New World Paleoepidemiology:

Epidemic disease in the Southeast

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Much postulation, research and debate have centered upon paleodemography and disease in the New World. Many explanations of the depopulation of the New World following European contact focus heavily, if not exclusively, on epidemic disease as the driving force. Other models, however, incorporate the effects of epidemic disease with factors of regional variability to construct models of depopulation. The estimation of native population counts prior to contact varies greatly in both method and technique. It is, then, no surprise that the numeric results vary greatly as well. The general consensus, however, is that Native American populations certainly experienced a population decline after European contact. Evidence of epidemic disease can be difficult to ascertain because it leaves no skeletal marker. Both archaeological and osteological methods are used to identify the impact of colonialism in the New World. In this paper, I will review the nature, spread, and evidence of epidemic disease in the New World, citing examples from the Southeastern United States.

Virgin Soil Epidemics

Explanations of the impact of Old World disease on the New World began to emerge during the contact period. The earliest explanations for this impact were assumed to be the result of innate differences between Old and New World populations (Crosby 1976). Crosby (1976:292) notes a similarity in a more recent hypothesis, which he calls the “genetic weakness hypothesis,” that cites genetic differences between the two populations as an explanation for the widespread and virulent effects of Old World disease on New World populations.

Another explanation, however, considers the effects of disease on previously unexposed populations. Diseases that spread to new areas in this manner are called “virgin soil epidemics” (Crosby 1976:289). Crosby (1976) defines a virgin soil epidemic as a disease that affects previously unexposed populations with no immunological defense to the newly introduced
disease. According to Crosby (1976), virgin soil epidemics were the primary factors in the Native American depopulation of the New World. Crosby (1976) cites two primary reasons for the high death toll of virgin soil epidemics, which are the individual characteristics of each disease and the reaction to such diseases by a previously unaffected population.

It is appropriate to note the various types of virgin soil epidemics that decimated Native American populations in North America. Early and more recent (Dobyns 1983) portraits of prehistoric Native Americans frequently idealized health and lifeways, assuming little to no prevalence of disease prior to contact. Research indicates, however, that prior to European contact the New World experienced a number of infectious diseases, namely tuberculosis and treponemal infections (Bogdan and Weaver, 1992; Braun et al., 1998). The diseases introduced by European contact include smallpox, measles, plague, typhus, diphtheria, chicken pox, malaria, cholera, yellow fever, dengue fever, trachoma, whooping cough, encephalitis, scarlet fever, helminthic infections and amebic dysentery (Crosby 1967; Merbs 1992). The origins of other diseases such as poliomyelitis are unclear (Merbs 1992). Many of the European introduced diseases have been identified through historic documentation. For example, Crosby notes (1967) that Spanish mission documents describe instances of “viruelas,” which are actually the pustules commonly resulting from smallpox. As Crosby (1967) points out, while helpful, historic accounts may inaccurately represent the frequency and type of disease within a region. Due to an author’s lack of precise medical knowledge, a historical document may incorrectly indicate disease type and symptoms. Also, personal factors such as the wish to be looked upon favorably by others may have lead an author of a historical document to either inflate or decease the impact of disease among a population (Crosby 1967).
Population Estimates

Estimates of native population size prior to contact exhibit a high degree of variability. This is not surprising, as estimating paleodemography is no easy task. However, a discussion of recent native population estimates is necessary to evaluate the deleterious effects of Old World disease on New World populations.

Currently, estimates of North American population prior to 1492 range from one to eighteen million (Daniels 1992; Dobyns 1983). Daniels (1992) provides an extensive compilation of population estimates. He categorizes three paradigms of population estimation, which are “bottom up,” “area modeling,” and “top down” (Daniels 1992:310). According to these models, the “bottom up” classification renders estimates of 500,000 to 2,500,000, the “area modeling” classification renders estimates of 2,500,000 to 7,000,000 and “top down” classification renders estimates form 7,000,000 to 18,000,000 (Daniels 1992: 310). According to Daniels (1992) the “bottom up” model of population estimation was most frequently used during the first half of the 20th century. Mooney, among others, relied almost exclusively on historical population accounts for these estimations (Daniels 1992). Between the 1950’s and 1980’s “area modeling” became the dominant model for population estimation (Daniels 1992). Unlike the “bottom up” model, “area modeling” also utilizes indirect lines of evidence and inference (Daniels 1992). The “top down” model emerged during the 1980’s and augmented “area modeling” by estimating population size using depopulation ratios and more lenient models of carrying capacity (Daniels 1992).

Within the Southeast, Ubelaker (1992) estimates native population size prior to European contact at 204,400, citing other minimum and maximum estimates for this region at 155,800 and 286,000 respectively. Additionally, Ubelaker (1992) estimates a population decrease of 47,000
within the Southeast between 1500 and 1600. However, Ubelaker does not clearly define the
Southeast for the reader. It should be noted, however, that Ubelaker’s estimates are based on a
total population estimate of slightly less than two million individuals within North America and
north of Mexico. Compared with the other estimates listed above, this is fairly conservative.

Thornton (1997) calculated the annual rates of population decline in North America
based on several well-known estimates of Mooney, Ubelaker, Thornton and Dobyns (see table
1). These rates were calculated based assumptions regarding nadir population of 375,000 in
1900, population stability and an exponential rate of decline (Thornton 1997). Based on this, the
difference between the annual rates of decline for the most conservative population estimate
(1,153,000; -0.28%) and the highest population estimate (18,000,000; -0.97%) is 0.69 %.
According to these rates of decline, a moderate population estimate of 7,000,000 would require
an annual decline rate of –0.73 % (Thornton 1997). The author notes the potential problems and
inaccuracies associated with such assumptions, but assumes an equilibrium of rates through time
and across populations (Thornton 1997). However, as evidenced below with the study cases,
none of these assumptions is accurate in regards to Southeastern depopulation. Despite the
shortcomings associated with analyses based on annual rates of population decline, they do
illustrate that over time changes as small as one percent or less annually can result in a large
demographic change within a population (Thornton 1997). However, because the three
assumptions used to calculate these rates are not valid for the Southeast, the rates of population
decline for this region may not be valid as well.

Models of Disease Spread

The rate of epidemic disease spread may generally be grouped into two paradigms, those
that indicate a rapid, widespread, and uniform rate of disease dispersal and those that indicate a
varied rate of dispersal based upon cultural, geographic and temporal factors. A number of estimates (Dobyns 1983, Ramenofsky 1987) view outbreaks of epidemic disease as uniformly and simultaneously affecting a large majority of the contiguous United States. According to Dobyns (1983), New World depopulation occurred prior to historic documentation during the sixteenth century and spread out of a Mexican epidemic. Other researchers, such as Larsen (1994), view the effects of epidemic disease as a “patchwork.” Larsen (1994) notes that while a commonality among Native American response to disease can be seen, such responses were also influenced by factors unique to each tribal group. These factors include settlement size, relocation, population density, trade, diet, slavery, and physiological stress (Larsen 1994; Ward and Davis 1999). Of these approaches, the latter is more realistic in its consideration of the variability of native groups both before and after contact.

Modes of Study

The primary modes of research regarding epidemic disease may be categorized as being either archaeological or osteological. Historic documentation is also a useful tool in determining the impact of epidemic disease on past populations (Crosby 1967). Used alone, however, it may be largely insufficient or inaccurate. Milner (1980) discusses the scarcity of historical accounts from the Southeast relating to epidemic disease prior to 1700 and the resulting need for archaeological analysis to fill this void. Because historical documents have the potential to skew population counts and misrepresent disease type, other modes of study may be necessary. Archaeological methods for the study of epidemic disease include settlement pattern analysis and mortuary analysis. Osteological methods are utilized to evaluate various forms of stress within a population.
A number of pitfalls await each of these modes of research in relation to epidemic
disease. Two of the primary dangers are a lack of representativeness in a sample population and
the lack of proper consideration for alternate explanations of change (Konigsberg and
Frankenberg 2002; Larsen 1994). Once these pitfalls have been satisfactorily sidestepped for a
particular dataset, a researcher can then consider epidemic disease as a primary factor.

The analysis of mortuary context can reveal the presence of epidemic disease within a
population. If a population were struck by such an affliction, one would expect to see not only
an increase in mortality in the archaeological record but also changes in mortuary practice.
Milner (1980) and others (Hill 1996; Margerison and Knusel 2002) list four expected criteria for
the effects of epidemic disease on mortuary context based on recent and historical data relating
to epidemic disease which are cultural change, burial change, mass burial and higher mortality of
among specific age and sex groups. Alternatively, some evidence suggests that in Europe the
bubonic plague attacked without regard to age and sex (Margerison and Knusel, 2002). I
mention this not to equate Europe’s bubonic plague to virgin soil epidemics of the New World,
but to provide an example of an epidemic disease that may not satisfy the above final criterion.
However, the presence of all of these criteria in a dataset would provide strong evidence of a
catastrophic event, such as an epidemic disease.

Settlement pattern analysis is another archaeological method of determining the impact of
epidemic disease on a population. One would expect to see changes in the size and frequency of
settlements if epidemic disease played a primary role in depopulation. However, unique issues
such as population relocation and other forms of depopulation such as warfare and famine must
be considered as well because they may also produce similar results in settlement pattern change.
Osteological analyses may also be conducted to determine the effects of epidemic disease on a population. Unfortunately, direct indication of epidemic disease is rare. Due to a frequently swift and fatal impact, epidemic disease rarely leaves an osteological marker (Larsen et al., 1992). This is not to say, however, that osteology cannot be used as a method of analysis in the study of epidemic disease. A number of researchers (Larsen, et al. 1992; Hill 1996) have examined osteological markers of diet and stress to extrapolate information regarding the impact of epidemic disease. Larsen’s research (1992; 1994; 2000) evaluates precontact and postcontact data from Spanish La Florida, providing strong evidence for the increase of nutritional, physical and health stresses during the contact period. The presence of physiological stresses prior to contact may lead to an increased susceptibility to epidemic disease.

Study Cases

Within the last thirty years, a vast amount of literature has been published on New World epidemic disease. While much of this literature focuses on population estimation and depopulation theory, an increasing amount of research provides evidence of epidemic disease. As a result, I have provided several representative and noteworthy examples of the various ways in which evidence of epidemic disease may be quantifiably ascertained and understood.

Provided below are three study cases that provide evidence of European induced epidemic disease within the Southeastern United States. These study cases are geographically concentrated on the Lower Mississippi Valley, west-central Alabama and Spanish La Florida (coastal Georgia). Ramenofsky’s research in the Lower Mississippi Valley, while somewhat dated (1987), provides an example of data supporting the uniform model of epidemic disease spread based upon archaeological analysis of settlement patterns. Archaeological and osteological evidence presented by Hill (1996) supports the influence of variation in the spread
of epidemic disease in the Southeast. Finally, Larsen’s (1992; 1994; 2000) ongoing osteological study in coastal Georgia also provides strong evidence for the opposing regional model of dispersal.

**Study Case I: Lower Mississippi Valley**

Ramenofsky (1987) examined settlement patterns in the Lower Mississippi Valley across three temporal periods to ascertain the frequency and changes in settlement across time (see figure 1). Temporal ranges were determined based upon radiocarbon dates, ceramic seriation, and amount of European trade goods (Ramenofsky 1987). Ranges were calculated for each period, with spans of 1400-1540, 1541-1682 and 1700-1750 for periods I, II and III respectively (Ramenofsky 1987). Within each period, settlement counts were collected and adjusted (using the formula \([Sc/(t/100)]\), where \(Sc\) is settlement count and \(t\) is period duration) to obtain minimum, maximum and mean counts (see table 2) (Ramenofsky 1987). A total of sixty-six settlement components from the region were used for this study.

Each of the settlement counts, both raw and adjusted, indicates a decrease in frequency between the periods I and II. Period I represents the prehistoric phase, immediately followed by the initial contact phase (Period II). A slight increase in frequency may be seen between periods II and III. In period I, the raw settlement count was 49 and the adjusted counts ranged from a minimum of 9.1 to a maximum of 123.0, with a mean count of 35.0. In period II, the raw settlement count was 10 and the adjusted counts ranged from a minimum of 6.3 to a maximum of 7.5, with a mean count of 7.1. Finally, in period III, the raw settlement count was 7 and the adjusted counts ranged from a minimum of 11.6 to a maximum of 23.3, with a mean count of 14.0. Ramenofsky (1987) attributes the decrease in settlement frequency between periods I and II to a disease induced population collapse and the latter increase in settlement frequency
between periods II and III to sampling bias, citing the increased ratio of known historic sites due to historical documentation. Such a bias is an important consideration and may have resulted in disproportionate numbers of prehistoric and historic sites in the Lower Mississippi Valley.

**Study Case II: Alabama**

Hill (1996) examined practices of change in mortuary tradition and mass interment in protohistoric Alabama. Historical documentation suggests that De Soto traveled through the area during this period (Hill 1996). A change in mortuary custom may be seen archaeologically through the shift to burial urn interment during the protohistoric period (Hill 1996). Evidence of multiple interments per urn also indicates significant changes in mortuary practice (Hill 1996). The protohistoric period, also known as known as Mississippian Decline, is characterized by a significant departure in culture, material remains and mortuary customs (1996).

Hill (1996) also examined osteological evidence and mortuary context from five archaeological sites in Alabama (see figure 2). Of these sample sites, two date to the precontact Mature Mississippian period (GR 2 and P 133) and three date to the protohistoric period (WX1, TU 4, and HA 19). Hill found an increase in the frequency of porotic hyperostosis lesions in the latter portion of the protohistoric period, indicating a prevalence of iron deficiency (see figure 3). Of the dataset from site 1HA19, approximately 67% exhibit evidence of porotic hyperostosis and 45% exhibit some form of infection (Hill 1996). The earliest site included in the sample, 1GR2, exhibits evidence of porotic hyperostosis in only approximately 5% of the sample, with infection representing about 8% of the sample population (Hill 1996). This indicates a 62% increase in porotic hyperostosis and 38% increase in infection between the two periods. The increase of porotic hyperostosis spikes significantly at site 1HA19. Conversely, the frequency of infection actually decreases slightly between Mature Mississippian and protohistoric periods and
then rebounds in the late protohistoric period. So, while the incidence of infection decreases at the onset of the protohistoric, the prevalence of porotic hyperostosis increases significantly after contact. Equally interesting is the evidence of rare, genetic traits seen in four individuals (Hill 1996). Hill (1996) notes the appearance of hydrocephalus and bifurcated incisor and canine roots. This evidence is isolated to sites 1WX1 and 1HA19, both of which date to the protohistoric period. Hill notes that such “dental anomalies” have been linked with homogenous populations, which may have contributed to genetic isolation and thus to susceptibility to disease (1996:24).

**Study Case III: La Florida**

Larsen’s (2000; 1994a; 1994b; 1992a; 1992b) research provides clear evidence of increased osteological stress experienced by Spanish mission inhabitants, the Guale, during the historic period (see figure 4). The author employs the widest temporal range of the cited studies, which is extremely helpful in determining change through time. Specimens were divided into four periods, which were “precontact preagricultural (<1150),” “precontact agricultural (1150-1500/1550),” “early contact (1607-1680),” and “late contact (1686-1702)” (Larsen 1996: 105) Larsen conducted osteological analyses of diet and health to ascertain the effects of Spanish contact on the Guale.

Analyses of bone chemistry and dental wear reveal changes in the dietary patterns of the Guale during the contact period. A comparison of stable carbon isotope ratios (\(^{13}\text{C} / ^{12}\text{C}\)) and stable nitrogen isotope ratios (\(^{15}\text{N} / ^{14}\text{N}\)) between the late prehistoric and contact periods illustrates an increase in the consumption of corn and a decrease in marine consumption during the mission period (see figure 5 and table 3) (Larsen 2000). For example, between the precontact agricultural and early contact periods, the mean stable carbon isotope ratio increases from −13.0
to −11.5 and the mean stable nitrogen isotope ratio decrease from 10.7 to 9.4, both of which indicate the above mentioned change in diet (see table 3 for additional values) (Larsen 1996). The mean increase in stable carbon isotope ratios from −14.5 to −13.0 and decrease in stable nitrogen isotope ratios from 12.8 to 10.7 between the precontact preagricultural and precontact agricultural periods illustrate earlier dietary changes as well (Larsen 1996). This indicates a directional change in diet across the four temporal periods. Conversely, an analysis of dental microwear indicates an increase in the consumption of soft foods during the contact period (Larsen 2000).

Changes in health were also demonstrated osteologically. An analysis of the increased frequency of porotic hyperostosis and cribra orbitalia indicated an increase in iron deficiency among the contact period populations (see table 4 and figures 3 and 6) (Larsen 2000). Approximately a six percent total prevalence of porotic hyperostosis is present in the each of the two precontact period samples, while approximately 27 percent of each of the two contact period samples exhibit such lesions (see table 4 for additional values) (Larsen et al., 1992). These data isolate an increased frequency of iron deficiency to the contact period.

Evidence of dental hypoplasia, however, does not indicate a significant shift in frequency between the precontact and mission periods (see table 5 and figure 7). This demonstrates continuity in the prevalence of physiological stress. The mean widths (mm) of the maxillary I₁, I₂, C and mandibular I₁, I₂, C for the precontact agricultural period are 0.70, 0.64, 0.68 and 0.57, 0.56, 0.79 respectively (Larsen et al., 1992). The mean widths (mm) of these same teeth in the early contact period are respectively 0.58, 0.70, 0.73 and 0.41, 0.46, 0.73 (see table 5 for additional values) (Larsen et al., 1992). While the frequency of hypoplasia did not significantly
increase during the contact period, the prevalence of such lesions in the precontact agricultural period demonstrates the existence of physiological stress prior to contact.

**Discussion**

Ramenofsky’s (1987) findings indicate a significant decline in settlement frequency in the Lower Mississippi Valley following initial contact. While the frequency of settlements rebounded postcontact, Ramenofsky attributes this to sampling bias rather than a population increase. The use of data dating to periods surrounding initial European contact provides a temporal frame wide enough to ascertain change in regional settlement patterns. The data span a range of roughly 350 years, placing initial Spanish contact (1541) close to the center of the range. Ramenofsky asserts that the evidence provided adheres to the model of temporally rapid and geographically extensive disease spread supported by Dobyns (1987). This model cites the Mexican smallpox epidemic of 1519 as the catalyst of depopulation in the New World (Dobyns 1983). The author notes an 80 percent decrease in the number of settlements during the relatively short period following contact (1992). The data indicates that the number of approximately fifty settlements between 1450 and 1550 decreased to ten settlements by 1600 (1992). Larsen (1992) notes that the dense population of the Lower Mississippi Valley may have contributed to rapid disease spread in this area.

Ramenofsky conjectures that the virgin soil epidemics affecting the New World usually resulted in a 30%-100% mortality rate, causing a significant population change in one or two generations (1987). The 80% decrease in settlement frequency between periods I and II certainly satisfies this condition. The author further notes that changes as significant as these that also fall between initial and full contact must be attributed to epidemic disease (1987). As illustrated by
Hill, however, native populations in the Southeast, and elsewhere, were not stationary prior to European contact.

However, there are a number of factors that discredit the representativeness of the Lower Mississippi Valley sample. First, the sample relies heavily on material collected by Phillips, Ford and Griffin during the first half of the twentieth century. Much of this is survey material, and subject to sampling bias due to the limited collection process and the subjective interests of the collector. Ramenofsky acknowledges such a bias, yet asserts that the findings are even more significant in the face of said bias. However significant the finding, a representative sample is still necessary to make inferences regarding a larger population. Additionally, the method of constructing settlement count components from ceramic seriation may have been affected by the sampling bias of survey material because the surface ceramics may not have been representative. This may have acted to either inflate or deflate the settlement counts, depending on the ceramic type and its prevalence within the seriation.

Hill examined osteological evidence from five protohistoric period sites in Alabama, said to be representative of the total population, although, specific sampling methods are not discussed. One of the limitations of Hill’s study is the lack of a wide temporal range for study. The study lacks a definitive contact period comparison. The issue of representativeness also emerges as the author does not describe the sampling methods. A total of five sites are examined in this study, two of which date to the earlier Mature Mississippian period, and three to the protohistoric. Because one site, 1HA19, represents a far greater percentage of infection and porotic hyperostosis it may not be representative of the population. However, the increase in infection and porotic hyperostosis may have acted to increase susceptibility to disease during the late protohistoric. Other research (Larsen et al., 1992) supports Hill’s findings regarding an
increase in porotic hyperostosis during the historic period in the Southeast. Hill provides evidence of physiological stress via an increase in the frequency of iron deficiency. Hill (1996) notes that both the frequency and the range of such lesions increase in the protohistoric, citing the prevalence across age ranges. Because these lesions are usually isolated in younger individuals, Hill (1996) infers nutritional stress among the protohistoric populations. Coupled with the evidence of genetic anomalies, she infers a general health decline with roots prior to contact (Hill 1996). The evidence presented by Hill indicates that a combination of factors including poor diet, homogenous population and epidemic disease may have influenced the increase in osteological stress markers among individuals in protohistoric Alabama.

While Larsen relied most heavily upon osteological data among the Guale, he also utilized historical documentation of disease outbreaks and archaeological evidence of decreasing settlement size. For example, Spanish mission documents indicate regional measles epidemic in the years 1613 and 1617 and a smallpox epidemic in 1655 (Larsen 2000). Settlement pattern analysis reveals that between 1670 and 1680, the number of sites decreases from six to four (Larsen 2000).

The archaeological record of coastal Georgia allowed Larsen to examine osteological change across a wide temporal frame. Such a broad range permitted not only the evaluation of contact stress but also allowed for the evaluation of precontact agricultural stress. As demonstrated by Hill (1996), an increase in porotic hyperostosis among native populations of protohistoric Alabama likely indicates nutritional stress related to diet. By comparing precontact preagricultural populations to precontact agricultural populations, Larsen (2000) was able to isolate osteological variation related to the agricultural switch prior to contact. The frequency of porotic hyperostosis increases significantly between the precontact and contact periods, which
indicates nutritional stress and a potential susceptibility to epidemic disease (Hill 1996; Larsen 2000).

According to Larsen, the specimens included in the two contact period samples represent accretional rather than catastrophic assemblages due to the disarticulation of the skeletons (2000). The author notes that mass interments are typically comprised of articulated skeletons (Larsen 2000). Thus, these assemblages do not provide evidence of massive mortality due to epidemic disease. They do, however, provide evidence of the increased variability within the populations both before and after contact, as seen through changes in diet and health.

Of the four expected criteria for epidemic disease presented by Milner (1980), only the study from protohistoric Alabama satisfies all conditions. This period of Alabama history is characterized by transitions in both cultural and burial practice. Hill (1996) found evidence of mass interment and higher rates of mortality among the young as well. Larsen’s research among the Guale did not indicate mass interment. While the mission period is characterized by changes in cultural and burial traditions, these are likely the result of Christian influence as opposed to epidemic disease (Larsen 2000). Because Ramenofsky’s study of the Lower Mississippi Valley did not analyze mortuary context, the results cannot be tested against Milner’s model.

**Conclusion**

While the rate of disease spread, demographic estimations of collapse and methods of analysis differ, all agree that native populations suffered from the effects of virgin soil epidemics. Analysis of osteological assemblages verifies the presence of epidemic disease in the Southeast prior to European contact (Bogdan and Weaver, 1992; Braun et al., 1998). This evidence rejects earlier hypotheses of idealized Native American life and health. Two primary models of the spread of epidemic disease emerge from the literature, which are the uniform
(Ramenofsky 1987; Dobyns 1983) and regional models (Larsen 1994). Evidence from the Lower Mississippi Valley indicates a reduction in the frequency of settlements following contact and appears to support the model of catastrophic depopulation (Ramenofsky 1987). However, the representativeness of the sample is questionable. Evidence from both Alabama and the Georgia coastline also signal population changes after contact. These studies focus on the osteological and mortuary evidence to infer information regarding health and activity both preceding and following contact. These data suggest that prehistoric populations were experiencing changes that may have influenced susceptibility to epidemic disease. More importantly, perhaps, these data reveal the inherent variability of native populations prior to contact. The existence of such evidence points to variability in the spread of epidemic disease in the New World. Also apparent, is the advantage of utilizing multiple forms of analysis in developing an accurate portrayal of Native American life during the contact period.

**Future Research**

An increasing amount of current epidemiological research focuses on the extraction and analysis of ancient DNA (aDNA) for evidence of tuberculosis (Braun et al., 1998; Mays et al., 2001; Nicholson et al., 2002; Taylor et al., 2001). With the recent increase in the interest of aDNA as an independent line of evidence for tuberculosis, it may be possible to detect the presence of other forms of epidemic disease genetically. Recent research even indicates potential contamination hazards in the analysis of specimens treated with bone glue as a preservation tool (Nicholson et al., 2002). Because most evidence of epidemic disease must be indirectly inferred, such an analysis would prove invaluable for paleoepidemiological research.
Appendix

Tables

Table 1
Native North American Population Estimates and Annual Rates of Decline (North of Mexico)

<table>
<thead>
<tr>
<th>Estimate</th>
<th>Annual Rate of Decline (to 375,000 in 400 years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1,153,000 (Mooney 1928)</td>
<td>-0.28 %</td>
</tr>
<tr>
<td>1,894,280 (Ubelaker 1988)</td>
<td>-0.40 %</td>
</tr>
<tr>
<td>7,000,000 (Thornton 1987)</td>
<td>-0.73 %</td>
</tr>
<tr>
<td>9,800,000 (Dobyns 1966)</td>
<td>-0.82 %</td>
</tr>
<tr>
<td>18,000,000 (Dobyns 1983)*</td>
<td>-0.97%</td>
</tr>
</tbody>
</table>

* North of Mesoamerica

Source: Thornton 1997

Table 2
Lower Mississippi Valley Settlement Counts and \[S/(T/100)\]

<table>
<thead>
<tr>
<th>Raw Period</th>
<th>Adjusted Counts</th>
<th>Adjusted Counts</th>
<th>Adjusted Counts</th>
<th>Adjusted Counts</th>
<th>Adjusted Counts</th>
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<tr>
<td></td>
<td>Counts</td>
<td>Min. (S_c)</td>
<td>Mean (S_c)</td>
<td>Max. (S_c)</td>
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<td></td>
</tr>
<tr>
<td>III</td>
<td>7</td>
<td>0.3</td>
<td>23.3</td>
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<td>II</td>
<td>10</td>
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Source: Ramenofsky 1987
Table 3
13C/12C and 15N/14N ratios in La Florida

<table>
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<tr>
<th>Period</th>
<th>13C Mean</th>
<th>13C Number</th>
<th>13C Range</th>
<th>15N Mean</th>
<th>15N Number</th>
<th>15N Range</th>
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<tr>
<td>&lt; 1150</td>
<td>-14.5</td>
<td>20</td>
<td>-18.6-13.4</td>
<td>12.8</td>
<td>20</td>
<td>10.6, 14.4</td>
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<tr>
<td>1150-1500</td>
<td>-13.0</td>
<td>9</td>
<td>-16.4-10.0</td>
<td>10.7</td>
<td>9</td>
<td>9.5, 13.3</td>
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<td>1607-1680</td>
<td>-11.5</td>
<td>22</td>
<td>-14.3-9.6</td>
<td>9.4</td>
<td>22</td>
<td>7.4, 10.8</td>
</tr>
<tr>
<td>1686-1702</td>
<td>-11.5</td>
<td>21</td>
<td>-12.6-10.0</td>
<td>10.0</td>
<td>22</td>
<td>8.3, 11.6</td>
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</table>

Source: Larsen 1992

Table 4
Porotic Hyperostosis Frequency in La Florida

<table>
<thead>
<tr>
<th>Age</th>
<th>&lt; 1150</th>
<th>&lt; 1150</th>
<th>1150-1500</th>
<th>1150-1500</th>
<th>1607-1680</th>
<th>1607-1680</th>
<th>1686-1702</th>
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<tbody>
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<td></td>
<td>%</td>
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<td>%</td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
<td>No.</td>
</tr>
<tr>
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<td>60.0</td>
<td>5</td>
<td>50.0</td>
<td>4</td>
<td>66.7</td>
<td>12</td>
</tr>
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<td>5-9.9</td>
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<td>4</td>
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<td>20.0</td>
<td>5</td>
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<td>4.2</td>
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<td>6.2</td>
<td>145</td>
<td>26.5</td>
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<td>27.2</td>
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</table>

Source: Larsen 1992

Table 5
Hypoplasia Width in La Florida

| Tooth | Width (mm) | No * | Width (mm) | No. * | Width (mm) | No.* | Width (mm) | No. *
<table>
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<td>.58</td>
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<td>.64</td>
<td>66</td>
<td>.70</td>
<td>77</td>
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<tr>
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<td>67</td>
<td>.68</td>
<td>81</td>
<td>.73</td>
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<td>.54</td>
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<td>.78</td>
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</table>

* “Number of Individuals with at least one hypoplastic event”
# “Partial Sample”

Source: Larsen 1992
Figures

Figure 1
Map of the Lower Mississippi Valley

Source: Modified from National Parks Service
(http://www.cr.nps.gov/nr/twhp/wwwlps/lessons/71hudson/71locate/.htm)

Figure 2
Map of Alabama sites used by Hill

Source: Modified from Alabama Maps
(http://alabamamaps.ua.edu/alabama/physical/)
**Figure 3**
Porotic Hyperostosis

Source: Larsen 1998
(http://www.cast.uark.edu/local/icaes/conferences/wburg/posters/cslarsen/html)

**Figure 4**
Map of Spanish La Florida

Source: Modified from Exploring Florida
http://fcit.coedu.usf.edu/florida/maps/1600/m053900.htm
Figure 5
Scatterplot $^{13}\text{C}/^{12}\text{C}$ and $^{15}\text{N}/^{14}\text{N}$ ratios in La Florida

Source: Larsen 2000, page 149

Figure 6
cribra orbitalia

Source: Larsen 1998
(http://www.cast.uark.edu/local/icaes/conferences/wburg/posters/cslarsen/html)
Figure 7
Dental Hypoplasia

Source: Larsen 1998
(http://www.cast.uark.edu/local/icaes/conferences/wburg/posters/cslarsen/html)
References

Alabama Maps. University of Alabama and Cartographic Research Laboratory. (September 23, 2002); http://alabamamaps.ua.edu/alabama/physical/.


National Parks Service: the siege of Port Hudson. (October 10, 2002); http://www.cr.nps.gov/nr/twhp/wwwlps/lessons/71hudson/71locate/.htm


