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A Critical Evaluation of the Frequency of Anemia in Native American Populations

Kristina Marie Stoeppler
Appalachian State University

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Analyzing anemia in paleopathological studies facilitates the understanding of past life-ways by clarifying the effects of disease and diet on human populations. By determining disease states that were frequent in particular populations, hypotheses can be developed as to why certain populations were more vulnerable to stress than others. The presence of anemia, in particular, can give insight into the adequacy of a population's diet as well as their susceptibility to particular diseases.

Anemia, literally meaning "no blood," is a condition characterized by a depressed concentration of hemoglobin or packed cell volume (Stuart-Macadam and Kent 1992). Red blood cells in anemic individuals are therefore not as efficient in carrying oxygen throughout the body. There is a wide range of symptoms associated with anemia, including; pallor, fatigue, poor appetite, gastrointestinal problems, depression of neurological function, menstrual disturbances, poor physical growth, lowered immunity, higher maternal morbidity, and child mortality during pregnancy (Ramakrishnan 2001; Underwood 2001).

In this study I examined a 16th century, North American skeletal collection from the Sully site in South Dakota. I illustrate the population's general health status by comparing the frequency of dietary anemia to available diet and to potential anemia producing pathogens. In addition, this study aims to gain a better understanding of the Sully data through comparisons of two contemporaneous populations: Libben and Mouse Creek. The purpose of these comparisons is to better understand the biological variability among these three cultures. In doing so, this study provides critical information for understanding the effects of anemia by examining the adequacy of the population's diet, sanitation practices, and association with parasites and pathogens.

PALEOPATHOLOGICAL DIAGNOSIS OF ANEMIA

A paleopathological diagnosis of anemia is established through the observation of porotic hyperostosis and/or cribra orbitalia. Mann and Murphy (1990) define porotic hyperostosis as 0.5 to 2.0 mm sieve-like holes affecting the outer table of

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the cranium and the diploe (space between the two bone layers of the cranium), accompanied by increased vault thickness (resembling a porous orange peel). Cribra orbitalia is defined as porosity and/or expansion of the superior orbital plates during childhood (Mann and Murphy 1990). This condition is usually bilateral and appears as small to large holes in the upper surface of the orbits. Both of these cranial lesions (porotic hyperostosis and cribra orbitalia) occur when red blood cells are inadequately functioning. To compensate for the inadequacy of the red blood cells to carry oxygen, the body generates more of them. Consequently, the bone marrow cavity in the cranium (the diploe) will expand in order to allow for production of additional red blood cells (Boyd 1984). As the diploic space expands, it is thought to wear through the outer table of the cranium forming lesions (Larson 1997). This process begins in the eye orbits and, as the condition becomes more severe, proceeds back towards the lambda (the point were the sagittal suture meets the lambdoidal suture). Therefore, the presence of cribra orbitalia alone indicates a milder case of anemia than if porotic hyperostosis were also manifested (Stoeppler, unpublished data).

This expansion process occurs before pubescence, when the bone is more supple and malleable. However, different stages of remodeled lesions (porotic hyperostosis and cribra orbitalia) can be observed in adult crania. The lesions will heal with the presences of an adequate hemoglobin concentration, or they will remain active if the individual continues to live with depressed iron stores (Todd 1994). The time frame in which adequate iron stores must be consumed before healing takes place is yet to be determined. In addition, menstruating females are less likely to be able to heal the cranial lesions because of their increased demand for iron, and therefore can cause an iron deficiency bias within the data. For these reasons, the analysis of prepubescent populations (when there is a large enough sample) is preferable.

CAUSES OF ANEMIA

When considering epidemiological studies, it is necessary to recognize that a correlation between two factors does not necessarily imply cause and effect. The body is a complex system, in which many factors synergistically affect the development of diseases. Global scenarios (such as anemia frequencies in current Sub-Saharan Africa) create innumerable variables that can all lead to the development of anemia. Therefore, the following research is specifically addressing anemia in the North American pre-contact context.

Anemia is most commonly thought to be the result of parasitization, bacterial, fungal, viral infections, inadequate intakes of iron, B12, and folate, as well as the presence of dietary factors that limit the absorption of the aforementioned minerals. (Factors such as sickle cell anemia, toxin exposure, and renal disorders of the elderly, which are well-known causes of anemia in some populations, are not included in this study, because they did not affect Native American pre-contact populations.)

Dietary stress:

Pernicious anemia results from vitamin insufficiencies that prohibit the methylation of DNA in the red blood cells preventing proper development. This, in turn, precipitates a shortage of hemoglobin and thereby leads to an anemic state. Vitamins B12, B6 and folate are responsible for DNA methylation in the red blood cells, and these vitamins are obtained through animal products (such as fish, eggs, beef and milk) and green plant foods (Sizer and Whitney, 2000).

While pernicious anemia seems to permeate strict vegan lifestyles, few cases are reported in the general population. However, iron deficiency anemia (IDA), which is anemia resulting from inadequate hemoglobin function due to insufficient iron intake, is the number one nutrient deficiency in the world (Sizer and Whitney, 2000). Iron intake recommendations for the United States suggest that men consume

10 milligrams per day of iron, and women should consume 18 milligrams per day (Sizer and Whitney, 2000). Most grains are low in iron, especially corn, wheat, and rice (often below 2 milligrams per serving). Underwood (2001) speculated that populations having excessive dependence on corn, wheat, or rice would show evidence of IDA. Beans and other legumes are somewhat higher in iron, but many servings are required to reach the suggested dosages. Prehistoric foragers would have had higher iron stores because of their heavy utilization of seeds, which typically contain around seven milligrams of iron per serving, as well as their consumption of higher quantities of meat (Underwood 2001).

Unlike the prehistoric foragers described by Underwood (2001), some agrarian populations consumed the majority of their calories in the form of corn, therefore their serum iron levels would tend to be depressed because of the decreased consumption of meat. For example, using cribra orbitlia and porotic hyperostosis as indicators of anemia, Boyd (1984) found that people in the Appalachian summit regions who were still using some hunting and gathering subsistence strategies showed very little evidence of anemia, while the cultures in the Mississippi river valley that had developed a predominately maize agriculture economy showed higher frequencies. Boyd (1984) attributed these differences to discrepancies in the diet between the two populations. In addition, Mensforth, Lovejoy, Lallo, and Armelagos (1978), based on their examination of prehistoric sites from Ohio, concluded that illness and dietary stress are important factors in causing anemia. Also, in a study from the southwestern United States conducted by El-Najjar et al. (1975), where the frequency of anemia was positively associated with dietary factors, it was concluded that anemia due to inadequate iron is the most likely causative factor for the deficiency to develop.

Another factor in determining dietary stress as the cause of anemia is iron absorption by the body. Iron occurs in two forms in foods. Some is bound into heme, which is the iron containing part of hemoglobin and myoglobin, and is found in poultry, fish, and other meats. Other iron is nonheme, such as iron found in plants and the nonheme iron in meats.

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The form of the iron affects absorption; heme iron is absorbed much more efficiently, an average of 23%, while only 2-23% of nonheme iron is absorbed. In addition, phytates, tannins, calcium and phosphates in milk, and fiber in whole grain cereals impair absorption (Sizer and Whitney, 2000). Therefore, even if people were eating beans and corn, their body would only be absorbing a small percentage of the iron they were consuming.

Given these relationships, studies cannot simply determine the amount of iron that was eaten. Absorption levels should be based on nutrients and minerals consumed with the iron. For example, Woodruff (1958) studied 272 infants with IDA and noted that the poor diets of several anemic infants were similar to those of a much larger number of infants who did not develop anemia to the same degree of severity. He concluded that factors other than diet are important in the etiology of IDA (cf. Stuart-Macadam 1992). However, Woodruff did not comment on any differences in fiber or calcium and phosphate intake, which theoretically, could cut iron absorption down to 2% (Sizer and Whitney 2001).

This interaction holds true for Native American populations as well. A large number of studies have suggested that the prolonged lactation practiced by Native American cultures, and conse-

quent high intake of calcium, would have significantly deterred iron absorption (Palkovich 1984). They base this conclusion on the presence of an iron-binding protein, lactoferrin, which has the potential to influence the development of IDA in children and infants. However, recent clinical studies are questioning the validity of this claim, as the benefits of extended breast-feeding seem to far out-weigh the risk of anemia. For example, a UNICEF, WHO, US AID project reported that some 2 million children under 1 year of age die every year from diarrhea associated with various forms of infant feeding (diarrhea can intensely affect absorption of nutrients into the blood stream and may therefore lead to IDA). Many researchers suggested that these deaths could be averted if children were exclusively breastfed through 4-6 months of age, thereby reducing their exposure to contamination while also reaping the nutritional immunological protection of breast milk through this period (Dettwyler and Fisherman 1992; Jelliffe and Jelliffe 1978).

Studies of Native American lactation indicate that children were traditionally breastfed until the age of one or longer (Gonzalez 1969), and early accounts of Native Americans also describe lengthy lactation periods (Waselkov and Braud 1984, Madanagd 1986). With extensive lactation periods, children would have had more protection from diarrheal diseases that decrease absorption of nutrients, while receiving adequate supplies of folate and perhaps iron. Gonzalez (1969) conducted extensive interviews and concluded that each of the different ecological zones in the United States adequately supplied the essential nutrients to Native Americans. She further acknowledges that the seasonal variations could have produced a "feast or famine" situation. However, Larson (1997) notes that even a season of nutrient deprivation would not cause chronic anemia unless it exceeded six months or more.

The hypothesis of diet-induced anemia does not have unanimous support. Stuart-Macadam (1985), for example, suggests that except in cases of outright malnutrition, diet plays a minor role, if any, in the development of IDA. She also found that even if iron intake was reduced to nil, which is virtually impossible even with the most frugal diets, it would still take at least two to three years to develop IDA. Likewise, several case studies have shown that iron is recycled continually in the body, making dietary attributed chronic anemia only obtainable after several years of deprivation (Underwood 2001). In addition, iron absorption by anemic individuals increases to over 35%, as opposed to 25% at best in healthy individuals (Stuart-Macadam 1985). Yet people, especially women and children, who rely on corn or other nonheme iron sources to supply their dietary requirements would have a hard time consuming, and absorbing, 18 mg of iron per day even with increased absorption.

Infectious disease:

Other possible causes of anemia in North American pre-Colombian populations are pathogens, specifically bacteria and parasites. Porotic hyperostosis is related to the overall pathogen load of a population; "a heavy pathogen load will increase the incidences of parasitic infections, viral and fungal diseases, which have the effect of reducing iron status of individuals" (Stuart-Macadem 1985; p. 4). With the development of agricultural complexes in North America, urbanization and human contact increased, causing a significant decrease in sanitation. Today, most of the world's populations live under these same conditions and the World Health Organization reported in 1975 that most people in the world lose one to four milligrams of iron per day due to parasites.

According to a study conducted by Masasme et al (1974), up to 80% of children, young adult women, and pregnant women in the tropics have anemia (cf. Stuart-Macadam 1985). The results of the study showed that patients with anemia have significantly fewer bacterial infections than patients

with adequate iron stores. Stuart-Macadam's explanation for this finding was as follows:

> ...prorotic hyperostosis has been referred to as a 'nutritional' stress indicator. Traditionally those groups with a higher incidence of porotic hyperostosis have been considered to be less successful in adapting to their environment or more nutritionally disadvantaged than other groups... there is new appreciation of the adaptability and flexibility of iron metabolism; as a result it has become apparent that diet plays a very minor role in the development of iron deficiency anemia. It is now understood that, rather than being detrimental, hypoferremia [anemia] is actually an adaptation to disease and microorganism invasion. When faced with chronic and or heavy pathogen loads individuals become hypoferremic as part of their defense against these pathogens.. (Stuart-Macadam 1992:1)

Nevertheless, there are many negative effects of anemia making it unlikely for the body to be benefited by an intentional decrease in iron stores. In addition, the immune system would be depressed as a result of IDA, making people more susceptible to infections (Schmidt and Roberts 2000). Stuart-Macadam (1992) argued that the body would develop an iron-deficiency response in order to defend itself from pathogens; therefore iron deficient people benefit by being less attractive to foreign invaders (parasites and pathogens). However, individuals with an iron deficiency are more susceptible to severe infections (Martin et al. 1985). Furthermore, Combes (2001) states that individual nutrition modulates the immune system; a properly balanced diet is one characteristic that most strongly influences the resistance of hosts to pathogens. This is due to the requirement for iron in the incorporation of hydroylysine and hydroxyproline into collagen (Aufderheide and Rodriguez-Martin 1998). Endt and Ortner (1982) examined the content of these amino acids in normal bone and compared them to individuals with porotic hyperostosis and found 5-25% less amino acids in the individuals with porotic hyperostosis. Iron deficiency anemia has also been found to adversely affect immune function and to increase susceptibility to infection under laboratory conditions. This is due to impairment of the T-cells to mitogens and the bactericidal activity of neutrophils (this study was conducted ***in vitro***) (Walter et al. 1997). Furthermore, evidence from recent controlled trials in juveniles indicates that short-term iron supplementation does not increase the incidence of common childhood infections such as diarrhea, respiratory illness, or increased parasite susceptibility (Ramakrishnan 2001). Also, Roberts and Manchester (1995) found an increased mortality in individuals with cribra orbitalia. In summary, anemia makes the body susceptible to infection – the body does not deter infection by becoming anemic.

Pathogens can potentially factor into the development of anemia by directly causing anemia due to decreased absorption of nutrients. Table 1 (located at the end of this article) provides a list of diseases that might have affected iron absorption of Native American Indian populations. These data are presented in terms of probable occurrence in the population, anemic and periosteal complications, and other skeletal markers that are manifested and interpreted by the paleopathologist as "diseased". Only four of the potential diseases possibly afflicting Native Americans lead directly to chronic anemia (anemia is a primary reaction): trauma, syphilis, sporotrichosis, and congenital cyonogalourus. Furthermore, all of these diseases that potentially cause anemia will manifest other osteological markers, such as bone deformations and abnormalities, or periostitis, of the long bones. Those diseases that produce anemia as a secondary response do so after the disease is in a chronic stage in which white blood cells are being produced instead of red blood cells, thereby causing anemia. It is only after the

disease has run a significant length of time that signs of anemia might develop (Kaschula 1998). In contrast, the diseases discussed above that directly lead to anemia do so at initial onset (Kaschula 1998).

Parasites:

Parasites can also lead to anemia. Stuart-Macadam and Kent (1992) cite malaria, hookworm, and osteomyelitis as pathogens that could lead to porotic hyperostosis. Larson (1997) argues that the high occurrence of porotic hyperostosis in El-Najjar's (1975) Southwestern population studies, from maize-dependent populations inhabiting canyon bottomlands, are a result of problems arising from poor drainage and contaminated water rather than maize consumption as suggested. Larson (1997) criticizes these studies by stating that they underplay the role of parasitism in IDA, especially in the case of Enterobius vermicularis (pin worm).

Table 2 indicates which parasites may or may not have been affecting Native American populations, the effect they would have on the population, and why they would not be a factor in the etiology of IDA. Although the list of parasites that could have affected Native American populations is quite extensive, tapeworms, Trichuris, and hookworm are the only parasites present on the continent that would have been of any relevance to the study population. In addition, it has been documented that even in the most severe cases of parasitic infestation, a sufficient diet will likely disguise all symptoms, including anemia (Schmidt and Roberts 2001).

Childhood diarrhea:

Another possible cause of chronic anemia is dysentery caused by Giardiasis and weanling diarrhea. These diseases are attributed to poor potable water and sanitation. Therefore, the probability of anemia caused by weanling practices must be made on a case-by-case basis. While these diseases are caused by diet and pathogens, they are recorded separately from dietary and pathogenic etiologies in that they are culturally manifested.

CONCLUSION: CAUSES OF CHRONIC ANEMIA

As noted above, all diseases that precipitate chronic anemia leave behind other skeletal markers in addition to cribra orbitalia and porotic hyperostosis. Therefore, it is possible to distinguish anemia caused by bacteria and viral infections from anemia caused by diet. It is also established that individuals must first be malnourished before they will show signs of chronic anemia from parasitic infections (Schmidt and Roberts 2001). Again, it is anemia that makes the body susceptible to infection – the body does not deter infection by becoming anemic.

While some studies have shown that populations with hyperostosis have consumed as much iron as those without, few studies have considered the absorption of iron in addition to consumption. Yet, high parasite and pathogen loads caused by crowding and poor sanitation can cause anemia as well. These findings illustrate the importance of separating the etiologies of anemia; where individuals displaying anemic lesions alone are classified as having severely deficient diets, and individuals displaying other diseased lesions in addition to anemic lesions are classified as having pathogen-induced anemia.

Much information can be gained from studying the frequency of cribra orbitalia and porotic hyperostosis in archeological settings if they are accurately calculated. I collected my data over a three month period (June through August 2002) at the Smithsonian Institution examining the skeletal remains of 173 juveniles from a pre-contact South Dakota Indian culture for evidence of IDA with the intent of uncovering a new understanding of the population's life-ways and, in particular, their diet. Upon analysis of my data, I have also found it necessary to re-evaluate previous studies in-order to compare my findings with accurate anemia frequencies. The two studies I was able to re-evaluate on the basis of full skeletal analysis, archaeological data,

and contemporary medical studies were from the Mouse Creek and Libben sites that were originally evaluated by Boyd (1984) and Mensforth et al. (1978) respectively.

BACKGROUND

Study site

I examined the skeletal remains from the Sully Site, a pre-historic community located 20 miles north of Pierre, South Dakota that was previously part of the Arikara culture of the Upper Missouri River. This skeletal collection, excavated by Bass in 1957, is currently housed in the National Museum of Natural History (Washington DC). Bass directed the excavation of four cemeteries (A, B, C and D), but suggested that Cemetery C, which contained burials in patterns that differed from the other cemeteries, was not associated with Sully and was instead associated with another unnamed village (Bass unpublished document). Consequently, cemetery C was not included in my study.

Jantz et. al. (1971), Lehmer (1971), Key (1983), Rodgers (1990), Johnson (1994), and Bass and Stephenson (unpublished manuscript), have been the main researchers involved in the excavation and interpretation of the site. While many aspects of the village occupation are under debate, there are several accepted facts. First, none of the early historic records of this portion of the Upper Missouri River had any reference to a Native American site in the location of Sully (Stephenson and Bass unpublished data). Although Lewis and Clark extensively described neighboring villages (both occupied and abandoned), they made no mention of the Sully site in October of 1804 (Lewis and Clark 1983). Furthermore, an 1866 military post on the west side of Sully Creek also made no mention of it (Billick 2002). Consequently, Bass and Stephenson (unpublished manuscript), and Rodgers (1990), date the site from 1650-1750. The cemetery population would have thereby avoided the recorded European derived epidemics that began in 1772. Craniometric data from Key (1983) support Bass's findings, but push the date of occupation further into contact periods. However, Key (1983) estimated that the grave contents were even later. While all cemeteries, except C, contain trade goods, trade routes had been established before direct contact would have occurred. Some French fur traders would have made their way to the Arikara, but their viability as disease vectors is not likely because the extensive journey would not have been possible for diseased individuals. Therefore, according to the majority of researchers, the Sully site would have had minimal direct contact with Europeans. Due to this fact, European diseases can be safely eliminated as causative factors in the development of periosteal lesions or anemia exhibited in the study collection.

Arikara Culture

The Arikaras were a settled agricultural group (Dorsey et al.1940, Wetfish 1965) that were thought to have separated from the Pawnee around AD 1400 and settled in the Dakota river valley (Rodgers 1990). The Arikara people eventually became part of the Mandan when they were forced onto reservations in the 19th century. Using historic and archaeological evidence, Rodgers (1990) dates the pre-contact period for the Arikara from approximately the late 1500's until 1680. The earlier date coincides with the first archaeological data that may reasonably be identified as Arikara. The later date is the time of the first direct contact with the village groups on the Upper Missouri. However, diseases introduced by Europeans may have had a significant impact on the Arikara population well before the eighteenth century (Ramenofsky 1987). According to Rodgers (1990), this time frame (1500 – 1680) was most affected by the introduction of diseases, however he notes that the disease vectors responsible for epidemics are tentative. From 1681-1724, there was little direct trade with French

explorers as other Native Americans served as intermediaries between the Arikara and the Europeans. At this time, there would have been few chances for the spread of European disease. Soon thereafter serious social stress and a dramatic life-way change ensued (Rodgers 1990). Yet, as noted above, the occupation of the Sully village ceased before this transition.

The other members of the Arikara culture, having existed into the contact period, have provided historical accounts of their life-ways. Particularly valuable to an anemia study are accounts of diet, agriculture, and sanitation. Agriculture and hunting were two primary subsistence activities, although the collection of roots, nuts, berries, and other naturally occurring resources formed an important supplement. Corn was the principal domesticate, although beans, squash, and sunflowers were also grown (Rodgers 1990). This wide variety of nutrients would have supplied a well-balanced diet for the Arikara. While corn was the main staple, there is evidence that hunting (particularly of buffalo) remained an important food source, thereby providing the Arikara with significant iron intake. Furthermore, B12, B6 and folate would have been available to the Arikara, which would have fended off pernicious anemia. Historical records (eg. Abel 1939) mention directly or imply periods of starvation and periods of feast. However, as previously stated, longer periods of nutrient deprivation would be necessary for development of chronic anemia.

Rodgers (1990) claims that no fertilizer was used on the fields and that they were periodically left fallow while others were utilized. It is significant that no human feces were used, making the spread of hookworms and tapeworms less likely. This is especially true in the case of the Sully site because of the locality on the swift Missouri River, which would have moved contaminated feces away. Other than hunting and trading, another source "of buffalo were the carcasses found floating in the Missouri River during the spring thaw...although the animals were often in an advanced state of decay, the Arikaras ate parts of the meat raw" (Rodgers 1990; p 45). While breaches of normal sanitation standards like the one recorded above might have provided sufficient opportunities for parasites to invade the Native Americans, their well balanced diet would have helped to ward off adverse effects from the parasites that they were exposed to during these situations.

Villages were well dispersed and consisted of several earthen lodges and shelters. While population estimates vary dramatically depending on the time period and the researcher, the limited size and dispersal of the villages would have helped to quell parasite infestations and sanitation problems because contact with people would be limited, especially when compared to higher density populations.

Overall, when taking into consideration the historical records of the Arikara life-ways, as well as the archeological data from the study site, the Arikaras at the Sully village had a healthy life-style. Their diets were well-balanced so that what pathogens they were exposed to could be warded off by a well functioning immune system. In addition, the exposure to pathogens was probably kept to a minimum because: (1) there was no direct contact with Europeans so epidemics would not have affected them; (2) the community was located on a moving body of water so improper dispensing of sewage was not as significant a health risk; (3) they did not use human feces as fertilizer so parasites were not as likely to contaminate the soil, and; (4) the villages were well dispersed so human contact would limit pathogen transmission.

MATERIALS AND METHODS

Skeletal material and diagnosis of IDA

The skeletal material excavated from the Sully site is in excellent condition and supplied a large sample population. In addition, there appears to be no time of excessive mortality, but rather the

individuals died at a steady rate (Billick 2002). There is a minimum of 500 skeletons from the cemeteries, of which 173 are mostly complete juveniles under the age of 10. This study focuses on juveniles in order to eliminate a possible bias caused by excessive iron loss of menstruating females. Age was assessed according to root and crown development of the dentition (Bass 1995). As noted in the introduction, the indication of other anemia causing diseases is just as important as the identification of anemia. The most common sign of nonspecific infection is periostitis. This bone infection has a characteristic look that is similar to a paper birch tree; the thin flaky layers of bone growth and odd striations are similar to the muscle attachment points in growing children (Hunt, David personal communication 2001). Larson (1997) describes the pathology as osseous plaques that arise from the osteoblastic stimulation caused by infection or trauma. The lesions can be localized or may involve several skeletal elements depending on the etiology of deformity. Because the majority of diseases listed in Table 2 cause chronic anemia after the onset of bone infection, it is very likely that when periosteal lesions occur with porotic hyperostosis the severity of the anemia was induced by infection rather than diet. In this way, it is possible to differentiate between various causes of anemia. If there are only cranial lesions found, then it is possible that they were caused by a deficiency in diet. Conversely, if there are additional postcranial skeletal lesions found with IDA lesions, then the infection was likely the primary cause of the chronic anemia state.

Complete skeletal analysis was not conducted for every juvenile, but the crania were all closely examined. If cribra orbitalia or porotic hyperostosis were observed, they were given a score that corresponded to the severity of the porosity. This decision was made according to the number of pores, size of the pores, and space that was affected by the pores. Those crania that exhibited porotic hyperostosis or cribra orbitalia were also examined for periostitis and other pathological bone changes. The surface area that was affected determined the severity of the bone infection. All scores were based on a scale of 1 to 10, 10 being the most severe.

RESULTS:

In my examination of the Sully juveniles (N=173), 11(6.3%) showed signs of cribra orbitalia (see Table 3). No porotic hyperostosis was conclusively determined, indicating that these were only minor cases of chronic anemia. When these 11 juveniles displaying cribra orbitalia were further investigated for evidence of periostitis, 7 were found to manifest additional postcranial lesions, while 4 (2% of the study population) displayed only cribra orbitalia.

Sully, Libben, and Mouse Creek Comparisons

I compared my findings from the Sully population with those of two contemporaneous studies of the Mouse Creek (Boyd 1984) and Libben (Mensforth 1978) skeletal collections to determine if anemia was occurring at similar rates, and if not, why was there a difference? Both of these studies provided data that separated individuals with only cribra orbitalia and porotic hyperostosis from those with anemic lesions and skeletal lesions. Of the 241 individuals included in the Libben collection, 24 (21%) displayed only cribra orbitalia and/or porotic hyperostosis, while 25 (23%) displayed cribra orbitalia and/or porotic hyperostosis with additional periosteal lesions (See Table 4). The Mouse Creek population contains the highest percent of anemia victims with 28 of 42 (66%) individuals exhibiting cribra orbtialia and/or porotic hyperostosis. None of the 28 individuals displayed additional periosteal lesions.

Figure 1 shows that Sully has the highest proportion of individuals with cribra orbitalia and/or porotic hyperostosis accompanied by periosteal lesions compared to individuals with only cribra orbitalia and/or porotic hyperostosis. Yet, Sully has

the lowest percentage of total anemic individuals. Mouse Creek has the highest percent of individuals with only anemic lesions, as well as the lowest number of individuals with periostits. In addition, Libben has the highest percentage of anemia victims with periosteal infections.

Following previous methodologies used to infer anemia in skeletal remains (Boyd 1984, Parham 1992, Todd 1994) the results presented by this study would have been interpreted by diagnosing the 11 (6.3%) individuals with cribra orbitalia as having nutritional deficiencies. However, when those individuals manifesting both cribra orbitalia and periosteal lesions are separated out of the population, even more information can be gained because the presence of infection indicates that the anemia was a secondary response to disease. If we exclude those cases of cribira that are associated with periostitis, then a different picture of the population's diet emerges (i.e. their adaptability to their environment). This is especially true when comparing anemia frequencies of populations in different locations and with different cultural practices.

As previously stated, studies of radiology, parasitology and anthropology (Ubelaker 1992, Shmidt and Roberts 2000, Ortner and Putschar 1981) subscribe to bacterial and viral infection being a primary cause of anemia. In fact, when looking at the levels of circulating serratin in the body, many studies of iron deficiency have shown infection to be a larger contributor to anemia than diet. Therefore, individuals displaying chronic signs of infection (i.e. periostitis) should be eliminated from the data when conducting a dietary profile of past populations.

In this study, I found only four juveniles (2%) who displayed cribra oritalia alone. Bearing in mind that the site had an approximately one hundred-year occupation, this finding indicates an adequate diet. This hypothesis is also supported by the analysis of the archaeological data (Jantz et al. 1971, Lehmer 1971, Key 1983, Rodgers 1990, Johnson 1994). While meat was not a primary nutrient in their diet, and there were times of famine, the wide variety of plant foods would have supplied adequate folate and iron to have prevented chronic anemia from occurring.

In addition, analysis of the descriptions of the Arikara culture (Jantz et. al. 1971, Lehmer 1971, Key 1983, Rodgers 1990, Johnson 1994, and Bass and Stephenson unpublished manuscript) shows that sanitation was relatively good so transmission of parasites would be restricted. They did not use fertilizer on their fields, so the absence of human feces would prevent fecal contamination of foodstuffs. Also, the villages were dispersed along a flowing body of water contributing to positive sanitation practices. These living conditions do not lend themselves to chronic infection of parasites, especially when added to the cold and dry climate of North Dakota. With good sanitation practices and adequate diet, the only etiolology of anemia left to analyze is weanling diarrhea in infants. Considering the small percentage of anemic individuals, the weaning practices of the Arikara communities probably included an extended lactation period that would have bolstered the child's immune system to significant levels, which would support Gonzalez's (1969) suggestion of lengthy lactation periods in early Native American populations that have been discussed previously.

My finding of a 2% rate of chronic anemia caused by dietary stress in the Sully population is more strongly supported by the archaeological and historic records than would the higher rate of 6% that would have been yielded had individuals with periosteal infections not been eliminated. Although much more information can be ascertained about a population's diet by adopting this practice, previous studies (Parham 1992) of IDA have failed to separate cribra orbitalia and porotic hyperostosis from other signs of disease and infection. In these previous studies, skeletons that exhibit cribra orbitalia and or porotic hyperostosis might also have periosteal lesions, thereby artificially inflating the instances of

IDA in a population. Therefore, where the raw data are available, I compare my results with those obtained from other sites.

The Libben site

The Libben Site from the Ohio River valley was occupied a few hundred years before the Arikara moved into the Missourri River valley. Their settlement was one of similar construction to the Sully site, consisting of small lodges placed along the riverbanks, although the Libben population was considerably larger than that of Sully. Floral and faunal analysis indicated a diet rich in animal protein but with little evidence of plant foods (Mensforth et al. 1978).

Mensforth (1978:20-21) and his colleagues analyzed 241 juveniles from the Libben cemeteries. They produced one of the only studies that took into account the manifestation of chronic anemia due to infectious disease because "most nutritional deficiencies are either precipitated by, or aggravated by infectious diseases in individuals with borderline nutrient supplies." Their investigation produced results in which anemia frequencies could be associated with specific causes (see Table 4). Compared with Sully, Libben has a higher number of anemia cases (18% more than Sully when specimens displaying periosteal lesions are removed) even though both populations have a diet rich in meat. There are two possible explanations for this difference: (1) that increased population leads to both crowding and sanitation problems, thereby increasing pathogen loads; (2) a folic acid deficiency (i.e., pernicious anemia). While pernicious anemia is downplayed in current studies, it is possible that red blood cells were less efficient because of a lack of folate, especially considering that Mensforth (1978) found little evidence for plant food staples in the Libben diet. If their diet was lacking in folate, pernicious anemia could have led to an increased susceptibility to diseases, just as IDA would, accounting for the increased periostitis frequency as well. The Libben population was nutritionally disadvantaged compared to Sully because there was a lack of plant food, not meat. Because the Libben peoples were anemic (or borderline anemic) they would have been more susceptible to infection, resulting in the higher amount of periosteal lesions accompanied by the cranial lesions.

Pernicious anemia seems to be the most valid argument for the difference. If an increase in population size led to higher frequencies of anemia, you would expect to see a higher number of periosteal lesions in proportion to the cribra orbitalia. This is because decreased sanitation leads to the increase in bacterial disease, which could eventually manifest symptoms of chronic anemia as a secondary response; the primary response would be other skeletal lesions. Therefore, the increased crowding would have led primarily to more periosteal lesions, and secondarily to anemic lesions. However, only half of the population displayed both anemic lesions and perisotitis. In addition, the population was malnourished enough for 21% of the children to develop chronic anemia without the presence of other diseases. This would indicate that diet is the primary contributor to the development of these anemic lesions. In summary, for bacterial or viral infection to be the main cause of the anemia, periosteal lesions would be found on more of the skeletons with cribra orbtialia and perotic hyperostosis because infection would be the primary cause of the anemia. Yet, the opposite was found to be true with the Libben site because there was a substantial increase (compared to the Sully site) of skeletons with only the cranial lesions.

The Mouse Creek site

The Mouse Creek skeletal collection is from the Appalachian Summit Region in eastern Tennessee. Corn is hypothesized to be a common domesticate among the Mouse Creek population. However, Lewis and Knedberg (1941:7) found "the bones of deer, rabbit, squirrel, wild turkey, turtle and

fish were abundant in the refuse of Mouse Creek communities" (cf. Boyd 1984). Although corn was utilized, there is evidence that other (iron supplying) resources were also consumed. Other than times of famine in the winter, which has been established as being customary (Gonzalez 1969), the village had both meat and plant foods available. Yet, the population has 64% more cribra orbitalia then Sully and no cases of periosteal lesions associated with the cranial lesions. Because there are no cases of periosteal lesions associated with cribra orbitalia and or perotic hyperostosis, a nutritional etiology can be deduced as the primary cause of the anemia. However, as noted above, according to the archaeological data the Mouse Creek people probably had sufficient nutrients in their diet. Therefore, anemia must be caused by a nutritional deficiency that occurs for a reason other then diet (such as parasites or weanling diarrhea). Little is known about the cultural practices of the Mouse Creek people, such as the use of human feces as fertilizer and lactation periods. Because we do not know if children were weaned with a corn gruel, or at what age they were weaned, weanling diarrhea could have been a significant contributor to the development of chronic anemia, possibly accounting for the variation in anemia frequencies.

What can be established is that the anemia of the Mouse Creek population is caused by a nutritional deficiency because none of the skeletons that manifested porotic hyperostosis and or cribra orbitalia had any sign of other infection or disease. When compared to Sully, there is a drastic difference in the frequency of anemia. Even though both communities appear to have sufficient food supplies and decent sanitation, Mouse Creek has many more victims of chronic nutritional anemia. Weanling diarrhea is the one factor that cannot be controlled for in this situation other than that, by deduction, we know that Sully children seemed to suffer little from it. Therefore, the probability that it was weanling diarrhea that caused the variation seems likely.

CONCLUSION

The bacterial and viral diseases that eventually cause chronic anemia initially cause other osteological manifestations (ie. periostitis, osteomylitis and pathologic bone deformities). Therefore, skeletons displaying evidence of chronic anemia and chronic infection should be eliminated from a dietary profile because it is probable that the infection led to chronic anemia rather than anemia caused by dietary insufficiency. Overall, when utilizing this method of deduction, 6.3% of the anemia cases are eliminated from the Sully site, from the Libben site 56% of the anemia cases are removed, and none from the Mouse Creek site are removed. Reducing (or sustaining) the number of individuals potentially afflicted by dietary anemia drastically changes the researcher's perspective on resources available to the study populations, as well as the differences in the various populations' life-styles that would have impacted their lives so differently.

A possible variation in life-style might have been exposure to parasites. However, they all were in well-dispersed villages that had a significant supply of running water, and if human feces were used at all, it was limited. These factors would have reduced the prevalence of parasites. Also, it is worth restating that in most cases it is only if diet is insufficient that a victim will display signs of chronic anemia due to a parasite infection.

Weanling diarrhea is probably the most difficult variation to address because there are no definitive ways of knowing the traditional lactation periods. However, this is something that must always be kept in mind when evaluating specific case studies. For example, it is not a significant contributor to the Sully population because there is not a high prevalence of anemia, however, in the case of Mouse Creek, because there are no periosteal lesions, and there is a high number of anemia victims, while diet is considered fairly balanced, weanling diarrhea might have been a large factor in the

development of chronic anemia.

Diet is the main etiology of anemia addressed in this paper. Archeological data from all of these sites suggest that everyone had a steady supply of iron, and that Libben was the only population that might have had a folate deficiency. For more information to be attained on the effects of dietary anemia, a differential study such as the one presented here must be used to analyze maize-dependent societies. Researchers like Parham (1998) have already conducted such skeletal analysis, however, the cases where anemia is a secondary response to infection have not been eliminated, that is anemia of chronic disease is included as evidence of dietary stress.

In this study, infectious lesions were included in the differentiation of anemia so that the possibility of infection causing the cribra orbitalia and porotic hyperostosis in the study populations can be eliminated giving a much more accurate estimate of dietary anemia in past populations. From the evidence presented here, I believe the Sully population was healthy as a result of adequate nutrition, while the Libben population was possibly affected by a folate deficiency (i.e. pernicious anemia), and the Mouse Creek population was possibly affected by weanling diarrhea. These conclusions can be drawn because anemia caused by chronic infection was compared to anemia caused by nutritional deficiencies, something that must be taken into consideration when analyzing future dietary profiles of Native American cultures.

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Table 1 Infectious Disease Leading to Anemia of Chronic Disease

Infection	Causes Anemia Primary/Secondary Response	Duration of the Disease long enough to develop bone modifications	Osteological Markers of Disease present (other then those caused by anemia)	Present in North America	Source
Osteomyelitis	Secondary	Yes	Yes	Yes	Kaschula 1995; Larson 1992; Ortner 1992
Trauma (Infected wound)	Can be Primary	No	Yes	Yes	Larson 1992; Ortner 1992
Syphilis	Can be Primary	Yes	Yes	Possibly	Larson 1992; Ortner 1992
Infantile Corticle Hyperostosis	Secondary	Yes	Yes	Possibly	Kaschula 1995
Hyperparathyroidisim resulting from hypocalcemia	Secondary	Yes	Yes	Yes	Kaschula 1995
Toxoplasmosis	Secondary	Yes	Sometimes	Extreme southern regions	Larson 1992; Ortner 1992
Sporotrichosis	Rarely, secondary if it occurs	Yes	Yes	Yes	Kaschula 1995
Coccidoidomycosis	No	Yes	Yes	No	Kaschula 1995
Baceraemia and Septicemia	Secondary	Yes	Yes	Yes	Kaschula 1995
Hypo and Hyperthyroidisim	Primary	Yes	Yes	Yes	Kaschula 1995
Absorbic Acid and Copper deficiency	Secondary	Yes	Yes	Yes	Kaschula 1995
Menningitis	Secondary	Yes	Yes	Yes	Kaschula 1995; Larson 1992; Ortner 1992
Periosteal lesterrosis	No	No	Possibly	Yes	Kaschula 1995
Congenital Cyonogalourus	Primary	Yes	Yes	Yes	Kaschula 1995
Tuberculosis	Secondary	Yes	Yes	Yes	Larson 1992; Ortner 1992

Table 2: Parasites Contributing to the Development of Chronic Anemia

Parasite	Will it contribute to IDA development?	Why/ Why not	Habitation of North America prior to 1700s	Possibly contribute to Native American IDA	Source
Plasmodium (malaria)	Yes	sickle cell, binds to hemoglobin	No, brought by first explorers	No	Larson 1997; Kelton 1998; Smith 1987
Enterobbius vermicularis (pinworm)	No	does not effect blood stream or lumen of the stomach or upper GI tract	Yes	No	Schmidt and Roberts 2000; Reinhard 1992; Larson 1997
Trichuris trichura	Yes	effects hemoglobin directly	Extreme southern regions of the continent – with poor sanitation	Yes, in southern areas	Schmidt and Roberts 2000
Ascaris lumbricoides (tapeworm)	Yes	feeds off of contents of GI tract	Yes	Yes	Stuart-Macadem 1995; Schmidt and Roberts 2000
Stronglyoides	only rarely - effects immunocompromised individuals	Does not effect blood stream	Possibly	Possibly	Larson 1997; Schmidt and Roberts 2000
Shistosoma	Yes	Effects blood stream directly	No – brought to North America with Slave trade	No	Schmidt and Roberts 2000
Necator americanus and Anclyostoma duodenale (Hookworm)	Yes	Feed directly on blood stream	Yes	Yes	Faulkner 1991; Schmidt and Roberts 2000; Allison et al 1974

Table 3 - Results From Sully Site

	Total Cases From Sully	Percent of Individuals Affected
Total Cribra Orbitalia and Porotic Hyperostosis	11	6.3
Only Cribra Orbitalia and Porotic Hyperostosis	4	2
Cribra Orbitalia, Porotic Hyperostosis and Periosteal Lesions	7	4

Table 4 Results from Mouse Creek and Libben Sites

	Total Cases From Libben	Percent Of Individuals Affected	Total Cases From Mouse Creek	Percent Of Individuals Affected
Total Cribra Orbitalia and Porotic Hyperostosis	107	44.4	28	88
Only Cribra Orbitalia and Porotic Hyperostosis	24	21.2	28	66
Cribra Orbitalia, Porotic Hyperostosis and Periosteal Lesions	25	23.2	0	0