anthropology*, 5(2): 23–32.

Pfeiffer, S. and Fairgrieve, S. I. 1994. Evidence from ossuaries: the effect of contact on the health of Iroquoians. In *In the Wake of Contact: Biological Responses to Conquest* (eds C. S. Larsen and G. R. Milner). New York: Wiley-Liss, pp. 47–61.

Pfeiffer, S. and King, P. 1983. Cortical bone formation and diet among protohistoric Iroquoians. *American Journal of Physical Anthropology*, 60: 23–8.

Pfeiffer, S., Stewart, K. and Alex, C. 1986. Growth arrest lines among Uxbridge ossuary juveniles. *Ontario Archaeology*, 46: 27–32.

Ramenofsky, A. F. 1987. *Vectors of Death: The Archaeology of European Contact*. Albuquerque: University of New Mexico Press.

Ramsden, P. G. 1990. Saint Lawrence Iroquoians in the upper Trent River valley. *Man in the Northeast*, 39: 87–95.

Reff, D. T. 1991. *Disease, Depopulation and Culture Change in Northwestern New Spain, 1520–1764.* Salt Lake City: University of Utah Press.

Saunders, S. R. and Melbye, F. J. 1990. Subadult mortality and skeletal indicators of health in Late Woodland Ontario Iroquois. *Canadian Journal of Archaeology*, 14: 61–74.

Saunders, S. R., Ramsden, P. G. and Herring, D. A. 1992. Transformation and disease: precontact Ontario Iroquoians. In *Disease and Demography in the America* (eds J. W. Verano and D. H. Ubelaker). Washington, DC: Smithsonian Institution, pp. 117–26.

Sioui, G. 1992. *For an Amerindian Autohistory.* Montreal: McGill-Queen's University Press.

Smith, M. T. 1987. *Archaeology of Aboriginal Culture Change in the Interior Southeast: Depopulation during the Early Historic Period*. Gainesville: University of Florida Press.

Snow, D. R. 1980. *The Archaeology of New England*. New York: Academic Press.

Snow, D. R. 1992. Disease and population decline in the Northeast. In *Disease and Demography in the Americas* (eds J. W. Verano and D. H. Ubelaker). Washington, DC: Smithsonian Institution, pp. 177–86.

Snow, D. R. 1995. Microchronology and demographic evidence relating to the size of pre-Columbian North American Indian populations. *Science*, 268: 1601–4.

Snow, D. R. 1996. Mohawk demography and the effects of exogenous epidemics on American Indian population. *Journal of Anthropological Archaeology*, 15: 160–82.

Snow, D. R. and Lanphear, K. M. 1988. European contact and Indian depopulation in the Northeast: the timing of the first epidemics. *Ethnohistory*, 35: 15–33.

Snow, D. R. and Starna, W. A. 1989. Sixteenth-century depopulation: a view from the Mohawk Valley. *American Anthropologist*, 91: 142–9.

Spiess, A. E. and Spiess, B. D. 1987. New England pandemic of 1616–1622: cause and archaeological implication. *Man in the Northeast*, 34: 71–83.

Sullivan, N. C. 1997. Contact period Huron demography. In *Integrating Archaeological Demography: Multidisciplinary Approaches to Prehistoric Population* (ed. R. R. Paine), Center for Archaeological Investigations, Southern Illinois University at Carbondale Occasional Paper No. 24. Carbondale: Southern Illinois University, pp. 327–42.

Thornton, R., Warren, J. and Miller, T. 1992. Depopulation in the Southeast after 1492. In *Disease and Demography in the Americas* (eds J. W. Verano and D. H. Ubelaker). Washington, DC: Smithsonian Institution Press, pp. 187–96.

Thwaites, R. G. (ed.) 1896–1901. *The Jesuit Relations and Allied Documents*, 73 vols. Cleveland: Burrows Brothers.

Trigger, B. G. 1976. *The Children of Aataentsic: A History of the Huron People to 1660*, 2 vols. Montreal: McGill-Queen's University Press.

Trigger, B. G. 1981. Ontario Native people and the epidemics of 1634–1640. In *Indians, Animals, and the Fur Trade: A Critique of 'Keepers of the Game'* (ed. S. Krech III). Athens: University of Georgia Press, pp. 19–38.

Trigger, B. G. 1985. *Natives and Newcomers: Canada's 'Heroic Age' Reconsidered*. Montreal: McGill-Queen's University Press.

Trigger, B. G. 1990. *The Huron: Farmers of the North*. Toronto: Holt, Rinehart & Winston.

Trudel, M. 1973. *The Beginnings of New France 1524–1663*. Toronto: McClelland & Stewart.

Ubelaker, D. H. 1992. North American Indian population size: changing perspectives. In *Disease and Demography in the Americas* (eds J. W. Verano and D. H. Ubelaker). Washington, DC: Smithsonian Institution, pp. 169–76.

Upham, S. (1992). Population and Spanish contact in the Southwest. In *Disease and Demography in the Americas* (eds J. W. Verano and D. H. Ubelaker). Washington, DC: Smithsonian Institution Press, pp. 223–36.

Warrick, G. 1988. Estimating Ontario Iroquoian village duration. *Man in the Northeast*, 36: 21–60.

Warrick, G. 1990. A population history of the Huron-Petun, A.D. 900–1650. Doctoral dissertation. Department of Anthropology, McGill University, Montreal.

Warrick, G. 2000. The precontact Iroquoian occupation of southern Ontario. *Journal of World Prehistory*, 14: 415–66.

Wrigley, E. A. 1969. *Population and History*. Toronto: McGraw-Hill.

Wrong, G. M. (ed.) 1939. *The Long Journey to the Country of the Hurons*. Toronto: The Champlain Society.