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Anemia Among Past Maya Populations: When Will We Have the Answer?

Katie Whitaker

Today it has been estimated that approximately 2.15 billion people worldwide are iron deficient, and almost 1.20 billion people suffer from various severities of iron-deficiency anemia (Wright & Chew 1999: 925). Anemia has also been a serious problem for several populations in the past, including those of the ancient Maya civilization. Although we cannot know for sure the actual prevalence of the disease among the ancient populations, with the available skeletal analysis from the osteoarchaeological record we can at least infer that it was a health problem for the Maya, as it is for the world’s populations of today. The Maya civilization flourished from approximately 900 BC to AD 900 with populations occupying areas including Guatemala, Honduras, Mexico and Belize. There have been many studies devoted to the rise and fall of the Maya, and almost every other aspect of their daily lives. However, studies on the prevalence of various diseases among the Maya have not been researched as much as their importance would warrant and, beyond this, the various causes or reasons for the prevalence of so many health related ailments have not been adequately researched. With the exception of diseases that the Spanish may or may not have inadvertently transmitted, such as small pox and syphilis, discussion of ailments that were not immediately life threatening, or infectious were generally ignored. This paper will examine the various models that attempt to explain the high prevalence of iron deficiency anemia among the ancient Maya civilization, as well as possible faults inherent in each.

Background

The Pathology of Anemia

Anemia can be present in the body as a result of a genetic inheritance, as in the case of sickle cell anemia, or by acquiring it, for example, by having an iron-deficient diet. In the past it was always assumed that acquired anemia could only be evident during life, but in the last thirty-five years it has become known that, in moderate to severe cases, it can still be seen post mortem in bone changes which most often manifest in the skull (Stuart-Macadam 1992: 154). In young children acquired anemia is usually apparent with varying degrees of severity. As such, the bony changes that are evident in skeletons are most often (unless they are children) evidence of survival of anemia and therefore, shed light on the overall health during growth and development. Welecker (1885, 1888) as quoted in Stuart-Macadam, was the first to notice cribra orbitalia, a form of porous lesions within the inner orbits of the eye (Figure I) although it was not until 1961, that it was actually linked to diet deficiencies, specifically anemia (Stuart-Macadam 1992: 153). In the skeletons of young children, the initial reaction to severe anemia was the pitting around the orbits, although evidence of this in older individuals by itself suggests a more moderate form of anemia (Wright & White 1996: 157). Williams (1929), as quoted in Stuart-Macadam, was the first to connect iron deficiency anemia with porotic hyperostosis, the bony evidence found in the cranium. In older children who are still dealing with anemia, the external table of the cranial vault becomes pitted in appearance, and often thicker, which heals and remodels during maturation and adulthood. The bones of the skull, including the parietals and the occipital bone take on a spongy, porous look, as evident in Figure 2. Both forms of iron-deficiency anemia are generally described first as lesions at various points of development of the infection, from active, to healed and remodeled, depending on the condition of the individual. Among the ancient Maya there is also a wide range of skeletal evidence of both types of lesions as well as various stages of bone remodeling. These will be described in more detail in the proceeding sections of this paper.

The Osteological Paradox

At this point it is necessary to briefly review the Osteological Paradox. Suggested by Wood et. al. (1992), the paradox brought to light the inherent problems in inferring past health from skeletal samples of the archaeological...
record. For instance, it has in the past, and to a certain extent today, been believed that there should be a common correlation between the disease we see in burials and the actual prevalence of that disease in past populations. Unfortunately this conclusion does not take into account selective mortality and hidden heterogeneity. Selective mortality, as described by Wood et al. (1992), means that the samples of the population that have been unearthed are just that, a sample, and as such would not indicate all of the people that were at risk for the disease, or even all of those who suffered from it, but rather just those who died, and only at their individual age. This means that when researching burials one can only infer health from those that are present Thus we cannot, to any great degree, deduce what the health of the rest of the population would have been. Hidden heterogeneity, another aspect of the osteological paradox, insists that the populations of the past were made up of “an unknown mixture of people with varying amounts” of susceptibility to disease, and as such the skeletal remains that we see are simply those with a vulnerability to anemia (Wood et al 1992: 334). This vulnerability could be due to genetics, socio-economic factors or even the time period in which they lived. Overall the paradox should be taken as a cautionary note for all those attempting to infer population health from the archaeological record. This study by Wood et al. has been cited frequently since its publication, and as such it is a truly groundbreaking theory that all archaeologists must take into consideration. It is therefore worth noting that any population generalizations contained within this paper are solely due to the original author, although every attempt has been made to exclude these conclusions unless used for the purpose of analogy.

The two models discussed below for the potential causes of anemia among the ancient and historic Maya are both accepted hypotheses within the archaeological community. Several ancient and historic sites are discussed below, and Figure 3 illustrates their locations throughout the Maya realm.

The Ancient and Colonial Period Maya

There appears to be a general consensus among many Maya specialists that the population, for its duration, were in less than ideal health. For example Freidel and Schele (1990) suggested Late Classic Copan was the site of Maya that dealt with malnutrition, sickness, infection and stress (Wright & White 1996). Hammond (1982), also suggested that during Post-Classic times there was a decrease in nutritional standards, and according to burials an “increased susceptibility to endemic diseases which may have become epidemic” (Wright & White 1996: 154). By these accounts the Maya appeared to be unhealthy, whether due to anemia, environmental factors, or infectious disease. Anemia however comes up over and over as a serious problem for the ancient Maya. In several sites, including Cuello, Chichen Itza, Iximche and Tancab the majority of the burials containing evidence of anemia were seen in children. This suggests that children were dying as a result of very severe anemia, as evidenced in many of their burials (Figure 4). The numbers for these sites, and others discussed below, are summarized in Figure 5. Below are two models that attempt to account for the prevalence of anemia among the ancient to historical period Maya.

Maize-Dependent Model

This model, as suggested by its title revolves around the hypothesis that the Maya were so dependent on maize, and it composed so much of their diet, that it led to health problems, specifically anemia. Williams quoted in El-Najjar, first proposed this in 1929 as the Dietary Model, but it was not until 1976 when El-Najjar reevaluated the idea that it came to specifically deal with maize. They both found that there was a link between anemia and a diet comprised of staples low in iron or substances that interfered with iron absorption, such as millet (Holland & O’Brien 1997: 184). Maize, a well-known staple of the ancient Maya, as well as the Maya of today, was found to be low in iron, with the added detriment of having the chelating effect of the phytic acid (ibid.). This meant that a lot of the iron that was present in the maize was nutritionally unusable. In fact, less than seven percent of the iron in maize is readily available to be absorbed. Several areas throughout the Maya realm have been characterized as having medium to high instances of anemia, caused by a maize dependent diet. Hooton (1940) as described in Wright and Chew, was the first to describe these high instances of anemia at the Cenote of Sacrifice at Chichen Itza, a Post Classic site. He speculated that since the majority of the infected burials were children (78% of child burials), there was a connection with diets that depended mainly on maize (Wright & Chew, 1999). At the Altar de
Sacrificios during the Classic period, Saul (1973) found an extremely high instance of porotic hyperostosis (up to 89% of the adults found) (Saul 1973: 316). He felt it was so significant that it became the basis for a nutritionally centred argument for an ecological collapse of the Maya (Wright & White 1996: 158). In Cuello, a Pre Classic site, there appears to be less incidence of porotic hyperostosis as compared to other Maya sites including Copan (Saul & Saul 1991: 146). According to Wright and Chew (1999), this indicates less maize consumption, which is confirmed also by stable carbon isotopic analysis. At Lamanai, which was also established by isotopic analysis, there is a greater incidence of anemia among the Post-Classic population then earlier periods, which corresponds to an increase in maize consumption. Unfortunately, there is also an increase found during the Historic period, for which White (1988: 14) has no explanation. Wright and Chew also speculated that since the majority of porotic hyperostosis incidences were generally less common in the late Prehispanic and Historic period, when maize consumption was lower, higher instances of porotic hyperostosis from the Classic period could be caused by the higher intake of maize.

Although abundant, these sites do not tell the whole story. Regrettably there has been a lot of debate about the validity of this model. One problem, according to Wright and White (1996) being that if indeed nutrition was the direct cause, there should be a change in the abundance of lesions over time. When the samples from Altar and Seibal were reanalyzed there was no noticeable decrease in the amount of lesions, suggesting that diet may not have played as large a role as Saul (1973) originally thought. Beyond this, at Iximche, a Post-Classic/Historic site, in which maize consumption was very high, when measured by isotopic analysis, porotic hyperostosis had one of the lowest frequencies compared to other sites throughout the Maya region (Wright & White 1996: 160). Although, as Armelagos et. al. (1991) pointed out, porotic hyperostosis does occur more often among agriculturalists than among foragers, suggesting that diet does play a key role. Overall it seems clear that there is no consensus on how much importance should be placed on diet.

**Parasitic Model**

First suggested by Hengen in 1971 (Stuart-Macadam 1992), the parasitic model suggests that contrary to the generally held belief, anemia is actually a positive adaptive response to environmental stress (Holland & O’Brien 1997: 184). In fact, proponents of this model contend that diet actually plays a minor role, if any at all, in the disease, and they believe anemia should be seen as a body’s defense against disease. The parasite (which will be discussed below) would have been more common in sedentary populations as opposed to migrants, in which unsanitary conditions would have been prevalent. The shift to agriculture would have contributed to the increase in parasite “quantity” and the chances of a person contracting an ailment (Armelagos et. al. 1991: 16).

Theoretically, a parasite, such as the tapeworm enters the body and causes massive blood loss, and therefore anemia. The hookworm, *Ancylostoma duodenale*, is known to have been present pre-Hispanically in both North and South America, so it would have been safe to assume it was present in Mesoamerica (White 1994). Many authors also cite that in Guatemala today parasitic infection has reached 100% in some areas (White 1988: 14). In fact White (1994) also believes that Lamanai, being in a tropical lagoon environment, was susceptible to parasites especially those that were insect-borne. Beyond this, as a result of the variety of environmental conditions in which porotic hyperostosis has been seen, it appears to be more likely that other causes besides diet, such as the parasite may account for the frequency of anemia. This hypothesis coincides with the variability in the amount of maize prevalent in various diets (Wright & White 1996: 157). The climatic inconsistency may control the distribution of the parasite, considering the larvae can only survive in temperatures between 70-85°F. Wright and White contend that this would explain the abundance of evidence of anemia at lowland sites, and the limitedness in highland sites, such as Iximche (1996: 161). Overall it appears few academics are willing to attribute evidence of porotic hyperostosis with parasites, and yet the theory is still circulating within academic circles.

There have been several major criticisms against the parasite model, many of which come from the work of Holland and O’Brien (1997). They state “we believe that anemia, whether it is caused by maize or nematodes, is a sign that something is seriously wrong” (Holland & O’Brien 1997: 185).
Therefore, it is difficult to understand how the presence of anemia could actually be a "good" thing. Anemia can take the form of a very debilitating illness, especially in terms of weakness and susceptibility to fainting. It is difficult to understand, with the ideas of natural selection, that this would be "selected for" (as nothing actually is), when it would obviously be a drawback for populations that generally based their days around some form of physical labour. If in some sites prevalence of anemia among the burials was 80%, how could daily activities possibly take place? Although just a sample, at the very least these people were probably unable to work. Also, to almost completely rule out diet as a causing factor appears to be lack of common sense, especially when in our modern times it has become common knowledge that diet plays such a major role. Although it is safe to say that diet may not be the sole cause, it is a stretch to say that parasites would be the major foundation. One problem with naming one attribute with the blame is the variety of populations and environments in which anemia has been actually and historically present. For example, Wright and White stated that the Maya series have shown pervasiveness of porotic hyperostosis among populations amounting to 77% in sub-adults and 65% in adults (1996:159). This has been found to correlate with Coastal Californians in which the cause has been attributed to parasitism, and yet also among populations of the Southwestern Pueblo at Canyon de Chelley and Chaco Canyon (where it is more prevalent) in which the cause has been found to be linked directly to maize agriculture (ibid.). There appear to be too many questionable results to actually solidify the parasite as a predisposition to anemia. If the presence of anemia among present populations were to be taken into account, it is difficult to see how a parasite could cause so many illnesses. Specifically, if parasites, most likely due to poor sanitary conditions are the cause, how can they be attributed to so many sufferers living in North America and Europe in which, in general, conditions are at the very least better than they were during ancient and historic Maya times. Anemia, although a very common ailment, appears to have a multitude of causes, but with a general understanding, especially among the medical community, that diet can and does play a key role.

The Maya of Today

Today's populations of Maya have been studied on several occasions in an attempt to construct an analogy whereby to compare the ancient and historic presence of anemia. Wright and Chew have attempted to use this analogy to not only explain the presence of anemia, but they have also, whether purposefully or not, cast more doubt on both the maize-dependent model and the parasite model. When looking at the diet of present Maya populations, Roys (1972), noted in Wright and Chew, that the diets of past and present Maya had very little difference (Wright & Chew 1999: 927). In fact, if any important difference exists, it is that the Maya of the past were actually healthier, having more access to meat and fish than the present poor peasants of modern Mesoamerica (Robin 2001: 19). This is puzzling for proponents of the dietary model, especially considering present incidents of anemia are equal to or less than that of past populations (Wright & Chew 1999: 928). If diet had indeed decreased in nutritional quality, one would expect to find an increase in anemia, and yet the results we see today are subjective as a result of the osteological paradox. Perhaps in reality, anemia was far more prevalent in past populations than we are willing to admit, and yet we cannot infer beyond the evidence we have. Evidence has also been suggested that children were weaned for longer periods of time during ancient and historical times: four years compared to only twenty-two months today (ibid.). It is not clear how this correlates with "healthier" children. Of course breast milk is today seen as better than bottled formula, and yet in terms of iron content, breast milk is actually lower then the new "iron fortified" brands. Had ancient and historic mothers been anemic themselves, their breast milk would likely have been even more deficient. What appears to be evident throughout the literature is that no single cause has been accepted wholeheartedly. More prevalent today is a third model in which a variety of causes have been attributed to the prevalence of anemia. These include diet, parasites, environment, and the presence of "outsiders", whether traders or later colonists, such as the Spanish. I think, however, that this is just an easy way out, but does not rule out the probability that there is an alliance of causes. Until more research is done, and perhaps when the medical community and the archaeological community can collaborate, this is what we are left with. I agree that this third model can explain every incidence of anemia among the ancient and historical Maya,
but until it can be solidified as a group cause it appears to just be a “band aid” solution, that needs to be studied in greater detail.

Conclusions

Anemia has reached pandemic proportions in our world population, so it would seem natural to assume that it has regularly been present in past populations. Unfortunately we only have a limited number of skeletons from which to learn, and inferring beyond that sample has its own inherent problems. From the available sample however, anemia was an important ailment and as such it is important to understand the causes. The maize-dependent model and the parasitic model have both been presented in the literature over the past quarter century and yet there is still no clear consensus. The new “third model” has tried to solidify the previous ideas by simply incorporating them in order to explain the vast majority of incidences, and yet maybe that should not be the goal. Just as every other pathology does not always manifest itself in the same manner, perhaps anemia simply has different causes related to genetics, socio-economic standing and the environment. Therefore, perhaps we should not try to create one causation but rather population or environmentally specific theories (as opposed to one all encompassing hypothesis) that would explain the varying degrees of the illness. Just as stature and age need to be looked at with population specific models, specific manifestations of anemia should also become specific to populations throughout the world. Until more research is done, and perhaps more work is conducted on analogy, we will have to rely on the questionable models that exist today. In my opinion it would appear that diet should still form the basis of any model, especially considering that the addition of iron, either as supplements or extra foods, can have such a reversal effect on the disease.

Bibliography


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