Publicación INCAP PCI/093

LORI E. WRIGHT
Department of Anthropology, Texas A&M University
College Station, TX 77843-4352

FRANCISCO CHEW
Institute of Nutrition of Central America and Panama
Apartado Postal 1188, Guatemala City
Guatemala

Porotic Hyperostosis and Paleoepidemiology: A Forensic Perspective on Anemia among the Ancient Maya

Porotic lesions caused by childhood anemia are commonly found on ancient Maya crania and have been cited as evidence for extremely poor nutrition during the Classic Period. We reconsider this characterization in the light of recent data on childhood anemia in rural Guatemala and the prevalence of porotic hyperostosis in crania of forensic skeletal remains of rural highland Maya from Plan de Sanchez, Baja Verapaz, which date to 1982. The abundance of porotic hyperostosis in adults from Plan de Sanchez fits well with the number of modern rural children suffering from anemia, but the lesions are very rare compared to archaeological series. Although some minor change in diet and infection may contribute to differences in porotic hyperostosis, it is likely that higher mortality leads to fewer anemic lesions in modern adult crania. We hypothesize that more anemic children survived to adulthood in the past than do today. *[iron-deficiency anemia, porotic hyperostosis, Maya, nutrition, forensic anthropology, osteological paradox]*

Paleopathology, the study of disease in ancient times, has grown to a mature field over the last two decades. Emphasis has changed from a case-study focus to a populational approach, which aims to characterize epidemiological change over time. This shift has been especially important to the emerging understanding of the evolution of several disease entities (especially tuberculosis and treponematosis) but also has allowed paleopathology to become an active player in the study of past cultural change. Skeletal evidence now contributes to a better understanding of prehistoric life and of the health implications of subsistence strategies and population density. However, recent work has shown that it is difficult to evaluate the biocultural consequences of pathological lesions at the population level, and of changes in their prevalence, because of the difficulty of identifying the effects of frailty and differential mortality on lesion abundance (Wood et al. 1992).

One approach that may provide a partial resolution to this dilemma in some world areas is through more explicit analogy with the health of living populations. Certainly, diagnosis of bone disease is carried out by comparison with clinical findings, and interpretations of prehistoric health status are based on analogy with health conditions in living populations. Often such inferences draw only on generally recognized epidemiological processes, such as the interaction of malnutrition and infection during early childhood. However, more direct, local analogy can also inform the interpretation of health more specifically. For instance, Roberts and Manchester (1995:2) suggest that such analogy may be useful where local modern cultures maintain strong indigenous cultural traditions and are little affected by modern technological change and medical intervention.

In this paper, we explore the use of ethnographic analogy as a tool to evaluate the broader implications of poor health on an archaeological culture. We focus on the skeletal evidence for anemia among the Maya of Central America. As a measure of health that reflects both diet and infectious disease, the abundant lesions of this condition on ancient Maya skeletons have led to a characterization of the prehispanic population as perilously stressed. We reconsider this interpretation in the light of (1) the epidemiology of modern Maya populations whose health status has been intensively researched and (2) the prevalence of skeletal lesions of anemia in forensic skeletal remains of the modern population.

Childhood Anemia and Its Skeletal Manifestations

Iron deficiency is the most common nutritional problem in the world and is a primary health concern of developing

nations. It is estimated that 2.15 billion people are iron deficient worldwide, while 1.20 billion suffer from anemia (Viteri 1993). Iron deficiency and anemia occur if the amount of iron absorbed from foods does not satisfy the organisms' requirements. Initially, depletion of body iron stores leads to an increase in serum ferritin, undersaturation of transferrin, an increase in transferrin receptors, and elevated erythrocyte protophorphyrin levels. Together, these changes indicate an iron deficient status. Anemia is a more severe condition and refers to replacement of normal cells by smaller blood cells (microcytic) that carry less hemoglobin (hypochromic) (Ryan 1997).

Both iron deficiency and anemia can be due to a variety of factors. One important cause may be the insufficient consumption of iron. In developing countries, economic factors limit the consumption of foods rich in iron, especially meat, in which iron is bound to heme compounds that are readily absorbed through the intestinal mucosa. Non-heme iron, which is found in cereals, legumes, fruits, and vegetables that constitute the primary diet of the lower socioeconomic classes in many countries, is much less bioavailable. Some food chemicals can bind iron into complex formations that cannot be absorbed by the cells of the intestinal lining. These include phytates, oxalates, polyphenols, and dietetic fiber, which are abundant in plant foods (Lynch 1997). In addition, it has recently been documented that calcium inhibits the absorption of dietary iron (Prather and Miller 1992). When ingested together with iron, Vitamin A improves iron nutrition, perhaps through a better mobilization and utilization of iron stores (Mejía and Chew 1988). Vitamin C stimulates the absorption of nonheme iron (Lynch 1997; Ryan 1997).

Disease also plays a role in iron status. Absorbed iron may not be available for use by the body in synthesis of hemoglobin despite normal or high body iron reserves. In inflammatory or infectious processes iron reserves may be sequestered as a defense against infectious pathogens, which require iron for growth. Such iron-withholding has been documented in patients with mycobacterial, mycotic, and bacterial infections (Weinberg 1992). Other research suggests that chronic anemia increases susceptibility to infection; at this time there is little consensus about the direction of causative arrows between anemia and infection (Ryan 1997; Walter et al. 1997).

Parasitic infection can play a substantial role in iron status. Infection by whip worm *(Trichuris)* and hookworms *(Necator* and *Ancylostoma)* cause intestinal bleeding, thus raising iron requirements for infected persons. Other parasites, such as giardia *(Giardia lamblia)* and roundworm *(Ascaris lumbricoides),* diminish the intestinal absorption of iron (de Vizia et al. 1985; Stoltzfus et al. 1997).

Infants require more iron than other age groups because of their high growth rate. For them, iron deficiency may be especially critical. Maternal breast milk is the ideal food during the first few months of life, but infant iron stores decline after 4 to 6 months of age. After this age, iron-rich solid foods should be introduced to maintain normal growth and prevent anemia. Because children are often fed bulky carbohydrate staples that contain little iron, early childhood is a critical period for iron status throughout the developing world (Ryan 1997). Moreover, childhood anemia has severe functional consequences that can persist after recovery of normal iron status. These include retardation in physical growth, delayed walking ability, decreased physical work capacity, and slower cognitive development and achievement (Lozoff et al. 1991; Pollitt 1994; Ryan 1997; Walter et al. 1989).

Porotic hyperostosis is the skeletal manifestation of prolonged childhood anemia (Hengen 1971; Moseley 1963; Stuart-Macadam 1987a). This term, coined by Angel (1966), refers to porotic lesions in the external table of the cranium. The lesions result from the expansion of cranial diplöe—a hemopoietic tissue—in response to anemia. Diplöe expansion puts pressure on the thin external table of the child's cranium, which becomes thinner and porotic. As the tissue expands further, new bone production occurs along the path of the expanding tissue, resulting in vault thickening and the pathognomic, radiographic "hair-on-end" appearance (Stuart-Macadam 1987a, 1987b).

In young infants, diplöic expansion is first evident in the orbital roof (called "cribra orbitalia"), but in older infants the lesions form in the parietal bosses, the occipital squama, and occasionally in the frontal (Stuart-Macadam 1989). Lesions that were active at the time of death show a porotic or corraline external table. Healed lesions show infilling of the pores with new, dense bone, and remodeling of the lesion as a whole (Figure 1). With maturity, the hemopoietic capacity of the diplöe is reduced by its partial involution to fatty yellow marrow, so cranial lesions do not form anew in adults. Instead, healed lesions from childhood may persist and remain visible in adult skeletons (Stuart-Macadam 1985).

Unlike Angel (1966), who diagnosed porotic hyperostosis as thalassemia in Mediterranean skeletons, paleopathologists working in the Americas attribute the lesions to iron deficiency anemia and emphasize a combination of low dietary iron and parasitic infection to explain the abundance of the lesions in prehistoric skeletons. Porotic hyperostosis has been found to occur with greater frequency in skeletal series of maize agriculturalists than among skeletons of foragers, a trend that suggests that diet explains the lesions (Cohen and Armelagos 1984; El-Najjar et al. 1976; Holland and O'Brien 1997). Yet early research in the Midwest (Lallo et al. 1977; Mensforth et al. 1978) demonstrated that porotic hyperostosis was linked to infectious disease—periostitis—and suggested that malabsorption of iron due to "weanling diarrhea" was a contributing factor. In several recent studies, researchers have found that diet alone cannot explain the distribution of porotic

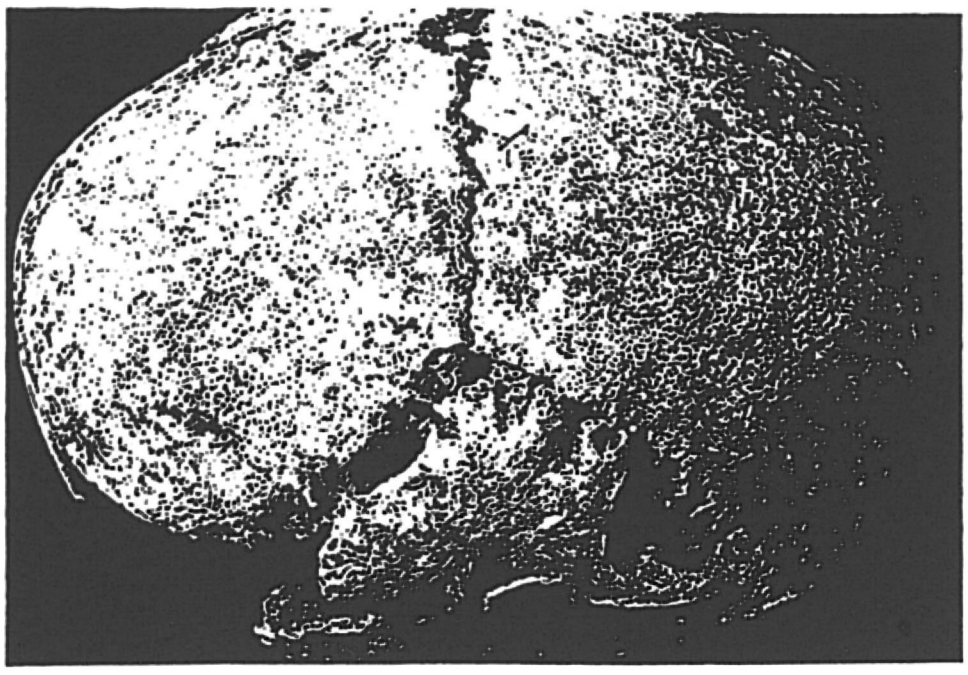

Figure 1. Healed porotic hyperostosis in an adult Classic Period Lowland Mayan skeleton, which also shows tabular oblique cranial deformation.

hyperostosis and draw attention to parasitic infection (Reinhard 1992; Ubelaker 1992; Walker 1986). Current consensus is that low dietary iron, weanling diarrhea, infectious disease, and intestinal helminthiasis act synergistically to produce childhood anemia and account for the varied occurrence of porotic hyperostosis in skeletal series (Hill and Armelagos 1991; Holland and O'Brien 1997; Palkovich 1987).

Stuart-Macadam (1992) argues that porotic hyperostosis might be caused by iron sequestering during infection (Weinberg 1992). She hypothesizes that a high rate of lesions indicates a population that was *well adapted* to a high pathogen (parasite) load, and in which iron-withholding contributed to anemia. Goodman (1994:167) points out the importance of distinguishing between anemia (a chronic condition) and iron-withholding (a short-term response) because some research shows anemia to compromise immune status and to increase susceptibility to infection. Holland and O'Brien (1997) characterize Stuart-Macadam's argument as a "parasite model" and emphasize the well-demonstrated link between maize agriculture and porotic hyperostosis. However, since the direction of causal arrows between anemia and infection continues to be debated by human biologists (Ryan 1997; Walter et al. 1997), Stuart-Macadam's argument cannot yet be dismissed.

Wood and colleagues (1992) have brought the effects of mortality and frailty on lesion abundance to the forefront of recent discussions (Cohen 1994, 1997; Goodman 1993; Jackes 1993). In "The Osteological Paradox," Wood et al. (1992) argue that the abundance of lesions in a skeletal population alone reveals little about the incidence of disease in the living population from which it came. Traditionally, paleopathologists have assumed that a high lesion abundance indicated an unhealthy population, and that an increase in pathology would parallel an increase in disease, and conversely. However, in any population, individuals vary substantially in their susceptibility to disease. Because frail individuals are prone to an early death, a low level of pathology may actually indicate high mortality in a relatively frail population. Conversely, a high level of pathology may indicate high survivorship in a population of low frailty. In short, "better health makes for worse skeletons" (Wood et al. 1992:356).

For porotic hyperostosis, this argument is especially relevant. Remodeled lesions of porotic hyperostosis persist into adulthood, providing evidence in adult skeletons of *survived* anemic experience. Under-enumeration of subadult skeletons, differential preservation, and excavation biases often limit paleopathological study to adult remains. However, it is difficult to extrapolate from the abundance of healed porotic hyperostosis among adults to a characterization of childhood anemia when the effects of childhood mortality on lesion prevalence are unknown. Exploring this issue further, in this article we compare the abundance of porotic hyperostosis in crania of ancient and modern Maya Indians. Comparative data on the incidence of childhood anemia and mortality in modern Maya inform our interpretation of the implications of porotic hyperostosis for prehispanic Maya health.

Anemia among the Ancient Maya

Porotic cranial lesions were first described in archaeological crania from Yucatan by Virchow (1887) and Boas (1890), at which time the etiology of the lesions was unknown. Ever since Hooton (1940) described dramatic "osteoporosis symmetrica" in the skulls of children from the Cenote of Sacrifice at Chichen Itzá, porotic hyperostosis has been interpreted to indicate that ancient Maya reliance on maize agriculture had negative implications for health and for the survival of ancient Maya civilization. Hooton speculated that the condition was caused by some form of nutritional insufficiency, due to "dependence upon a diet consisting mainly of maize" (Hooton 1940:276).

Subsequent work by Saul (1972, 1973, 1977) at Altar de Sacrificios and Seibal established the diagnosis as iron-deficiency anemia. The abundant porotic hyperostosis he observed contributed to his view that the Maya "were exposed to an unhealthy ecological setting within whose confines survival was difficult and decline was always a possibility" (Saul 1972:75). Saul's work was published at a time when archaeologists were grappling with the apparent contradiction between the high settlement density of the Classic Period (AD 250–900) and the low productivity of extensive slash-and-burn agriculture (Haviland 1969; Sanders 1973). Moreover, iron-deficiency anemia was becoming known as a major health concern in Central America (World Health Organization 1968).

As in North America, paleopathologists working with Maya remains have emphasized a low dietary intake of iron in the etiology of iron-deficiency anemia, but also consider the likely role of parasitism. The hookworm

Ancylostoma duodenale is known to have been present prehispanically in both North and South America (Horne 1985; Reinhard 1990). A second hookworm species, *Necator americanus*, is currently endemic in Guatemala, but may have been introduced from the Old World (Scrimshaw and Tejada 1970).

A high prevalence of healed porotic hyperostosis has been reported in adult crania of prehispanic Maya skeletons. These data are summarized in Table 1. Figure 2 illustrates the locations of the sites mentioned. The wide range may be partly due to scoring differences among observers, but real differences in diet and pathogen load presumably also occurred. At some sites the lesions are more common in subadults than in adult remains, which suggests that anemic children were more susceptible to early mortality.

At Classic Period inland sites, some 60 to 90% of adult skulls show healed scars, indicating survival through chronic anemia during childhood. Lower levels of porotic hyperostosis are observed in Belize at Preclassic Cuello. This is likely due to less maize consumption at Cuello, as measured by stable carbon isotopic analysis of human bone (Tykot et al. 1996). At Lamanai, also in Belize, porotic hyperostosis is more abundant during the Postclassic Period than earlier, which corresponds to an increase in maize consumption documented isotopically (White et al. 1994). Despite dietary stability, the lesions are more abundant in Colonial Period skeletons than Postclassic ones at Lamanai, presumably due to an increase in Spanish-borne infectious disease (White et al. 1994). The lower incidence of porotic hyperostosis at Postclassic Lamanai versus Classic inland sites (where isotopic data indicate a similar diet) is in part because White et al. (1994:140) scored only more severe active cases, not well-healed ones.

Both sites on the Caribbean coast (Playa del Carmen, Tancah) show fairly abundant lesions. With the exception of coastal Tancah, we also observe that porotic hyperostosis is generally less common in late Prehispanic and early Colonial skeletal series (Tipu, Lamanai, Iximché) than during the Classic Period at inland sites where maize consumption was high (the Pasión, Copán). For Iximché, Whittington (personal communication, 1998) notes a low frequency (16.1%) in a sample that he believes had high social status. Yet maize consumption (measured isotopically) was high in this sample (Whittington and Reed 1994).

Ethnographic Analogy and Ancient Maya Health

Why were so many prehispanic Maya iron deficient as children? Whether laying blame on diet, parasitism, or a combination, bioarchaeologists have either explicitly or implicitly based their interpretations on analogies with modern health conditions in the Maya area. Likewise, archaeological reconstructions of past diet also rely heavily on ethnohistoric and ethnographic analogies.

A broad variety of cultigens are described in the colonial texts, providing evidence that they were also consumed prior to the Spanish incursion (Marcus 1982; Tejada Valenzuela 1970). Studies of flora and fauna exploited in the early twentieth century also form a basis for inference about prehispanic diets (Lundell 1939; Shattuck 1933). For instance, citing data on Yucatec Maya food consumption (Benedict and Steggerda 1937; Steggerda 1941), Roys (1972:44) argued that modern diets are "probably not very different, on the whole, from . . . [the diet] . . . of the pre-Spanish Maya."

Until the recent growth of paleopathology, the most productive approach to characterization of prehispanic health was by analogy with modern health. Shattuck's (1933, 1938) studies in Yucatan and Guatemala showed anemia to be quite common and documented a high rate of hookworm infestation. In the 1950s, research conducted by the

Table 1. Occurrence of porotic hyperostosis in Maya skeletal series (Modified after Wright and White, 1996: Table 1).

Skeletal Series	Chronological Period	Environmental Location	Subadults %	Subadults *N*	Adults %	Adults *N*	Reference
Cuello	Preclassic	Belize	12.5	8	3.6	28	Saul and Saul, 1991
Copán	Classic	inland	58.8	17	60.0	30	Whittington, 1989
Pasión (combined) [a]	Classic	inland	55.5	18	65.4	81	Wright, 1994
Altar de Sacrificios	Classic	inland	—		89.3	28	Saul, 1972, 1973
Chichen Itzá cenote	Postclassic	inland	77.8	18	52.9	17	Hooton, 1940
Playa del Carmen	Postclassic	coastal	—[b]		48.0	28?	Márquez Morfin, 1982
Lamanai	Postclassic	Belize	—[b]		9.0	53	White, 1988
Lamanai	Historic	Belize	—[b]		17.0	100	White, 1988
Tipu	Historic	Belize	35.8	106	19.4	185	Cohen et al., 1994
Iximché	Postclassic/Historic	highland	0.0	1	16.1	31	Whittington, p.c. 1998
Tancah	Historic	coastal	100.0	3	77.8[c]	9	Saul, 1982

[a] Combined sample includes Dos Pilas, Aguateca, Itzan, Altar de Sacrificios, and Seibal.

[b] Subadult data are included with adult statistics for these samples

[c] Rate is recalculated from data in Saul (1982) to include scorable crania only.

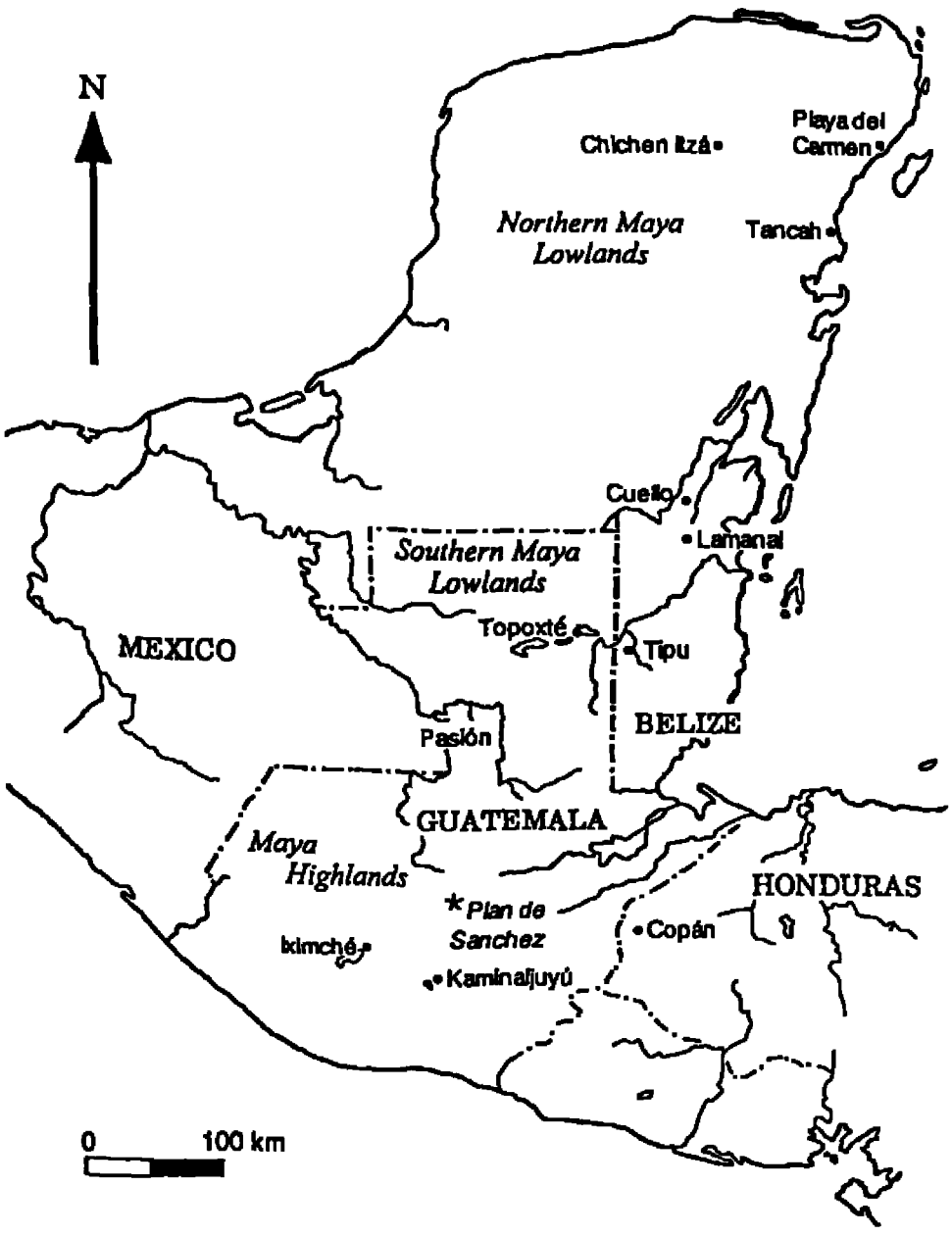

Figure 2. Map of the Maya area, indicating Plan de Sanchez and archaeological sites mentioned in the text.

newly founded Institute of Nutrition of Central America and Panama (INCAP) began to reveal the complexity of malnutrition among highland Maya populations, an understanding that continues to evolve today (Martorell 1992). Scrimshaw and Tejada (1970) summarized this research, providing a handy reference for inference about prehispanic health by archaeologists. The early INCAP studies found that heavy hookworm infestation was common in Guatemalan patients with anemia, but that low dietary iron intake was a confounding etiological factor. Citing these studies, Saul (1972:42) argued that prehispanic anemia had a similar etiology, and subsequent researchers have followed his lead (e.g., White et al. 1994; Whittington 1989; Whittington and Reed 1997; Wright 1994).

Early discussions of ancient Maya health by human nutritionists drew parallels between modern and ancient lifeways but suggested that the prehistoric Maya were *healthier* than their modern descendants. For instance, Tejada Valenzuela (1970) noted that the ancient Maya could have been malnourished like their descendants, given the importance of maize in their diets, but ultimately concluded that the state of health must have been better in the past because more (hunted) animal protein was available to them than to the impoverished modern Maya, who rarely purchase beef. Likewise, Behár (1968) argued that the prehispanic diet would have been superior to that consumed by rural indigenous communities today. Further, he suggested that the recent trend to earlier weaning and the replacement of breast milk with inadequate processed weaning foods are responsible for a higher level of infant morbidity and mortality than would have pertained in prehistory.

By contrast, archaeologists have used modern analogy to support a diagnosis of extremely *poor* prehispanic health. For instance, Santley cites modern nutritional studies to infer that malnutrition may have been extreme among the Late Classic Maya (Santley 1990; Santley et al. 1986). Similar arguments are made by Shimkin (1973) and Saul (1973) with regard to the collapse.

Certainly, modern Maya peoples do provide the most appropriate analogs for inference about prehispanic Maya health. We have direct evidence that indicates similarities in the composition of the diet of past and present, and we can infer broad similarities in parasite load. Most indigenous peoples in Guatemala remain subsistence farmers in rural areas, where they have little access to modern medicine, and where traditional culture and beliefs remain strong. Herein, we attempt to bridge the gap between analogy from modern disease rates to paleopathology by examining the abundance of porotic hyperostosis in a collection of recent forensic skeletons from rural villages in the Department of Baja Verapaz, Guatemala. We situate these data in their historical and epidemiological context. This modern analogy provides a comparative perspective from which we can further evaluate the impact of anemia on ancient Maya populations.

Anemia in Rural Guatemala

In 1995, the Guatemalan Ministry of Public Health, in cooperation with the Institute of Nutrition of Central America and Panama, conducted a survey of anemia in Guatemala as one component of a broad nutritional survey, the Encuesta Nacional de Micronutrientes (MSPAS 1996). Hemoglobin levels were measured in 1,712 children between the ages of 1 and 5 years. Children with hemoglobin levels lower than 11g/dl were considered to be anemic (corrected for variation in altitude). The survey found low hemoglobin in 23 to 30% of children in different regions of the country (Table 2). Baja Verapaz is included in the "Northeastern" zone where, overall, 23.8% of children are anemic. However, our Baja Verapaz forensic sample is from the western extreme of this zone and is largely indigenous, unlike much of the Northeast. Dietary and epidemiological characteristics of the Baja Verapaz villages more closely resemble those of the "Highland" zone, where 30.7% of children are anemic. Unfortunately, lowland Petén is not among the studied regions. The data also show that anemia is most prevalent among rural children (29%), lower among those living in large towns (24%), and lowest in children from the capital city (15%) (MSPAS 1996).

Anemia does not affect children at all ages equally. Younger children more often suffer from anemia than do older ones (Figure 3). Age-related dietary trends may contribute to this pattern because older children may eat more diverse foods than toddlers. An additional factor may be survivorship, which we address below. The survey does indicate a link between anemia and morbidity. Among children who had suffered from acute diarrhea or acute respiratory infection during the two weeks prior to the survey, a larger proportion were anemic (32.8% and 27.5%, respectively) than among those who had not been sick (21.7 and 22.8%, respectively) (MSPAS 1996:61). Diarrhea and respiratory infection are common causes of death among Guatemalan children. Among children aged 12 to 24 months, breastfed infants more commonly suffer from anemia (57.3%) than do those who no longer nurse (44.9%) (MSPAS 1996). Although reasons for this finding were not addressed in the study, it is likely that healthier, faster-growing children are weaned earlier than frail, slow-growing children. Moreover, frail children who are weaned early tend to suffer higher mortality, so their deaths deceptively raise the "healthy" appearance of the weaned group (Dettwyler 1999; Mølbak et al. 1994).

This distribution is best attributed to diet, not to parasitism. Maize consumption is highest in the rural Maya villages of the Highlands, and this is where more children are anemic. Recent national survey data indicate that ingestion of iron is insufficient for all groups, averaging 25% below adequate levels (MSPAS 1996). Among children aged one to two years, iron intake is especially low, only 56% of requirements. Moreover, black beans are the primary source of iron (17–31%) in all regions except for the Highlands, where maize contributes slightly more iron (20%). Meat, the source of iron with highest bioavailability, provides less than 5% of iron to all regions surveyed (MSPAS 1996). Hypovitaminosis A also contributes to inadequate utilization of iron stores in Guatemalan children (Mejía and Chew 1988). Due to lime processing (Bressani et al. 1958), Maya maize foods are rich in calcium carbonate, which inhibits intestinal absorption of ingested iron (Prather and Miller 1992). The importance of these factors likely vary ethnically and with socioeconomic status. For instance, tortillas may provide up to 50% of iron intake among the Kek'chi of Alta Verapaz (Krause et al. 1992), a value much higher than the national estimate.

Although hookworm infection can be an important contributor to anemia in Guatemala as elsewhere, patterns of infection do not match the patterns of anemia. In theory, hookworm larvae can only survive in the soil at temperatures between 70 and 85°F (Beck and Barrett-Connor 1971:103). Temperatures often drop well below this in the highlands, which may limit the spread of the infection. Thus, were blood loss due to hookworm a major factor precipitating anemia, we would expect less anemia at higher altitudes; however, this is precisely where anemia is most prevalent today. Moreover, a recent study of Guatemalan Ladinos found that hookworm infestation increases with host age. Both the proportion of individuals infected and the intensity of infection (number of eggs per gram of feces) *increase* through childhood (Anderson et al. 1993). Yet the prevalence of anemia *decreases* through childhood. This indicates that diet is more critical to the distribution of anemia today in Guatemala than is parasite load.

A Forensic Analogy: Plan de Sanchez

Since the 1950s, Guatemala has seen persistent combat between its military and guerrilla forces. Violent conflict peaked during the early 1980s, when a large number of civilians died, often in coordinated military massacres. Peace was not formally achieved until the end of 1996. Since 1992, the Forensic Anthropology Team of Guatemala (EAFG) has been exhuming clandestine graves from these conflicts. Their work begins with detailed interviews of surviving relatives, in order to collect data that might aid in identification. The remains are excavated using careful archaeological procedures, recovering clothing, shrapnel, bullets, and other evidence in addition to the skeletons. Osteological work aims to identify the remains and to document any perimortem pathology that might indicate cause and manner of death. After skeletal analyses are complete, the remains are returned to surviving relatives for reburial with appropriate ritual (EAFG 1997; Moscoso Moller 1994).

The valley of Rabinal lies in the western part of the Department of Baja Verapaz. The village of Plan de Sanchez, where one massacre occurred, is located on the northern

Table 2. Prevalence of anemia in Guatemalan children between 1 to 5 years of age (MSPAS 1996).

Region	*N*	% Anemic[a]
Highland Zone	515	30.7
South Coastal Zone	424	23.1
Department of Guatemala	523	25.1
Northeastern Zone	311	23.8
Total	1773	26.0

[a] Hemoglobin <11g/dl, adjusted for altitude

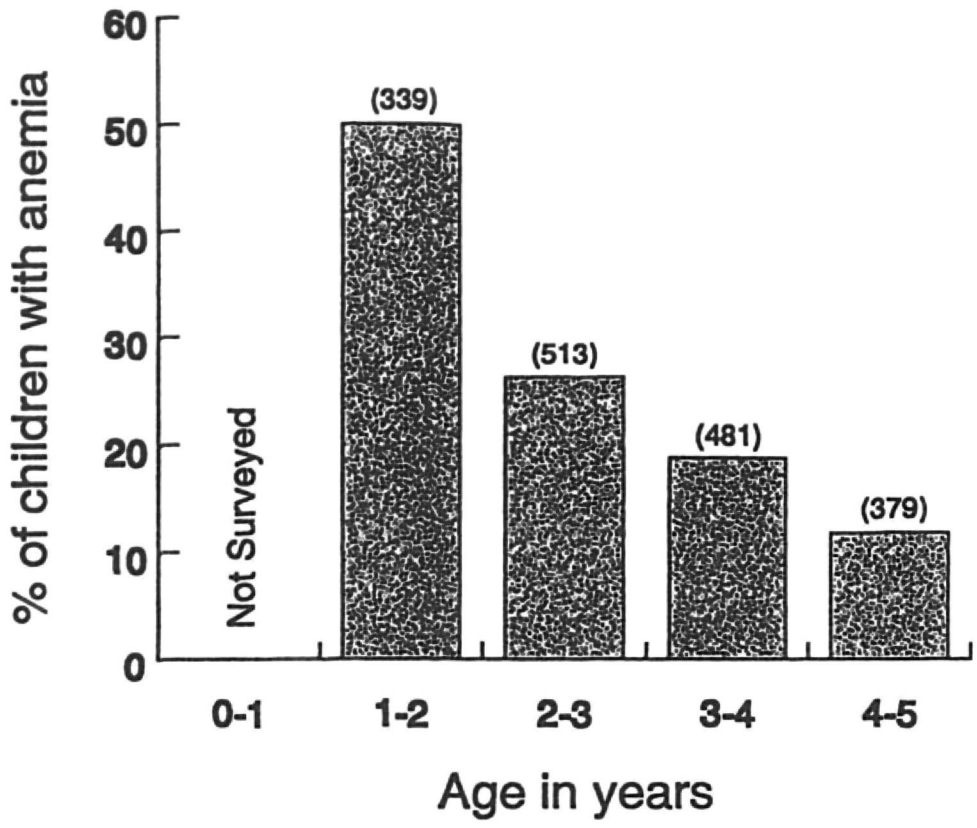

Figure 3. Prevalence of anemia (Hb <11g/dl) in Guatemalan children by age in 1995. Number of children surveyed is given above each age bar. Data are from the Encuesta Nacional de Micronutrientes (MSPAS 1996).

face of the Sierra de Chuacús, which forms the southern rim of the valley of Rabinal (Figure 2). The village lies approximately 9 kilometers from the municipal seat, at an elevation of roughly 1,700 meters above sea level. Today, most occupants of the valley are Achi Maya, speaking a Mayan dialect closely related to K'iche'. Rabinal is well known for its strong Achi traditions; the population of the municipio is approximately 82% indigenous. The rural population is primarily engaged in subsistence agriculture, but most own very small plots of land that are inadequate to support a family. Domestic craft production raises income for some families and others migrate seasonally to the Pacific Coast to work as agricultural laborers (EAFG 1997).

The massacre at Plan de Sanchez occurred on July 18, 1982. Rural people from some 13 mountain villages who had attended the Sunday market in Rabinal were intercepted by the military on the road that runs from Rabinal, through Plan de Sanchez to the larger village of Concul. The army escorted their captives to a house on the outskirts of Plan de Sanchez and collected remaining inhabitants of the village from their homes. Skeletal evidence and witness testimony indicate that some of the people were shot individually, but many died when the house was grenaded and burned. Several days later, neighboring villagers hastily buried the bodies in narrow shallow pits, most of which contained several skeletons (EAFG 1997).

The 84 skeletons recovered include 32 females, 28 males, and 21 children under 13 years of age. They include very young infants through elderly adults. The team has positively identified 25 skeletons on the basis of dental work and other unique skeletal identifiers that match antemortem data. Firearm wounds were evident on 39 of the skeletons, 34 of which show a gunshot wound to the cranium. Other indicators of perimortem trauma include fractures, machete cut marks, shrapnel, bullets, and burning (EAFG 1997:180–184).

Most of the dead are ethnically Achi. This is evident from the surnames provided by relatives and by indigenous clothing found in many women's graves. Of the 25 skeletons positively identified by the EAFG, 11 have indigenous surnames (EAFG 1997:190–191). Of course, many Achi now have fully Spanish names. A small number would be classified as Ladino by having foregone use of traditional Maya clothing and by learning Spanish. For the most part, in Rabinal, rural Ladinos are genetically indigenous and continue to practice a traditional lifestyle in terms of agricultural and dietary choices.

We believe that the Plan de Sanchez skeletal series is broadly representative of the rural population, since both old and young, healthy and infirm commonly attend markets. Also, those who had remained at home in Plan de Sanchez are included in the series. The most obvious difference between the forensic remains and archaeological skeletal collections is that the forensic series is a catastrophic mortality sample instead of a natural one. The catastrophic origin of the cemetery is most likely to affect the prevalence of lesions on subadults, many of whom would otherwise have survived to adulthood. Accordingly, we focus on porotic hyperostosis in adult remains.

Unfortunately, many of the remains were calcined and fragmentary. Only 47 crania of people over 15 years at death could be scored for either cribra orbitalia or porotic hyperostosis. Since the lesions are usually bilaterally symmetrical, we required only one observable side. For porotic hyperostosis, we scored those crania that had more than two-thirds of the parietal bones and occipital squama fully observable, including the region between the parietal boss and lambda, which is typically affected. Most could be scored for either porotic hyperostosis or cribra orbitalia, but not both.

Of these, 3 of 26 skulls with observable orbits show cribra orbitalia and 3 of 28 show porotic hyperostosis. These are not the same 3 individuals. The orbital lesions are extremely mild. A young adult female, PS-VIII-8, shows remodeled porosity with coalescence of the foraminae in the orbital roof that can clearly be identified as cribra orbitalia, but the other two exhibit extremely slight porosity and surface irregularity that might also have been the result of a healed infectious process (PS-XIII-3, a middle-adult female; and PS-IX-3, a middle-adult male).

The three cases of porotic hyperostosis are likewise very mild. The skull of one male aged 15 to 20 years at death, PS-II-3, which shows sclerotic porosity near lambda on both the parietal and occipital, is the most evident case (Figure 4). Two others show very slight porosity, which can be identified as porotic hyperostosis with somewhat less confidence. These are skeleton PS-X-1, a 17-year-old

female with very slight porosity of the occipital squama, and PS-XX-2, a middle-adult male with isolated porotic lesions of the parietal. None of the skeletons show thickening of the cranial vault or formation of a coral-like network of hyperostotic bone, features that are observed in prehistoric Maya skeletal remains.

Whether we consider each of these cases as bona fide anemic scars or not, it is clear that dramatically fewer modern skeletons show bone changes due to anemia than in archaeological Maya series, and those few lesions are much less severe. For porotic hyperostosis, a maximum of 10.7% of forensic skulls show anemic changes while 11.5% show cribra orbitalia. By comparison, porotic hyperostosis occurs on more than 5 times as many archaeological crania from Classic inland sites. The rarity of porotic hyperostosis at sites from around the beginning of the Colonial Period (Iximché, Lamanai, Tipu) is similar to that seen among the forensic series. Although the forensic sample is small, so too are many of the prehistoric samples. Chi square tests confirm that the abundance of porotic hyperostosis at Plan de Sanchez is dramatically less than at Copán (Yates-corrected χ^2, $p = 0.0003$) or in the Pasión region (Yates-corrected χ^2, $p = 0.0001$), but is comparable to the abundance at Iximché (Yates-corrected χ^2, $p = 0.86$). Certainly data from additional forensic samples are desirable to confirm this pattern.

Discussion

Although the etiology of porotic hyperostosis is well documented, little research has addressed the severity and duration of anemia required to initiate the bone changes. Theoretically, we can expect bone changes to occur only in patients whose compensatory erythropoietic mechanisms are activated by chronic anemia. The 30% of rural Guatemalan children classified as anemic is more than double the incidence of porotic hyperostosis in adult skulls from Plan de Sanchez. But only about 12% of children 5 years old have anemia. This matches the 11% of Plan de Sanchez adults showing porotic hyperostosis surprisingly well and suggests substantial congruity between the measures. Recall that the age-specific anemia prevalence is not stratified for region or ethnicity. Hence, a slightly larger proportion of Rabinal 5-year-olds likely suffer anemia. It is also likely that anemia was slightly more prevalent prior to 1967 (when the Plan de Sanchez adult skeletons were children) than it is today, since other measures of childhood health show marked improvement over the last 30 years (MSPAS 1995). Porotic hyperostosis in adult skeletons likely underestimates the prevalence of anemia in older children.

By comparison, up to 65% of prehispanic children who survived to adulthood had experienced chronic anemia. This dramatic difference in porotic hyperostosis between the present and past cannot be put down to cemetery sampling bias. Since all segments of the population are included in the forensic sample (including many who did not attend the market), the adult sample is unlikely to be biased toward especially "healthy" individuals. Moreover, had the members of the Plan de Sanchez adult population lived out their natural lives, the lesion frequency might be lower due to remodeling of some lesions in elderly skeletons, but could not be higher. Some confirmation of the generality of the trend comes from Haley's (1975) survey of radiologists in Guatemala, none of whom had observed radiological signs of anemia in their practices.

Were Classic Period children dramatically less healthy than modern children are? This discrepancy might be explained by change in three different factors: (1) diet, (2) infection, (3) mortality, or by a combination of them.

(1) Dietary Continuity?

Since nutrition is the most important factor explaining modern anemia, we turn first to evaluate the analogy between modern and ancient diets. Dependence on maize is a key dietary factor implicated in low iron intake and absorption. Data from the 1960s indicate that maize consumption provided 70% of calories and 62% of proteins ingested by adults in indigenous communities in Sacatepequez. For preschool children, consumption of milk products and processed sugar was greater, so that cereals provided 62% of calories and 54% of protein (Flores et al. 1964). These are the best data available for comparison with the population of Plan de Sanchez prior to 1970. A 1991 national survey (SEGEPLAN 1993) found that maize provided 45% of calories to rural households in the country as a whole. Maize consumption is now offset by wheat and processed sugar, which provide 24.6% of calories. Together, plant foods contribute 82% of protein in rural diets, with meat providing only 18% (SEGEPLAN 1993). Although maize consumption may have declined recently, it does not appear to have been replaced with animal foods that would have improved iron status.

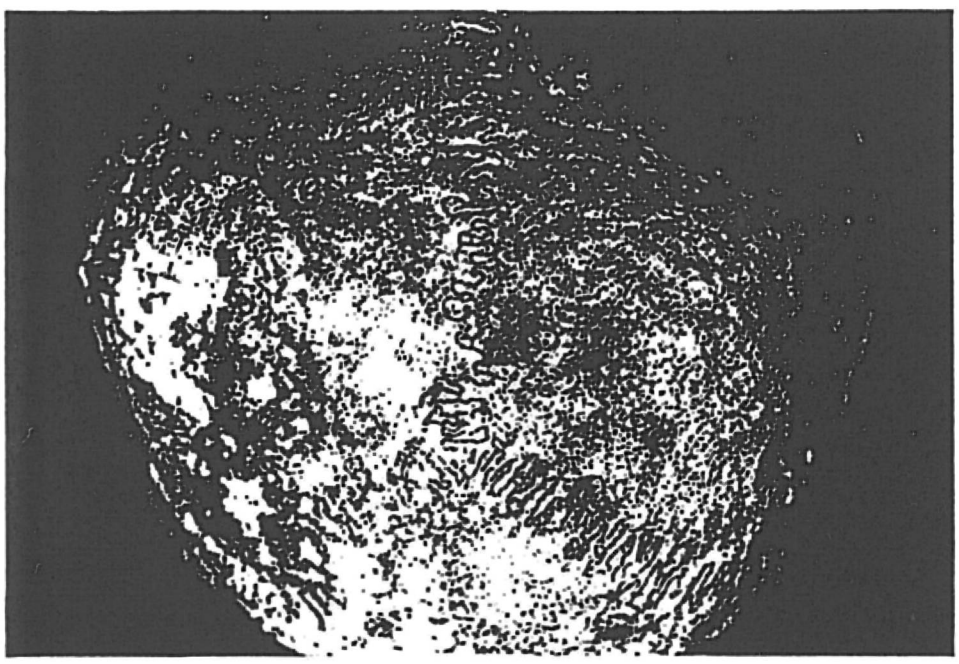

Figure 4. Healed porotic hyperstosis in Plan de Sanchez skeleton PS-II-3.

For the prehispanic Maya, we can estimate maize consumption from the stable carbon isotope ratios of bone. The relative abundance of the two stable isotopes of carbon, ^{12}C and ^{13}C, in plants are determined by the photosynthetic pathways used. Maize, which uses the C4 pathway, incorporates proportionately more ^{13}C than do plants using the C3 pathway. These isotopic signals, designated as $\delta^{13}C$ and measured in units of permil (‰) relative to the standard Pee Dee Belemnite (PDB), are passed on to the tissues of consumers and can be measured in archaeological bone. Since maize was virtually the only C4 food consumed in the Maya area, $\delta^{13}C$ ratios serve as a measure of maize consumption. Classic Period Lowland Maya skeletons have bone collagen $\delta^{13}C$ averaging -17 to -8‰, indicating heavy reliance on maize (Wright and White 1996). Because collagen is constructed with emphasis on dietary proteins (Ambrose and Norr 1993), these data indicate that maize provided up to 70% of protein consumed. Bone mineral carbonate tracks the weight % of all sources of ingested carbon (Ambrose and Norr 1993) and provides a better measure of maize consumption, if not contaminated by soil carbonates. Carbonate $\delta^{13}C$ of adult bone ranges from -7.5 to -3.0‰ (Gerry and Krueger 1997; Wright and Schwarcz 1996), indicating that an average of 53 to 81% of carbon atoms came from maize.[1]

For children, dental enamel records diet $\delta^{13}C$ during the development of teeth. The $\delta^{13}C$ of third molar enamel, developing between ages 9 and 12 years, has been measured to be -2.0‰ at Postclassic Topoxté ($N = 14$) (Acevedo et al. 1997), -2.6‰ at Classic Tikal ($N = 9$) (Wright, unpublished data), and -3.1‰ for Classic "royal" burials from Copán ($N = 7$) (Wright and Buikstra, unpublished data). These would correspond to 88%, 84%, and 80%, respectively, of carbon atoms obtained from maize. At highland Kaminaljuyu, premolars average -2.9‰ ($N = 36$) (Wright and Schwarcz 1998), indicating that 82% of carbon in the diets of children aged 3 to 6.5 years was from maize. These data imply that maize contributed 80 to 90% of the carbon atoms ingested, an estimate that is remarkably close to the 80% consumed today. Albeit a crude comparison, ancient diets did parallel modern diet in terms of the total bulk of maize eaten.

Perhaps more significant than the absolute consumption of maize (and iron obtained from maize) is the inhibitory effect of calcium carbonate in lime-processed maize on the intestinal absorption of iron (Prather and Miller 1992). This recently discovered chemical interaction means that very little of ingested iron—whatever its source—can be absorbed if eaten together with lime-processed maize foods, such as tamales or tortillas. Given that alkaline processing is practiced by all maize-reliant modern cultures (Katz et al. 1974), we expect that maize was also treated with lime prehistorically (e.g., Nations 1979).

Other foods also contribute substantially to iron status. The INCAP data find poultry eggs and bread to be significant sources of dietary iron in all regions of the country, and these sources were unavailable in the past. Presumably these have slightly offset consumption of other foods, such as wild game, fish, and snails. Most meat consumed today is beef, usually a cash purchase, that provides about 7% of protein consumed by both adults and children (Flores et al. 1964), which supplies 1 to 4% of children's iron outside the capital city (MSPAS 1996:82).

Prehistoric protein consumption can be examined using stable nitrogen isotopes that show trophic enrichment during digestion (DeNiro and Epstein 1981). Prehistoric Maya adult bone collagen ranges in $\delta^{15}N$ from 7 to 11‰, overlapping the $\delta^{15}N$ of both herbivore and carnivore collagen (Wright and White 1996). Hence, many individuals ate significant quantities of meat that would have been good sources of iron. However, on average, it is likely that meat provided less than half of dietary proteins. Although difficult to estimate with any precision, the $\delta^{15}N$ of prehistoric bone raises the possibility that meat consumption was perhaps greater in the past than today.

Changing consumption of plant foods is unlikely to have dramatically improved iron status in the twentieth century. The most significant change may be in Vitamin C sources. Old World citrus fruits and bananas provide nearly half of Vitamin C intake today (Flores et al. 1964) and probably raised Vitamin C intake above prehispanic levels, improving iron absorption slightly. Although vegetables that were introduced to Maya diets after the conquest may have elevated Vitamin A intake slightly over prehispanic levels, when Vitamin A came primarily from chili peppers and greens, absorption of betacarotenes like Vitamin A is poor unless eaten with adequate fats. Modern Maya diets, like prehispanic ones, remain very low in fat.

Given the broad parallels between the past and present diets, the slight variations in diet between past and present seem inadequate to account for the dramatic difference in porotic hyperostosis. At worst, prehispanic diets seem to lie within the range documented across the country today and are insufficient to account for the large discrepancy in porotic hyperostosis.

(2) Parasitism and Chronic Infection

Were parasitism an important contributor to anemia, we should expect a heavier toll of hookworm at the Classic Lowland sites where porotic hyperostosis is so abundant. We would also expect more intestinal parasitism in the densely populated Classic Lowland cities than in the small mountain villages. Yet, *Ascaris* and *Trichuris* are also abundant in the highlands and infect younger children with greater intensity than hookworm, so they may contribute to early childhood anemia (Anderson et al. 1993; Scrimshaw and Tejada 1970). Evidence for the prehispanic presence of *Ascaris* in the New World is tenuous, but *Trichuris* is well documented (Horne 1985; Reinhard 1990). As *Necator*

is thought to have been introduced from the Old World and is today the predominant hookworm in Guatemala, one could hypothesize that its introduction led to a decline in *Ancylostoma,* the indigenous species. Since *Ancylostoma* causes greater bleeding (Stoltzfus et al. 1997), this shift might have reduced the impact of hookworm on anemia. Alternately, the addition of the new species may have exacerbated anemia. Unfortunately, we have no way to test such changes.

If, indeed, chronic infection produces anemia through iron-withholding, other infectious conditions may contribute to overall iron status. Prehispanic Maya skeletons often show bilateral periostoses that compare well with those expected from an endemic treponematosis (Saul 1972; Whittington 1989; Wright 1994). Today, yaws is seldom diagnosed in Guatemala, but it was endemic in lowland Suchitepequez during the 1950s (Scrimshaw and Tejada 1970:216). But porotic hyperostosis and periosteal reactions are not correlated in archaeological skeletons (Wright 1994:331–332), so there is little evidence to suggest that such chronic infections had complications for iron status in the prehispanic lowlands.

(3) Mortality and Anemia

The decline in anemia with increasing age shown in Figure 3 might be interpreted to indicate that children get healthier as they grow up. But, if we take into account that many children die during the first 5 years of life, one probable explanation of the trend is that anemic children die young, leaving healthier children in the older age groups. Anemic children often suffer from diarrhea and respiratory infections. Respiratory disease accounted for 19% of deaths in children under 15 years that were documented by INCAP in 1956–57 in 4 indigenous villages in Sacatepequez. Kwashiorkor accounted for a further 19% of deaths (Scrimshaw and Tejada 1970:205). Mortality likely affects anemic and nonanemic children differentially; malnourished anemic children are frail and may be removed from the population before they reach age 5. We suspect that if all one-year-old children were to survive to adulthood, porotic hyperostosis would be much more abundant on modern remains.

Therefore, we raise the possibility that differences in mortality levels might be responsible for the large difference in porotic hyperostosis. In 1995, childhood mortality (from birth to 5 years) in Guatemala as a whole is estimated to have been 79 deaths per 1,000 live births, or 7.9%. Indigenous childhood mortality is estimated to have been 9.4% (MSPAS 1995). However, these values represent a very recent improvement, with infant mortality rates (under 1 year) cut in half since 1970 (MSPAS 1995; Population Council 1990). For the Highland Maya, Early (1982) suggests that the decline in deaths during the second and third year of life, which characterizes Phase II of the demographic transition, only began in the 1960s. The Plan de Sanchez adults were born prior to 1967, when total childhood mortality was probably on the order of 20% to 25%. If anemic children die first, early childhood mortality today may account for much of the documented decline in anemia with age.

We can suggest two reasons why childhood mortality might have been lower in prehispanic times than it was for most of the twentieth century. Most obvious is that children today contend with a host of infectious diseases brought from the Old World by the Spanish conquistadors (Lovell 1985; Merbs 1992). Although accounting for fewer deaths than respiratory infection and malnutrition, these pathogens are important causes of death today. The WHO reports that measles and whooping cough—two Old World diseases—accounted for 19% and 4% of deaths between ages 1 and 4 years in 1979, respectively, and 10% and 3% of deaths in 1981 (WHO 1983:52, 1985:100). Moreover, Old World bacteria and viruses likely contribute to infantile diarrhea and respiratory infection. Although some of the children who ultimately die from these imported pathogens might well have died from indigenous infections, it is likely that the arrival of Old World pathogens made survival of early childhood even more challenging to Maya children than it had been before the conquest.

The second cause of higher mortality today is a result of the recent trend to shorten the period of breastfeeding, as first suggested by Behár (1968). Studies conducted around the world have documented that breastfeeding provides immunological benefits to toddlers (Ogra and Losonsky 1984), in addition to nutrients that augment solid foods. Once they stop nursing, children no longer receive this immune boost, and they also take in a higher bacterial load by drinking contaminated water and eating contaminated food instead of clean breast milk. Many studies have shown that breastfeeding reduces the risk of infantile diarrhea, a major cause of death in developing countries. In Guinea-Bissau, West Africa, Mølbak et al. (1994) have shown that children aged 12 to 35 months who were not breastfed suffered more frequent and longer duration diarrheal episodes than did breastfed children. Significantly, weaned infants suffered 3.5 times higher mortality than did their breastfed peers (Mølbak et al. 1994).

Ethnohistoric and ethnographic data suggest that the duration of lactation has been dramatically reduced during recent centuries. Today the average duration of breastfeeding in rural indigenous communities in Guatemala is 22 months (Pérez-Escamilla 1993; Population Council 1990). But for the sixteenth-century Yucatec Maya, Bishop Diego de Landa observed that children often continued to nurse until 4 years of age (Tozzer 1941:125).

Information on prehistoric weaning patterns is more difficult to glean. Breastfed infants essentially feed at a trophic level above their mothers, so the supplementation of breast milk with protein from solid foods can be inferred

from the $\delta^{15}N$ of bone collagen of infants who died young (Fogel et al. 1989). For the Maya, few studies have presented detailed age-at-death information for juveniles analyzed. At Topoxté, Acevedo et al. (1997) found 2 skeletons with enriched $\delta^{15}N$ aged 9 months and 4 years at death, indicating that breast milk provided most protein to these infants until their deaths. However, one 18-month-old infant had $\delta^{15}N$ comparable to adult skeletons, indicating that it had begun to consume significant quantities of solid foods prior to its death. These data demonstrate considerable variability in infant feeding patterns at this site and, of course, are biased toward *deceased* children, not survivors. $\delta^{15}N$ analyses of larger numbers of infant skeletons are needed before the average age of supplementation can be estimated for the lowland Maya.

Stable oxygen isotope ratios of tooth enamel shed light on weaning age for individuals who survived to adulthood (Wright and Schwarcz 1998). Because body water is enriched in ^{18}O over drinking water, breastfed infants show heavier $\delta^{18}O$ than weaned infants (Roberts et al. 1988). Thus, tooth enamel deposited while children are nursing is heavier than that formed after weaning. Comparing the $\delta^{18}O$ of teeth in 35 skeletons from Kaminaljuyu in the Guatemalan highlands, Wright and Schwarcz (1998) have shown that breastfeeding continued perhaps until 5 years of age there, although infants began to consume solid foods by 2 years of age.

Although sparse, the stable isotope data do support the ethnohistoric suggestion that lactation for 4 years was probably common among the prehispanic Maya and was perhaps as much as 2 or 3 years longer than the mean duration of breastfeeding today. Ongoing research on Classic Period Lowland skeletons will help to confirm this point. Given that so many modern infants die from complications of malnutrition, reduction in weaning age from 4 to 2 years among the Maya may have come with a substantial cost in mortality.

Unfortunately, attempts at paleodemographic reconstruction in the Maya area are severely compromised by poor preservation, subadult underenumeration, and excavation biases. Even at Copán, which boasts perhaps the largest and most representative Classic Maya skeletal series, osteological data do not allow adequate estimation of childhood mortality (Paine 1992). It is interesting to note that Paine (1992:228–233) uses pretransition highland Guatemalan mortality statistics as a point of departure for his paleodemographic analysis and then assumes that Classic Copán mortality must have been equal to or higher than these modern levels, but does not consider the possiblity that it might have been lower. While we suggest lower prehistoric mortality than in the first half of this century, it may have been higher than it is today, and we cannot infer an exact level from the data at hand. Note that our hypothesis does not necessarily conflict with environmental models of the Maya collapse which posit a demographic crisis at the end of the Classic Period, because such a crisis could have arisen rapidly. As discussed elsewhere, there is little paleopathological evidence for a long, gradual deterioration in health over the span of Maya history (Wright and White 1996).

Conclusions

We conclude that much of the large difference that we see in the percent of adults with scars from childhood anemia is not due to substantially greater malnutrition or parasitism among the ancient Maya than their modern descendants. Instead, in the past, a greater proportion of children survived their childhood than they do today, albeit with anemia. In this case, the "osteological paradox" (Wood et al. 1992) may well ring true. The rarity of porotic hyperostosis on the skulls of modern forensic skeletons may be due to a heavier burden of infectious disease, earlier weaning, and thus higher childhood mortality when compared to the prehistoric condition. Although it seems counterintuitive, this implies that ancient Maya children lived in a *healthier environment* than do Maya children of the twentieth century, or at least one in which they were exposed to fewer diseases that might have killed them. Moreover, they were better protected, by continued breastfeeding, to cope with the pathogens they encountered in early childhood. Paradoxically, this lower disease burden in the past resulted in greater survival of frail children and more abundant evidence of healed skeletal pathology among adults.

The argument that childhood health is more compromised in the present than it was in the past is supported by data on stature reduction. As Stewart (1949, 1953) observed, modern Maya peoples are shorter than were their prehispanic ancestors. In a recent review, Danforth (1994) has shown that this stature reduction occurred in later colonial times. For instance, the colonial Maya of Tipu, like those of the Classic Period, are taller than modern Maya (Cohen et al. 1994; Danforth 1994). In addition to withstanding anemia and fending off early death, prehispanic children were better able to reach their growth potential than are Maya children today.

However, mortality is unlikely to be the sole reason for the difference in porotic hyperostosis between past and present Maya skeletons. Certainly, nutritional and parasitic differences also play some role. Although maize and meat consumption were in the range of modern diets, vegetable sources of Vitamins A and C have changed, perhaps ameliorating iron deficiency. High population density in the prehispanic lowland cities may have compounded the effects of infection and parasites on iron status more than they do today. Moreover, we recognize that the Plan de Sanchez skeletal series is small; analyses of additional forensic samples may force us to rethink our conclusions.

The contrast between the epidemiological circumstances in which Modern Maya peoples live and those of

the past has a profound consequence for the abundance of skeletal lesions in archaeological series. An explicit use of analogy with modern skeletal health has helped to bring the role of mortality into focus for comparative studies of prehistoric health. Our results highlight the extent to which epidemiological circumstances and mortality may affect the abundance of skeletal lesions and illustrate that the arguments of Wood and colleagues (1992) are indeed sage cautions. While we find support for a "paradoxical" interpretation of the prevalence of porotic hyperostosis for this specific case, we do not believe that comparable interpretations would be valid for many health changes that paleopathologists have described to date. Instead, multiple sources of evidence must be brought to bear when interpreting differences in lesion abundance between skeletal series, as for instance, in the case of the health consequences of the adoption of sedentism and agriculture by foragers (Cohen 1994, 1997; Goodman 1993). Such profound epidemiological differences as between the Maya present and past might not occur in most paleopathological comparisons undertaken in routine bioarchaeological work; however, the potential magnitude of this osteological paradox should not be ignored by researchers documenting health changes over human history. Although local indigenous populations may not be available for many world areas, or may be inappropriate analogs due to marked subsistence change or medical intervention, we encourage paleopathologists to explore such forensic opportunities where they are available.

Notes

Acknowledgments. This research was conducted in collaboration with the Forensic Anthropology Team of Guatemala, who encourage other osteologists to explore the research potential of ongoing forensic investigations. We thank the members of the team for their enthusiasm, generosity, and dedication. Pathological data were collected from the Plan de Sanchez skeletons with financial assistance from the Hannah Institute for the History of Medicine in Toronto, Canada. This project was approved by the Ethics Committee at McMaster University during Wright's tenure of a SSHRC Postdoctoral Fellowship there. We thank Kathy Dettwyler, Jane Buikstra, Joan Baker, and three *AA* reviewers for their critical comments, but remain responsible for any errors herein. We dedicate this paper to the people of Rabinal, in memory of their past and with hope for the future.

1. Assuming recent atmospheric depletion of 1.5‰ in $\delta^{13}C$ (Tieszen and Fagre 1993), end-member food compositions are calculated to be -25.5‰ for C3 and -9.5‰ for C4. Using a diet-carbonate offset of +9.5‰ (Ambrose and Norr 1993), the estimates assume foods to be either C3 or C4 and do not take into account foods of intermediate values, such as are expected for animal meats and CAM plants. Thus, these should be taken as maximal estimates of maize consumption.

References Cited

Acevedo, Renaldo, Lori E. Wright, Bernard Hermes, and Henry P. Schwarcz
1997 La dieta Maya después del colapso: un estudio isotópico del sitio topoxté. *In* X simposio de investigaciones arqueologicas en Guatemala, 1996, vol. 2. J. P. Laporte and H. L. Escobedo, eds. Pp. 587–600. Guatemala City: Ministerio de Cultura y Deportes.

Ambrose, Stanley H., and Lynette Norr
1993 Experimental Evidence for the Relationship of the Carbon Isotope Ratios of Whole Diet and Dietary Protein to Those of Bone Collagen and Carbonate. *In* Prehistoric Human Bone: Archaeology at the Molecular Level. J. Lambert and G. Grupe, eds. Pp. 1–37. Berlin: Springer-Verlag.

Anderson, Timothy J. C., Claire A. Zizza, Guisela M. Leche, Marilyn E. Scott, and Noel W. Solomons
1993 The Distribution of Intestinal Helminth Infections in a Rural Village in Guatemala. Mem. Inst. Oswaldo Cruz 88(2):53–65.

Angel, J. Lawrence
1966 Porotic Hyperostosis, Anemias, Malarias and the Marshes in Prehistoric Eastern Mediterranean. Science 153:760–762.

Beck, J. Walter, and Elizabeth Barrett-Connor
1971 Medical Parasitology. St. Louis: C.V. Mosby Co.

Behár, Moises
1968 Food and Nutrition of the Maya before the Conquest and at the Present Time. *In* Biomedical Challenges Presented to the American Indians. Pp. 114–119. Scientific Publication no. 165. Washington, DC: Pan American Health Organization.

Benedict, Francis G., and M. Steggerda
1937 The Food of the Present-Day Maya Indians of Yucatan, vol. 3, No. 18. Pp. 155–188. Washington, DC: Carnegie Institution of Washington.

Boas, Franz
1890 Cranium from Progreso, Yucatan. Proceedings of the American Antiquarian Society 6:350–359.

Bressani, Ricardo, Ramiro Paz y Paz, and Nevin S. Scrimshaw
1958 Chemical Changes in Corn during Preparation of Tortillas. Agric. Food Chem. 6(10):770–774.

Cohen, Mark Nathan
1994 The Osteological Paradox Reconsidered. Current Anthropology 35(5):629–631.
1997 Does Paleopathology Measure Community Health? A Rebuttal of "The Osteological Paradox" and Its Implication for World Prehistory. *In* Integrating Archaeological Demography: Multidisciplinary Approaches to Prehistoric Population. R. R. Paine, ed. Pp. 242–260. Center for Archaeological Investigations, Occasional Paper No. 24. Carbondale: Southern Illinois University.

Cohen, Mark N., and George J. Armelagos
1984 Paleopathology at the Origins of Agriculture. New York: Academic Press.

Cohen, Mark N., K. O'Connor, M. Danforth, K. Jacobi, and C. Armstrong
1994 Health and Death at Tipu. *In* In the Wake of Contact: Biological Responses to Conquest. C. S. Larsen and G. Milner, eds. Pp. 121–133. New York: Wiley-Liss.

Danforth, Marie Elaine
1994 Stature Change in Prehistoric Maya of the Southern Lowlands. Latin American Antiquities 5(3):206–211.
de Vizia, B., V. Poggi, P. Vajro, S. Cucchiara, and A. Acampora
1985 Iron Malabsorption in Giardiasis. Journal of Pediatrics 107:75–78.
DeNiro, Michael J., and Samuel Epstein
1981 Influence of Diet on the Distribution of Nitrogen Isotopes in Animals. Geochimica Cosmochimica Acta 45:341–351.
Dettwyler, Katherine
1999 Evolutionary Medicine and Breastfeeding: Implications for Research and Pediatric Advice. The 1998–99 David Skomp Distinguished Lecture in Anthropology. Bloomington, IN: Department of Anthropology, Indiana University.
EAFG (Equipo de Antropología Forense de Guatemala)
1997 Las masacres en Rabinal: estudio histórico antropológico de las masacres de Plan de Sánchez, Chichupac y Río Negro. Guatemala City: Editorial Serviprensa.
Early, John D.
1982 The Demographic Structure and Evolution of a Peasant System: The Guatemalan Population. Boca Raton: University Presses of Florida.
El-Najjar, Manmoud Y., Dennis J. Ryan, Christy G. Turner II, and Betsy Lozoff
1976 The Etiology of Porotic Hyperostosis among the Prehistoric and Historic Anasazi Indians of the Southwestern United States. American Journal of Physical Anthropology 42:477–488.
Flores, Marina, Berta García, Zoila Flores, and Marta Yolanda Lara
1964 Annual Patterns of Family and Children's Diet in Three Guatemalan Indian Communities. British Journal of Nutrition 18:281–293.
Fogel, Marilyn L., Noreen Turross, and Douglas Owsley
1989 Nitrogen Isotope Tracers of Human Lactation in Modern and Archaeological Populations. *In* Annual Report of the Director, Geophysical Laboratory, Carnegie Institution of Washington, 1988–1989. Pp. 111–116. Washington, DC.
Gerry, John P., and Harold Krueger
1997 Regional Diversity in Classic Maya Diets. *In* Bones of the Maya: Studies of Ancient Skeletons. S. Whittington and D. Reed, eds. Pp. 196–207. Washington, DC: Smithsonian Institution Press.
Goodman, Alan
1993 On the Interpretation of Health from Skeletal Remains. Current Anthropology 34(3):281–288.
1994 Cartesian Reductionism and Vulgar Adaptationism: Issues in the Interpretation of Nutritional Status in Prehistory. *In* Paleonutrition: The Diet and Health of Prehistoric Americans. K. D. Sobolik, ed. Pp. 163–177. Center for Archaeological Investigations, Occasional Paper No. 22. Carbondale: Southern Illinois University.
Haley, Harold B.
1975 Bone Disease in Contemporary Maya. 41st International Congress of Americanists 41(1):67–70.
Haviland, William A.
1969 A New Population Estimate for Tikal, Guatemala. American Antiquities 34:429–433.
Hengen, O. P.
1971 Cribra Orbitalia: Pathogenesis and Probable Etiology. Homo 22:57–75.
Hill, M. Cassandra, and George J. Armelagos
1991 Porotic Hyperostosis in Past and Present Perspective. *In* A Life in Science: Papers in Honor of J. Lawrence Angel. J. E. Buikstra, ed. Pp. 52–63. Scientific Papers No. 6. Kampsville, IL: Center for American Archaeology.
Holland, Thomas D., and Michael J. O'Brien
1997 Parasites, Porotic Hyperostosis, and the Implications of Changing Perspectives. American Antiquities 62(2):183–193.
Hooton, Earnest A.
1940 Skeletons from the Cenote of Sacrifice at Chichen Itzá. *In* The Maya and Their Neighbors. C. L. Hay, ed. Pp. 272–280. New York: D. Appleton-Century.
Horne, Patrick D.
1985 A Review of the Evidence of Human Endoparasitism in the Pre-Columbian New World through the Study of Coprolites. Journal of Archaeological Science 12:299–310.
Jackes, Mary
1993 On Paradox and Osteology. Current Anthropology 34(4):434–438.
Katz, S. H., M. L. Hediger, and L. A. Valleroy
1974 Traditional Maize Processing Techniques in the New World. Science 184:765–773.
Krause, V. M., K. L. Tucker, H. V. Kuhnlein, C. Y. Lopez-Palacios, M. Ruz, N. W. Solomons
1992 Rural-Urban Variation in the Calcium, Iron, Zinc and Copper Content of Tortillas and Intake of These Minerals from Tortillas by Women in Guatemala. Ecology of Food and Nutrition 28:289–297.
Lallo, John W., George J. Armelagos, and Robert P. Mensforth
1977 The Role of Diet, Disease, and Physiology in the Origin of Porotic Hyperostosis. Human Biology 49(3):471–483.
Lovell, W. G.
1985 Conquest and Survival in Colonial Guatemala: A Historical Geography of the Cuchumatan Highlands, 1500–1821. Montreal: McGill-Queen's University Press.
Lozoff, Betty, E. Jimenez, and A. W. Wolf
1991 Long-Term Developmental Outcome of Infants with Iron Deficiency. New England Journal of Medicine 325:667–694.
Lundell, Cyrus L.
1939 Plants Probably Utilized by the Old Empire Maya of Petén and Adjacent Lowlands. Papers of the Michigan Academy of Sciences 24(1):37–56.
Lynch, S. R.
1997 Interaction of Iron with Other Nutrients. Nutrition Reviews 55:102–110.
Marcus, Joyce
1982 The Plant World of the Sixteenth- and Seventeenth-Century Lowland Maya. *In* Maya Subsistence: Studies in Honor of Dennis Puleston. K. V. Flannery, ed. Pp. 239–273. New York: Academic Press.
Márquez Morfin, Lourdes, M. E. Peraza, J. Gamboa, and T. Miranda
1982 Playa del Carmen: una población de la costa oriental en el postclásico (Un Estudio Osteologico). Colección

Cientifica 119. Mexico: Instituto Nacional de Anthropología e Historia.
Martorell, Reynaldo
1992 Overview of Long-Term Nutrition Intervention Studies in Guatemala, 1968–1989. Food and Nutrition Bulletin 14:270–277.
Mejía, Luis A., and Francisco Chew
1988 Hematological Effect of Supplementing Anemic Children with Vitamin A Alone and in Combination with Iron. American Journal of Clinical Nutrition 48:595–600.
Mensforth, Robert P., C. Owen Lovejoy, John W. Lallo, and George J. Armelagos
1978 The Role of Constitutional Factors, Diet, and Infectious Disease in the Etiology of Porotic Hyperostosis and Periosteal Reactions in Prehistoric Infants and Children. Medical Anthropology 2(1):1–57.
Merbs, Charles F.
1992 A New World of Infectious Disease. Yearbook of Physical Anthropology 35:3–42.
Mølbak, Kåre, A. Gottschau, P. Aaby, N. Højlyng, L. Ingholt, and A. P. J. da Silva
1994 Prolonged Breast Feeding, Diarrhoeal Disease, and Survival of Children in Guinea-Bissau. British Medical Journal 308:1403–1406.
Moscoso Moller, Fernando
1994 Contribuciones de la arqueología y antropología forense a la evolución del sistema legal en Guatemala. *In* VII Simposio de Investigaciones Arqueologicas en Guatemala, 1993. J. P. Laporte and H. L. Escobedo, eds. Pp. 41–46. Guatemala City: Ministerio de Cultura y Deportes.
Moseley, J. E.
1963 Bone Changes in Hematologic Disorders. New York: Grune and Stratton.
MSPAS (Ministeria de Salud Publica y Asistencia Social)
1995 Encuesta nacional de salud materno infantil 1995. Guatemala: Government of Guatemala.
1996 Informe de la encuesta Nacional de Micronutrientes, Guatemala, 1995. Guatemala: Government of Guatemala.
Nations, James D.
1979 Snail Shells and Maize Preparation: A Lacandon Maya Analogy. American Antiquities 44:568–571.
Ogra, Pearay L., and Genevieve A. Losonsky
1984 Defense Factors in Products of Lactation. *In* Neonatal Infections: Nutritional and Immunologic Interactions. P. L. Ogra, ed. Pp. 67–87. Orlando: Grune and Stratton.
Paine, Richard R.
1992 Population Dynamics at Copan, Honduras, AD 450–1250: A Study in Archaeological Demography. Ph.D. dissertation, Department of Anthropology, Pennsylvania State University.
Palkovich, Ann M.
1987 Endemic Disease Patterns in Paleopathology: Porotic Hyperostosis. American Journal of Physical Anthropology 74:527–537.
Pérez-Escamilla, Rafael
1993 Breast-Feeding Patterns in Nine Latin American and Caribbean Countries. Bull. Pan American Health Organization 27(1):32–42.
Pollitt, E.
1994 A Development View of Cognition in the Undernourished Child. Nestle Foundation Annual Report 1994: 88–105.
Population Council
1990 Guatemala 1987: Results from the Demographic and Health Survey. Studies in Family Planning 21(1):55–59.
Prather, T. A. and D. D. Miller
1992 Calcium Carbonate Depresses Iron Bioavailability in Rats More Than Calcium Sulfate or Sodium Carbonate. Journal of Nutrition 122:327–332.
Reinhard, Karl J.
1990 Archaeoparasitology in North America. American Journal of Physical Anthropology 82(2):145–163.
1992 Patterns of Diet, Parasitism, and Anemia in Prehistoric West North America. *In* Diet, Demography and Disease: Changing Perspectives on Anemia. P. Stuart-Macadam and S. Kent, eds. Pp. 219–258. New York: Aldine de Gruyter.
Roberts, Charlotte, and Keith Manchester
1995 The Archaeology of Disease. Ithaca, NY: Cornell University Press.
Roberts, Susan B., W. A. Coward, G. Ewing, J. Savage, T. J. Cole, and A. Lucas
1988 Effect of Weaning on Accuracy of Doubly Labeled Water Method in Infants. American Journal of Physiology 254(4 Pt 2):R622–R627.
Roys, Ralph L.
1972 The Indian Background of Colonial Yucatan. Norman: University of Oklahoma Press.
Ryan, Alan S.
1997 Iron-Deficiency Anemia in Infant Development: Implications for Growth, Cognitive Development, Resistance to Infection, and Iron Supplementation. Yearbook of Physical Anthropology 40:25–62.
Sanders, William T.
1973 The Cultural Ecology of the Lowland Maya: A Reevaluation. *In* The Classic Maya Collapse. T. P. Culbert, ed. Pp. 325–366. Albuquerque: University of New Mexico Press.
Santley, Robert S.
1990 Demographic Archaeology in the Maya Lowlands. *In* Precolumbian Population History in the Maya Lowlands. T. P. Culbert and D. S. Rice, eds. Pp. 325–343. Albuquerque: University of New Mexico Press.
Santley, Robert S., Thomas W. Killion, and Mark T. Lycett
1986 On the Maya Collapse. Journal of Anthropological Research 42:123–159.
Saul, Frank P.
1972 The Human Skeletal Remains of Altar de Sacrificios: An Osteobiographic Analysis. Papers of the Peabody Museum of Archaeology and Ethnology, vol. 63, no. 2. Cambridge, MA: Harvard University Press.
1973 Disease in the Maya Area: The Pre-Columbian Evidence. *In* The Classic Maya Collapse. T. P. Culbert, ed. Pp. 301–324. Albuquerque: University of New Mexico Press.
1977 The Paleopathology of Anemia in Mexico and Guatemala. *In* Porotic Hyperostosis: An Enquiry. E. Cockburn, ed. Pp. 10–15. Monograph No. 2. Detroit, MI: Paleopathology Association.

1982 The Human Skeletal Remains from Tancah, Mexico. *In* On the Edge of the Sea: Mural Painting at Tancah-Tulum, Quintana Roo, Mexico. A. G. Miller, ed. Pp. 115–127. Washington, DC: Dumbarton Oaks.

Saul, F. P., and J. M. Saul
1991 The Preclassic Population of Cuello. *In* Cuello: An Early Maya Community in Belize. N. Hammond, ed. Pp. 134–158. Cambridge: Cambridge University Press.

Scrimshaw, Nevin S., and Carlos Tejada
1970 Pathology of Living Indians as Seen in Guatemala. *In* Handbook of Middle American Indians. T. D. Stewart, ed. Pp. 203–225, Vol. 9. Physical Anthropology. Austin: University of Texas Press.

SEGEPLAN, CADESCA, and INE
1993 Encuesta nacional de consumo aparente de Alimentos 1991. Guatemala City: Secretaria General de Planificacion Economica.

Shattuck, George C.
1933 The Peninsula of Yucatan. Publication 431. Washington, DC: Carnegie Institution of Washington.
1938 A Medical Survey of the Republic of Guatemala. Publication 499. Washington, DC: Carnegie Institution of Washington.

Shimkin, Demitri B.
1973 Models for the Downfall: Some Ecological and Culture-historical Considerations. *In* The Classic Maya Collapse. T. P. Culbert, ed. Pp. 269–300. Albuquerque: University of New Mexico Press.

Steggerda, Morris
1941 Maya Indians of Yucatan. Publication 531. Washington, DC: Carnegie Institution of Washington.

Stewart, T. Dale
1949 Notas sobre esqueletos humanos prehistoricos hallados en Guatemala. Antr. Hist. Guat. 1:23–34.
1953 Skeletal Remains from Zaculeu, Guatemala. *In* The Ruins of Zaculeu, Guatemala, vol. 1. R. B. Woodbury and A. S. Trik, eds. Pp. 295–311. New York: The United Fruit Company.

Stoltzfus, Rebecca J., Michele L. Dreyfuss, Hababuu M. Chwaya, and Marco Albonico
1997 Hookworm Control as a Strategy to Prevent Iron Deficiency Anemia. Nutrition Reviews 55(6):223–232.

Stuart-Macadam, Patty
1985 Porotic Hyperostosis: Representative of a Childhood Condition. American Journal of Physical Anthropology 66(4):391–398.
1987a Porotic Hyperostosis: New Evidence to Support the Anemia Theory. American Journal of Physical Anthropology 74(4):521–526.
1987b A Radiographic Study of Porotic Hyperostosis. American Journal of Physical Anthropology 74(4): 511–520.
1989 Porotic Hyperostosis: Relationship between Orbital and Vault Lesions. American Journal of Physical Anthropology 80(2):187–193.
1992 Porotic Hyperostosis: A New Perspective. American Journal of Physical Anthropology 87(1):39–47.

Tejada Valenzuela, Carlos
1970 Nutrición y prácticas alimentarias en Centroamérica. Universidad de San Carlos, Publicación Anual 1:74–111.

Tieszen, Larry L., and Tim Fagre
1993 Carbon Isotopic Variability in Modern and Archaeological Maize. Journal of Archaeological Science 20:25–40.

Tozzer, Alfred M.
1941 Landa's Relacion de las Cosas de Yucatan. Papers of the Peabody Museum of Archaeology and Ethnology, vol. 18. Cambridge, MA: Harvard University Press.

Tykot, Robert H., Nikolaas J. van der Merwe, and Norman Hammond
1996 Stable Isotope Analysis of Bone Collagen and Apatite in the Reconstruction of Human Diet: A Case Study from Cuello, Belize. *In* Archaeological Chemistry. Organic, Inorganic and Biochemical Analysis. M. V. Orna, ed. Pp. 355–365. ACS Symposium Series, vol. 625. Washington, DC: American Chemical Society.

Ubelaker, Douglas H.
1992 Porotic Hyperostosis in Prehistoric Ecuador. *In* Diet, Demography and Disease: Changing Perspectives on Anemia. P. Stuart-Macadam and S. Kent, eds. Pp. 201–217. New York: Aldine de Gruyter.

Virchow, Rudolf
1887 Schaedel von Mérida, Yucatán. Zeitschrift für Ethnologie 29:451–455.

Viteri, F. E.
1993 Guide for the Global Control of Nutritional Anemias and Iron Deficiency. Geneva: World Health Organization Nutrition Unit.

Walker, Phillip L.
1986 Porotic Hyperostosis in a Marine-Dependent California Indian Population. American Journal of Physical Anthropology 69(3):345–354.

Walter, T., I. de Andraca, P. Chadud, and C. G. Perales
1989 Iron Deficiency Anemia: Adverse Effects on Infant Psychomotor Development. Pediatrics 84:7–17.

Walter, Tomás, M. Olivares, F. Pizarro, and C. Muñoz
1997 Iron, Anemia, and Infection. Nutrition Reviews 55(4):111–124.

Weinberg, Eugene D.
1992 Iron Withholding in Prevention of Disease. *In* Diet, Demography, and Disease: Changing Perspectives on Anemia. P. Stuart-Macadam and S. Kent, eds. Pp. 105–150. New York: Aldine de Gruyter.

White, Christine D.
1988 The Ancient Maya from Lamanai, Belize: Diet and Health Over 2,000 Years. Can. Rev. Phys. Anthrop. 6(2): 1–21.

White, Christine D., Lori E. Wright, and David M. Pendergast
1994 Biological Disruption in the Early Colonial Period at Lamanai. *In* In the Wake of Contact: Biological Responses to Conquest. C. S. Larsen and G. Milner, eds. Pp. 135–145. New York: Wiley-Liss.

Whittington, Stephen L.
1989 Characteristics of Demography and Disease in Low Status Maya from Classic Period Copan, Honduras. Ph.D. dissertation, Department of Anthropology, Pennsylvania State University.

Whittington, Stephen L., and David M. Reed
1994 Los esqueletos de Iximché. *In* VII simposio de investigaciones arqueologicas en Guatemala, 1993. J. P. Laporte

and H. L. Escobedo, eds. Pp. 23–28. Guatemala: Ministerio de Cultura y Deportes.

1997 Commoner Diet at Copán: Insights from Stable Isotopes and Porotic Hyperostosis. *In* Bones of the Maya: Studies of Ancient Skeletons. S. L. Whittington and D. M. Reed, eds. Pp. 157–170. Washington, DC: Smithsonian Institution Press.

WHO (World Health Organization)

1968 Nutritional Anaemias: Report of a WHO Scientific Group. No. 405. Geneva: World Health Organization.

1983 World Health Statistics Annual. Geneva: World Health Organization.

1985 World Health Statistics Annual. Geneva: World Health Organization.

Wood, James W., George R. Milner, Henry C. Harpending, and Kenneth M. Weiss

1992 The Osteological Paradox: Problems of Inferring Prehistoric Health from Skeletal Samples. Current Anthropology 33(4):343–370.

Wright, Lori E.

1994 The Sacrifice of the Earth? Diet, Health, and Inequality in the Pasión Maya Lowlands. Ph.D. dissertation, Department of Anthropology, University of Chicago.

Wright, Lori E., and Henry P. Schwarcz

1996 Infrared and Isotopic Evidence for Diagenesis of Bone Apatite at Dos Pilas, Guatemala: Paleodietary Implications. Journal of Archaeological Science 23(6):933–944.

1998 Stable Carbon and Oxygen Isotopes in Human Tooth Enamel: Identifying Breastfeeding and Weaning in Prehistory. American Journal of Physical Anthropology 106(1): 1–18.

Wright, Lori E., and Christine D. White

1996 Human Biology in the Classic Maya Collapse: Evidence from Paleopathology and Paleodiet. Journal of World Prehistory 10(2):147–198.