The paleopathology of pellagra: investigating the impact of prehistoric and historical dietary transitions to maize

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Summary – This article outlines interdisciplinary research avenues that can aid in reinterpreting and expanding our understanding of the skeletal biology of maize intensive diets. We suggest that new and innovative perspectives can emerge from hypotheses concerning the impact of diet and health on skeletal manifestations of pellagra. Pellagra, a niacin/tryptophan deficiency disease, is prevalent in populations that have high maize/low protein diets. Historically it has been essentially a disease of undernutrition and social inequality. We offer a unique analysis of both macro- and microstructural skeletal indicators from 31 individuals known to have died from pellagra (n=14) and non-specific general malnutrition (n=17). These cases are part of the Raymond Dart Skeletal Collection, housed at the University of Witwatersrand Medical School, Johannesburg, South Africa. This sample was drawn from a mid-to-late 20th Century Black South African population. In earlier reports we demonstrated that these individuals were found to exhibit a high incidence of alveolar bone loss, dental caries, enamel hypoplasias, periostitic lesions, osteomyelitis, cribra orbitalia, and cranial pitting. The frequency of specific pathological indicators separated the pellagrins from those with non-specific general malnutrition; however the indicators were not pellagra specific. We strongly argue that pellagra does affect bone in a noticeable way at both the macro- and micro-level, and therefore as a diet-related disease can not be excluded as a possible cause for lesions seen in maize dependent populations. We have also reported on the histological findings for rib samples taken from a subset of pellagra (n=10) and general malnutrition (n=14) cases. The critical difference between pellagrins and non-specific malnutrition cases was a decreased cortical area for pellagrins. Given its implications for interpreting the paleopathology of prehistoric and historical transitions to maize intensive diets we present a checklist of macro- and micro-level indicators for investigating a signature pattern for the skeletal biology of pellagra. The overall synthesis of our findings provides new interdisciplinary insights into skeletal-based interpretations of nutrition and micronutrient-related health problems for populations undergoing dietary transitions.

Keywords – pellagra, dietary deficiencies, skeletal system.

Introduction

Across the globe at various times and places the consumption of maize has led to a nutritional deficiency disease called pellagra. Historically, wherever maize became an important dietary staple pellagra often followed. Roe (1973) aptly titled her classic social history on this disease “A Plague of Corn,” which highlights its often insidious impact on human populations. Pellagra has been most strongly linked to high-maize and low-protein diets, which are usually deficient in both niacin (a...
water soluble B-vitamin) and tryptophan (an essential amino acid). New World indigenous peoples were thought to be protected from the disease by ubiquitous alkali processing techniques, using lime (CaOH) or plant ash (e.g., KCl, MgCl), which helped to increase the bioavailability of niacin and tryptophan in maize (Katz et al., 1974). This however may or may not have been true in many prehistoric contexts. Consequently, the first descriptions of pellagra were in the Old World during the 18th century. It was especially prevalent and became endemic among peasant populations from Italy and Spain, who were consuming large amounts of maize, without the benefit of an alkali culinary tradition. Pellagra has essentially been a disease of under-nutrition and social inequality around the world (Brenton, 1998, 2004).

Our main objective for this article is to outline research issues and methods that will aid us in reinterpreting and expanding our understanding of paleopathology associated with prehistoric and historical transitions to maize intensive diets. This is an interdisciplinary effort that brings together the fields of paleopathology, bone physiology, nutrition, epidemiology, demography, medical history and archaeology. From this holistic approach we believe that new and innovative interpretations about the diet and health of maize dependent populations can emerge from hypotheses concerning skeletal manifestations of pellagra. Although a great deal of research has focused on the paleopathology and isotopic signatures of maize intensification, to date no critical discussion has emerged related to the potential affect of pellagra on prehistoric peoples (Brenton & Paine, 1998, 2000, 2002, 2005; Paine & Brenton, 2002, 2004). For all ....... on purpose it is essentially non-existent in the paleopathology and paleonutrition literature. This includes both skeletal indicators as well as any in-depth study of the archaeology of alkali or other food processing technologies that might have developed to prevent it.

As an introduction to the issue we provide an overview of pellagra as a nutritional deficiency disease and the impact of pellagra on skeletal tissue. This is followed by a summary of our own research on both macro- and micro-skeletal indicators recorded from a skeletal sample of Black South African pellagrins. We propose that a focus on micronutrient deficiencies such as this adds an important dimension to our understanding and assessment of the diet and health of peoples both past and present.

Overall this paper is a case study in paleopathology that exemplifies a state of the art assessment of the impact of pellagra and malnutrition on human bone. Not only is it a report on the only skeletal sample of pellagrins known to date, our work and assessment provides a critical link to the pioneering work of Gilman and Gilman (1951) completed over 50 years ago. This 21st century bridge to their research continues an investigation in to the impact of health and nutritional status on bone physiology and human well-being.

**Pellagra as a Nutritional Deficiency Disease**

Pellagra is generally characterized as a niacin and/or tryptophan deficiency disease. A dietary deficiency in niacin (also known as the water soluble vitamin B3) is manifest as a disruption in the maintenance of cellular processes resulting primarily from decreased levels of the coenzymes NAD (nicotinamide adenine dinucleotide) and NADP (nicotinamide adenine dinucleotide phosphate) (Henderson, 1983). These biochemical alterations contribute to pellagra’s classic symptoms discussed below. If dietary protein intake is adequate, however, the essential amino-acid tryptophan can be converted to niacin at a ratio of 60:1 (National Research Council, 1989). Other factors that inhibit this conversion or the bioavailability of niacin and tryptophan can also contribute to the etiology of pellagra. This can include high levels of the amino acid leucine; zinc and iron micronutrient deficiencies; as well as hormone and drug interactions.

The medical literature on the nutritional and social history of this disease is extensive and provides many classic studies on vitamin and nutrient deficiencies (see Carpenter, 1981; Etheridge, 1972; Roe, 1973; Terris, 1964). Again, the common denominator for developing pellagra is usually an association with eating maize as the major dietary staple, with few supplemental foods, and the lack of an alkali or equivalent food processing technology (e.g., tortillas, hominy) (see
In many communities pellagra was endemic, occurring seasonally when stored maize was the primary or only source of nutrients. Therefore, the disease is common when one consumes a high-maize and low-protein diet. We suspect that this may have also been the case amongst prehistoric New World indigenous peoples that were maize intensive horticulturists. Even though the introduction and intensification of maize production has an extensive archaeological record throughout the Americas, there is little discussion on the development of related food processing technologies or the role of pellagra.

The word pellagra can be literally translated as rough skin. Classic soft tissue and behavioral symptoms include the four “D’s”: dermatitis, diarrhea, and dementia, with the fourth “D” being death. More specifically, tissue symptoms are often characterized by dry, scaly and atrophic cutaneous skin lesions on the hands, arms and legs. Patterned symmetrical erythema or reddening of the epidermis may occur on the face in a butterfly shape, and around the neck. The later form is often referred to as Casal’s necklace after the physician who first recognized pellagra in 18th century Spain (Carpenter, 1981; Roe, 1973). Inflammation of the mouth and swelling of the tongue (stomatitis and glossitis) along with diarrhea are seen as the disease progresses. Later stages of pellagra are signaled by nausea, vomiting, bloody stools and neurasthenia, which can be expressed as a loss of appetite, physical weakness, depression and an increased level of anxiety (Harris and Harris, 1941).

It is also important to note that in many cases of pellagra anemia and immuno-suppression are common, leading to the onset of a host of infectious diseases. This reaction is expected as a nutrition-disease synergism. In short, the worse case scenario would progress as follows: the severe burning and itching of the dermatitis is later accompanied by bloody stools. This is followed by periods of dementia and if left untreated results in inevitable death. It should be emphasized that more often than not pellagrins did not die. If their diet was altered to include sources of niacin or tryptophan, often unknowingly, the signs of pellagra disappeared. These symptoms demonstrate the biocultural nature of the disease. Pellagra is the biological outcome of inadequate or unbalanced diets usually associated with the cultural dimensions of social inequality and differential access to nutrient-rich foods (Brenton, 1998).

Impact of pellagra on bone

Published research on skeletal manifestations of pellagra is very limited. A passing mention of the “possibility” of pellagra in prehistoric skeletal collections has been briefly noted by Wells (1964: 119), Brothwell (1967: 63), Buikstra (1992: 88), and Larsen et al. (2002: 411). During the first few decades of the 20th century the high rate of endemic pellagra in southern U.S. populations led to a number of medical and epidemiological studies and publications attempting to elucidate the etiology of pellagra. Explanations ranged from toxic molds in corn, to insect-borne pathogens, to bad genes. By the 1920’s researchers like Joseph Goldberger of the U.S. Public Health Service claimed a dietary factor as being involved, linking pellagra to maize consumption and poverty (Terris, 1964). Yet, his work was not widely accepted for almost 20 years, by which time niacin had been recognized as the deficient element, and cereals were being fortified. Unfortunately, most of this American research had little relevance to skeletal tissue.

The fragility and brittleness of ribs and long bones related to pellagra were observed early on by European physicians from the late 18th through 19th centuries (Marie, 1910). Early 20th century publications usually cite works in Italian by Lombroso (1892). He noted that the fragility of bones in pellagrins depended on the eccentric atrophy of the compact substance (hard bone) with hypertrophy of the medullary substance. This process produced thinning of the outer portion of the bone, which could be demonstrated under the microscope (see Funk, 1922; Lavinder, 1911; Marie, 1910; Niles, 1912; Roberts, 1914). Today we would generally refer to this phenomenon as osteoporosis. Roberts (1914) presents x-ray illustrations of the hands of pellagrins which show the rarification (increased porosity) of the phalangeal ends of the bones. In addition, changes analogous to those wrought by osteomalacia;
delayed healing of fractures; demineralization; and lesions characteristic of experimental scurvy, have been noted in association with pellagra (Harris, 1919; Roberts, 1914).

An invaluable yet rarely cited publication from 1951, “Perspectives in Human Malnutrition” by Joseph and Theodore Gillman, provides some of the best evidence for skeletal manifestations of pellagra and the reaction of bone in malnutrition. As physiologists working with populations of South African Blacks, this team of brothers combined data from skeletal samples, clinical studies, and autopsy reports, to give an in-depth picture of the physiological impact of pellagra and malnutrition. In fact it was the Gillmans’ research that led us to the skeletal sample of pellagrins we’ve analyzed and have reported on below.

Their work related to skeletal tissue and pellagra can be summarized as follows (Gillman & Gillman, 1951): (1) Radiological studies of adult pellagrins demonstrated marked osteoporosis; (2) A negative mineral balance in pellagrins was noted which indicated active mobilization and excretion of endogenous mineral substances, and undoubtedly impacted the turnover of bone; (3) Extensive dental caries were present in over half of the pellagra patients. In most cases caries were associated with severe gingival retraction, sepsis, exposure of the cementum, and loosening of the teeth; and (4) An examination of costo-chondral junctions removed from autopsied infant pellagrins demonstrated varied reactions that were a) similar to scurvy with the fracturing of the trabeculae and chondroperiosteal angle, b) unmistakably rickets, and c) created a zone of ossification where the trabeculae were seen to be covered with osteoid.

Additional research on pellagra over the last half-century highlights other micronutrient factors involved in niacin and tryptophan metabolism, which may have an influence on skeletal tissue. The essential minerals iron and zinc can affect the tryptophan to niacin conversion. For example: (1) An inadequate intake of iron reduces the utilization of tryptophan for conversion to niacin although it does not effect niacin utilization (Oduho et al., 1994); (2) Low levels of niacin will demand more tryptophan and therefore more iron; and (3) If tryptophan is deficient, this can lead to low plasma zinc levels and low bone zinc concentrations (Krieger & Statter, 1987). Conversely low intakes of zinc may inhibit the tryptophan to niacin conversion. Therefore, iron and zinc deficiencies can contribute to and must be understood in the larger context of the etiology of pellagra.

Many essential minerals, including iron and zinc, are known for example to bind to phytates in maize (Lynch, 1997; Reddy, 1989). The phytates form insoluble complexes with minerals making them physiologically unavailable in the diet. This can lead to mineral deficiencies, which are further exacerbated by the synergisms of infectious and parasitic diseases. Iron deficiency from a number of stressors (diet, infection, etc.) can manifest itself in the form of abnormal bone metabolisms such as porotic hyperostosis and cribra orbitalia. Zinc deficiency has been associated with infections and skeletal manifestations of spina bifida (although greater attention has been given recently to the role of folic acid in this birth defect). We suggest a reassessment of these highly recognized bone pathologies in light of their micronutrient interaction with pellagra.

Unfortunately, the focus of this article cannot concern itself with all the physiological and biochemical relationships between nutrients and their impact on bone. It should be clear, however, that whereas no specific diagnostic skeletal criteria can be established for pellagra from this literature; it does strengthen the argument for the inclusion of pellagra in discussions related to the manifestation of several paleopathologies observed in maize dependent populations. The following sections report on our own analysis of a skeletal sample of pellagrins that will provide further insights in to the paleopathology of pellagra and is used to strengthen our argument that many of the skeletal pathologies routinely associated with maize intensification, such as increased dental caries and forms of porotic bone (e.g., Goodman et al., 1984), may also be attributed to pellagra.

We will show that pellagrins do exhibit skeletal lesions in dry bone and as a dietary disorder must be considered along with iron deficiency anemia when examining the burial remains of population known to be maize dependent. Specifically, we will make the case that niacin deficiency should also be added to this list of potential factors affecting the diet and health of prehistoric and historical...
populations highly dependent on maize.


**Skeletal Analysis of a Known Sample of Pellagrins**

**Materials and Methods**

We examined thirty-four autopsied 20th century Black South African remains from the Raymond Dart Skeletal Collection, housed at the University of Witwatersrand Medical School, Johannesburg, South Africa (Table 1). Age at the time of death and cause of death are recorded for each case. All of the individuals in our sample died from dietary deficiencies, either pellagra, non-specific general malnutrition, or scurvy. Individuals diagnosed with pellagra were collected between the years of 1940-82 while those who died of non-specific general malnutrition and scurvy were collected during the period of 1926-90.

Macro-skeletal data reported here were collected on fourteen pellagrins, seventeen cases of non-specific general malnutrition and two cases of scurvy. Based on a review of the literature and skeletal collection listings available to us, this sample of fourteen pellagrins represents the only sample of autopsied skeletal remains with a cause of death by pellagra known to us to date from anywhere in the world. The two cases of scurvy represent all of the cases listed in the Raymond Dart Skeletal Collection.

These well documented specimens provide us with a unique opportunity to test our hypothesis that pellagrins exhibit skeletal lesions normally associated with other maize intensive dietary deficiencies and at similar rates. The preservation of the skeletal material used for this purpose was excellent and most of the individuals had a complete inventory of skeletal elements. We undertook a skeletal inventory of bone available for examination and a survey of skeletal lesions (periostitis, dental caries, cribra orbitalia and cranial pitting) for each individual. This was conducted using methods suggested by (Buikstra & Ubelaker, 1994). Following Goodman & Martin’s (2002) suggestion, cranial pitting and cribra orbitalia lesion were lumped together as a single indicator of dietary health. In addition, calipers were used to measure alveolar bone loss while cortical bone loss for the 4-6th rib was measured under a microscope.

**Results**

An examination of the fourteen pellagrins from the Raymond Dart Skeletal Collection (Table 2) clearly shows that they exhibit the type of lesions in

<table>
<thead>
<tr>
<th>Cause of Death</th>
<th>Sample Size</th>
<th>Age Range</th>
<th>Mean Age</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pellagra Female</td>
<td>14</td>
<td>27-29</td>
<td>51</td>
</tr>
<tr>
<td>Pellagra Male</td>
<td>5</td>
<td>27-70</td>
<td>43</td>
</tr>
<tr>
<td>Non-Specific Malnutrition Female</td>
<td>6</td>
<td>39-10</td>
<td>53</td>
</tr>
<tr>
<td>Non-Specific Malnutrition Male</td>
<td>11</td>
<td>15-65</td>
<td>46</td>
</tr>
<tr>
<td>Beri-Beri Male</td>
<td>1</td>
<td>25</td>
<td>25</td>
</tr>
<tr>
<td>Scurvy Male</td>
<td>2</td>
<td>39-40</td>
<td>39.5</td>
</tr>
<tr>
<td>TOTAL</td>
<td>34</td>
<td>16-89</td>
<td>48</td>
</tr>
</tbody>
</table>
high frequencies which are often attributed to scurvy and iron deficiency anemia in maize dependent populations. For pellagrins periostitic lesions of the lower leg are very common and are seen in 71% of the cases. In addition, dental caries are seen in 35.7% of the cases while cribra orbitalia and/or cranial pitting occur 28.5% of the time. The most extreme skeletal/dental problems for the pellagrins are associated with alveolar bone loss which is on average 0.50cm of bone loss per pellagrin and extreme cortical bone loss of the rib (13.6mm2). More dental caries were noted in male pellagrins (44.4%) than females (20%). Non-specific malnutrition cases had 58.8% with dental caries. There are no significant differences in the frequency of these skeletal lesions between the cases of pellagrins and the non-specific general malnutrition which includes individuals with scurvy and iron deficiency anemia.

Additional research on bone histology from this collection by the authors (Paine and Brenton, 2006) has demonstrated that pellagra and non-specific general malnutrition also affects the micro-anatomical features of the rib by lowering the bone turnover rates resulting in very low osteon population densities. To summarize our histological findings, the rate of secondary osteon production appears to be extremely low for individuals suffering from pellagra, 72% to 36% slower in production than expected. The mean rate of the lower osteon population density for our 10 pellagra samples is 59%; the mean for the nonspecific malnutrition samples is 49%. These results are based on expected Osteon Population Density (OPD) for a given age. OPD is the number of intact and fragmentary secondary osteons found per mm2 area of bone (Stout & Paine, 1992; Stout & Paine, 1994). For example, a pellagrin with an age at death of 38 years would have an expected OPD of 25.44; we found that this individual actually had an OPD of 7.84, showing 69% less than expected OPD formation. It is clear that extreme micro-nutrient deficiencies do affect the remodeling process of bone. OPD rates may be one of the several micro-anatomical key indicators to elucidate dietary problems of historic and prehistoric communities.

Table 2. Frequency and metric data for dental and skeletal lesions.

<table>
<thead>
<tr>
<th>Cause of Death</th>
<th>Sample Size</th>
<th>Mean Age</th>
<th>Periostitic Lesions</th>
<th>Dental Caries</th>
<th>Alveolar Bone loss</th>
<th>Cribra/Cranial Pitting</th>
<th>Periapical Abscess</th>
<th>Rib Cortical Area*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pellagra</td>
<td>14</td>
<td>51</td>
<td>71%</td>
<td>35.7%</td>
<td>0.50cm</td>
<td>28.5%</td>
<td>42.8%</td>
<td>13.6 mm2</td>
</tr>
<tr>
<td>Male</td>
<td>5</td>
<td>43</td>
<td>60%</td>
<td>20.0%</td>
<td>0.45cm</td>
<td>20.0%</td>
<td>40.0%</td>
<td>14.22 mm2</td>
</tr>
<tr>
<td>Female</td>
<td>9</td>
<td>55</td>
<td>77%</td>
<td>44.4%</td>
<td>0.51cm</td>
<td>33.3%</td>
<td>44.4%</td>
<td>13.41 mm2</td>
</tr>
<tr>
<td>Non-Specific Malnutrition</td>
<td>17</td>
<td>48</td>
<td>58%</td>
<td>58.8%</td>
<td>0.38cm</td>
<td>35.3%</td>
<td>52.9%</td>
<td>17.49 mm2</td>
</tr>
<tr>
<td>Male</td>
<td>6</td>
<td>53</td>
<td>50%</td>
<td>50.0%</td>
<td>0.25cm</td>
<td>50.0%</td>
<td>50.0%</td>
<td>15.37 mm2</td>
</tr>
<tr>
<td>Female</td>
<td>11</td>
<td>46</td>
<td>63%</td>
<td>63.6%</td>
<td>0.43cm</td>
<td>27.3%</td>
<td>54.5%</td>
<td>18.67 mm2</td>
</tr>
<tr>
<td>Beri-Beri</td>
<td>Male</td>
<td>1</td>
<td>25%</td>
<td>0%</td>
<td>0.15cm</td>
<td>0%</td>
<td>0%</td>
<td>No Data</td>
</tr>
<tr>
<td>Scurvy</td>
<td>Male</td>
<td>2</td>
<td>39.5%</td>
<td>50%</td>
<td>0.40cm</td>
<td>0%</td>
<td>0%</td>
<td>24.4 mm2</td>
</tr>
</tbody>
</table>

*Rib cortical area is based on the following data: pellagra = 10 (3 females, 7 males); non-specific malnutrition = 14 (5 females, 9 males); scurvy = 2 males.
Discussion

Knowledge of skeletal indicators associated with pellagra has been available to anthropologists for well over a century but was never noted. Our work shows that most of the clinical expectations made by past physicians and researchers (Harris, 1919; Roberts, 1914; Gillman & Gillman, 1951) can be seen as macro-anatomical indicators in dry bone. For example, we found that pellagrins do exhibit, cortical bone loss of the rib, periostitic lesions, dental caries, and extreme alveolar bone loss which are the same general lesions that have been linked to iron deficiency anemia, scurvy and other mineral or vitamin deficiencies described by most paleopathologists. And yet, it appears that there are few if any specific macro-skeletal lesions associated with pellagrins that separate them from non-specific malnutrition or from specific dietary problems like iron deficiency anemia or scurvy. There is no difference in the frequency of lesions between the two groups. Pellagrins do not exhibit a higher or a lower frequency for periostitis, cribra orbitalia, or dental caries than those individuals suffering from other dietary problems. They show the same basic lesion potential.

The critical point here is that pellagra, like scurvy and iron deficiency anemia, does affect bone in a noticeable way and can be viewed at both the macro- and micro-level. Ultimately, as a dietary problem pellagra, can not be excluded as a possible cause for lesions seen in maize dependent populations. In light of our findings, it seems likely to us that some of the reported examples of dietary deficiencies and other nutrition-infection synergisms for maize dependent populations may not be just the result of a lack of iron or vitamin C but the result of niacin deficiency, or in some related combination with other micronutrients.

Pellagra and its associated health problems are well documented in historical populations (Brenton, 1998) and this certainly could be the case for some of the early Native American populations under going the transition from foraging to maize production or populations undergoing culturally determined stress from contact with other peoples. Our position is well supported by the rate of skeletal lesions in pellagrins from the Raymond Dart Skeletal Collection. The pellagrins far exceed the lesion frequencies of Native American populations who became increasingly dependent on maize as their stress levels increased due to contact with Europeans (see Larsen et al., 2002 for background information on these communities). This could also reflect a technological buffer from the ubiquitous use of alkali processing.

Given that pellagrins have the potential to greatly surpass the frequency of lesions often associated with iron deficiency anemia and other dietary problems, we strongly suggest that while observing the paleopathological lesions of the skeletal systems of maize dependent populations, an expanded model be used taking into account the etiology of pellagra and its skeletal alterations. This would then include niacin along with iron and vitamin C as potential micronutrient deficiencies with skeletal manifestations. In doing so we increase the potential for accurately interpreting the biocultural impact that diet had on the health of past populations. These models must also be prepared to account for the complex nutrition-infections synergisms that are impacting the skeletal system, too.

Bone histology can also further aid the assessment of how pellagra may have affected the health of individuals from the past. We suggest that attention be paid to the cortical bone of ribs, both in terms of cortical area and micro-anatomical features (Paine & Brenton, 2006). Work done by Sedlin (1964) supports this suggestion, and has shown that rib cortical bone is sensitive to bone loss due to metabolic problems including dietary deficiencies. It appears that pellagra certainly fits this expectation (Figure 1); in fact, it has produced a greater degree of cortical bone loss of the rib than non-specific malnutrition and scurvy (Table 2).

The measurement of the cortical area of the 4-6th rib can be easily done and may offer a means for separating out pellagra as a dietary problem from scurvy in light of the fact that vitamin C deficiency does not seem to affect cortical bone area. Micro-anatomical features like the turnover rate of secondary osteons may also prove to be very useful in the separation of dietary problems from other health causes (Brenton & Paine, 1998, 2005; Paine & Brenton, 2004, 2006). The two cases of scurvy showed normal cortical bone area for the rib (Table 2) but had 54% less OPD than expected for their ages of 39 and 40 years.
Conclusions

We have identified several critical points to bear in mind while interpreting skeletal lesions seen in maize dependent communities. They are:

1. Niacin deficiency does lead to skeletal lesions that can be observed in dry bone (cortical bone loss in the rib, periostitic lesions, dental caries, extreme alveolar bone loss, cribra orbitalia and cranial pitting).

2. Pellagrins exhibit considerably more cortical bone loss of the rib than non-specific malnutrition cases (Figures 1 and 2). Most of our samples came from males. Surprisingly, the two scurvy cases show considerable cortical bone retention compared to the other dietary cases.

3. Periostitic lesions were found in a slightly greater frequency in pellagrins compared to non-specific general malnutrition cases within the Raymond Dart Skeletal Collection.

4. Dental indicators do no clearly differentiate pellagra from non-specific general malnutrition, although alveolar bone loss is greater in pellagrins.

5. Dental caries are high in pellagrins, yet not as frequent as nonspecific malnutrition cases.

6. Pellagra and general malnutrition also affects the micro-anatomical features of the rib by lowering the bone turn-over rates resulting in very low osteon population densities (OPDs) and this indicator may be one of the keys to understanding micro-nutrient problems via skeletal remains (Paine & Brenton, 2006). Even the individuals suffering from scurvy showed considerably lower than expected OPD rates (54% less than expected).

In a final consideration, it should not be a surprise that pellagrins show the same macro-skeletal lesions that are typically associated in skeletal remains suspected to have been subjected to other dietary deficiencies like iron deficiency anemia or scurvy, or related to infectious disease. Overall, we need to remember that pellagra is a form of micronutrient malnutrition and the skeletal system has only a few macro-skeletal means by which it can respond to a dietary deficiency; bone deformation, the formation of new bone, and bone resorption.

In the end, we have not identified pellagra specific skeletal lesions to be used to separate

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Fig. 1 - A photo of rib microstructure, secondary osteons of a female who died from pellagra. Notice the thin cortical bone and the relatively low osteon population densities. See supplementary material for colour figures.
skeletal cases of pellagra from others, this was not our intent. What we have however demonstrated is that niacin deficiency needs to be included as a potential dietary problem by bioarchaeologists and paleopathologists as they attempt to create a biocultural model for explaining the causes of skeletal lesions seen in prehistoric and historical peoples who were maize dependent. By pointing this out we hope to reduce the dogma of paleopathology by having researchers add pellagra to their interpretive menu as a possible consideration for the presence of periostitic lesions, extreme alveolar bone loss, cortical loss in ribs, high dental caries frequencies and the presence of cribra orbitalia and cranial pitting seen in maize dependent prehistoric and historical skeletal populations.

Finally, the synthesis of our findings offers new insights into skeletal-based interpretations of nutrition and micronutrient-related health problems. It is a highly relevant holistic and interdisciplinary integration of research that advances our knowledge of the health and nutritional status of populations undergoing dietary transitions both past and present.

Info on the Web

**General Information about Pellagra**

www.en.wikipedia.org/wiki/Pellagra  
www.healthatoz.com/healthatoz/Atoz/ency/pellagra.jsp

**Medical and Social History of Pellagra**

www.healthsystem.virginia.edu/internet/him/pellagra.cfm  
www.mnwelldir.org/docs/history/vitamins.htm  
www.eufic.org/web/article.asp?cust=1&lng=en&sid=4&did=16&artid=103
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