NUTRITIONAL ANAEMIA:
A MULTIPLE NUTRIENT HYPOTHESIS CONCERNING
IRON, VITAMIN C, FOLIC ACID AND
VITAMIN B_{12} IN THE DICKSON MOUNDS
# MISSISSIPPIAN PERIOD SKELETAL COLLECTION

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NUTRITIONAL ANAEMIA: A Multiple Nutrient Hypothesis Concerning Iron, Vitamin C, Folic Acid and Vitamin B_{12} in the Dickson Mounds Mississippian Period Skeletal Collection

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"Under the conditions in which nutritional deficiencies occur in the population, they are rarely single" (Scrimshaw 1964:115).

"The third moon is that of the Small Corn. This moon is often impatiently looked for, their crop of large corn never sufficing to nourish from one harvest to another" (Du Pratz 1972:320).

ABSTRACT

In the realm of nutritional anaemia, iron deficiency anaemia is the one well known and most often cited cause of the skeletal alterations cribra orbitalia and porotic hyperostosis. However, this thesis has put forward the possibilities of including several other dietary factors that, when lacking in the diet, can cause anaemia. These other nutrients include vitamin C, folic acid and vitamin B_{12}. By presenting information concerning the sources, metabolism and the physiological roles each of these four nutrients play in the human body, it becomes apparent that an interrelationship exists among them. This synergism is compounded by the fact that when one of these nutrients is lacking in the diet a concomitant deficiency in another can occur. Iron, vitamin C, folic acid and vitamin B_{12} are essential for proper maintenance of hematopoiesis. As a result, when they become deficient in the diet, the quality and quantity of mature red blood cells are altered and anaemia ensues.

In order to extrapolate medical and nutritional literature to a paleopathological study, the Mississippian Period (A.D. 900-1500) skeletal collection from Dickson Mounds, central Illinois was analysed. This sample was utilized due to the wealth of previously published material concerning many facets of their existence, but most importantly due to the presence of cribra orbitalia and porotic hyperostosis already noted for this population. One hundred ten individuals from the 367 possible burials from this time period were used for this study due to their completeness, time constraints and available funds. A demographic profile of the population was first constructed and then a narrow focus was concentrated on the

manifestations of cribra orbitalia and porotic hyperostosis. This smaller population was isolated even further via the macroscopic assessment of characteristic skeletal alterations for each specific nutrient for each individual.

In the end, a small cohort of seven individuals possessed evidence of pathologies indicative of deficiencies and anaemia caused by the four previously mentioned nutrients used in this study. An additional sixteen individuals manifested deficiencies in at least two different nutrients. As a result, this study demonstrated that multiple nutrient deficiencies causing anaemia *can* be identified in one individual. The etiologies of these deficiencies may be caused by frank dietary insufficiencies related to a diet that relied heavily on maize, the pressures of sedentism, population expansion and the concomitant presence of diseases that may have altered the nutritional status of the people buried at Dickson Mounds. In effect, single nutrient hypotheses are no longer warranted for any paleonutritional study as many nutrients are synergistic and deficiencies of isolated nutrients are extremely rare.

ACKNOWLEDGMENTS

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CHAPTER 1: INTRODUCTION

Anaemia may be caused by a number of genetic, environmental and nutritional factors. When studying human skeletal remains from past populations, paleopathologists must take all of these variables into account in order to identify the morbidity, and possibly the mortality, of a population. As a result, stating the exact etiology of anaemia becomes cumbersome and tedious, as all factors including skeletal manifestations, biological, geographical and nutritional statuses must be taken into consideration. Even with this in mind, it appears that researchers have limited their view to single nutrient hypotheses concerning nutritional anaemias and have not fully investigated the possibility of a multiple nutrient hypothesis. By being so focused, archaeological remains that possess skeletal alterations caused by anaemic episodes may have been restricted.

Within the realm of nutritional anaemias, iron deficiency anaemia has been studied most extensively and is the most cited cause of the skeletal lesions of cribra orbitalia and porotic hyperostosis (Lallo *et al.* 1977; Stuart-Macadam 1989a and others). However, anaemia may be caused by a multitude of nutritional factors such as deficiencies in protein, essential amino acids, vitamins A, E, riboflavin [B2], pyridoxine [B6], pantothenic acid and probably niacin, copper, cobalt and various other trace minerals. However, only a few of these nutrients produce known skeletal changes. Therefore, this thesis will concentrate only on four: iron, vitamin C, folic acid and vitamin B_{12}.

When iron, vitamin C, folic acid and vitamin B_{12} are deficient in the diet for prolonged periods of time, iron deficiency anaemia, scurvy and megaloblastic anaemia can

ensue. By being closely intertwined metabolically and within the areas of hematopoiesis and bone development, it is hypothesized that these four nutrients, when lacking in the diet of one individual, will produce common manifestations of cribra orbitalia and porotic hyperostosis. This is due to the fact that they all cause some form of anaemia when deficient in the diet and thus, they all can cause hyperplastic marrow in certain situations. This commonality of skeletal alterations helps tie these four dietary factors together. However, each nutrient also affords other specific characteristic skeletal manifestations that allow the observer to differentiate among them. Consequently, by being aware of these similarities and differences, paleopathologists may be able to formulate a multiple nutrient hypothesis where nutritional anaemia exists. Nutritional and environmental situations of past populations are pertinent to this hypothesis and should also be taken into consideration.

The physical anthropological literature concerning the possibilities of a multiple nutrient hypothesis for nutritional anaemia possesses many strengths and weaknesses. On the one hand, virtually no studies exist that describe the manifestation of multiple deficiencies. Only a concentration of single dietary insufficiencies has occurred (e.g., iron deficiency anaemia, scurvy and rickets) in the literature (e.g., Stuart-Macadam 1989a). The strength in nutritional anaemia literature lies in the acknowledgment that single nutrient deficiencies are rare in nature and more often than not, multiple deficiencies are occurring. However, the recognition of the commonality of multiple deficiencies is hindered by the fact that little is said or data provided for the reader concerning this area. For example, Huss-Ashmore, Goodman and Armelagos (1982:399) realize that in animals "single nutrient

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deficiencies are rarely encountered outside the laboratory setting" and "single deficiency diseases are also relatively rare in human populations". They go on to say that

> "even in clear-cut cases of deficiency disease, however, it is doubtful that only a single nutrient is lacking. An actual diet so restricted as to cause frank vitamin deficiency may be limited in protein, energy or minerals as well. As in animal studies, the degree of interaction of multiple nutritional factors is difficult to assess. Insofar as specific effects of nutrients can be identified in bone, they reflect the portions of the growth and repair process mediated by that element"(1982:400).

However, only statements are being provided by these authors of the possibilities, but virtually no paleopathological research has been performed to provide the evidence.

Steinbock (1976:232) also states that "it should be emphasized that malnutrition is rarely selective for only one dietary component. Malnutrition (including malabsorption and excessive loss of nutrients) is almost always multiple, resulting in deficiency of several or many nutrients to varying degrees". It is understood that some single nutrient deficiencies can manifest themselves on skeletal material, but their occurrence is rare. It then becomes quite ironic that Huss-Ashmore, Goodman and Armelagos (1982:408-410) present a section in their paper titled "*Specific* Deficiencies Identified in Archaeological Samples" (emphasis mine), when they state at the beginning of this section, that it is difficult to ascertain *specific* nutritional deficiencies from archaeological material. Therefore, it seems odd that single nutrient deficiencies are pursued so intently, when researchers acknowledge that it should not and cannot be done on archaeological skeletal samples. By devoting a section to specific deficiencies, the authors are perpetuating the idea that *only* single nutritional deficiencies can be identified on skeletal remains. Paleopathologists can no longer afford to do this and

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should begin exploring the possibilities of evidence pointing toward multiple nutrient deficiencies.

Through medical and clinical literature research, it has become apparent that such a simplistic view of nutritional anaemia is no longer warranted. In fact, a more complicated view exists. Many multiple nutrient deficiency studies, particularly pertaining to the anaemia of scurvy, are abundant in the medical literature (Barratt and Summers 1996; Becerra *et al.* 1998; Bothwell *et al.* 1964; Bronte-Stewart 1953; Goldberg 1963; May *et al.* 1953; Proehl and May 1952; Scully *et al.* 1995; Szarfarc and de Souza 1997; Velez *et al.* 1966; Zalusky and Herbert 1961). These studies provide evidence of the interrelationships between iron, vitamin C, folic acid and vitamin B_{12} within the human body concerning their mobilization, activation and storage.

A study conducted by Wixson and Griffith (1986) is an excellent example demonstrating that interrelationships do exist between ascorbic acid, vitamin B_{12}, folic acid and iron. They believe that the complex interrelationships between these nutrients provide evidence that "pure" spontaneous deficiency of any one nutrient is not a common occurrence (Wixson and Griffith 1986:235). Their study involved the review of past research in which anaemias occurred spontaneously in nonhuman primates due to the feeding of inadequate diets or by inducing anaemia by diets deficient in a nutrient. By providing information of not only the previously mentioned nutrients in this research, but also several other nutrients, Wixson and Griffith present a well-rounded but complicated picture of the nutritional etiologies of anaemias. They also present the possibility of using nonhuman primates as

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biological models for humans because of the similar absorption and retention of nutrients (Wixson and Griffith 1986).

Another study that utilizes nonhuman primates to exemplify the presence of multiple deficiencies in the etiology of nutritional anaemias is that of Eisele *et al.* (1992). This research involved young rhesus macaques (*Macaca mulatta*) housed in outdoor corn cribs, who were fed commercially prepared primate diets. They eventually developed skeletal lesions characteristic of scurvy as well as anaemic conditions. With the presentation of anaemia in scorbutic circumstances, these authors hypothesize that this manifestation may be attributed to vitamin C's role in the absorption, mobilization, and use of iron; folic acid metabolism; and maintenance of vascular integrity. As a result, this study provides evidence that with the deficiency of one nutrient in the body, a concomitant deficiency of several others may also occur.

Isolated cases of multiple nutrient deficiencies causing anaemia have also been observed in human populations. For example, a case of a seven-year-old child presented with iron deficiency anaemia, folic acid deficiency and scurvy were recorded by Clark *et al.* (1992). These insufficiencies were only alleviated with the treatment of not only iron supplements, but also folic acid and vitamin C. This study illustrates the important role of all three nutrients in the development of anaemia.

Unfortunately, this complicated picture of nutrition has not been realized or addressed appropriately within paleopathological research. Instead, researchers have narrowly focused on single nutrient deficiencies when discussing the presence of cribra

orbitalia and/or porotic hyperostosis. For example, a study performed by Lallo *et al.* (1977) provides information about the presence of porotic hyperostosis in four very diverse skeletal populations that range from a hunting and gathering population from Late Woodland (A.D. 900-1050) Dickson Mounds to agricultural populations from Middle Mississippian (A.D. 1150-1350 and A.D. 1490 +/- 55 years) from Dickson Mounds and Eiden in Lorain, Ohio. This study revealed that the frequency of porotic hyperostosis in the agricultural groups was significantly higher ($p < 0.01$) than in the hunting and gathering group and the transitional group. It is concluded that the presence of porotic hyperostosis in the agricultural populations was due to the lack of iron and the presence of high iron-inhibiting absorption substances, such as phytates and phosphorus, found in maize.

At no point in their discussion do these authors suggest that other nutrients, besides iron, may have caused the characteristic anaemic skeletal manifestations. However, they do propose that the presence of chronic infectious disease in these populations may have lowered iron levels within the blood. This recognition of other etiologies is important in paleopathology, but the authors still focused on the lack of only one nutritional element. Evidence of the effects of infectious disease in lowering nutrient levels for several different dietary elements is well recognized in the nutrition and immunological literature and should be utilized by paleopathologists.

Only recently, has the recognition of multiple nutrient deficiencies in causing anaemia been reported. Fairgrieve and Molto (n.d.), propose that the potential cause of cribra orbitalia in Dakhleh Oasis skeletal samples from Egypt may not necessarily be solely

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attributable to iron deficiency. He suggests that folic acid and possibly vitamin C deficiencies may be the sources in presenting cribra orbitalia, as these people relied on goats' milk. Goats' milk lacks folic acid, but is high in ascorbic acid. Therefore, he rules out a deficiency in vitamin C, but extends the possibility that another nutrient along with iron may be causing these lesions.

Janssens (1981) also proposes that folic acid deficiency is the nutritional entity, and not iron deficiency, causing the manifestation of cribra orbitalia and porotic hyperostosis in a young female (16-18 years of age) from a cemetery in Donk, Belgian Limburg. Janssens believes that by relying heavily on goats' milk, which contains relatively little folic acid, megaloblastic anaemia would ensue and probably cause the presence of hyperplastic bone marrow. By demonstrating that bone marrow hyperplasia is a result of any anaemic condition, it should not be bound only to iron deficiency. Janssens' proposed etiology of cribra orbitalia and porotic hyperostosis helps to further the research that more than just iron deficiency can cause anaemic conditions and skeletal alterations. Such research is needed to support the concept of a multiple nutrient hypothesis where nutritional anaemias are concerned. However, it is unfortunate that virtually no other literature presents such a fresh, new perspective on nutritional anaemias.

This study proposes to provide information concerning four nutrients, namely iron, vitamin C, folic acid and vitamin B_{12}, their roles within the body, in causing nutritional anaemia and the possibility of identifying multiple deficiencies via skeletal alterations on one individual. By utilizing 110 individuals from the Mississippian Period skeletal

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collection from the Dickson Mounds cemetery complex from central Illinois, a possible multiple nutrient hypothesis will be demonstrated pertaining to these four nutrients. The Dickson Mounds skeletal remains were observed for this study due to the wealth of information already recovered concerning the presence of anaemia in this population (Goodman *et al.* 1984; Lallo *et al.* 1977; Milner 1992), as well as the multitude of other studies performed on it, such as stable isotope analysis (Buikstra 1992), subsistence base changes (Cook 1979), infectious disease (Lallo *et al.* 1978), childhood morbidity (Clarke 1980), mortality trends (Clarke 1977), dentistry/teeth (Cook and Buikstra 1979; Ditch and Rose 1972; Rose 1977; Rose 1979; Rose *et al.* 1978;), degenerative joint disease (Martin *et al.* 1979), osteoporosis (Saitta 1981), mortuary behaviour and social organization (Rothschild 1979), population genetic analysis (Wolfe Steadman 1997) and comprehensive studies (Goodman *et al.* 1984; Lallo and Rose 1979). In several of these publications, iron deficiency anaemia along with infectious diseases, parasitism, sedentism and an increase in population have all been a part of the etiology concerning the skeletal manifestations of cribra orbitalia and porotic hyperostosis.

Analyses of the Mississippian Period skeletal collection from Dickson Mounds included a demographic profile, i.e., age at death and sex, of the 110 individuals; and macroscopic identification of cribra orbitalia and porotic hyperostosis. Characteristic skeletal alterations for each specific nutrient were then identified for each individual. Statistical analyses were conducted in order to determine if the relationships occurring within this population were significant. In the end, a small cohort emerged which appears

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to possess evidence of multiple deficiencies in iron, vitamin C, folic acid and vitamin B_{12}. These deficiencies may have been related to an inadequate diet, or malfunctions possibly in nutrient absorption and/or retention. Also, the presentation of infectious diseases, probably caused by sedentism, increased populations and unhygienic conditions, may have exacerbated the sub-optimal nutrient stores. Clinical skeletal evidence and medical literature provide evidence of the existence and manifestation of nutritional anaemia. By presenting this information in conjunction with skeletal evidence from several Dickson Mounds burials, this thesis proposes the possibility that a multiple nutrient hypothesis should be followed by paleopathologists when considering nutritional anaemias. The time has come to formulate multiple nutrient hypotheses for not only nutritionally induced anaemias, but also for other areas of paleonutrition.

CHAPTER 2: NUTRITION AND DISEASE

To understand the pathological manifestations of nutritional deficiencies, it is necessary to discuss the metabolism and sources of each nutrient in isolation so that a general understanding is developed about the importance of each one to the body. It will become apparent that iron, vitamin C, folic acid and vitamin B_{12} are crucial to the development and maintenance of bone via different mechanisms. As a consequence, deficiencies in any of these four nutrients can cause alterations in the skeletal matrix. Also, synergistic interrelationships exist between these four nutritional factors which increases the likelihood of multiple deficiencies as each one depends on the other to some respect. The following detailed summary of the consequences of nutritional deficiencies on the immune response and the effects of parasitism and mircoorganism invasions on the nutritional status of individuals will provide an understanding of the importance that nutrition plays within the areas of immunity and successful attacks on disease. The interrelationships between iron, vitamin C, folic acid and vitamin B_{12} and their roles in hematopoiesis also provides information concerning their importance within this biological realm.

2.1 NUTRIENT SOURCES AND METABOLISM

2.1.1 Iron:

Ingestion of the trace element iron can occur via the consumption of various foodstuffs. Iron can be found in considerably large amounts in meats with residual blood or muscle cells such as, liver. Meat is considered a very good source of iron because it

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contains an important chelating agent that allows for more rapid absorption than non-heme iron, or iron in plant-derived foods (Linder 1991:219; Roberts and Manchester 1995:166; Stuart-Macadam 1989a:212). Iron can also be ingested via vegetable foodstuffs, such as spinach, black beans, corn (maize), wheat and soybean. Once consumed, iron is absorbed by the intestine and then transported to the liver, bone, red blood cells and reticuloendothelial cells (Finch 1989; Linder 1991; Roberts and Manchester 1995:166; Stuart-Macadam 1989a:212). Essentially, iron can be found in all cells of the body, but is more concentrated in the liver, spleen and bone marrow. However, body iron is most concentrated in the iron porphyrin complexes, hemoglobin (65%) and myoglobin (10%) (Dallman 1986:12), and therefore, most of the body's requirements of iron can be acquired via the breakdown of these cells. The remainder of the body's iron requirements must be satisfied by exogenous sources.

In order for iron to be metabolized by the body it requires the presence of three proteins: transferrin, which transports iron through extracellular fluids; specific membrane receptors for transferrin, which help determine the amount of iron taken up by individual tissues; and ferritin, which stores intracellular iron (Finch 1989:57). By utilizing these three proteins, iron functions in the body as a transport system for oxygen within blood and muscle, electron transfer in relation to energy metabolism (Linder 1991:217), immunocompetence and possibly neurotransmission, and collagen synthesis (Stuart-Macadam 1989a:212).

Although transferrin and ferritin help transport and store iron, these processes can be

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hindered or accelerated depending upon the presence of certain elements and conditions within the body when consuming a mixed diet. This is mainly due to the fact that the chemical form of iron varies considerably in different foods. As such, substances in food may inhibit the absorption of iron via chemical interactions in the lumen of the gut. Iron absorption can be blocked via the presence of oxalates (spinach), phytates (maize), tannins (tea) and other phenolic compounds that can be consumed from potentially iron-rich foods (Ensminger *et al.* 1983; Linder 1991:219-220; Roberts and Manchester 1995:166; Stuart-Macadam 1989a:212; Wadsworth 1975:100). All of these compounds that are found in various plant foods present iron as an insoluble precipitate, thus rendering it less available for absorption.

An example of the importance of the combination of foods eaten by an individual and how it affects the amount of iron absorbed by the body is given by Layrisse and Martinez-Torres (1971:147). According to information gathered by these two authors, veal iron absorption was decreased by about 20% when veal was consumed with vegetable foods, but when either corn or black beans were combined with animal food, the iron absorption from these vegetables was almost doubled that when they were eaten in isolation. Therefore, with a diet high in substances that reduce the bioavailability of iron, the absorption of this element by the body will be inhibited even with the consumption of iron-rich foods. Obviously, problems also arise with iron deficient individuals who consume these compounds in the presence of low iron content foods.

On the other hand, the absorption of iron can be facilitated by dietary iron-chelating

and reducing agents, such as ascorbic acid (vitamin C), sugars (e.g. fructose), fumarate (salt or ester of fumaric acid which is an intermediate found in the Krebs cycle), citrate, and certain amino acids (Linder 1991:219; Roberts and Manchester 1995:166; Stuart-Macadam 1989a:212; Wadsworth 1975). Vitamin C in the diet allows for a better absorption of iron because it helps to keep iron in the ferrous or reduced state. According to Ensminger *et al.* (1983:84) the acidic environment afforded by the presence of hydrochloric acid from the stomach helps to neutralize some of the alkalinity in the small intestine which consequently increases the solubility, and thus the absorption, of iron. In the absence of these vital substances, consumed iron will not be as readily available, meaning that if these nutrients are deficient in the diet, a deficiency of iron may result in extreme circumstances.

It is important to note that not only is the form of iron found in foods significant, but that the method in which foods containing iron is prepared is also crucial. For example, Sanford (cited by Wadsworth 1975:100) observed that when liver was cooked 40% of the iron was released by acid-peptic digestion, but when it was eaten raw, only about 28% of the iron was released and made available to the body. In contrast to this, Callender *et al.* (cited by Layrisse and Martinez-Torres 1971:142) found that more iron was absorbed from raw rabbit hemoglobin than cooked rabbit hemoglobin (11 % verses 8%).

The recommended daily allowance (RDA) for iron is 6-30 mg (Pennington 1998). If the lowest level is not met, whether it is for reasons of ingesting compounds that reduce the bioavailability of iron, by eating foods that are void of iron or that contain low levels of the trace element, or a combination of these circumstances for an extended period of time

by certain individuals, iron deficiency anaemia can result. Iron deficiency anaemia in the form of a nutritional deficiency, is considered to be the least important type of anaemia in terms of its prevalence on a world wide scale (Stuart-Macadam 1989a:213). This is mainly due to the fact that the body can adapt to the decreased iron intake by increasing its absorption accordingly during this time (Linder 1991:219; Stuart-Macadam 1989a:213) and by proportionally decreasing its absorption when large enough iron stores are already present (Linder 1991:219), or when the level of iron in the diet is raised (Wadsworth 1975:107). In this respect, the body responds more to iron excesses than with a deficiency. As a result, a dietary deficiency in iron may not always be the culprit in iron deficiency anaemia.

Iron deficiency anaemia is influenced or caused by many other important factors that can occur simultaneously or independently. Iron deficiency anaemia can be the result of blood loss due to hemorrhaging, parasitic infestations of the gut or intestines, injury and menstruation (Finch 1989:62-3; Klepinger 1992; Linder 1991; Roberts and Manchester 1995; Stuart-Macadam 1989a). When blood is lost, iron is lost as well due to its association with red blood cells. Chronic diseases, such as cancer, can also cause iron deficiency anaemia. The presence of infectious diseases has also been linked to iron deficiency anaemia due to the fact that during times of infection the body reacts by decreasing the amount of its accessible iron (refer to section on Nutrition and Immunity in this chapter for a more in depth discussion) (Klepinger 1992:123; Linder 1991:225; Palkovich 1987; Stuart-Macadam 1989a:212-3; Weinberg 1974). Iron can also be lost from the simple act of sweating (Layrisse and Roche 1964:296; Linder 1991).

According to Dallman (1986:17) there are three overlapping stages of the progression of iron deficiency anaemia. The first stage is characterized by a decrease in the concentration of serum ferritin, which ultimately reflects the declining concentration of iron stores in the liver, spleen and bone marrow. The second stage includes an increase in the iron-binding capacity of the body as a response to lower levels of iron consumption in conjunction with a declining concentration of serum iron. The final stage is the only stage in which harmful affects are first observed because the supply of transport iron decreases considerably as to restrict the concentration of hemoglobin and/or other iron compounds that are used for other physiologic functions.

2.1.1.1 Functions of Iron:

Iron is used in small amounts for electron transport and is associated with several enzymes, particularly the heme-containing cytochromes, and oxidative phosphorylation in all cells. It is also associated with the enzyme ribonucleotide reductase which means that it is involved in the synthesis of DNA. However, the prime functions of iron and its importance to the body deal with oxygen transport within blood and muscle (Ensminger *et al.* 1983:77) and electron transfer in relation to energy metabolism (Linder 1991:217-218). It has also been linked with some aspects of the immune defense system whereby iron is associated with the enzyme myeloperoxidase of leukocytes used to kill bacteria, along with the production and disposal of oxygen radicals.

2.1.2 Vitamin C:

Humans cannot synthesize vitamin C because, through evolution and genetic mutation, we have lost the microsomal enzyme, L-gulonolactone oxidase (Garrow and James 1993:671; Nobile and Woodhill 1981:17) which can transform a sugar, like glucose or galactose, into ascorbic acid (Nobile and Woodhill 1981:17). According to Nobile and Woodhill (1981:18), humans have lost the gene responsible for producing the enzyme system and as such, humans must rely on exogenous food sources in order to provide the body with a sustainable amount.

Vitamin C can be found in large quantities in citrus fruits, and most fruits in general, rosehips, acerola berries, cranberries, tomatoes, cabbage, potatoes and other vegetables (Linder 1991; Nobile and Woodhill 1981). Vitamin C is also present in liver and glandular tissues of animals that when eaten in sufficient amounts, will provide an adequate intake of this vitamin (Steinbock 1976:254). Therefore, this form of vitamin C is extremely important for those populations living in northern climates where fresh fruits and vegetables are unobtainable. Vitamin C can also be found in small amounts in milk and animal foods, especially tissues of high metabolic activity (e.g., eyes, adrenals, ovaries, spleen, lungs, liver, brain, kidneys, heart muscle, spinal fluid, and all blood cells). However, the content of vitamin C in all of these tissues does not remain stabilized throughout life because as one gets older, the content of vitamin C in these tissues decreases. It is also recognized that differences in vitamin C content in body tissues can also occur due to individual variations and differences between sexes (Nobile and Woodhill 1981:59).

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Although vitamin C can be found in appreciable amounts in these foods, the concentration of it can decrease rapidly via modern storage methods, processing and cooking practices. Vitamin C content in fruits and vegetables can also be affected by climate, conditions in the soil, seasonal variations, degree of maturity, the freshness of the food and the part of it that is actually consumed. For example, more vitamin C can be found in the skin of an apple than in the pulp; during maturation, peas can lose a considerable amount of vitamin C, but such losses do not occur in tubers (potatoes), nor leafy vegetables like spinach; during storage after harvesting, vitamin C content in fruit and vegetables decreases depending on the time and the temperature conditions of storage and the type of food; soft berries and green vegetables rapidly lose their vitamin C content due to bruising (Nobile and Woodhill 1981:32) and vitamin C and folate levels can decrease with ultraviolet light exposure (Linder 1991). Vitamin C content can also decrease when sliced, cut or bruised fruit and vegetables are left standing for an extended period of time or washed with water (Nobile and Woodhill 1981:32). This latter point is due to the fact that vitamin C is a water soluble vitamin. This characteristic also allows vitamin C to readily diffuse throughout the body, but it also means that appreciable amounts can be excreted in the urine, sweat and feces.

Cabbage can be a good source of this vitamin, but when it is shredded, the cell walls are damaged and an enzyme which is normally present in the cells, called ascorbic acid oxidase, is released. This enzyme oxidizes vitamin C beyond the dehydroascorbic acid stage and all that remains are the oxidized products of vitamin C which no longer possess activity

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(Nobile and Woodhill 1981:32). The rate of enzymatic oxidation also increases when cooking temperatures increase, but then the enzyme is destroyed before the temperature reaches the boiling point (Nobile and Woodhill 1981:32). Therefore, by placing vegetables into boiling water or blanching them the enzyme ascorbic acid oxidase is destroyed, thus preserving vitamin C. Bruising vegetables will also activate this enzyme. For example, bruised tomatoes would contain less vitamin C than unblemished ones (Nobile and Woodhill 1981:34).

When ingested, vitamin C can be absorbed through the mucous membranes of the mouth, stomach and upper part of the small intestine, and from there, it passes readily into the blood stream. This general ease of absorption by several different environments in the body is due to its water solubility. However, intestinal absorption of ascorbic acid and its entry into cells may be facilitated by the conversion of this vitamin to dehydroascorbic acid: this form penetrates membranes better than the reduced form at physiological pH (Shils *et al.* 1994:435). After its entry into the intestinal epithelium or tissue cells, dehydroascorbic acid can be readily reduced back to ascorbic acid.

When consumption levels of vitamin C are inadequate and fall to trace or zero levels in scurvy, the excretion of vitamin C decreases rapidly (Nobile and Woodhill 1981:64). Conservation of ascorbic acid is accomplished via such metabolic adaptations as increased renal tubule reabsorption (Shils *et al.* 1994:436). However, when taken in excess of tissue saturation, much vitamin C is eliminated rapidly from the kidneys (Nobile and Woodhill 1981:58-59; Shils *et al.* 1994:436).

2.1.2.1 Functions of Vitamin C:

In the body, vitamin C functions as a redox system, also known as an oxidation-reduction system. As such, it has the general importance of being an antioxidant by affecting the body's 'redox potential' (Linder 1991:143). Due to this characteristic, vitamin C has the ability to give up hydrogen and become oxidized, or accept hydrogen and become reduced. This translates into vitamin C being a very unstable molecule, which was previously exemplified with the changing content of vitamin C in foods under certain circumstances. By providing the body with a redox system, vitamin C participates in essential and sensitive pathways.

Vitamin C is related to protein metabolism as it is used in the hydrolysis of certain amino acids. For example, it is involved as a reductive cofactor, in conjunction with molecular oxygen, iron and alpha-ketoglutarate, for the normal synthesis of collagen (Garrow and James 1993:240; Shils *et al.* 1994:436). This process involves the hydoxylation of proline and lysine to the hydroxyproline backbone and the hydroxylysine cross-linkage, respectively. Consequently, vitamin C is also a part of the intercellular substances necessary for bone growth, tissue repair and wound healing. It is believed that ascorbic acid is involved in the final stage of collagen formation whereby hydroxyproline molecules add stability to the collagen molecule (Basu and Schorah 1982:42). During the proline hydroxylation reaction, the enzyme-bound iron is oxidized to ferric iron (Fe^{3+}). It is during this particular reaction that ascorbic acid is required in order to reactivate the enzyme by reducing iron back to the ferrous state.

When the body is deficient in vitamin C, as in scurvy, these processes are affected and the general matrix of bone, mucous membranes and skin become compromised. As a result, gum and bone changes, and the failure of wounds to heal could arise from the reduction in the amount of collagen formed. The tendency of blood vessels to hemorrhage can also be explained by the weakness in connective tissue structure (Basu and Schorah 1982:43). A decrease in the urinary excretion of hydroxyproline and hydroxylysine molecules, with a concomitant increase in proline and lysine molecules has been observed in individuals with vitamin C deficiency, thus supporting the claim that vitamin C plays a physiological role in the hydroxylation of these amino acids (Basu and Schorah 1982:43).

As previously mentioned, vitamin C also participates in iron metabolism via the reduction of ferric ions to ferrous ions upon the transfer of iron from the plasma transferrin to the liver transferrin (Garrow and James 1993:240; Linder 1991:143,146; Nobile and Woodhill 1981:65; Shils *et al.* 1994:438). This structural modification ultimately enhances iron absorption from the diet. Vitamin C functions either to maintain iron in the ferrous state or, along with ferrous ions, it can induce the production of a highly active form of oxygen, such as superoxide, which can directly attack substrates in certain reactions (Basu and Schorah 1982:41).

Vitamin C is also necessary for folic acid metabolism by converting folic acid to folinic, or tetrahydrofolic acid, and the protection of these substances in body stores from excretion in the urine (Nobile and Woodhill 1981:66; Shils *et al.* 1994:438). It is also known that vitamin C is involved in the chelation of calcium which means that it has a function in

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bone mineral metabolism as well (Linder 1991:146).

2.1.3 Folic Acid:

The active form of the folic acid molecule consists of three major subunits: a pteridine ring linked by a methylene bridge to para-aminobenzoic acid, together forming pteroic acid, which is joined by a peptide linkage to glutamic acid (Shils *et al.* 1994:404; Garrow and James 1993:249). The term folic acid actually refers to pteroylglutamate, which is a monoglutamyl form of the vitamin. The circulating form of this vitamin is known as 5-methyl-H_4 folate which is produced via reductions and substitutions in the pteridine ring of the folic acid molecule (Garrow and James 1993:250). This conversion is dependent on vitamin B_{12} and ascorbic acid. The addition of up to seven glutamates at the gamma-carboxyl linkage helps to enhance folate-dependent reactions by producing polyglutamyl folate, the major form in which both dietary and intracellular folates occur (Garrow and James 1993:250).

A dietary source of folic acid is essential to humans because it cannot be synthesized by the body. It can be found in large quantities in yeast, liver and other organ meats (muscle meats are considered poor sources), leafy green vegetables such as alfalfa and spinach, mushrooms, nuts, grains and some fresh fruits, particularly citrus (e.g., oranges, strawberries). Even though food folate can be absorbed from the entire length of the small bowel, it is absorbed primarily from the proximal third of the small intestine. Dietary folates exist primarily in polyglutamated, reduced and substituted forms of pteroylglutamate (folic

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acid) which have to be hydrolyzed to free forms (i.e., each molecule of folic acid with one or a small number of glutamic acid residues attached) before being actively transported into the absorbing enterocyte (Garrow and James 1993:250; Shils *et al.* 1994:406). As a result, chemical alterations of the parent molecule must be performed in order for folate to become available to the body. However, some folates are more accessible in different forms of food than others.

A study performed by Tamura and Stokstad (1973:513) revealed that folate residues are more available in bananas, lima beans, liver and Brewer's yeast, while poor availability of folate occurred in foods such as, orange juice, romaine lettuce, egg yolk, cabbage, defatted soybean and wheat germ. As with vitamin C, the bioavailability of folic acid can be altered or even destroyed via heating, and thus, foods containing this vitamin should not be overcooked. In fact, losses from 50% (Ensminger *et al.* 1983:85) to 95% (Herbert cited by Malin 1975:214) of folic acid have been estimated during cooking and storage due to oxidation of the nutrient. The situation can also be intensified when the food is finely chopped and cooked in water for extended periods of time. For example, losses ranged from 46% in halibut to 95% in pork chops for meat, and average losses for vegetables such as, cauliflower and carrots, were from 69% to 78%, respectively (Cheldelin *et al.* 1943).

The bioavailability of folate monoglutamates is much greater than that of folate polyglutamates which provides a significant reason for the hydrolysis of the latter molecules. Folic acid absorbed from the intestine, at physiologic concentrations, is largely converted to reduced forms and then methylated or formylated, whereas at higher concentrations, it is

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transported through the enterocytes without modifications (Shils *et al.* 1994:407). However, according to Shils *et al.*(1994:407), transfer of folate from the intestine to circulation will occur at a faster rate with reduced and formylated or methylated forms, than with folic acid.

The conjugase action of folate hydrolases may be specifically inhibited by food factors described in yeast and beans, and they may also be nonspecifically impaired at acidic pH levels (Campbell 1995:457; Shils *et al.* 1994:407). It is also possible that the activity of the brush border folate hydrolases may be altered in certain diseases (see later discussion in Nutrition and Immunity section). This alteration of folate hydrolases plays a significant role in causing malabsorption and sometimes deficiencies in folate. The impaired mucosal transport of monoglutamyl folates after deconjugation also accounts for many cases of folate malabsorption (Shils *et al.* 1994:407).

Absorption of folate can be enhanced via several mechanisms. For example, the active mucosal transport of this vitamin can be enhanced by the presence of glucose and galactose (Shils *et al.* 1994:407). Absorption of folate may also be enhanced via folate-binding proteins found in milk which protect the vitamin from intestinal bacteria by directly delivering it to mucosal carriers (Linder 1991:140). Studies have also provided evidence that folate may be absorbed more rapidly by the jejunum than the duodenum (Linder 1991:140).

In a healthy individual, total body folate stores can range from 5-10 mg, of which half is stored in the liver (Garrow and James 1993:250; Shils *et al.* 1994:410). Most of the stored folate in the body is in the polyglutamate form. For transport it must be hydrolysed

into smaller molecules, such as monoglutamate. Folic acid, in both metabolically active and inactive forms, is excreted in the bile and urine (Garrow and James 1993:251; Linder 1991:140; Shils *et al.* 1994:410). According to Garrow and James (1993:251), approximately equal amounts of the body folate pool are excreted daily in the stool and urine, with a biological half-life of labeled stores of approximately 100 days. Therefore, in a span of approximately three to six months folate stores can be depleted if it is not consumed in the diet.

Daily requirements of folic acid vary from country to country, and from individual to individual. For instance, Canadian median folate intakes are between 150-200μg/day; in the UK, folate intakes range around 200pg/day (0.0002μg/day); and in the USA, folate intakes range around 3pg/Kg (0.000003μg/Kg) body weight for adults of all ages (Garrow and James 1993:251). Daily folate requirements will increase when factors that increase one's metabolic rate increases and when cell turnover increases (i.e., during pregnancy, lactation and growth spurts). Metabolic rates increase during bouts of infection and during hyperthyroidism, and cell turnover increases during bouts of hemolytic anaemia, during rapid tissue growth in the fetus, and with malignant tumors (Shils *et al.* 1994:412).

2.1.3.1 Functions of Folic Acid:

Folate is required for the synthesis of thymidylate, and thus, is required for DNA synthesis. The reaction requires a vitamin B_{12}-containing enzyme which removes a methyl group from methyl folate and delivers it to homocysteine, thereby converting homocysteine

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to methionine (methyl-homocysteine) and regenerating tetrahydrofolic acid (THFA) from which the 5, 10-methylene THFA involved in thymidylate synthesis is made (Goldsmith 1975:593; Shils *et al.* 1994:414). According to Shils *et al.* (1994:414), "because methyl folate may only return to the body's folate pool via a vitamin B_{12}-dependent step, a patient with vitamin B_{12} deficiency has much of his folate "trapped" as methyl folate, which is metabolically inactive". As such, this "folate trap" hypothesis helps explain why the hematologic impairment of vitamin B_{12} deficiency is not clinically distinguishable from that of folate deficiency. Therefore, in both instances, the hematologic defect results from a decrease during the S, or "synthesis", phase of DNA production caused by the lack of adequate 5, 10-methylene THFA which is used to deliver its methyl group to deoxyuridylate to convert that substance to thymidylate. This means that the lack of DNA synthesis can cause many hematopoietic cells to die in the bone marrow because of their inability to complete the S phase of cell proliferation. In effect, this is a form of "ineffective erythropoiesis".

The disturbed DNA synthesis results in megaloblastosis: the presence of giant germ cells (Herbert 1989:47; Shils *et al.* 1994:416,651). Megaloblasts are identified microscopically by their large size and the presence of stippled sieve-like open chromatin which suggests a defect in nuclear maturation (Shils *et al.* 1994:416). The precise cause of megaloblastic maturation is still obscure, but according to Shils *et al.* (1994:416), it may have something to do with poor thymidylate synthesis. Whether poor thymidylate synthesis is due to folate or vitamin B_{12} deficiency, it ultimately results in the failure to promote

elongation of DNA chains even with a relatively normal capacity to initiate DNA synthesis. Shils *et al.* (1994:416) state that "this process occurs, presumably, because lowered thymidylate concentrations remain adequate to serve as a substrate for 'initiating' but not for 'elongating' the DNA chain by polymerase". On the other hand, the mechanism of the defect may be an "illicit" incorporation of thymidylate precursors, such as deoxyuridylate, into DNA, with a subsequent cleavage of DNA containing the illicit molecule (Shils *et al.* 1994:416).

Many cellular changes occur during folate deficient states in the human body due to defective DNA synthesis (Steinberg *et al.* 1983:605). For instance, multilobation, or hypersegmentation, of neutrophils can occur along with chromosome breaks in lymphocytes (Cooper 1995:90; Exton-Smith 1979:130; Krieger 1982:229). Changes are also observed in epithelial cells which are in essence, similar to changes seen in erythroblasts of the marrow. For example, when the nucleus and the cytoplasm increase in size, the tendency for multiple nuclei and the clumping of chromatin in the nucleus can occur (Chanarin 1969:373). These changes have been demonstrated in many human cells (e.g., buccal mucosa, urinary tract, stomach, and jejunum cells) (Chanarin 1969:373) and thus, they may also be manifested in the skeletal matrix.

Folate enzymes are also required in mammalian metabolic systems involving the transfer of one carbon units (Chanarin 1969:247; Shils *et al.* 1994:418). These metabolic systems include *de novo* purine synthesis, pyrimidine nucleotide biosynthesis and three amino acid conversions (e.g., the interconversion of serine and glycine, the catabolism of

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histidine to glutamic acid, and conversion of homocysteine to methionine), as well as the methylation of small amounts of transfer RNA (Chanarin 1969:247-254; Shils *et al.* 1994:419). In folic acid deficient states, DNA synthesis is reduced due to the lack of methylene THFA that is needed for thymidylate synthesis, and methenyl and formyl THFA for purine biosynthesis (Linder 1991:143). A study has provided evidence that the ultimate capacity for cell division was directly proportional to the intracellular polyglutamyl folate levels (Steinberg *et al.* 1983:610). As a result, DNA synthesis is governed by the amount of folate within each cell and can become compromised when insufficient amounts are present.

According to Shils *et al.*(1994:413), four stages of folate depletion can be identified via its progress throughout these stages. Stage 1, the early stage, occurs when a negative nutrient balance can be characterized by a fall in serum folate (<3ng/ml), but lower red cell folate levels are not yet detected. In Stage 2, folate depletion is indicated by low serum folate, and characteristically, by a fall in erythrocyte folate levels. In Stage 3, folate deficient erythropoiesis occurs, which indicates defective DNA synthesis, abnormal diagnostic deoxyuridine synthesis and granulocyte nuclear segmentation. Finally, in Stage 4, clinical folate deficiency is manifested by gross macro-ovalocytosis, elevated mean corpuscular volume and anaemia. It is noted by Shils *et al.* (1994:413-414) that more than half of the folate depleted individuals who have not yet reached the stage of anaemia, will be missed by screening tests which do not recognize that folate depletion may precede anaemia by many months.

2.1.4 Vitamin B$_{12}$:

Vitamin B$_{12}$ is another vitamin that cannot be synthesized by humans, and as such, must be consumed in the diet. Foods high in vitamin B$_{12}$ (cobalamin) are organ meats, such as lamb and beef liver, kidney and heart, bivalves, some seafood such as crab, salmon and sardines, and dairy products such as cheddar cheese and cottage cheese. As a result, fruit, vegetables and grains are not good sources of this vitamin, which means that true vegetarians could become deficient. The vitamin B$_{12}$ molecule consists of a nucleotide and a structure resembling the porphyrin-like ring which contains four reduced pyrrole rings linked with a central cobalt atom (Garrow and James 1993:245; Goldsmith 1975:598).

Ingested cobalamin is absorbed by a more complex system than that of other vitamins. Absorption across the brush border of the ileum requires the presence of ionic calcium, intrinsic factors and a physiologic pH above six (Linder 1991:137; Shils *et al.* 1994:406). Also, when circulating in the blood, vitamin B$_{12}$ requires different proteins called transcobalamin I, II and III for its transportation (Garrow and James 1993:245-246; Linder 1991:139; Shils *et al.* 1994:406). Normal stores of 1-10mg occur in the body with the liver containing the bulk of them (50-90%). On a daily basis approximately 0.6 to 6μg of the vitamin are excreted in the bile, but is consequently reabsorbed by the ileum (Shils *et al.* 1994:408). This translates into an almost total conservation of this vitamin and thus, deficiencies can take an extremely long time to develop.

As with other nutrients in the diet, cobalamins can be destroyed by extraneous factors, such as strong oxidizing and reducing agents. For example, when ascorbate is

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placed in solution with cobalamins, it can destroy cobalamins by converting them to various analogues which can ultimately block their metabolism (Shils *et al.* 1994:404). Essentially, vitamin B_{12} is resistant to heat unless it is exposed to an alkaline medium in temperatures in excess of 100°C. For example, when muscle meat is boiled at 170 °C for forty-five minutes, a loss of 30% of the vitamin can occur (Shils *et al.* 1994:412).

2.1.4.1 *Functions of Vitamin B_{12}:*

Cobalamin functions as a coenzyme in the catabolism of odd chain length fatty acids (conversion of methylmalonyl-Coenzyme A to succinyl-Coenzyme A) and some amino acid carbons. It also functions as a reducing agent for sulfhydryl (SH) groups that are required for many SH-activated enzyme systems (Shils *et al.* 1994:419). The most important role vitamin B_{12} plays in the body centers around its involvement in DNA synthesis (see previous discussion under Folic Acid Functions for a more detailed explanation). In this process cobalamin is involved in the transfer of a methyl group from methyl tetrahydrofolic acid to homocysteine in order to convert cysteine to methionine (DNA constituents). This reaction also entails the conversion of folic acid into a more bio-active form so that folic acid can be utilized by the body (Linder 1991:138,139). As such, cobalamin is involved with folic acid during the transfer of 1-C units in certain biochemical reactions which means that it may be very difficult to distinguish one deficiency from another.

When vitamin B_{12} is deficient in the body, nerve degeneration due to the lack of myelination can cause neuropathological disorders. Vitamin B_{12} deficiency can also cause

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disruptions in the release of alkaline phosphatase and osteocalcin from osteoblasts (Linder 1991:140). As a result, the production of bone is altered. Due to the interrelationship between folic acid and vitamin B_{12} and their role in DNA synthesis, production of RNA proceeds as normal, but DNA replication and cell division are blocked when vitamin B_{12} (and/or folic acid) is deficient in the diet, thus causing the synthesis of megaloblasts. Consequently, vitamin B_{12} deficiency can also produce megaloblastic anaemia. As such, vitamin B_{12} deficiency will be categorized as megaloblastic anaemia in the rest of this thesis, albeit a deficiency of vitamin B_{12} can also cause pernicious anaemia.

Deficiencies in the aforementioned nutrients are not only caused by inadequate diets, but also many other factors within the human body can alter nutrient levels. For example, a deficiency can result from poor absorption and activation of the nutrient (e.g., consumption of nutrient inhibitors), a temporary increase in its requirement (e.g., disease, febrile illnesses), excessive body losses (e.g., sweating, diarrheal diseases) and possibly a genetic malfunction in certain enzymes that render the nutrient unavailable to the body.

2.2 DISEASE

2.2.1 Nutrition and the Immune System:

This brief introduction to host defense mechanisms will enable the reader to identify terminology used in the proceeding sections dealing with iron, vitamin C folic acid and vitamin B_{12} and their roles in the immune system. Nutritional status of an individual, as well as age and immunologic status, before the onset of disease and infection is crucial to the human body because it can alter the virulence of the pathogen and the ability of the individual to fight it. It has become apparent that a synergism between the loss of nutrients secondary to infection can occur, albeit the pathways to deficiencies can be different and can be modulated by such factors as the type and severity of infection, physiological status of the host and several external factors, such as dietary management (Brown and Black 1981). However according to Scrimshaw (1964), the presence of a pathogenic agent and a deficiency of an essential nutrient alone cannot determine whether or not disease will occur. As such, many different factors govern the onset of disease and infection within the triad of host, agent and environment, i.e., stressors and limiting resources, meaning that the right situations must be encountered for the body to be affected. Although, it must be pointed out that when exposure to pathogens does exist, nutritional status becomes important because both acute and chronic infections can deplete the body of crucial stores of nutrients, which inadvertently can result in nutritional deficiencies rendering a patient more susceptible to secondary or superimposed infections (Beisel 1979). Also, the severity of the macro- and micro-nutrient deficiency can have an affect on the severity of the immunologic impairment

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(Chandra 1990).

Immunity to an infection can be influenced by nutrition in several ways: deficiencies can determine the progress of infection either via action on the host to facilitate the initial invasion of the infectious agent; through an effect on the agent once it is established in the body tissues; through favoring secondary infection; or by delaying convalescence after infection (Scrimshaw *et al.* 1959:369). Many of these areas have been researched, but confusion to their full effect on the virulence or duration of infection is still acknowledged. The relationship between nutrition and infection is complicated, but an effect of either nutrition on infection, or infection on nutrition, has been observed and is well documented.

One primary reason for a loss in nutrients is that consumption is often depressed in sick individuals (Brown and Black 1981; Robinson *et al.* 1986; Scrimshaw 1964 and 1977). When consumption is decreased, the body must rely on stored elements that could be exhausted before healing. Also, there is a tendency to withdraw solid foods and replace with watery infusions and gruels that are less nutritious. A secondary factor deals with the decrease in absorption of nutrients during bouts of infection. This is usually caused by impaired intestinal absorption of both macro- and micro-nutrients during enteric infections and with clinically defined diarrheal diseases (Rosenberg cited by Brown and Black 1981; Robinson *et al.* 1986; Scrimshaw 1977). Malabsorption of nutrients occurs because of the rapid facilitated intestinal movement during diarrheal episodes, a decrease in intestinal enzymes, injury to mucosal epithelium and metabolic consequences of bacterial overgrowth of the small intestine. Impaired absorption of nutrients can also occur with acute conditions

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such as tuberculosis by its direct affects on the gastrointestinal epithelium (Scrimshaw 1977:1537). Another factor that can cause a loss of nutrients is via an increase in catabolic reactions (Beisel 1979; Scrimshaw 1977). This becomes important during febrile illnesses as basal metabolic rates increase and energy stores are compromised. It is also important to note the site, severity, duration and microbial agent causing the infection as these too can alter the nutritional status. Consequently, all of these factors can be heightened by the presence of malnutrition as well as the original immunological response.

The immunological response by the host can occur via two different routes: nonspecific and specific. The nonspecific immune response is essentially the first line of defense against infection as it tries to prevent the entry of the antigen into the immune system and/or the establishment of overt infections. This mechanism can develop independently of the presence of infections or antigenic stimuli and includes: the complement system, phagocytic cells (e.g., polymorphonuclear neutrophils, macrophages, and monocytes), anatomic barriers such as skin, mucous membranes and cilia, mucus, interferon and lysozyme (Kuvibidila *et al.* 1993; Robinson *et al.* 1986). The specific immune response opposes the nonspecific immune response as it is dependent on invasion first. The specific host defense mechanism develops following antigenic stimulation, which includes both cell-mediated immunity and humoral immunity. Both of these forms involve different T-cell subsets, B cells, antibodies or immunoglobins (IgG, IgA, IgM, IgD and IgE), macrophages/monocytes and their secretory products (e.g., cytokines and immunoglobins) (Kuvibidila *et al.* 1993).

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Consequently, which mechanism is activated depends on the type of infection invading the host. For example, extracellular bacteria and viruses will trigger phagocytic cells, complement and antibody production; intracellular parasites and fungi will trigger T lymphocyte production and secretion of lymphokines that will activate monocytes and macrophages; and intracellular viruses will activate cytotoxic T-cells which stem from suppressor (CD8) T-cell lineage and natural killer cells (Kuvibidila *et al.* 1993).

2.2.1.1 Iron and the Immune System:

Conflicting evidence exists as to whether iron has a beneficial role pertaining to its presence or its absence from the immune system when microorganisms and/or parasites attack (see Stuart-Macadam and Kent 1992 and those authors cited within Bhaskaram 1988; Chandra 1990 and 1992; Dallman 1987; Keusch 1990; Scrimshaw 1990; Sherman 1984, 1990 and 1992; Sherman and Spear 1993). Many studies present results that point to iron's importance in fending off disease, while others provide contradictory results which implicate that in its absence, iron cannot be sequestered by invading parasites and/or microorganisms and thus, they cannot grow and proliferate. For example, many researchers believe that anaemic patients exhibit a lower incidence of infection because data suggests that 64-83% of patients with adequate iron stores suffered from various bacterial infections, while only 7% of the anaemic patients suffered (Masawe *et al.* study cited by Sherman and Spear 1993). However, an overwhelming number of studies report that iron deficiency is generally detrimental to the immune system whereby an increase in infection is usually noted

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(Sherman 1990 and 1992; Sherman and Spear 1993). For example, in two independent studies reported by Sherman (1984:255), mean hemoglobin levels signifying iron deficiency were observed to be negatively associated with rates of meningitis and pneumonia in a retrospective study in Papua, New Guinea. Respiratory infections, in general, were reported by parents to be increased during the state of iron deficiency throughout the first eighteen months of life for their children.

Conflicting evidence is partly caused by a number of variables that have not been controlled or properly designed within each experiment, i.e., dose and type of antigen, inoculation route, type, severity of malnutrition or concurrent deficiencies, degree of the infection and presence of a concomitant infection, as well as uncontrollable environmental differences. Inter- and intra-population variations also exist in defining what constitutes "anaemic" levels for groups, i.e., what may be normal for one group may not be normal for another. Problems also arise in using animals to model human responses to certain experimental situations. Therefore, only corollaries can be produced as most human experimentation is deemed highly unethical and immoral. However, some human cases have been identified, but lack of homogeneous experimental situations and unknown medical history, have also plagued these results. Therefore, it becomes very complicated as to which results provide the most viable and plausible explanation for the role of iron in the immune system. With this in mind, both view points are presented so that a clear understanding of the implications, both positive and negative, of iron deficiency on the immune system can be gained.

During an acute infection, iron uptake from the serum is accelerated by the liver so that synthesis of acute-phase reactant glycoproteins, i.e., haptoglobin and fibrinogen, can be increased (Beisel 1979). As such, iron is needed for synthesis of vital proteins that are required for the immune response. Not only is iron required for the synthesis of erythrocytes, but it is also necessary within the immune system for synthesis of certain proteins, as well as its role as an enzyme in certain reactions. For example, interleukin 1 production has been found to be impaired during episodes of iron deficiency (Sherman 1990:144). This has a widespread effect on the body's immune system as impairments in cell-mediated immunity, humoral immunity, nonspecific immune functions and other defenses against infection can all be affected by a diminished interleukin 1 action. Therefore, when iron is deficient in the body, immunity can be inhibited and a predisposition to infections can result.

Animal studies report that iron deficiency can impair the humoral immune response, (see studies cited by Sherman and Spear 1993). For example, a study observed a dose-response relationship between the degree of iron deficiency in rats and their ability to produce an antibody to a tetanus toxoid which was possibly attributed to impaired protein synthesis for antibody production (Nalder *et al.* study cited by Sherman and Helyar 1988:177). The nonspecific immune response is also known to be altered (e.g., a decrease in natural killer cell activity is noted) (Dallman 1986; Suskind *et al.* 1977) in iron deficient children as one study cites that bactericidal capacity of polymorphonuclear leukocytes (PMN) against *Staphylococcus aureus* was reduced in anaemic children (Chandra cited by

Sherman and Spear 1993). It has also been observed that splenic natural killer cell function is reduced by 50% in severely and moderately iron deficient rat pups that were virally challenged four days prior to an assay (Sherman and Lockwood study cited by Sherman and Helyar 1988:176). Cell mediated immunity is markedly impaired (Beisel 1982; Sherman 1984 and 1992; Sherman and Helyar 1988; Suskind *et al.* 1977) and T-lymphocyte count is also decreased significantly during iron deficiency because activation and cell division progression are diminished (Beisel 1982; Dallman 1986 and 1987; Kuvibidila *et al.* 1993; Sherman 1992; Sherman and Spear 1993).

To date, knowledge gained from animal studies have allowed researchers to postulate about the mechanisms that impair immunity due to iron deficiency. With the lack of iron in the body, the activity of the iron-containing enzyme, known as ribonucleotide reductase (needed for DNA synthesis), is reduced thereby inhibiting DNA synthesis of cells including those involved in the immune response which are required to undergo complex differentiation and quick proliferation (see studies cited by Beisel 1982; Dallman 1987; Kuvibidila *et al.* 1993; Sherman 1984 and 1990; Sherman and Helyar 1988; Sherman and Spear 1993). Iron is also necessary for the activation of myeloperoxidase (enzyme responsible for bactericidal activity of PMN), serum peroxidase, lysozyme and lactoferrin (Beisel 1982; Chandra 1992; Sherman 1984; Sherman and Helyar 1988; Sherman and Spear 1993). Therefore, when iron is lacking, the capacity of these compounds to function in the immune system is most likely reduced.

Also, lesions in certain immunologically important organs and tissues, such as the

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spleen and thymus, have an effect on their functions within the immune system (Sherman 1992; Sherman and Spear 1993). An example of the crucial importance of the role that iron plays in the immune system was exemplified in studies where newborn pups of iron deficient pregnant dams showed severe fatty degeneration of the liver and lesions in the spleen and thymus (Rothenbacher and Sherman, and Sherman and Tissue cited by Bhaskaram 1988:151). Also, when iron deficiency was prolonged during the critical periods of gestation and lactation, a potential risk factor for the development of the immune system of the offspring was noted.

However, some might also say that the decrease in serum iron concentration, as in the hypoferremic state, is also beneficial because it helps prevent invading microorganisms and/or parasites from obtaining the iron it needs for growth and multiplication. For example, a study of African patients with iron deficiency anaemia produced malarial attacks that often developed after iron therapy was initiated (Masawe *et al.* as cited by Beisel 1982:445). This evidence promotes the idea that inducing iron deficiency anaemia or lack of parenteral iron administration to iron deficient individuals will be advantageous, but in fact it creates immunological incompetence and susceptibility to a variety of other diseases (Scrimshaw 1990). In response to an iron-restricted environment, bacterial membranes and metabolism are altered so that a stronger affinity to host iron-bound proteins can occur. These induced changes are directly related to the virulence and the resultant infection produced by the invading organisms (Sherman and Spear 1993). Also, Keusch (1990) states that there is evidence from both human infections and experimental animal infections that

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bacteria can proliferate *in vivo* under iron-limiting conditions, but it is also observed that iron administration can increase the incidence of infection rates.

Only with more research and technological advancements will a possible answer be formulated pertaining to the apparent paradox between the positive and negative benefits of iron deficiency and immunity. However, in light of the evidence presented here, iron deficiency does have a deleterious effect on the immune system and its abilities to ward off disease and infection.

2.2.1.2 Vitamin C and the Immune System:

The role of vitamin C in the immune system is an important one, especially pertaining to nonspecific immunity. Due to vitamin C's role in collagen synthesis, when this vitamin is lacking in the diet, the integrity of skin and mucous membranes is compromised. As a result, the first line of defense is affected. Another nonspecific line of defense, phagocytosis, is also diminished during episodes of deficient vitamin C (Ulijaszek 1990:140). According to Siegel (1993:167), nutritional deficiencies of vitamin C have been shown, in general, to lead to an increased susceptibility to infection. Vitamin C's role in the immune response has been suggested by its high concentration in leukocytes, its rapid utilization during infection and its depression in clinical situations often associated with diminished immunologic function (Siegel 1993). The immuno-enhancing effects of vitamin C have been demonstrated via the enhancement of *in vitro* peripheral blood lymphocyte responses to mitogens, antigens and allogeneic peripheral blood lymphocytes (Siegel 1993).

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As a result, it has been demonstrated that cell-mediated immune mechanisms may be severely impaired during the scorbutic state (Anderson *et al.* 1990; Bendich 1990; Kuvibidila *et al.* 1993).

Once again, conflicting evidence burdens the exact effect of vitamin C insufficiency on the immune system. For example, it has been noted in neutrophils isolated from scorbutic guinea pigs that a decrease in phagocytosis of *Bacillus subtilis* occurs and is reversed by vitamin C supplementation, but evidence also exists for the contrary (Chatterjie *et al.* study cited by Siegel 1993:179). However, there is a general agreement that ascorbate plays a positive role in the reduction of symptoms following infection of common colds (studies cited by Siegel 1993:168) and microbial infections via the maintenance of cellular immune functions (Anderson *et al.* 1990). It is believed that vitamin C's role as a free radical within the body enables this vitamin to inactivate bacterial viruses or bacteriophages (Murata *et al.* cited by Siegel 1993), such as the herpes virus, rabies virus and the tobacco mosaic virus. This attack of ascorbate on viruses helps protect cellular energy metabolism via the neutralization of granulocyte-derived hypochlorous acid, thus helping to maintain the functions of the phagocytes and the functional integrity of the lymphocytes (Anderson *et al.* 1990). It has been noted that phagocytic activity of neutrophils and macrophages are not impaired, but their locomotion both *in vitro* and *in vivo* is definitely diminished (Anderson *et al.* 1990; Beisel 1982; Kuvibidila *et al.* 1993).

It has also been observed that the manifestation of delayed-type hypersensitivity (DTH) responses is most likely due to impaired cell-mediated immune responses (Beisel

1982) seen *in vivo* caused by defective migration of macrophages accompanying ascorbate deficient episodes (studies cited by Siegel 1993:174). The diminished hypersensitivity response that has been demonstrated in guinea pigs can be attributed to an impaired ability to develop an inflammatory response (Beisel 1982:425). Chemotactic responses to animal and human polymorphonuclear leukocytes as well as macrophages are observed to be related to the vitamin C status of the individual. For example, vitamin C increases the motility of macrophages due to vitamin C's role in the hexose monophosphate shunt activity (Beisel 1982; Siegel 1993). It has also been suggested that vitamin C protects the body against viral infections via the enhanced production of interferon and an increased T-lymphocyte responsiveness (Siegel 1993) and thus, protection becomes related to the length and severity of the deficiency (Beisel 1982). Unlike iron, the activity of natural killer cells is not affected by vitamin C deficiency and neither is the humoral immune response (Beisel 1982).

Although some experiments appear to be contradictory toward vitamin C's protective role in the immune response, a growing body of evidence does point to its fundamental role in phagocytic cell functions. Also, during disease-related episodes, vitamin C has been observed to fluctuate in accordance and thus, a possible cause and effect relationship is affordable (Beisel 1982). Febrile illnesses have been known to increase requirements of vitamin C (Robinson *et al.* 1986:474) and therefore, with insufficient levels of this vitamin, deficiencies can occur at a faster rate. Infection can also precipitate scurvy in those individuals who have sub-optimal levels or a borderline nutritional status with respect to vitamin C.

2.2.1.3 Folic Acid, Vitamin B_{12} and the Immune System:

Both folic acid and vitamin B_{12} also play important roles within the immune system. As a result, when they become deficient in the body, immunocompetence becomes affected. Due to the role of both of these nutrients in cell proliferation, it becomes apparent that T and B lymphocytes and thus, cell-mediated immunity would be altered by their insufficiencies (Gross et al. 1975). For example, studies by Gershwin et al. (cited by Kuvibidila et al. 1993:139), state that lab animals experience spleen and thymus atrophy, reduced T cell numbers, cytotoxicity of T cells and splenic lymphocyte proliferation, as well as impaired lymphocyte responses during folic acid deficiencies (Bendich 1992; Beisel 1982; Newberne 1977). According to Lindenbaum (1979:23), there is an association between respiratory and inflammatory disorders, i.e., tuberculosis and arthritis, and folate deficiency as infections seem to precipitate megaloblastic anaemia (Scrimshaw et al. 1968). Therefore, it is important to note the presence of superimposed infections that may play a part in precipitating deficiencies.

Experiments with rats with reduced intakes of folic acid often developed more severe signs of disease via a higher incidence of diarrhea and higher intestinal rotaviral titer (Bendich and Cohen 1988:112). Deficiencies in folic acid also produce a reduced host resistance and impaired lymphocyte functions for both humans and experimental animals (Beisel 1982). A diminished humoral immunity is also exemplified in folate deficiency via a decreased mitogenic response of splenic lymphocytes (Beisel 1982) as well as a reduction in cytotoxic T cell function (Shils et al. 1994:651).

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A study performed by Gross *et al.*(1975), discovered that cell-mediated immunity is depressed in megaloblastic anaemia due to folate deficiency and the lack of DNA synthesis. As such, researchers have noticed that leukocyte folate levels decreased in proportion to the severity of the megaloblastic marrow changes (Gross *et al.* 1975:225). Studies have noted a delayed hypersensitivity response to dinitrochlorobenzene in patients with megaloblastic anaemia associated with folic acid deficiency (Shils *et al.* 1994:651). A study by Boles *et al.*(cited by Shils *et al.* 1994:651), reported significant impairment of phagocytosis by neutrophils obtained from patients with low serum levels. Impairment of antibody responses to diphtheria toxoid and human red blood cells has also been reported in individuals with folate deficiency (Shils *et al.* 1994:651).

As observed in iron deficiency, vitamin B_{12} deficiency diminishes the bactericidal capacity and phagocytosis (Ulijaszek 1990) of neutrophils toward *Staphylococcus aureus*. This same observation is not found with folic acid deficiency (Beisel 1982; Gershwin *et al.* cited by Kuvibidila *et al.* 1993:139). However, a study performed by Skacel and Chanarin (cited by Bendich and Cohen 1988:109) states that bactericidal activity against this organism was impaired as well during folic acid deficiency. Therefore, contradictory studies do exist for these nutrients as well. Apparently, with administration of vitamin B_{12} and folic acid, this nonspecific immune function will improve (Bendich and Cohen 1988). An experiment conducted by Thomaskutty and Lee (cited by Bendich and Cohen 1988:110), notes that when rats were placed on vitamin B_{12} deficient diets, after thirty days a lowered resistance to infection occurred when they were inoculated with the parasite *Trypanosoma lewisi*. This

result was believed by these authors to be a product of the role of vitamin B_{12} in the transportation of folic acid into bone marrow cells and/or the uptake of folate by peripheral lymphocytes.

From the previous discussion of each nutrient analysed for this thesis, it should become evident that infections *can* create an increased demand for nutrients which may have been in limited supply originally. Even if these nutrients were not originally limited in the human body, infection will decrease their amounts and thus, a vicious cycle is created whereby the body is in constant flux with trying to fend off infection and/or trying to increase the nutritional elements within the body. Also, the nutritional status of an individual prior to the onset of disease and infection is an important aspect to consider when discussing the presence of hyperostosis and other nutritionally related pathologies.

As can be seen, a synergistic relationship exists between nutrition and infection: nutritional deficiencies lower resistance and infection aggravates existing nutritional deficits. If the nutritional status were in a deficit, then contact with carriers of infectious disease, along with unhygienic conditions, can lead to debilitating episodes and possibly premature death that would not have otherwise occurred in a nutritionally healthy individual.

2.3 HEMATOPOIESIS

2.3.1 The Interrelationship and Roles of Iron, Vitamin C, Folic Acid and Vitamin B_{12} in Hematopoiesis:

Research into the metabolism and the physiological role which each of these nutritional elements play in the human body, especially pertaining to erythropoiesis, skeletal growth and bone remodeling, has revealed that they are interdependent to some extent (Figure 2.1). Erythropoiesis is defined as the production of red blood cells (RBCs) in the bone marrow and blood which is triggered by a decrease of oxygen in both the tissues and the blood. In response to the lowered oxygen levels in the body, the liver releases a protein substrate and the kidneys release an enzyme-like complex, known as the renal erythropoietic factor, which helps convert the protein substance to erythrocyte stimulating factor (Ensminger *et al.* 1983:76). During times of anaemia and hemorrhaging, the production of this last factor increases and consequently decreases when enough red cells are circulating the body.
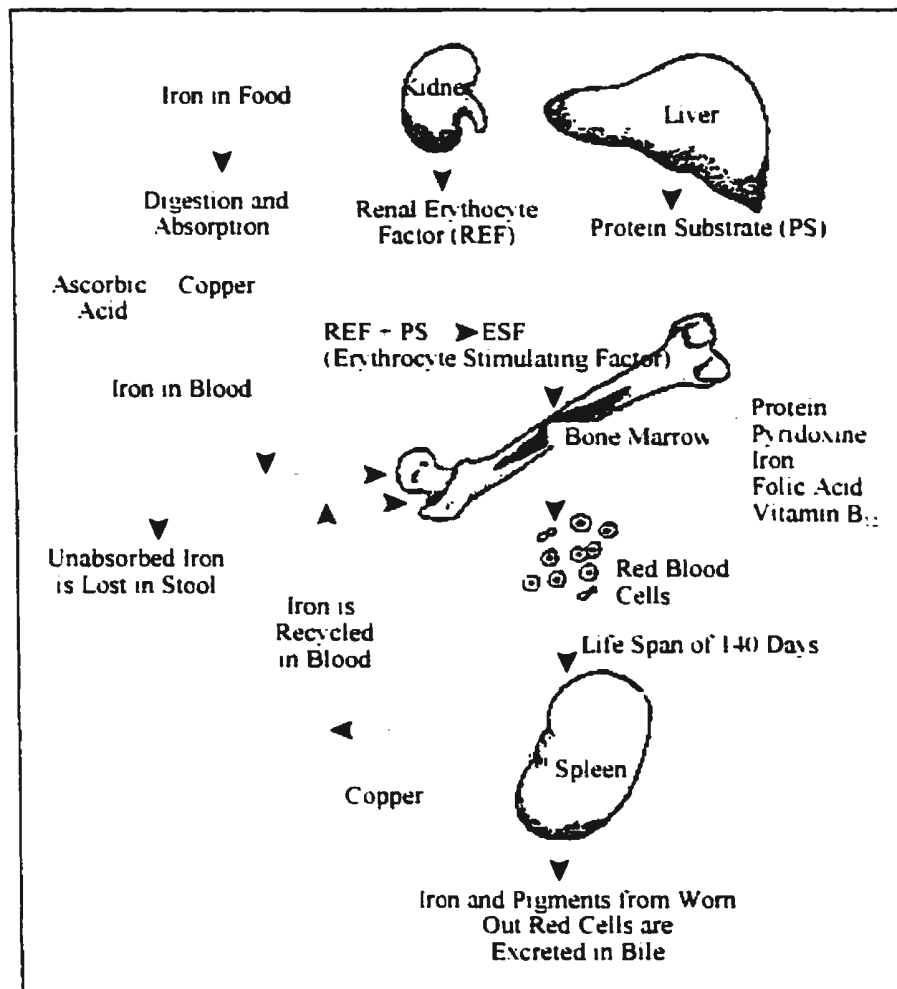
Figure 2.1: Nutrients Needed for Erythropoiesis (Adapted from Ensminger *et al.* 1983:76)

    I. MINERALS:

        Iron: Core of hemoglobin molecule

        Copper: Part of enzyme ferroxidase which converts iron to ferric form for release from tissues into blood

    II. VITAMINS:

        Ascorbic Acid: Reduces iron to ferrous form for absorption

        Pyridoxine: Cofactor in synthesis of hemoglobin

        Folic Acid and Vitamin B_{12}: Control division, growth, and maturation of red cells

    III. PROTEIN:

        Raw material for hemoglobin and red cells

A pool of stem cells found in the bone marrow are stimulated by the erythrocyte stimulating factor to ultimately become erythrocytes. However, several stages are involved in this transformation of stem cells to mature RBCs. These different stages undergo "maturational divisions" in which nuclear and cytoplasmic maturation takes place starting from stem cells and resulting in mature red cells (Figure 2.2) (Beck 1973:14-17; Ensminger *et al.* 1983:76).
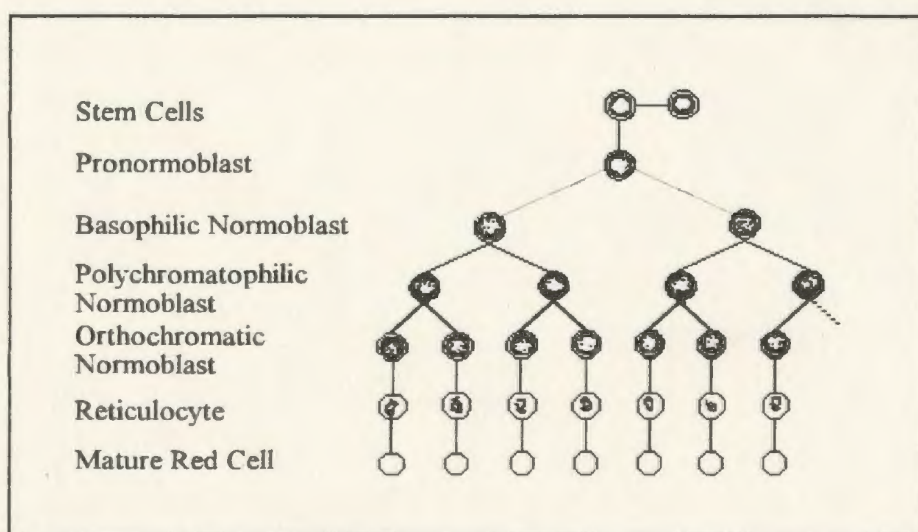
Figure 2.2: Maturation of Red Blood Cells
(**Note:** In disease, the number of divisions may be increased or decreased) (Beck 1973:15).

In effect, it is in these many stages where nutritional deficiencies can alter the quality and quantity of mature erythrocytes. For example, lack of iron will limit the synthesis of hemoglobin, which in effect is affected by the amount/presence of ascorbic acid available to the system, as this vitamin helps to convert iron in order to promote its absorption from the diet. With the limitation of hemoglobin synthesis, the production of abnormally small,

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or microcytic cells, characteristic of iron deficiency anaemia ensues. Also, with the lack of folic acid and vitamin B_{12}, DNA synthesis is altered, thus having an affect on cell division, growth and maturation of RBCs by consequently causing the production of megaloblasts. The presence of these nutrient deficiencies will decrease the average life span of 140 days of a RBC due to the production of defective erythrocytes. As a result, marrow hyperactivity is required in order to keep up with the demands of a sustained blood supply for the body.

Due to this complicated picture of erythropoiesis, other nutrients, besides iron, should not be excluded from the presentation, analysis and diagnosis of blood disorders causing anaemia.

CHAPTER 3: SKELETAL MANIFESTATIONS OF NUTRIENT DEFICIENCIES

The pathologic lesions ascribed to each of these nutritional anaemias effects each cohort of a population differently. Infants and children are more prone to deficiencies because of increased nutrient requirements during episodes of rapid growth. Infants and children are also more affected than adults because of the many growth, repair and maintenance processes that are deterred by the absence of these four nutrients. Fortunately, at the time of birth of a regular term infant, reserves of iron, vitamin C, folic acid, vitamin B_{12} and hemoglobin are usually substantial enough to prevent deficiencies for several months during inadequate diets. For example, according to Garrow and James (1993:592), "folate levels decline rapidly in neonates, reaching their nadir at 7-10 weeks postnatally in prematures and 11-12 weeks in full term babies".

Studies on contemporary populations have suggested that during the first few months after birth, infants divert the storage of iron to that of erythropoiesis (Wadsworth 1975:117) in order to compensate for increased growth rates. However, premature infants do not have these large stores at birth and consequently, are even more susceptible to developing anaemia. As a result, these individuals have smaller stores to call upon during the 'catch-up' period of growth and thus, the stores can be exhausted at a faster rate with low or deficient nutrient intakes. Also, when infants are part of a twin birth, the nutrients must be shared.

The process of weaning infants on to 'adult' foods can be nutritionally traumatic. Adult foods fail to provide infants with sufficient diets as most often the food presented to them is inadequate in nutrients and usually overcooked. Biologically, their bodies may not

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be prepared to digest foods properly at such young ages and also adult foods can expose infants to bacteria and parasites. With the introduction of pathogens, infants are more likely to suffer from 'weanling diarrhea' which can exacerbate the situation by increasing the amount of nutrients excreted and not made available to the body. Infestation of pathogens, worms, chronic infections and diarrheal diseases provide stress factors that when accompanied with an inadequate diet can substantially debilitate reserves of nutrients required for hemoglobin production.

Adolescents can also be prone to nutrient deficient states causing anaemia because, like infants and children, needs are elevated due to increased growth demands. Female adolescents also have to contend with menstrual blood losses and early pregnancy that can make nutrient levels critically low in the body with insufficient consumption.

Females, especially pregnant and lactating females, are also more likely to develop deficiencies because of their role in supplying sufficient nutrient stores for the growing fetus and nutrients via milk for breast-fed infants. According to Ensminger *et al.* (1983:81), during the course of the pregnancy, the blood volume increases by approximately 50% of that required during non-pregnant states. If a female is not consuming enough nutrients in her diet, then during states of pregnancy and lactation her stores will become exhausted resulting in nutritional deficiencies in not only herself, but possibly in the infant as well. Nutritional deficiencies are compounded by multiple gestation (multiparity), chronic illness and menstrual losses when poor diets are consumed.

Nutritional deficiencies are also more apt to occur in the older cohort, especially if

they are edentulous. Edentulous individuals usually have poor diets due to the limited resources available to them. Also, in order to make food more consumable, overcooking is usually required, resulting in substantial nutrient loss. Inadequate diets are most often consumed by this cohort because of decreased energy requirements. Also, as age increases some nutrient reserves decrease, which is not necessarily due to changes in metabolism, but as a result of differences in dietary habits between the young and the elderly (Basu and Schorah 1982:67). Not only is an inadequate diet attributed to low nutrient levels, but also to a decrease in absorption and poor utilization of many nutrients have also been cited as possible contributors (Jacob *et al.* 1988:1439). According to Ensminger *et al.* (1983:81), older persons have higher incidences of anaemia because of atrophy of gastric secretory cells resulting in deficiencies in hydrochloric acid and intrinsic factor, which aids in the absorption of iron and is required for vitamin B_{12} absorption, respectively. The presence of chronic diseases can also predispose the elderly as nutrients are required for immunocompetence.

As a result, all of these cohorts of a population require more iron, vitamin C, folic acid and vitamin B_{12} in their diets to meet elevated needs for the increased production of hemoglobin, myoglobin and enzymes that require these nutrients, normal growth and the maintenance of gestation and most important, to compensate for daily losses. If these needs are not met over a prolonged period of time, then anaemic states will ensue, possibly predisposing individuals to skeletal alterations.

In the anaemic state, either the quantity and/or the quality of the red blood cell

population are diminished. Apparently, the signs and symptoms of anaemia can be masked by the body by adaptations that allow for an increased oxygen-carrying capacity of a decreased amount of hemoglobin (Beck 1973:22). Therefore, by adapting to an anaemic state, the body can tolerate a 50% decrease in red cell mass without any symptoms. In order to compensate for abnormalities in the size, shape and overall amount of red blood cells (RBCs) in the body, overstimulation of RBC production occurs because of their shortened life span. As previously mentioned, RBC production originates in the red bone marrow. Replacement of red marrow by non-hematopoietic fatty yellow marrow occurs from about four years of age on (Stuart-Macadam 1985:393). This marrow involution starts in the extremities and then proceeds axially until only red marrow can be located in the vertebrae, sternum, ribs, clavicles, scapulae, bones of the skull and pelvis for those individuals over the age of 20 years (Stuart-Macadam 1985:393). Therefore, infants will hardly have any yellow marrow to draw on to convert to red marrow in an anaemic situation. As a result, a different distribution of anaemic bone changes will occur for infants and subadults in comparison to adult skeletons because expansion of the existing red marrow must be performed.

During periods of anaemia and high turnover rates of abnormal RBCs, hypertrophy of the red bone marrow ensues. This results in expansion of the marrow or diploë area, erosion and thinning of the outer table of bone and consequently, disclosure of the inner trabecular bone (Martin *et al.* 1985:266; Moseley 1965 and 1974; Stuart-Macadam 1985). This sequence of events manifests itself on the skeleton in the form of a sieve-like, pitting or coral bone presentation, known as porotic hyperostosis. This pitting of the compact bone

is also associated with an increase in the thickness of adjacent diploïc bone. Porotic hyperostosis is most prominent in the cranial vault, particularly frontal, parietal and occipital areas and is usually bilateral in presentation (Steinbock 1976:231,233; Stuart-Macadam 1989a:215).

This same process of diploïc expansion is also manifested bilaterally on the orbital portion of the frontal bone and is commonly referred to as cribra orbitalia. Apparently, a vascular net of blood vessels occupies the spaces in the spongy bone apertures connecting the veins of the diploë with those of the orbit (Steinbock 1976:239). In the presence of cribra orbitalia, an increased thickness in the orbital roof occurs (Stuart-Macadam 1987a and 1987b). Erosion in the orbital roof occurs more readily because of the presence of a thin outer table, in comparison to the skull vault, that can be acted upon by the processes of diploïc hyperplasia. It has been suggested that lesions in the orbits are the result of a mild or limited anaemic episode and those lesions found on the rest of the skull are the result of a more prolonged case of anaemia (Lallo *et al.* 1977:477; Stuart-Macadam 1989b:191). As such, it has been suggested that cribra orbitalia and porotic hyperostosis usually occur in unison with evidence showing that their paired incidence is high, but the discrete presence of only one of them at a time is also common (Stuart-Macadam 1989b). However, one must keep in mind that the degree of involvement of the skull is in no way related to the severity of the anaemia (Moseley 1974:169).

Given that deficiencies in iron, vitamin C, folic acid and vitamin B_{12} all produce some form of anaemia, they would all have the potential to share the commonality of

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presenting marrow hyperplasia and thus, porotic hyperostosis and cribra orbitalia in skeletal material. However, some of the etiologies may be different. When both porotic hyperostosis and cribra orbitalia are presented, the author has chosen to define this occurrence as cribra cranii for reasons of convenience. The discussion that follows only presents macroscopic evidence of skeletal alterations of each nutritional deficiency. It is realized that an abundance of pathological characteristics exists for each nutrient, such as histological and radiographic evidence, but due to the experimental design, they will not be discussed here.

3.1 IRON DEFICIENCY ANAEMIA

It is thought that children are more affected skeletally than adults because of the plasticity, susceptibility and size of their bones. This is believed to be related to the fact that the bones of children are less mineralized (Stuart-Macadam 1985:394). It has also been suggested that "marrow hyperplasia as a result of an exaggerated erythroid response invoked by the anaemia is more likely to cause expansion of bone and roentgenographic changes at an early age before the cortices are firm enough to resist expansion" (Lanzkowsky cited by Stuart-Macadam 1985:394). Infants and children also have smaller red marrow spaces than adults and as a result, the bone surrounding it will be altered more easily. As mentioned previously, infants and subadults also predominantly possess red marrow spaces instead of yellow fatty marrow. This translates into the utilization of all available red marrow spaces to produce red blood cells because little yellow marrow exists. Therefore, due to an increase

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in RBC production, the body of a child reacts by creating pressures which induce marrow expansion and destruction of compact bone.

Bony lesions are not as common in adults with iron deficiency anaemia because it is believed that an increase in RBC production can occur by increasing parenchymal cellularity (Stuart-Macadam 1985:394). This process will result in loss of fat in areas of normal hematopoiesis, expansion of hematopoietic marrow into previously inactive fatty yellow marrow cavities, shortening of cellular maturation time, and perhaps by shortening of generation time (Wintrobe cited by Stuart-Macadam 1985:394). This means that by avoiding the marrow space, skeletal alterations are no longer present in the adult. This exclusion of adults from the manifestation of skeletal lesions acquired from iron deficiency anaemia suggests that this form of anaemia is representative of a childhood condition (Stuart-Macadam 1985).

In iron deficiency anaemia, skeletal manifestations of this disease do not really involve the infracranial skeleton (Steinbock 1976:231). According to Maroteaux *et al.* (1979:295), in babies suffering from nutritional hypochromic iron deficiency anaemia, the face and long bones are never involved. However, according to Stuart-Macadam (1989a:215) and her analysis of known modern medical cases of iron deficiency anaemia, infracranial changes can occur (e.g. metacarpals and phalanges), but with less severity and in lower frequency than those of genetic anaemia. Along these same lines, Steinbock (1976:231) states the most common bone changes that can be affected in this region of the body pertain to marked osteoporosis and course trabecular striations in the distal end of the

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humerus and the proximal ends of the radius and ulna. Even though minimal signs of osteoporosis of the vertebral bodies can occur, there is always the complete absence of vertebral compression (Steinbock 1976:232).

Although Steinbock (1976) states that alteration of the sinuses is not involved in iron deficiency anaemia, Stuart-Macadam provides evidence that the paranasal sinuses can be affected. By utilizing a study performed by Reimann *et al.* in 1975 (cited by Stuart-Macadam 1989a:215), of the 88 patients, 38% showed deficiencies in form, size, or aeration of the frontal sinus, and 50% showed abnormalities of the maxillary sinuses. This contrast demonstrates that conflicting evidence and/or interpretation exists due to the difference in access of modern day medical cases. It also reveals that with more research on this deficiency, more insight and better information will be attained.

3.2 VITAMIN C DEFICIENCY: SCURVY

If vitamin C is lacking in the diet for prolonged periods of time, scurvy can result. The main symptoms of this vitamin deficiency are the presence of hemorrhages anywhere in the body; changes in structure of teeth and gums; changes, deformities and falling apart of bones due to the loss of supporting cartilage; enlargement of the heart and damage to heart muscles; degeneration of muscle fibers; anaemia due to destruction of blood-forming cells in bone marrow and loss of blood through hemorrhages; loss of calcium through degeneration of bone matrix; degeneration of sex organs; and an increase in metabolic rate or increased oxygen consumption (Sherman cited by Nobile and Woodhill 1981:68). As

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such, metabolism of the entire body is affected since vitamin C is present in tissues of high metabolic activity and, since the vitamin takes part in so many biochemical pathways, many essential processes will be seriously affected.

3.2.1 Infants and Children:

Infantile scurvy commonly occurs in infants between the ages of five and 24 months (Ortner and Putschar 1997:270; Stuart-Macadam 1989a:202). However, scurvy can become clinically apparent as early as four months, with manifestations peaking at approximately eight months (Resnick 1995:3346; Stuart-Macadam 1989a:202).

One of the most characteristic signs of infantile scurvy is the presence of hemorrhages anywhere in the body, especially in subcutaneous tissues, in the intestinal tract, in the gums (if the teeth have erupted), and the presence of subperiosteal hemorrhages that are usually found at the ends of the rapidly growing long bones of the lower limbs, the ribs and occasionally the skull vault and orbits (Stuart-Macadam 1989a:202). These hemorrhages and bone changes are also most often observed in a symmetrical pattern. Infantile scurvy can also include disruption of the dentition, inactivity, prominence of ribs at the costochondral junction, also known as 'beading' or 'rosary', particularly in the fifth, sixth, and seventh ribs and enlargement of joints, especially at the wrists and ankles (Maroteaux *et al.* 1979:202-203; Ortner and Putschar 1997:270-272; Resnick 1995:3346; Stuart-Macadam 1989a:202,204). Beading, or rosary, appearance on the ribs of scorbutic infants is quite similar to rickets, but is usually more angular and less knobby, with a

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concomitant presence of subperiosteal hemorrhages along the shafts of the ribs (Steinbock 1976:256).

The skeletal changes manifested in this disease all result from the depression of normal cellular activity with the continued resorption of the bone matrix and the presence of hemorrhages in the bones (Resnick 1995:3346; Stuart-Macadam 1989a:203). The reduction of osteoblastic activity then leads to a decrease in the formation of bony matrix. Both of these processes are most pronounced in areas of active endochondral bone growth such as, the ends of tubular bones and costochondral junctions. The metaphyseal region of bones experience extensive resorption of cortical and spongy bone augmenting the tendency to fracture through this zone which could lead to the evidence of fresh and remote hemorrhage (Resnick 1995:3346). As such, the increased brittleness of the metaphyseal region may result in infraction (incomplete fracture) (Maat 1982) and the presence of metaphyseal cupping (Stuart-Macadam 1989a:204). Small spicules or 'excrescences' of the metaphysis are produced by the combination of the lateral extension of the heavy provisional zones of calcification with elevation and the stimulation of the adjacent periosteal membrane (Resnick 1995:3346). In advanced cases, infraction and fractures at the metaphyses of long bones can occur (Stuart-Macadam 1989a:202).

Diaphyseal changes of infantile scurvy are manifested in the form of atrophy of the spongiosa in the shafts of the tubular bones. Therefore, the cortices become very thin with the presence of sparse trabeculae, which is accounted for by bone resorption and deficient periosteal new bone formation (Steinbock 1976:256). Cortical reduction is common and

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most times severe, even though fracture of the shafts is notably unusual (Resnick 1995:3348). According to Resnick (1995:3348; Steinbock 1976:254), subperiosteal hemorrhage is most frequent in the larger tubular bones, such as the femur, the tibia and the humerus. As a result, the presence of small or large shells of ossified hemorrhages can be found in the periosteal region of these bones.

Infantile scurvy can also affect the dentition of humans via cyst formation and hemorrhage in the enamel (Jaffe 1972:457) and interruptions of the lamina dura (Resnick 1995:3348). Gingival and subperiosteal hemorrhages causing spongy changes in the gums will frequently occur when teeth have erupted in infants (Maroteaux *et al.* 1979:202; Shils *et al.* 1994:915). Studies have shown that vitamin C deficiency can affect both tooth development and eruption (Shils *et al.* 1994:1011). This can entail minute pulpal hemorrhages in both deciduous and permanent teeth of scorbutic infants. Odontoblastic degeneration and aberrant dentin can also be observed in scorbutic infants (Shils *et al.* 1994:1010). In older children, the dental pulp undergoes hyperemia, edema, necrosis and aberrant degeneration, and also irregular formation of teeth can occur (Shils *et al.* 1994:1011). According to Shils *et al.* (1994:1011), "although it is likely that the primary mechanism of vitamin C deficiency-induced tooth, gingival, and bone disease is mediated through the disruption of collagen biosynthesis, no study has clearly demonstrated the relationship between scurvy and dental caries".

It has been suggested that in its most severe form, ocular lesions can be found in the skull (Nobile and Woodhill 1981:68). These lesions are the result of bleeding into the orbit

from hemorrhages and can cause exophthalmos in living scorbutic infants (Silverman 1985:74). Other lesions of the skull caused by the presence of a scorbutic environment have been described by Ortner and Ericksen (1997) and Ortner *et al.* (1999). These include bilateral presentation of porous and hypertrophic lesions on the greater wing of the sphenoid, the zygomatic, the palantine process and bone and the posterior, or infratemporal, surface of the maxilla. These authors suggest that these lesions are caused by the direct association of chronic hemorrhaging of the temporalis muscle and inflammation of the underlying periosteum of these two anatomical locations in relation to chewing. Hemorrhaging from the palatine branches of the third part of the maxillary artery would also contribute to lesions found on the alveolus and palatine. In another publication, Ortner (1984) describes other lesions of the skull in a Metlatavik child. He states that porous, but non-expansive lesions, occur on the anterior maxilla, the bone surrounding the inferior portion of the nasal aperture and the alveolar bone of the anterior portion of the mandible all the way up to the first deciduous molars. Loss of teeth, especially of single roots, has also been noted in scurvy.

When infantile scurvy is treated with megadoses of vitamin C, healing of bony alterations occurs. An increased thickening of the cortex, an increased definition of trabeculae of the metaphysis, the presence of massive subperiosteal bone formation, which later merges with the underlying cortex and spontaneous shifting of the diaphysis to realign with the displaced epiphysis occurs with healing (Resnick 1995:3348; Stuart-Macadam 1989a:204; Silverman 1985:676). According to Jaffe (1972:466), "even in very severe cases of infantile scurvy, it can confidently be expected that the skeletal changes will completely

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disappear", but that the "time required for restoration of the skeleton to a normal status varies and could conceivably be as long as several years". Therefore, bouts of infantile scurvy may not necessarily be recognized by paleopathologists in the archaeological record, if complete recovery has occurred.

3.2.2 Adults:

Skeletal changes in adult scurvy are of a minor nature in comparison to infants and children because of slower rates of growth and/or bone remodeling. According to Steinbock (1976:256), adult scurvy is similar in appearance to infantile scurvy except with the absence of the involvement of the epiphyseal ossification centers because they have closed. Pathological fractures are more common in the diaphyses rather than the metaphyses because of generalized deossification without the production of new bone (Steinbock 1976:256). A general osteoporotic appearance is also indicative of adult scurvy (Maroteaux et al. 1979:202).

Classic gum changes observed in scurvy are only associated with permanent teeth or buried roots, and can be enhanced by poor dental hygiene and advanced caries (Shils et al. 1994:915). However, it has been suggested by Jaffe (1972:452) that gingivitis and bleeding of gums can fail to develop if careful oral hygiene is practiced. Scurvy can cause interdental papillae to swell, become purple and bleed with trauma (Shils et al. 1994:915). Interruptions in the lamina dura around teeth have also been observed in scorbutic adults (Jaffe 1972:458). In advanced cases, the gums become spongy and friable, bleed freely, and

with the presence of secondary infections, loosening of the teeth to gangrene can occur (Shils *et al.* 1994:915-916). The presence of fistulae in the alveolar regions of both the maxilla and mandible have been diagnosed in scorbutic individuals. Also, jaws in advanced states of scurvy may show traces of osteitis (Živanović 1982:119).

Hemarthrosis, which is associated with the collapse of subchondral bone, especially in the femoral head, and synchondroses are also observed in adult scurvy. According to Resnick (1995:3349), this collapse of the subchondral bone may be related to ischemic necrosis of the bone which is produced by a vascular compromise effected by the hemarthrosis, or to osteopenia and subsequent fractures of subarticular bone. Hemarthrosis of the knee can be the result of interstitial hemorrhage and little collagen in this area (Bevelaqua *et al.* 1976:1874). Femoral head destruction can also occur as a rare complication of scurvy as a result of the loss of articular cortex, dissolution (osteolysis) and an effusion of the femoral head and acetabulum (Shettey *et al.* 1988:1878; Sudbury and Ford 1990:1109).

Both the axial skeleton and the long bones of the lower limb are affected by osteoporosis (Resnick 1995:3349; Stuart-Macadam 1989a:204). Osteoporotic skeletal changes in the vertebral column are manifested in biconcave deformities of the vertebral bodies, condensation of the bone at the superior and inferior margins and central rarefaction (Resnick 1995:3349). In the appendicular skeleton, cortical thinning can be associated with slight periosteal proliferation. According to Resnick (1995:3349), the cranium of adults suffering from scurvy may also be osteoporotic.

3.3 FOLIC ACID & VITAMIN B$_{12}$ DEFICIENCIES: MEGALOBLASTIC ANAEMIA

Research into the skeletal manifestations of these two nutrients has been limited. Archaeological evidence of megaloblastic anaemia is nonexistent because to this date, no skeletal alterations have been discovered and recorded. This could be due to both the lack of knowledge of how this nutritional deficiency affects the bone and to the current focus on and understanding of neural tube defects (NTDs) in association with maternal folic acid deficiency which have predominated in the literature. NTDs result from the failure of the neural tube to close during early embryogenesis. The most common types of NTDs are open lesions of the spinal column, known as spina bifida cystica and anencephaly. They are caused by environmental influences on the mother, her nutritional status with regard to folic acid, and partly by the hereditary constitution of the fetus (Garrow and James 1993:653; Post 1966:341; Roberts and Manchester 1995:32). When folic acid is deficient in the diet prior to conception and within the first few weeks following conception, NTDs are more apt to form in the fetus. This focus on NTDs has led to personal hypotheses as to other ways in which folic acid and vitamin B$_{12}$ deficiencies, often associated with megaloblastic anaemia, may affect the skeleton. Due to vitamin B$_{12}$'s close association with folic acid (as previously discussed in Chapter 2), it will be understood by the reader during this discussion that any references of folic acid deficiency and megaloblastic anaemia will also concern vitamin B$_{12}$ deficiency, even if it is not stated. As such, hypotheses and corollaries will be made where these two vitamins are concerned.

When late stage folate and/or cobalamin deficiencies are experienced by individuals, several biological occurrences can be manifested. People will become generally anaemic and have abnormally large red blood cells, hence the name, megaloblastic anaemia. In very late stages of the deficiencies, the total white cell and platelet counts are both low, therefore affecting the immune response. Marrows of these individuals become megaloblastic with serum and red cell folate levels being low. Deficiency in folate can also cause marked growth retardation and delay the onset of puberty (Garrow and James 1993:591).

When folate and cobalamin are deficient from the body for an extended period of time, megaloblastic changes to cells, especially RBCs, occur. Megaloblastic cells are essentially 'giant' cells in the marrow that have sufficient cytoplasm, but insufficient chromatin to divide. Those tissues, like the hematopoietic marrow, that are most dependent on cell proliferation will be the first affected by a deficiency in tetrahydrofolic acid and vitamin B_{12} (Linder 1991:142-143).

The morphologic changes that are most striking in bone marrow cells, with the "ineffective hematopoiesis", results in peripheral blood pancytopenia: an abnormal reduction in the number of RBCs, or anaemia, in white blood cells, or leukopenia, and an abnormal reduction in blood platelets, or thrombopenia (Krieger 1982:229; Shils et al. 1994:416). In megaloblastic anaemia, the red cell increases in size which entails an increase in the hemoglobin content, but the number of red cells decreases in the blood (Chanarin 1969:340).

According to Chanarin (1969:350), changes in the morphology of red cells are

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present at all stages in development, from the most primitive cell to the late pyknotic [('pyknosis' = a degenerative condition of a cell nucleus marked by clumping of the chromosomes, hyperchromatism, and shrinking of the nucleus)] erythroblasts. However, according to Shils *et al.* (1994:416), megaloblastosis is also present in all other duplicating cells of the body and may be most strikingly noted in the epithelial cells of the entire alimentary tract, producing glossitis [((inflammation of the tongue)] and variable degrees of megaloblastosis along the entire alimentary tract epithelium.

As a result, megaloblastic anaemia affects all cells within the body, but those cells most closely associated with the marrow spaces should be more predominantly influenced. Due to the fact that trabecular bone is associated with the marrow and cancellous spaces of bone, any changes manifested in the marrow might be seen in the closely associated trabecular bone. This may be exhibited by the presence of hyperplastic marrow and the expansion of hematopoietic areas, such as cribra cranii. With trabecular bone being most concentrated at the ends of long bones in adults, this is where possible megaloblastic anaemic changes of the marrow might affect trabecular bone the most. However, it is unknown as to *what* these alterations may consist of.

It has been observed in patients with severe metabolic defects of folate metabolism that homocysteinemia and homocysteinuria are frequent (Cooper 1995:89; Swain and St. Clair 1997:142). According to Cooper (1995:89), homocysteine infusions can induce arterial thrombosis in baboons and that homocysteinemia is statistically associated with deep arterial thrombosis in humans (also cited in Swain and St. Clair 1997). Swain and St. Clair

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(1997:142) also state that high homocysteine levels have been known to cause premature atherosclerosis and with corresponding low levels of folic acid in the serum, myocardial infarctions are also common. Due to the fact that thrombosis can occur in folate deficient patients, possible bone effects from this condition may be recognizable in archaeological populations, such as infarctions or areas of necrosis. In the presence of blood clotting and the persistent decrease in the number of blood platelets that is often associated with hemorrhagic conditions (thrombopenia), certain bony alterations might be noticeable. These bony alterations are not discussed in any of the literature thus far, but hemorrhagic conditions similar to scurvy, such as the subperiosteal hemorrhaging of long bones and the cranium causing subperiosteal bone formation upon healing, might be seen in folate deficiency (von Hunnius 1997a). Thrombosis can prevent nutrients from reaching areas of the bone, and in the condition of sickle cell anaemia this can cause lesions in the skull to resemble 'punched out' lesions known to occur in multiple myelomas (von Hunnius 1997b:15). It is quite possible that these skeletal alterations could be seen in folate deficient individuals, thus providing physical evidence on bones that could be used for diagnostic purposes in archaeological human remains.

Both folate and vitamin B_{12} are involved in the synthesis of DNA and cell proliferation. When these nutrients are deficient in the diet, these processes are altered. This would be most apparent in growing bones of infants, children and adolescents. As such, marked growth retardation should be observed in those bones of individuals suffering from megaloblastic anaemia. However, comparisons of growth were not performed in this

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study as that would have involved another thesis.

The skeletal manifestations afforded by deficiencies in each of these four nutrients have been summarized in Figure 3.1. This figure illustrates that each nutritional deficiency presents distinct skeletal changes. However, presentation of anaemia via cribra orbitalia and/or porotic hyperostosis also provides evidence of their similarities. By taking this information into consideration, identification of multiple nutrient deficiencies causing anaemia is plausible.

Fractures
Fistulae in gums
Loss of teeth
Osteitis along alveolar ridge
cupping & flaring of costochondral rib segments (infants)
Vitamin C Deficiency

▲

Osteoporosis

**POROTIC
HYPEROSTOSIS
&
CRIBRA ORBITALIA**

Hemorrhaging &
Subperiosteal New
Bone Formation

▶

**Iron Deficiency
Anaemia**

Retardation or pneumatization
of maxillary sinuses

◀

**Megaloblastic Anaemia: Folic Acid
& Vitamin B$_{12}$ Deficiencies**

Atherosclerosis: Thrombosis,
Necrosis, & Calcification

Figure 3.1: The Interrelationship Between Iron, Vitamin C, Folic Acid and Vitamin B$_{12}$
Deficiencies.

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CHAPTER 4: RESEARCH SAMPLE: DICKSON MOUNDS

In discussing the archaeology of the Dickson Mounds collection, emphasis is given to the biogeography of the surrounding area, especially pertaining to the resources that were available to the aboriginals during the Mississippian Period. The environment and the natural resources it provided are fundamental to this investigation, as the effect of nutrition on these individuals is the prime focus of the analysis. By realizing the potential of the resources available to the Dickson Mounds inhabitants from their surrounding environment, a better understanding about the possibilities of suffering from multiple deficiencies can be accomplished.

The Dickson Mounds collection comes from the Dickson Site #11F10 located in Fulton County, Illinois (Map 1). This site is located approximately one mile from the confluence of the Spoon and Illinois Rivers, three miles southeast of Lewistown. Illinois in the Central Illinois Valley. Fulton County is well known for having the greatest number of prehistoric archaeological sites within its boundaries of any county in the State of Illinois (Harn 1980:1). In fact, every phase of prehistoric cultural occupation known to the State of Illinois, can be found within the confines of this county and it becomes understandable why massive amounts of time, money and effort have gone into the archaeology of this area.

Historical accounts suggest that the Mounds on the Dickson property were known by the local residents to contain burials and the site was also an identified place for collectors of both skeletons. pottery and other mortuary items. Therefore, not only was the skeletal population decreased because of this. but also the specimens of pottery and other

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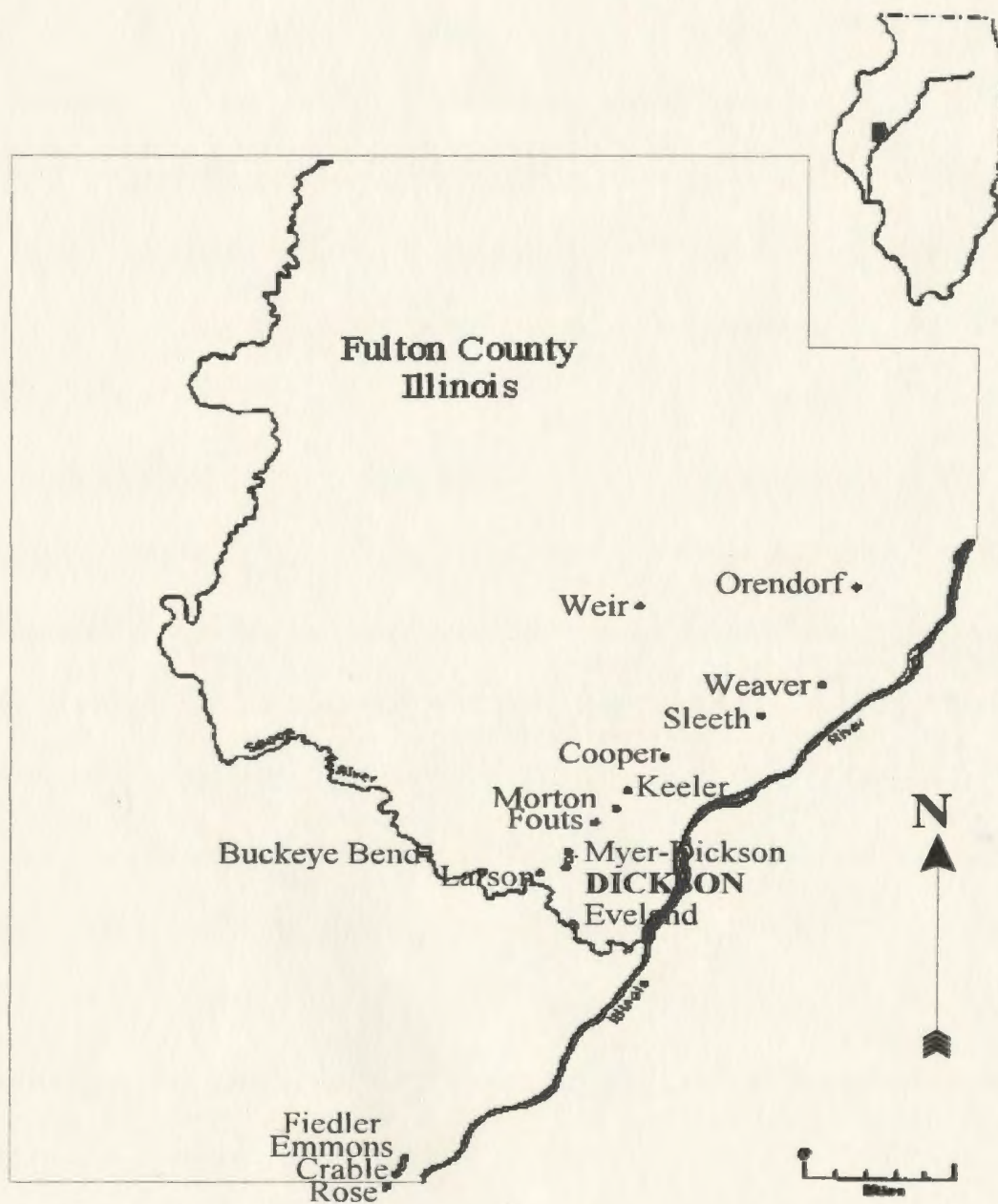
Map 1: The location of Dickson Mounds in Fulton County, Illinois (Adapted from Harn 1980).

personal grave items. Interest in the archaeology of Dickson Mounds was not truly expressed until 1927. An amateur archaeologist, Don F. Dickson, whose father happened to own the land, was the first known excavator of the Mound burials. Those burials unearthed by the Dickson family and others were kept *in situ* as an exhibit for the public to see and for the rare opportunity it presented to researchers, with the chance of observing approximately 200 individuals in their original positions and layout (Dickson cited by Morse 1978:83; Harn 1980).

The original partial excavation of a Middle Mississippian burial mound on family land began as an amateur's personal interest and hobby, but it quickly progressed into further research by professional archaeologists in the immediate vicinity. In the 1930's, a team of archaeologists from the University of Chicago unearthed approximately 200 burials (i.e., those left *in situ*) from one of the Mounds and also identified a number of settlement sites in a 100-square-mile radius (Buikstra and Milner 1989; Goodman and Armelagos 1985). However, it was not until 1966-1968 that massive excavations of other regions of Dickson Mounds were directed by Alan Harn, an archaeologist for the state of Illinois. During these excavations more than 800 burials were discovered, along with several local habitation sites (Goodman and Armelagos 1985:12; Harn 1980:2; Lallo and Rose 1979). The specimens from these burials were then loaned to the University of Massachusetts and under the supervision of Dr. George Armelagos, a substantial amount of biocultural and biological information was generated by both undergraduate and graduate research (e.g., Gilbert 1975; Lallo 1973) (Buikstra and Milner 1988:5; Goodman and Armelagos 1985).

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So far, excavations in this particular area have led to the unearthing of twelve burial mounds (Map 2), lending to a multitude of research and inevitably, an incredible wealth of knowledge about those individuals who inhabited this environment for several centuries. This wealth of knowledge is still being increased today with new forms of technological advances and new insights put on previously published ideas, as in this particular thesis, that have allowed researchers to keep going back to the same skeletal collection.
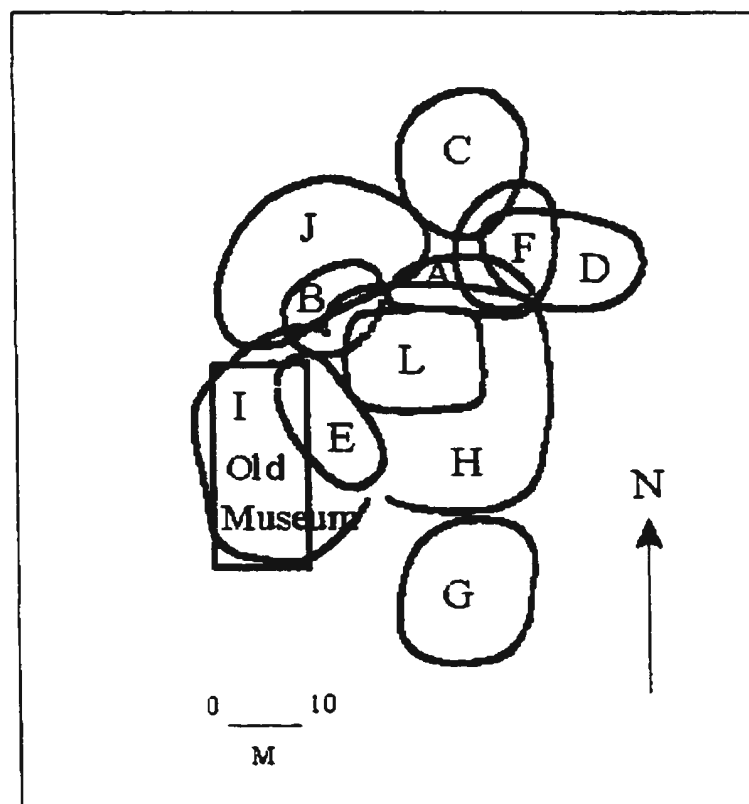
Map 2: Layout of Burial Mounds at Dickson Mounds (Adapted from Buikstra and Milner 1989).

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The twelve mounds form a complex crescent-shaped position on top of the Illinois River Bluff. Not all of the Mounds are burial mounds, but also included are two other mortuary features: a ceremonial platform mound and a premound cemetery (Buikstra and Milner 1989:2). As can be seen by Map 2, the mounds are not distinct and several of them are superimposed upon one another, which has led to the intrusion of burial units into several different mounds. Also, the loess soil has induced erosion and leveling off of the mounds, thereby adding to their amorphous structure. In accordance with the alphabetical labeling of the mounds, the earlier labels represent burials from an earlier time period. As such, Late Woodland individuals form a component of Mounds A-E and Mississippian burials are more a part of Mounds F-K. However, Eveland phase Mississippian Indians (ca. A.D. 1000-1150) did use the same bluff as the Late Woodland populations and thus, are also a part of several burial units found in Mounds A-E. Therefore, in order to classify each burial unit into its respective cultural horizon, characteristic burial furniture and skeletal positions were relied upon (Buikstra and Milner 1989; Harn 1980).

Analysis of the provenience of the one hundred ten individuals utilized in this thesis is presented below (Graph 1). Following what was just stated about the temporal use of the cemetery and the alphabetical categorization, those mounds most often used for burials in this population are in the Mississippian time line. This graph reveals that a large percentage of the burials were found in Mound J (37.50%), Mound F (20.54%) and 12.50% had no mound association. Therefore, use of these mounds coincides with the Mississippian time period and they are also relatively close in geographic position (Map 2).

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Graph 1: Provenience of Burials From Dickson Mounds
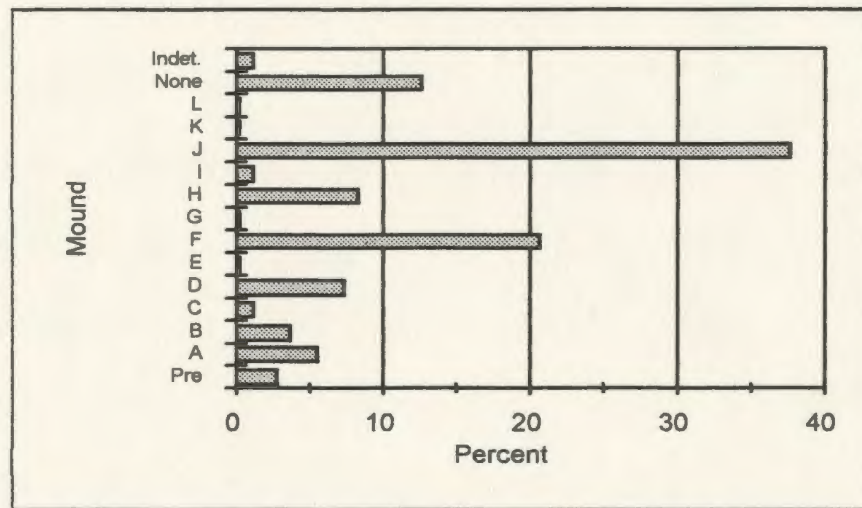
4.1 THE MISSISSIPPIAN PERIOD

Dickson Mounds is a multicomponent habitation-burial complex that spans three cultural horizons: Late Woodland, Mississippian Acculturated Late Woodland and Middle Mississippian (Goodman *et al.* 1984:272) with an occupation of approximately four centuries (A.D. 1000-1350). Even though several cultural horizons exist at this site, only the Missippian Period is concentrated on due to important characteristics that are more applicable to this thesis.

The Mississippian Tradition was incorporated into the indigenous Late Woodland populations of the American Bottoms area around 800 A.D. (Fowler 1978). A predominantly hunter-gatherer subsistence base with small populations (75-125) and seasonal occupations of camp sites, characterizes the Late Woodland occupation. This evolved into, or was completely absorbed by, a more horticultural-based subsistence (maize-based) with larger, sedentary populations, with its concomitant transformation in other

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cultural attributes. which came to be known as the emergence of the Mississippian Tradition (A.D. 900-1500). Prehistoric Mississippian sites can be found from the southern end of the Mississippi Valley and surrounding southeastern states north to Wisconsin, east to Tennessee. Georgia and the lower Ohio River Valley, and west to Oklahoma (Brown 1984). By A.D. 1050 the Mississippian Tradition was fully immersed into the lower and central regions of the Illinois valleys (Ham 1978).

The Mississippian culture can be characterized as having diverse forms of technology, social symbols/status markers and settlement patterns (Goldstein 1980:13). Their subsistence patterns were based mainly on maize horticulture with some exploitation of local flora and fauna. They belonged to stratified societies and had large population centers with pyramidal mound complexes usually located in riverine environments (Goldstein 1980; Ham 1980; Jennings 1989; Peregrine 1996:xx). The large earthen platforms served as substructures for temples, residences and council buildings (Thomas 1998:377). The dependence on maize horticulture during this period allowed for populations to substantially increase. Even though maize horticulture had existed in the southwest United States about 2700 B.P.. intense cultivation was not evident until early Mississippian cultures of approximately 900-1000 A.D. of the eastern United States (Jennings 1989:258-259). By relying on a single crop, seasonal deficiencies could have been encountered. However, by harvesting maize and utilizing other natural resources from their environment, dense Mississippian populations could be supported by different storage techniques. As such, the vessels produced were characteristic to the purposes they served

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and to the stylistic changes that evolved during this era.

Even though Mississippian pottery is characterized by its varied and elaborate technology and styles, a predominance of shell-tempered ceramics exists. This latter quality is a hallmark of this time period (Goldstein 1980: Jennings 1989). Two types of vessels were common during this era: vessels that were primarily of utilitarian use (e.g., smooth, plain and undecorated, incised or cordmarked) and those vessels that may have served a ceremonial function (e.g., more elaborate and decorative) (Goldstein 1980:15).

Many of these elaborately decorated forms of pottery were found associated with burials. The type of burial furniture often associated with skeletons and the placement of individuals in specific burial mounds were characteristic of the hierarchical mortuary practices of the Mississippian Period. Generally, these differential qualities would become more elaborate and more stratified with larger and more complex sites. Positions of burials would be either extended primary interments, semi-flexed, flexed, bundle or other forms that may be interred singly or in groups (Goldstein 1980:15). It is believed that status was ascribed by virtue of birth as many infants have been discovered with an elaborate burial ceremony and grave goods found in specific mounds. However, according to Jennings (1989:268) many burials with rich grave goods have been found in or near towns in large cemeteries which are supposed to be for lower status individuals.

Even settlements were organized in a hierarchical manner in which large urban regions would be marked by several temple mounds arranged around a plaza and house structures. In the surrounding area of this urban center there would be several smaller towns

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each equipped with at least one temple mound and a plaza. On the next level, surrounding these towns would be villages, hamlets and farmsteads that had the lowest denominator of status, but most likely served the larger establishments (Goldstein 1980). Of course, each level of society was some distance from each other, therefore creating a series of clusters along the landscape.

In the Central Illinois Valley, the Spoon River area settlements are the most well known for their affiliation with the Mississippian settlement and cultural traditions. This occupation apparently began in the eleventh century A.D., according to dates produced from the Eveland Site (A.D. 1050 and A.D. 1085) (Map 1) (Harn 1978). It is believed that like other northern Mississippian Tradition sites, diffusion and migration of some of the ideas from the southwestern locale of the Mississippi River Valley occurred. For example, the ceramic complex at Dickson Mounds includes approximately 72% of the shell-tempered plain type of vessel which is indicative of Mississippian traditions of the Cahokia (Harn 1980:19). However, the Central Illinois Valley Mississippians also developed other lithic and ceramic traditions that were independent from those of the American Bottom. For example, the Dickson and Orendorf ceramic series were produced in the Central Illinois Valley, but were not present in the southern Mississippian locales (Conrad 1991; Harn 1991).

The Myer-Dickson Site (Map 1) is one of seven hamlets, because of size and inferred function, that is considered Mississippian and closest to the Dickson Mounds site (Harn 1978). Houses found at this site are arranged in rows with an open plaza that covers

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approximately 4-6 acres and may have been in use for at least fifty years. Also associated with this site are thirty-one surrounding support camps, extractive sites, and work stations connected to a local ceremonial center 11km to the southwest, called the Larson site. This site included extensive habitation deposits, a pyramidal mound, a plaza and a stockade that were enclosed by a palisade. As such, all sites connected with this larger ceremonial complex are collectively known as the Larson community.

During the habitation of these sites associated with the Larson community, trade networks extended as far south as the Gulf of Mexico and Florida (Goodman and Armelagos 1985). Trading such great distances allowed for the incorporation of beautiful marine shell necklaces into the Spoon River area. It is also suggested that the Spoon River inhabitants traded their animal hides and/or meat for such ceremonial items, thereby decreasing their consumption of protein and other important nutrients. The Larson community Mississippian Aboriginals also exploited the Illinois River bluff as a burial ground, especially Mounds F, G, and H, and Mounds I and J by the late thirteenth century (Buikstra and Milner 1988:6). As a result, a hierarchy exists among the burials. Apparently, the use of Dickson Mounds as a cemetery terminated by ca. A.D. 1300.

4.2 THE ENVIRONMENT: PHYSICAL GEOGRAPHY AND BIOGEOGRAPHY

The site is located on a section of the Illinois River bluff that is approximately 27 m (90 feet) above the valley floor (Harn 1980:4). The bluff is made up of a thick, 10m (30 feet) layer of yellow to yellow-brown loess with an underlying 'tacky, glacial drift of the

Buffalo Hart moraine' (Harn 1980:4). The Illinois River Valley itself, is a remnant of the old Mississippi River. Geologically, the Valley affords sandstone, which is particularly useful as smoothing and abrading stones, hard stones such as granite, diorite and basalt, used for manufacturing tools and other domestic items, and low-grade chert, with its possible use in smaller tool production. Different forms of clays with montmorillonite, illite and kaolinite, most suitable for pottery fabrication, were also available in this area (Harn 1980:4).

The position of Dickson Mounds and its associated sites in the Spoon River area are at a prime location in which abundant and diverse flora and fauna can be found, thus providing an ideal environment. The many different forms of fauna available in the surrounding area and their nutritional composition pertaining to iron, vitamin C and B_{12} and folic acid are presented in Table 1.

The flora consists of a variety of nuts, fruits and berries, seeds, and roots and tubers that can be found in three distinct vegetation zones. The first zone consists of both highland prairie and forested areas normally characterized as the Upland Zone. The second area is denoted as the Transition Zone, which includes hillside talus-slope forest and transitional forest. The final area consists of floodplain prairie, forest and lakes and sloughs denoted as the Floodplain Zone (Harn 1980:6). Each zone affords characteristic flora which were important sources of food that are summarized along with their respective nutritional composition in Table 2. Those spaces that are empty in both of these tables mean that no nutritional compositions are available in the literature.

Table 1: Nutrient Composition of Fauna Available in Prehistoric Illinois

Mammals:

Type	Scientific Name	Wt. (g)	Vitamin C (mg)	Vitamin B_{12} (μg)	Folic Acid (μg)	Iron (mg)
deer, white-tailed (roasted)	Odocoileus virginianus	100	0			4.47
raccoon (roasted)	Procyon lotor	100	0	8.3	11	7.1
opossum (roasted)	Didelphis marsupialis	100	0	8.3	10	4.64
woodchuck	Marmota monax					
porcupine	Erethizon dorsatum					
squirrel (roasted)	Sciurus spp.	100	0	6.51	9	6.81
gopher	Geomys spp.					
rabbit (roasted)	Sylvilagus spp.	100	0	8.3	11	2.27
rabbit (stewed)		100	0	6.51	9	2.37
wild rabbit (stewed)		100	0	6.51	8	4.85
elk (roasted)	Cervus canadensis	100	0			3.63
muskrat (roasted)	Ondatra zibethica	100	7	8.3	11	7.1
beaver (roasted)	Castor canadensis	100	3	8.3	11	10
variety of rodents	Rodentia					

(Continued)

Waterfowl:

Type	Scientific Name	Wt. (g)	Vitamin C (mg)	Vitamin B$_{12}$ (μg)	Folic Acid (μg)	Iron (mg)
turkey (pre-basted, roasted)	Meleagris gallopavo	100	0	0.32		0.66
chicken (light & dark w/ skin)	Tympanuchus spp					
roasted		100	0	0.3	5	1.26
stewed		100	0	0.2	5	1.16
pigeon (passenger) (raw) w/o skin	Ectopistes migratorius	100	7	0.47	7	4.51
bobwhite	Colinus virginianus					
duck w/skin (roasted)	Anatidae	100	0	0.3	6	2.7
goose w/ skin (roasted)	Anatidae	100	0	0.41	2	2.83
swan	Cygnus					

Extraneous:

Type	Scientific Name	Wt. (g)	Vitamin C (mg)	Vitamin B$_{12}$ (μg)	Folic Acid (μg)	Iron (mg)
mollusk	Mollusca					
fish	Pisces					
e.g., northern pike* (raw)	Esox lucius	85	3			0.47
northern pike* (dried)		85	3			0.6
e.g., white sucker* (raw)	Catostomus commersonii	85				1.11
e.g., white sucker*(dried)		85				1.42
e.g., channel catfish* (raw)	Ictalurus punctatus	85				0.83
turtle, green (raw)	Chelonia	100				
turtle, green (canned)		100				
frog legs (raw)	Anura	100				1.5

w^1 -- with   *faunal remains taken from Lower Illinois Valley (Styles 1981), the rest are from Harn (1980).

wo^2 = without   All nutrient composition values taken from Pennington 1994 and 1998.

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Recommended Daily Allowances:

Vitamin C = 30-90 mg/day
Folic Acid = 25-400 μg/day

Vitamin B_{12} = 0.3-2.6 μg/day
Iron = 6-30 mg/day

Table 2: Nutrient Composition of Flora Available in Prehistoric Illinois

Fruit and Vegetables:

Type	Scientific Name	Wt. (g)	Vitamin C (mg)	Vitamin B_{12} (μg)	Folic Acid (μg)	Iron (mg)
persimmon (raw)	Diospyros virginiana	25	17			0.63
persimmon, Jap. (dried)		34				0.25
crabapple (raw)	Pyrus fusca	110	9			0.39
plum (raw)	Prunus americana	66	6		1	0.07
cherries, sweet (raw)	Prunus	68	5		3	0.26
strawberries (raw)	Fragaria americana	149	85		26	0.57
raspberries (raw)	Rubus (idaeus)	123	31			0.7
grape, American, slip skin (raw)	Vitis spp.	92	4		4	0.27
gooseberries (raw)	Ribes spp.	150	42			0.47
blackberries (raw)	Rubus pennsylvaticus	72	15			0.41
elderberries (raw)	Sambucus canadensis	145	52			2.32
mayapple	Podophyllum peltatum					
black raspberry (Blackberries, raw)	Rubus occidentalis	72	15	0.00	24	0.41
maize-yellow corn (boiled)	Zea mays	82	5		38	0.5
wild potato-vine						

(Continued)

Grass & Weed Seeds:

Type	Scientific Name	Wt. (g)	Vitamin C (mg)	Vitamin B$_{12}$ (μg)	Folic Acid (μg)	Iron (mg)
ragweed	Ambrosia spp.					
reed canary-grass	Phalaris arundinacea					
sunflower seeds (dried)	Helianthus spp.	28	0	0	43	1.40

Trees:

Type	Scientific Name	Wt. (g)	Vitamin C (mg)	Vitamin B$_{12}$ (μg)	Folic Acid (μg)	Iron (mg)
black haw	Viburnum prunifolium					
Kentucky coffee tree	Gymnocladus dioica					
mulberry berries (raw)	Morus rubra	140	51	0	8	2.59

Roots & Tubers:

Type	Scientific Name	Wt. (g)	Vitamin C (mg)	Vitamin B$_{12}$ (μg)	Folic Acid (μg)	Iron (mg)
hog-peanut	Amphicarpa comosa					
wild onion	Allium tricoccum					
onion (raw)		80	5	0	15	0.18
onion (boiled)		105	5	0	16	0.25
arrowhead (boiled, 1 med. corm, 1" dia.)	Sagittaria latifolia	12	0	0	1	0.15
bulrush	Scirpus (lacustris)					
Jerusalem artichoke (raw)	Helianthus tuberous	75	3	0	10	2.55
wild ginger (fresh)	Asarum reflexum	11	1			0.05
ginger (ground)		2				0.21

Several types of herbs: spikenard (*Arabia racemosa*), pokeweed (*Phytolacca americana*), common milkweed (*Asclepias*) and butterfly weed (*Asclepias tuberosa*), as well as a vine called bittersweet (*Celastrus scandens*) were also used by these people, but considering their limited nutritional value they were not listed in the chart above. Also available to the people of Dickson Mounds were several types of miscellaneous vegetation used in manufacturing:

Indian hemp (*Apocynum cannabinum*)
red cedar (*Juniperus virginiana*)
nettle (*Urtica dioica*)
red elm (*Ulmus rubra*)
pawpaw (*Asimina triloba*)
dogbane (*Apocynum androsaemifolium*)
basswood (*Tilia americana*)

Tables 1 and 2 provide important information suggesting that the surrounding environment provided the inhabitants of the Dickson Mounds site with a mixed and potentially well balanced diet. The landscape and the natural resources that were offered by the Illinois River Valley presented hunter-gatherers with an optimal place to settle. However, as time progressed, populations increased and inevitably competition among them would have most likely strained the available resources. Also, with the advent of more sedentary cultures in the area, these natural resources would also have become quite drained. At the same time that populations grew, so did the evidence of the introduction of maize via broken hoes and other agricultural implements. along with the floral remains of this cultigen.

According to Harn (1971:6), archaeological evidence reveals that the relative frequency or proportion of wild flora and fauna remains declined during the transitional period from Late Woodland to Mississippian and the use of domesticated maize increased. In accordance with this shift to maize agriculture. the frequency of points and scrapers

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decreased while grinding stones and agricultural implements characteristic of the Mississippian Acculturated Late Woodland increased. As a result, it is believed that with the change in tool use came a change in subsistence practices.

A similar trend in the decreased amount of both wild floral and faunal remains continues into the Mississippian Period at the Myer site as their appearance in refuse or storage pits was rare (Lallo 1973). Also, the Myer site presented a heavier reliance on maize via an increase in storage pits and pottery vessels. The tool assemblage also changed dramatically as the frequency of scrapers, points and blades decreased at this site, while hoes and grinding implements increased to such an extent that they appeared to dominate the tool assemblage. Even though the Myer site is located relatively close to both the Illinois and Spoon Rivers, little use of its resources had been demonstrated. In fact, Harn (1971:24) points out that only a few fish hooks were recovered in the area with no evidence of other marine resources being unearthed.

The evidence from the surrounding sites of Dickson Mounds has illustrated the trend noted in other areas where Late Woodland cultures progressed into the Mississippian Tradition, especially pertaining to nutrition. One notices a decrease in local wild floral and faunal resources with a concomitant increase in maize horticulture. Not only is this evidenced by a decreased frequency in remains, but also in their tool assemblage. In the height of the Mississippian Period there is the culmination of all these trends that is demonstrated by a heavy reliance on maize and an increase in population as well. As a result, the diet changed from one of a very well balanced and mixed diet where most likely adequate amounts of different nutrients were being consumed, to one that became dependent

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on a single cultigen which might not have been supplemented adequately. For example, maize is devoid in lysine, an essential amino acid, and it contains phytic acid which inhibits the bioavailability of iron. The nutritional composition of maize is also limited with only 5mg of vitamin C, $38\mu g$ of folic acid, 0.5mg of iron and no vitamin B_{12}. Therefore, with inadequate nutrition, the people of the Mississippian Period at Dickson Mounds had a higher chance of exhibiting skeletal alterations that are characteristic of nutritional deficiencies.

CHAPTER 5: THE OSTEOLOGICAL COLLECTION

The Dickson Mounds skeletal population chosen for this thesis is currently housed at the Research and Collections Center for the Illinois State Museum in Springfield, Illinois. This collection totals 1050 individuals, which is approximately one third of the estimated 3000 interments thought to have been buried in the cemetery (Harn 1980:76). One hundred ten individuals of an estimated 367 from the Mississippian time period (A.D. 900-1500) were analysed as the primary focus for this thesis. Further definition of the actual categorization of these burials into Early, Middle or Late Mississippian Periods could not be ascertained and as such, only the label of 'Mississippian' can be used for these burials. Only a portion of the burials could be analysed due to money and time constraints. However, the biggest factor in decreasing the sample dealt with the restriction of only looking at burials with 'complete' individuals and those with 'cranial and postcranial' segments (see section 5.3 in this chapter for further definition). Individuals who only had 'postcranial' bones stated in the database were not observed because of the importance of anaemic alterations to the cranium. Therefore, the sample was considerably decreased even before analysis of the Mississippian burials began.

The state of preservation of these skeletons is excellent because of the non-acidic soil conditions. The Mounds consist of loess soil, which has a high calcium oxide content and very few of the acids that normally break down bone. In spite of almost perfect conditions, the skeletal series has not totally escaped postmortem destruction. The biggest problem dealing with the causation of postmortem destruction is the weight of the mounds on the

skeletons. In effect, many crania, especially subadult, have been crushed by the weight of the soil. Also, postmortem disturbance of the burials occurred as evidence from rodent and groundhog burrowing into the soft loess soil could be found. Loss of whole and/or segments of individuals and grave goods also occurred with historical looting of the site.

The Illinois bluffs are deeply incised by many small stream valleys and washes that cut back into the uplands (Harn 1980:4). Also, small springs and seeps are frequent along the slopes of many of the valleys and as a result, slumping from the continuous wetting of the plastic clays and shales has occurred. Water is an important agent in the process of decay for bones as it can cause varying amounts of leaching in addition to hydrolysis of peptide bonds between adjacent amino acids in the protein chain and racemization (Hare 1980:212; Henderson 1987). Therefore, it might be suggested that the preservation of the skeletons at Dickson Mounds may have been altered by the presence of water in the burial environment. However, according to Keith (1981) and her study on cortical bone loss in juveniles from Dickson Mounds, this successive wetting and drying (i.e., the exposure to water) did not seem to have a significant effect on the bones.

5.1 MATERIALS AND METHODS

Prior to assessing the presence of skeletal alterations indicative of nutritional anaemia, it was necessary to determine the demographic profile of the Dickson Mounds population. Age and sex were determined by incorporating both subjective and objective methods to decrease any biases that might be encountered when using only one form of aging or sexing technique. When possible, aging and sexing techniques based on pre- and protohistoric Native American populations were practiced during this investigation in order to provide the closest estimate possible for this particular population. However, this was not always achieved due to the shortage of skeletal research performed on this biological group.

5.1.1 Sex Analysis:

Sex assessment of the Dickson Mounds population was determined by an amalgamation of techniques proposed by Krogman and İşcan (1986) and Bennett (1993). By utilising several different criteria from different areas of the skeleton, a more reliable estimate of sex can be interpreted. All gender assessments were based on observable macroscopic criteria. Standard pelvic and cranial observations were used, but since these criteria are generally subjective, other more objective methods were employed. Measurements of the proximal and distal tibial breadth and the circumference around the nutrient foramen were employed to overcome possible subjective biases. The tibia was chosen for metric sex analysis because of its ability to preserve well in the postmortem

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environment. By combining both subjective and objective criteria, a better sex estimation is produced. However, it was noticed during this investigation that many females had robust tibial measurements for this population, so that in some cases, the pelvic and cranial methods had to be more heavily relied on. For a sample of the sex estimation record form used in this thesis, which includes the characteristics observed in the pelvic and cranial regions, refer to the Appendix.

When possible, the sex of an individual was determined as definitely Male or definitely Female for all adult skeletons. However, in some instances sex characteristics were mixed. As a result, four sex categories were developed for this research: Male, Male?, Female and Female?. No unknown or equivocal sexes were identified because of the use of fairly complete skeletons. It is realized that Subadults manifest no typical sex characteristics on the skeleton prior to puberty, but for a more comprehensive view of the Dickson Mounds population, it was decided to define them. Therefore, an additional category of No Gender/Subadult was produced. In several cases late subadults were sexed when sexually dimorphic attributes were found.

5.1.1.1 Sex Categories:

MALE OR FEMALE: An individual was determined as Male or Female when all of the possible observable morphological and metric characteristics indicated one of the genders.

MALE? OR FEMALE?: When mixed sex traits occurred, the predominant sex characteristics were utilized for assessing sex. Gender possibilities were also encountered when sexually apparent skeletal regions, i.e., cranial, pelvic or long bone metrics, were missing from the burial. As a result, several individuals could not be recorded as definitely either Male or Female and had to be placed in this category.

NO GENDER/SUBADULT: An individual was recorded as being a Subadult when the individual had not yet reached skeletal sexual maturity and thus, no gender could be estimated. This category was produced due to the lack of distinct sexual differences found prepubescent in the skeleton. As a result, all subadults were placed within their own sex category.

5.2 AGE ANALYSIS

Specific appropriate and published age at death methods were employed for each skeleton in order to calculate a more defined age, which are outlined below in separate sections. All age estimations were based on observable morphological attributes. Once again, completeness of the skeletal remains aided in assigning relatively specific age at death estimations for each individual studied. A checklist of the methods and attributes used in this analysis is presented in the Appendix.

As discussed in the section on sex determination, subjectivity was overcome by

employing multiple criteria on each skeleton. By employing several methods more criteria could be used to assess age, which consequently allowed for an overall estimation. In most cases, age estimates were within +/- 5 years of age for adults and within months or several years for subadults, depending on the methods used. In the end, a simple average of all age techniques was performed.

SUBADULT (fetal to peri-pubescent): Specific age estimations within this category were determined by using three different techniques. Maximum diaphyseal length of long bones without epiphyses, primarily the humerus, radius, ulna, femur, tibia, fibula and the maximum breadth of the ilium (without the iliac crest epiphysis), were used in determining the specific age of individuals from birth to 11.5 years of age (Merchant and Ubelaker 1977). In older adolescents above the age of 11.5 years, the authors do not believe that the comparisons providing age estimations from diaphyseal lengths are meaningful due to their small sample sizes. Tooth eruption and growth charts, as presented by Steele and Bramblett (1994:102), were used for assessing age from fetal up to the age of 21. The final technique used for this age category was the assessment of development, maturation and fusion of the epiphyses (Johnston 1961; W.E.A. 1980). This provided age estimations from 11.5 to 24 years of age.

ADULT (Middle to Old Age): Six different techniques were employed for age estimation for the adults of this collection. Several of these methods only allowed for a short age window of opportunity for their assessment. As a result, only certain techniques could be used on certain individuals. Techniques used also depended on the presence of specific bones.

1. The characteristic changes of the sternal end of the clavicle, which occurs between the eighteenth and thirtieth year of age (Szilvássy 1980), were used to estimate age from this particular piece of skeletal anatomy. This allowed for the placement of individuals into three age categories: 18-20, 21-25 and 26-30 years of age.

2 and 3. The pelvic region was also relied on for an individual's age determination. Chronological changes in both the auricular surface of the ilium and the pubic symphysis were employed in this research. As a result, a combination of techniques presented by Lovejoy *et al.* (1985) pertaining to the auricular surface and Bennett's (1993) description of the Suchey-Brooks pubic symphysis system were used. However, after several applications of the latter method it was realized that relatively broad age estimations for the pubic symphysis were being calculated (e.g., 23-57, 27-66 and 34-86 years of age for males). As a result, the Todd pubic symphysis scoring system as presented by Buikstra and Ubelaker (1994:22) was used instead. Standards concerning the auricular surface chronological changes, as presented by Buikstra and Ubelaker (1994:25-32), were also used in conjunction with the Lovejoy *et al.* (1985) method.

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4. The analysis of the fourth sternal rib end as presented by Bass (1995:141-146) was used as another age at death estimation. Unfortunately this method could not be used extensively due to preservation and the inability to precisely identify the fourth rib. On occasion, several different sternal rib ends were used to obtain an overall estimation of age.

5. Ectocranial suture closure, as presented by Meindl and Lovejoy (1985), was also used as an age estimator. On its own, this method is not deemed as a reliable age indicator, but it can be very useful as a *component* of age determination procedures.

6. The final skeletal age indicator employed for this research pertains to chronological dental attrition of both the maxilla and the mandible (Lovejoy 1985). Although attrition varies from individual to individual within a population and from culture to culture, a relatively reliable estimation can be computed. By observing the amount of tooth wear each individual exhibits for upper and lower teeth, an age estimate can be calculated.

On their own, each one of the techniques used in this research does not present a reliable age indicator at death, but when used in conjunction with one another, they exhibit dependable estimates.

5.3 ASSESSMENT OF PATHOLOGIES POSSIBLY CAUSED BY NUTRITIONAL ANAEMIA

Following determinations of age at death and gender for an individual, each bone that was present was closely observed for any macroscopic alterations which might signify the presence of a nutritional deficiency causing anaemia. As described in Chapter 2, four nutrients causing nutritional anaemia were concentrated on: iron, vitamin C, folic acid and vitamin B_{12}. Each nutrient, when deficient, affords very distinct and characteristic alterations in bone (refer to Chapter 3 and Figure 3.1). However, in theory, they also should present similar changes in bone because of their roles in the production and maintenance of red blood cells in the human body. Listed and described briefly below are the pathologies distinct for each nutrient, as well as the similarities that they share which were used for the macroscopic investigation for this research.

5.3.1 Anaemic Bone Changes Shared by All Four Deficient Nutrients:

It has been postulated in Chapter 3 that when any of these four deficient nutrients cause anaemia in the human body, cribra orbitalia and/or porotic hyperostosis will likely be observed. This is due to the hypertrophy of the bone marrow that must compensate for the inadequate quality and/or quantity of RBCs within the human body. As a result, in those skeletal regions where thin cortical bone is present, as in the orbits and calvarium, hypervascularization of the bone ensues.

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Diagnosis of porotic hyperostosis and cribra orbitalia involved utilizing the criteria set forth by other researchers, such as Stuart-Macadam (1985:392). These two pathologies were assessed for the presence of active, i.e., new lesions that expressed spicules within the spongy bone (foramina), or healing lesions, i.e., presence of smooth margins, and whether they were light (scattered fine foramina) (refer to Plates 1, 5, 32, 33, 34, 39 and 42 for active and Plates 10 and 11 for healing versions), medium (large and small isolated foramina and foramina that have linked to form a trabecular structure) (refer to Plates 42 for active versions) or severe (outgrowth in trabecular structure from the normal contour of the outer table) (refer to Plates 32, 33 and 34 for active versions). In order to describe these lesions as such, a hand-held magnifying glass was used. Therefore, the first area of the skeleton that was addressed for the presence of anaemic pathologies was the skull. Once this had been performed, other areas of the body were macroscopically assessed for their respective skeletal alterations due to specific nutritional deficiencies in either iron, vitamin C, folic acid or vitamin B_{12}. However, in some cases poor preservation of the skeletons limited the assessment of these lesions. In others, ambiguous pathological manifestations have made the diagnoses ambivalent.

5.3.1.1 Iron Deficiency Anaemia:

Osteoporosis and course trabecular striations in the distal end of the humerus and the proximal ends of the radius and ulna are often found in association with iron deficiency

anaemia. Since radiographs or cross-sections of bones are necessary to determine the presence of osteoporosis, it was only possible to note it when bones were broken. Most upper limb bones were intact and did not preferentially exhibit broken metaphyses or diaphyses that would enable internal viewing of this area of the skeleton. However, if this circumstance presented itself, then cortices were observed. Osteoporosis was also not recorded as often because of its subjective nature in determining 'lightness' of the bones that could have been caused by postmortem destruction instead of osteoclastic over-development. However, when extreme cases of light bones were discovered, it was noted.

Alterations of the paranasal sinuses were also not assessed as the postmortem environment had destroyed and/or altered many nasal regions.

5.3.1.2 Vitamin C:

For both subadults and adults, vitamin C deficiency can result in the presence of hemorrhages and subperiosteal hemorrhages anywhere in the body. Although these are usually manifested in the soft tissues, in extreme cases they can become ossified and affect the neighbouring bone. Normally they are exhibited in a bilateral fashion, but this is not always the case. Fracturing, especially in the metaphyseal region of bones for infants and the diaphyseal region for adults, was also assessed for both infants and adults, with their state of healing recorded. Diagnosis of the presence of porous, but non-expansive, and hypertrophic lesions on the greater wing of the sphenoid, anterior maxilla, the bone

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surrounding the inferior portion of the nasal aperture and the posterior, or infratemporal, surface of the maxilla were performed. As well, the presence of osteitis (increased porosities caused by infection/inflammation of the compact bone) of the alveolar bone of both the maxilla and the mandible was examined. Loss of teeth, especially single-rooted teeth, was examined for their presence in each individual, as well as the presence of a fistula in the alveolar regions of both the maxilla and mandible.

Infantile scurvy can also present irregularities and disruption in tooth formation, but this was not observed in detail as many teeth were lost postmortem. Infraction and spicules of the metaphyses and thin cortices and sparse trabeculae of the diaphyses were also looked for during this investigation. Cupping and flaring of the costochondral rib segments were examined, but none were found due to the lack of preservation of this area of bone.

Hemarthrosis, synchondroses and femoral head destruction were examined in adults, however, none of these complications of scurvy were observed. Osteoporosis, in the form of biconcave deformities, condensation of the superior and inferior margins and central rarefaction of the vertebral bodies, is another pathology that was considered. Beyond this, osteoporosis and cortical thinning of other skeletal regions were diagnosed via the 'lightness' of the bone when bones were fully intact, even though this is a subjective form of pathological assessment.

5.3.1.3 Megaloblastic Anaemia:

In this form of anaemia, thrombopenia can cause hemorrhagic conditions similar to scurvy. For example, hemorrhages may calcify causing new subperiosteal bone formation and necrotic lesions may be evident. These conditions are most likely the result of atherosclerosis and thrombosis exhibited by megaloblastic anaemic individuals.

Although the above listing of skeletal alterations is specific and when identified could ultimately point toward a diagnosis of a nutritional deficiency, other things had to be taken into consideration. For example, postmortem degradation of bones and normal aging processes had to be taken into consideration prior to the final assessment. However, emphasis was placed on the identification of these deficiency-related skeletal manifestations for this study. In essence, all abnormalities were recorded so that a differential diagnosis could be afforded in certain circumstances. For example, an older individual may exhibit porosities in the cranium, but show characteristic age-related changes in other areas of the skeleton (e.g., degenerative osteoarthritis of the spinal column or long bone joints). As a result, a possible diagnosis of porotic hyperostosis may be influenced toward hyperporosity of age-related conditions. Therefore, it was important to take as many skeletal pathologies into consideration that could be identified on the skeleton.

5.4 DATA TREATMENT

All information that could be recorded for each individual, including the inventory of the skeleton for each burial, age, sex and the presence of pathological lesions were assessed for 110 individuals from the Dickson Mounds Mississippian Period collection. Although 367 individuals have been associated with the Mississippian time period, only those burials which contained complete specimens and those with both cranial and infracranial skeletal regions were observed. The presence of a skull within a burial was the most important element for this research as anaemic changes, i.e., cribra orbitalia and porotic hyperostosis, are always manifested in this skeletal region. As a result, if only infracranial skeletal elements were present in a burial these remains were not observed. This segregation of skeletal material was accomplished by looking at the data base already compiled by the Research and Collections Center in Springfield, Illinois which stated whether burials contained 'complete' individuals, cranial/postcranial elements, just cranial elements or just postcranial elements. It was important to study only the most complete individuals available in this collection because of the expansive nature of those nutrients analysed in this study and their specific manifestation on different areas of the entire skeleton.

Time and monetary constraints only allowed for a sample of the Mississippian burials to be analysed and for a macroscopic observation of grossly presented skeletal manifestations of four nutrient deficiencies causing anaemia. In the end, a sample of 201 skeletons, which included complete individuals and those burials with cranial/postcranial

elements, could be used from the Dickson Mounds collection. Out of this sample, only 159 individuals were available as fifteen burials could not be located to a specific box and twenty-seven of the observed sample were not appropriate for use because only a few cranial and/or postcranial elements were present. Thirty-eight complete specimens and seventy-two burials including cranial/postcranial elements were observed for this sample (N = 110). Therefore, 110 out of 159, or 69.2%, of the entire possible Mississippian population from the Dickson Mounds collection were utilized for this research.

The final working sample used for this research involved segregating those individuals who had either cribra orbitalia and/or porotic hyperostosis present in the skull, as these two manifestations are thought to be indicative of anaemic situations for the four nutrients chosen for this thesis. Of the 110 individuals assessed for this study, forty (36.36%) manifested these two pathologies. Only these specimens were analysed for the presence of other skeletal manifestations outlined above which are characteristic of anaemias caused by deficiencies in iron, vitamin C, folic acid and vitamin B_{12}.

5.4.1 Database Analysis:

All information gathered for the individuals observed from this collection was entered into a Quattro Pro 8.0 spreadsheet. From here, descriptive statistics were performed in which frequencies, ranges and other attributes were calculated in order to create a better understanding of the population. In those instances where significance of certain relationships was needed, then Chi-Square analysis was used.

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5.5 DESCRIPTIONS OF PATHOLOGICAL INDIVIDUALS

The following are descriptions of those individuals who exhibited some form of cranial anaemia, i.e., cribra orbitalia and/or porotic hyperostosis, and possible signs of cranial and infracranial lesions associated with nutritional deficiencies causing anaemia. Where possible, diagnoses of pathological lesions not associated with anaemia are presented as well.

Burial # 236: The individual from this burial was an infant, aged approximately 12 +/- 6 months. Cribra orbitalia is bilaterally presented in both orbits and is of the active and severe type (similar to Plate 32: B-442). The right orbital lesion measures 24.06x25.22mm; the left is broken, but 11.52x20.96mm can be measured. Porotic hyperostosis is also observed on the parietals and the occipital, especially centered near the lambdoidal suture. It is considered active and of medium severity with a more extensive lesion measuring 13.42x17.24mm on the left parietal; the right parietal only involves 8.6x21.02mm of the bone; and the occipital involves 16.46x32.9mm of mostly the left side. Active and medium porotic hyperostosis is also observed bilaterally on the frontal, with a lesion measuring 8.6x21.02mm on the right and 13.42x17.24mm on the left (Plate 1).

Circumscribing these lesions are areas of periostitis and/or reactive bone. Also found on the skull are endocranial porosities that are quite severe and similar to those found ectocranially, but there is the possibility that these apertures are due to postmortem damage or an extension of the hypervascularization already present in this individual.
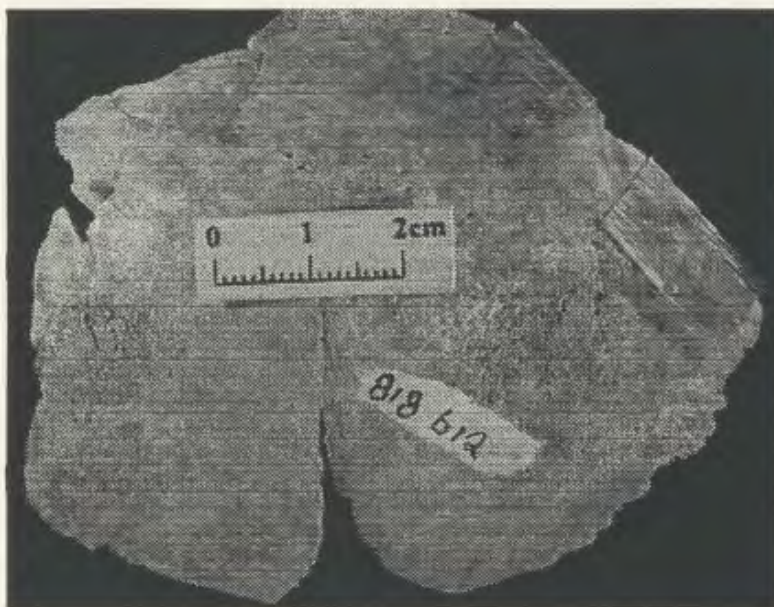
Plate 1: B-236
Porotic Hyperostosis of Frontal Bone: Active and Medium Severity. Subadult, 12 +/- 6 months of age. Note two areas of density, positioned on left and right sides of this bone.

The right tibial shaft presents growth in a concentric ring-manner in which the laying down of bone coincides with that of tree growth. It is most likely associated with irregular deposition of new pathological bone to the periosteum. The etiology of this pathology is unknown. It was also observed in Burial Number 391.

Burial # 243: The individual from this burial was a male aged approximately 50+ years. Cribra orbitalia may be present in this individual as a very light case of porosities can be observed bilaterally. A similar presentation of porotic hyperostosis is found on the occipital (36.16x60.2mm) and parietals (L=91.54x47.38mm; R=72.24x48.12mm). It is quite possible that these porosities are age, rather than nutritionally related, as this individual also exhibits several characteristics associated with older age. For example, degenerative osteoarthritis is present on the left navicular and calcaneus, along with all long bone joints (patellae included). Lipping is manifested on both the right and left medial tuberosities of the

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calcaneus, as well as the right talus. Marginal lipping is also found on the lumbar and cervical, with some vertebrae exhibiting lateral-medial indentation of the body. Superior and inferior vertebral condensation is located at thoracic #11, but this is most likely due to age-related processes. Bone spicules are associated with healed fractures found on left ribs #9 and #10 and right rib #9 (Plate 2) which may be in response to the fractures and/or age-related growth processes. The mandible of this individual also has lost most of its teeth via antemortem processes, with exception of the left I2, C and PM1 and the right C and PM1.
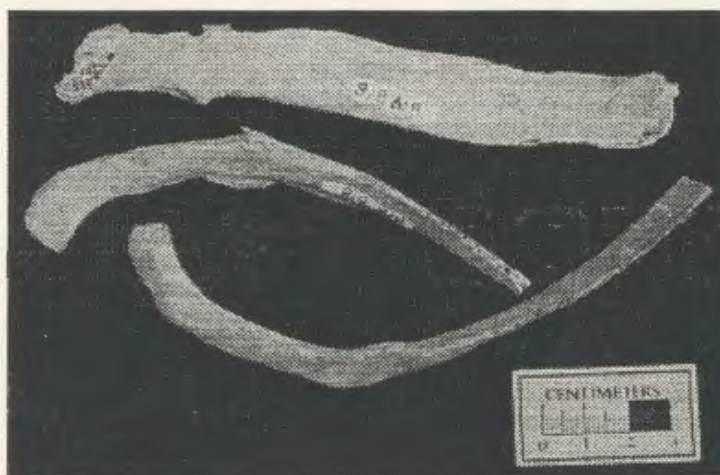
Plate 2: B-243
Healed Rib Fractures: Male, 50+ years of age. Top rib in the picture is the pleural side of right rib #9; middle is the superior side of left rib #10; and at the bottom of the picture is the inferior side of left rib #9.

Burial # 256: The individual in this burial was a female approximately 16-18 years of age. Cribra orbitalia is bilaterally presented in both orbits as active and of light severity (similar to Plate 5: B-310). A light case of porotic hyperostosis is only found on the parietals around the sagittal suture, as the occipital is not present. The lesion on the right parietal measures

87.9x36.68mm and the left is 88x26mm. Multiple lytic holes can be found along all of the medial and lateral aspects of the vertebral bodies, revealing cancellous bone. This may be the result of degenerative arthritis or the beginning of tuberculosis-like lesions.

Left and right metatarsals #5 have active and porous periostitis along their entire shafts. Periostitis is also located on the left metatarsal #1 along the superior side of the shaft, but this appears to be healed. A light and active case of periostitis is observed on the anterior side of the medial aspect of the left ilium. A similar example of periostitis is presented on the right and left femora by the proximal ends of the shafts on the lateral side just below the lesser trochanter, approximately midshaft. Two-thirds of the right and left tibiae have healed periostitis along the anterior crest. An active section of approximately 80.44x9.76mm in area is found on the right fibula midshaft, while the left fibula presents a slightly less severe case of approximately 10.26x5.34mm in area.

Burial # 270: This burial is of a subadult approximately 3 years +/- one year in age. This individual has a light case of cribra orbitalia that is essentially only present in the left orbit (9.6x20.3mm), as the right appears to be much lighter and most of the orbit is missing due to postmortem destruction. Lytic holes can be found in one lower thoracic vertebra and one upper lumbar with some woven bone present on the interior circumference of the holes with the exposure of cancellous bone (Plate 3). This lesion of the lumbar vertebra has actually destroyed a section of the centrum margin, while the thoracic vertebral lesion is located more centrally in the centrum body.

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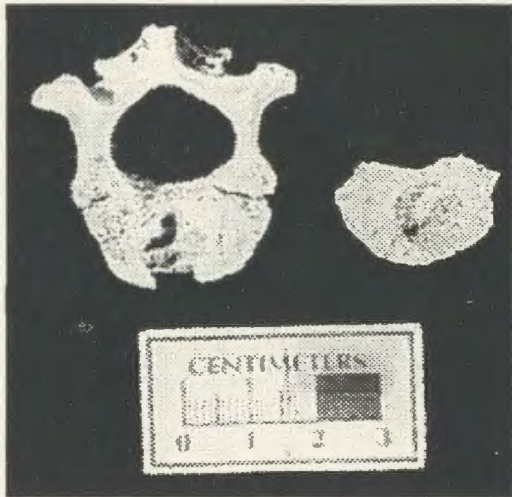
Plate 3: B-270
Lytic Lesions of Vertebrae: Subadult, 3 +/- 1 years of age. Lower thoracic and upper lumbar vertebrae shown here. Note exposure of cancellous bone.

The left ilium presents an interesting pathology that Lallo (1973) diagnosed as a hemangioma (Plate 4). According to Ortner and Putschar (1997:376-378), this pathology is a neoplasm formed by proliferating blood vessels and is a rare manifestation on any part of the skeleton. Lesions of this type are usually found in the spinal column, but can also occur in the cranial vault whereby they are usually round with a lytic margin and both the inner and outer tables are destroyed. Approximately 50% of the bone in this specimen is missing, but the pre-adult iliac border is still present. It appears that sclerotic and woven bone are holding the ilium together which is indicative of this type of pathology. One might suggest that this pathology is congenital due to the complete nature of the iliac border, the age of the individual and the way the ilium has almost grown around something, possibly a tumor.

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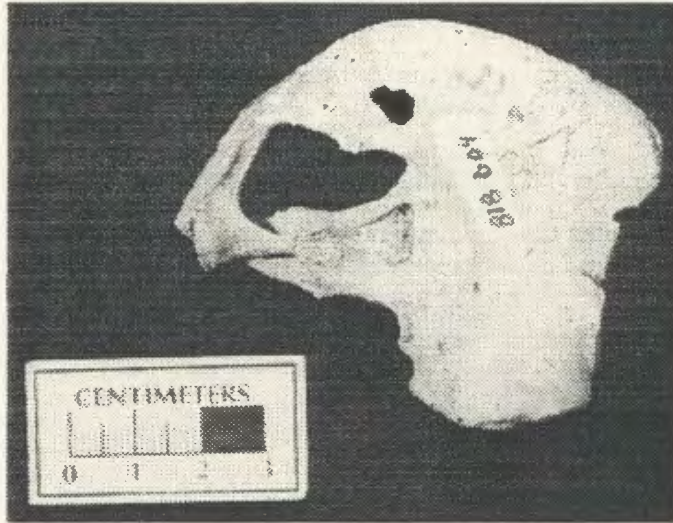
Plate 4: B-270
Hemangioma of Left Ilium: Subadult, 3 +/-
1 years of age.

Burial # 283: Burial Number 283 was a male of approximately 40 +/- 5 years of age. This specimen only has porotic hyperostosis of the occipital and the parietals. Both the occipital (48.72x56.86mm) and the parietals (R=38.7x30.5mm; L=28.62x9.28mm) are of medium severity and healing. Increased porosity can be found along the temporal lines as well. This individual exhibits age-related changes, i.e., osteophytes are found on the internal neural arches, especially of the lower thoracic, and the right patella has an osteophyte on the medial side with osteoarthritis present on the medial condyle and therefore, the hyperostosis of the cranium may also be age-related. A left tarsal medial phalange has some extra subperiosteal growth at the distal end. The mandible has lost all of its left molars and right molars #1 & 2 to antemortem processes and the right third molar is carious. An active area (27.19x10.58mm) of very porous periostitis can be found on the left tibia near the distal end of the shaft. Also associated with this long bone is an area of osteitis along the tibial crest from the midshaft to the proximal end.

Burial # 310: The individual from this burial was a subadult aged 3 years +/- 6 months. A light and active case of cribra orbitalia is found bilaterally. However, only a very small section of the right orbit is present, therefore only the left could be observed and measured (15.5x6.24mm) (Plate 5). The rest of the skull has been altered by postmortem conditions and thus, porotic hyperostosis could not be observed. A lytic lesion (8.52x6.9mm) is presented on a lumbar centrum which is exposing cancellous bone (Plate 6).

Plate 5: B-310
Cribra Orbitalia: Active and Light. Subadult, 3 years +/- 6 months of age.

Plate 6: B-310
Lytic Lesion of Lumbar Centrum: Subadult, 3 years +/- 6 months of age.
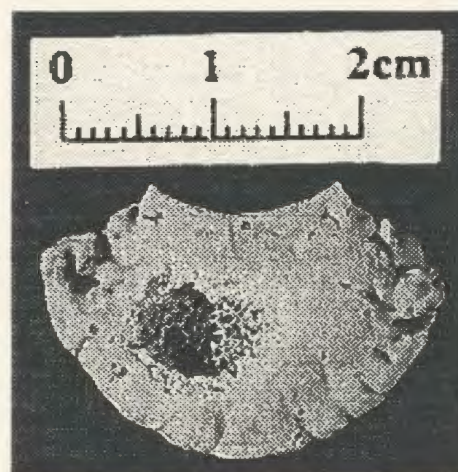
Burial # 348: Burial Number 348 houses a female between the age of 15-20 years. This individual only has a few, relatively large perforations on the orbital wall representing a minimum case of cribra orbitalia with healing lesions (L=8.76x14.82mm; R=7.22x14.38mm). Medium, healing porotic hyperostosis is present on the parietals by the sagittal suture (L=70.6x26.88mm; R=67.32x22.82mm) and another section is found by the lambdoidal suture (L=25.54x20.82mm). The occipital has mixed small and some larger foramina that are healing (21.46x60.76mm). Cradle boarding is very evident as the occipital is flattened. As a result, it is possible that the increase in porosities on this bone may be the result of this cultural practice.

A medial tarsal phalanx has extra subperiosteal bone on the lateral side close to the distal end. At the proximal end of the anterior crest of the left tibia is a section of healed periostitis. Vertebra L1 has medial-lateral indentations possibly associated with osteoporosis. Also associated with the vertebrae, is the presence of minimal osteophytosis. On the right rib #9, on the pleural side of the shaft, is a deposit of button osteoma-like bone.

Burial # 357: The individual associated with Burial Number 357 was a female, aged between 35 and 40 years. Only the right orbit is present in the burial unit and thus, cribra orbitalia is only observed for this side. Very small apertures reveal a light and healing form of this pathology measuring 9.06x13.36mm in area (similar to Plate 11: B-381). Porotic hyperostosis is noted as tiny apertures on the parietals (R=80.82x68.54mm; L=71.1x67.86mm) with slightly larger foramina found on the occipital (48.26x51.76mm). Although the cortical changes are minimal, leading toward elimination of this as a case of

porotic hyperostosis, the fact that a section of the left parietal exposes severe thickening of the diploë reinforces its presence (Plate 7).
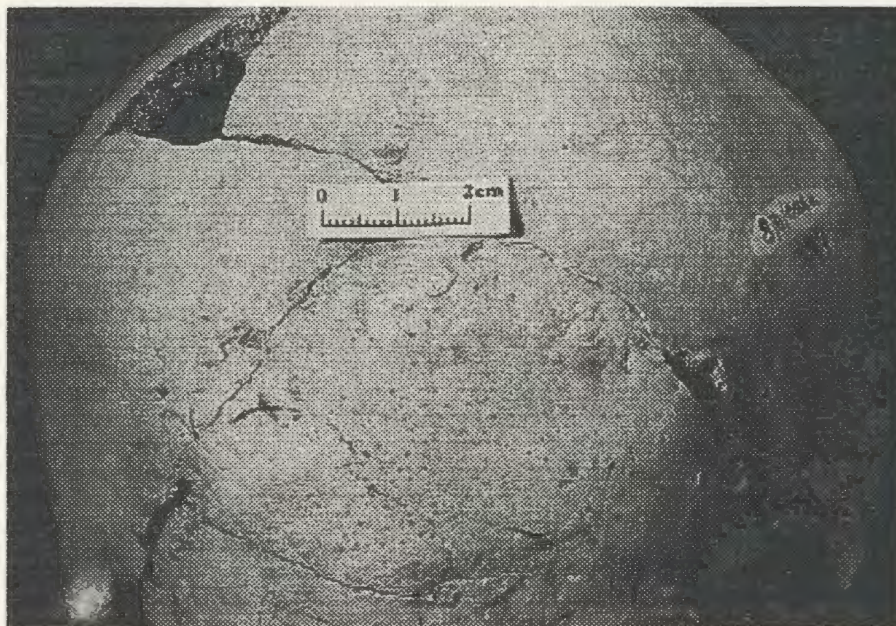
Plate 7: B-357
Thickening of Left Parietal Diploë: Female, 35-40 years of age.

However, several age-related pathologies are also noted on this specimen. Osteoarthritis is located at the sternal end of the first right rib, on the thoracic vertebral articular facets, i.e., rib facets, inferior articular facets and superior articular process facets, on the superior transverse process articular facet of cervical vertebra #3, and on the lateral condyle of the left humerus. There is a lytic hole (5.52x6.2mm) that is exposing cancellous bone on the superior side of a thoracic centrum. There is antemortem loss of the right PM2 and possibly M1 and 2, and also the left PM2, M2 and 3 of the maxilla. A large abscess/fistula is present around the right molars of the maxilla that extends up into the sinus

(Plate 8). In this area the maxilla is indented, which was only observed unilaterally, suggesting that it is not a variant, but instead is a result of the collapse of bone from the abscess/fistula.
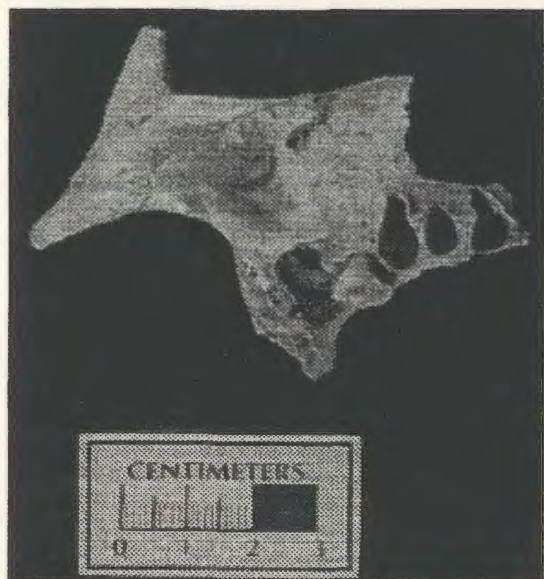
Plate 8: B-357
Possible Abscess or Fistula of Right Maxilla: Female, 35-40 years of age.

Burial # 360: Burial Number 360 was a subadult aged 3 years +/- 1 year. Bilateral cribra orbitalia is present in this individual and it is medium and healing. Porotic hyperostosis of medium severity and healing is located on the parietals and the occipital by the lambdoidal suture, whereby its concentration is more to the right on the occipital, and patches can be found on of the frontal bone. Reactive/active periostitis is found on the front of the right tibial shaft, the pleural side of one right rib, a piece of unidentified long bone shaft and on the front of the femoral shafts. Also, a very large reactive lytic lesion is noted on a piece of slightly flat unidentified bone.

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Burial # 366: The individual associated with Burial Number 366 was a female of the age 50+ years. Light and healing cribra orbitalia is observed bilaterally, but most of the right orbit is missing due to postmortem destruction (L=18x15.1mm) (similar to Plate 11: B-381). The skull is poorly preserved and therefore, it is hard to note any porosities. However, the diploïc space, which could be seen in cross-section of the fragments, is definitely expanded on the parietals and the occipital (16.06x26.62mm) (similar to Plate 7: B-357).

Periostitis is associated with the right and left metatarsals #5. Active periostitis, with striations and some pores interspersed, is also found on both the right and left tibial shafts, with the left being more severe, and the right and left fibular shafts (Plate 9).
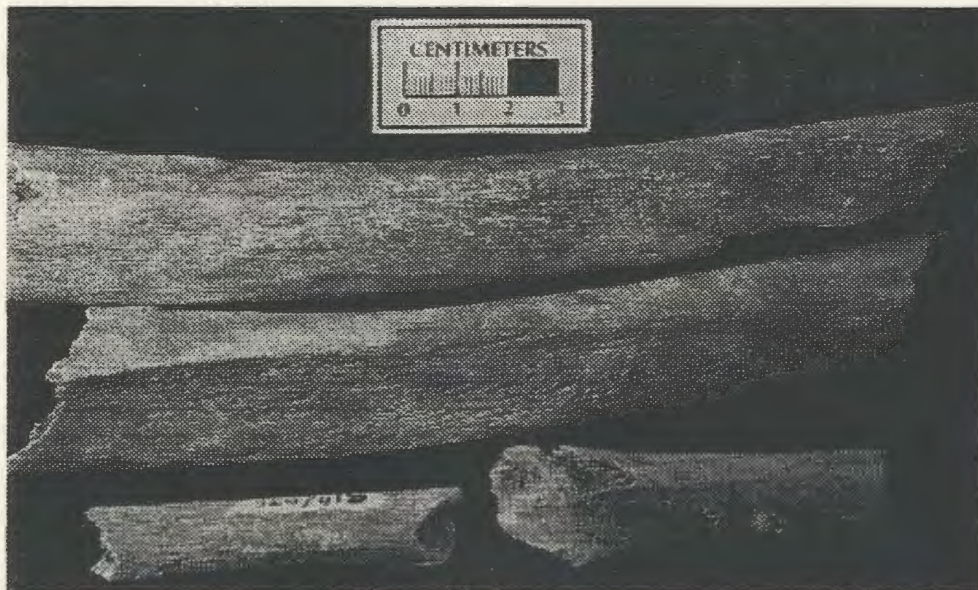
Plate 9: B-366
Variations of Periostitis on Tibiae and Fibulae: Female, 50+ years of age. Striations and pores on all bones. Active spongy deposition on left tibia (second bone from top), left fibular shaft (left bottom corner) and on left distal fibula (right bottom corner).

Two left ribs have been fractured and are healed. A left proximal phalanx and a medial phalanx of the foot are ankylosed together. Osteoarthritis is present on the margins of the centra of most lumbar vertebrae and superior articular facets of two thoracic. An osteophyte can be found on the superior olecranon process of the right proximal ulna. Antemortem loss of the right mandibular M2 is noted for this individual.

Burial # 369: Burial Number 369 was a subadult aged 18 +/- 6 months. Poor preservation is noted for this entire skeleton, but cribra orbitalia and periostitis are noted. Only the right orbit is present with active and of medium severity cribra orbitalia (8.86x9.78mm) (similar to Plate 43: B-464). At both the proximal ends on the posterior side of the tibiae, active periostitis is found.

Burial # 379: The individual associated with Burial Number 379 was a female, aged 21 years. Cribra orbitalia is bilaterally presented in both orbits in a healing and light manner (L=14x19.98mm; R=10.84x18.88mm) (similar to Plate 11: B-381). Light and healing porotic hyperostosis is found on the occipital near the lambdoidal suture (23.22x41.32mm) and on both the right (97.3x53.48mm) and left (100.3x48.32mm) parietals (Plate 10). Some spicules are found in several foramina on the parietals which suggest that the lesions are not totally healed. Some slight sections of striated periostitis are located along the distal shaft of the right ulna, especially by the styloid process. This is similarly presented on the right radius, but it is not as extensive. A section of plaque-like, polished area can be observed on the right mandible just beneath M1 and 2 (~14x5mm).

Plate 10: B-379
Porotic Hyperostosis of Left and Right Parietals: Healing and Light. Female, 21 years of age.

Burial # 381: The individual associated with this burial was a female approximately 25 +/-

5 years of age.   Cribra orbitalia is bilaterally presented (R=18x31.78mm;

L=17.28x28.66mm) in a light and healing manner (Plate 11).
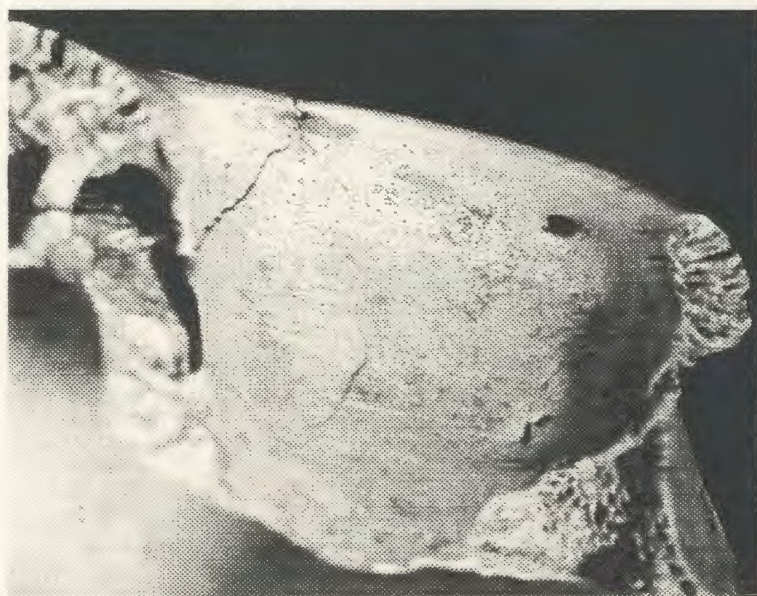
Plate 11: B-381
Cribra Orbitalia: Healing and Light.
Female, 25 +/- 5 years of age.

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Porotic hyperostosis is categorized as light and healing for both parietals (R=47.4x23.34mm; L=73.4x38.52mm), especially found along the sagittal suture, the frontal bone (38.96x32.46mm) and slightly larger perforations endocranially on the occipital bone (similar to Plate 10: B-379). Endocranial porosities are also noted along the sagittal suture. In the last third of the left clavicle, by the acromion end, is a healed fracture. A left rib also has a healed fracture and two right ribs have tiny button osteomata on the pleural sides of the shafts. This latter manifestation may be indicative of the presence of respiratory disease.

By the distal end and along the medial aspect of the interosseous crest of the left radius is a healed oval raised area of periostitis and/or osteitis. Periostitis is also noted in conjunction with a healed fracture on the distal end of the left ulna (Plate 12). It is possible that this manifestation is associated with the periostitis found on the adjacent left radius, as both lesions are present at nearly the same location at the distal ends of the bone.
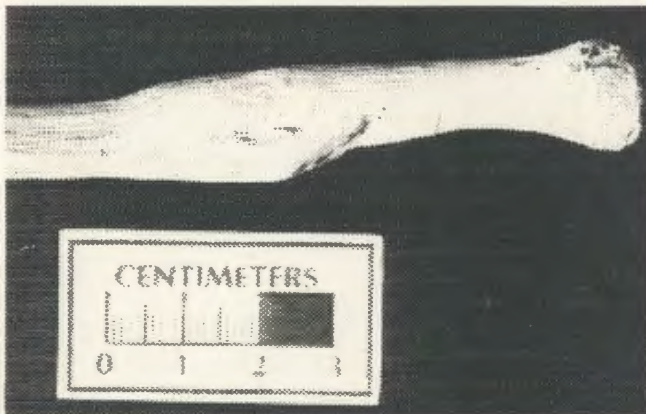
Plate 12: B-381
Periostitis and a Healed Fracture of the Left Ulna: Female, 25 +/- 5 years of age.

The left femur presents an anomalous oblong raised section on its medial side of the midshaft area which could be attributed to either an ossified hemorrhage or a healed section of periostitis and osteitis. Osteoarthritis is found on the posterior lateral condyle of the right

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femur. The posterior shaft of the left tibia has a section of healed periostitis that could also be considered a manifestation of osteochondroma, i.e., a benign bone tumor, although these are usually associated with the metaphyseal region and this is not. The left tibia also presents an enthesophyte just inferior to the condyles on the lateral side, which is indicative of excess muscle use. The right tibia also manifests porosities characteristic of periostitis along the medial side of the shaft.

The lumbar vertebrae exhibit lateral-medial indentation of the bodies. Marginal lipping of the proximal facets of the proximal carpal phalanges is also noted. There is also an unknown bone deformity of a left tarsal medial phalanx that is located on the lateral side of the distal end in which some extra subperiosteal bone growth is observed. Antemortem loss of the left M1 and right M2 can be observed on the mandible, along with an increase in porosities around the left M1 and right premolars of the mandibular body. A healing lesion, or an area with increased porosity, is found on the posterior side above the incisors of the right maxilla (Plate 13). There is also another smaller section found around the painted ID #. What is interesting about this individual is that most of the pathologies are situated/clustered on the left side of the skeleton.

Burial # 383: Burial Number 383 represents a female individual aged 21-24 years. This individual has many different and very severe pathologies that may be associated with a tuberculosis-like disease. To begin the discussion of this individual, it should be noted that cribra orbitalia is present bilaterally, with it being more severe on the right orbit than the left. The right orbit (6.54x16.8mm) is considered to be severe and active (similar to Plate
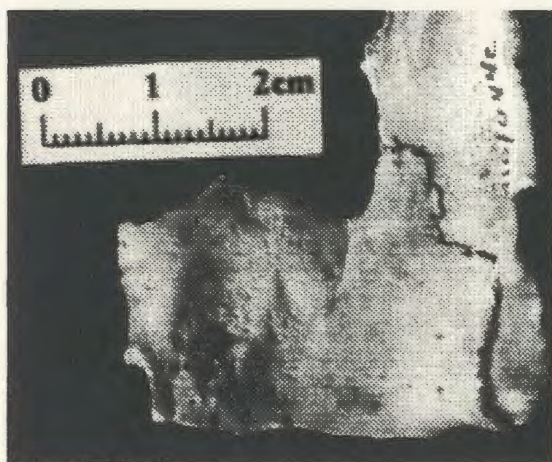
Plate 13: B-381
Hyperporosity of Right Maxilla: Female, 25 +/- 5 years of age. Possibly caused by scurvy and the placement of muscle attachments and chronic inflammation from hemorrhaging.

32: B-442) while the left (5.6x18.74mm) is light and active (similar to Plate 5: B-310). Some of the apertures on the right orbit are impressively large, with one measuring 2.22x1.85mm in area. Differential presentation of the orbital lesions may indicate possible hemorrhaging that was more severe in one eye, rather than the other. Porotic hyperostosis can be found on the right and left parietals via the presence of expanded diploïc areas. In fact, the thickest measurement on the right parietal is 11.44mm and the left is 11.52mm, which were taken at a point along the sagittal suture close to the lambdoidal suture (Plate 14).

Another measurement was taken internally from the sutures in the posterior section of the right and left parietals with values of 9.67mm and 10.38mm, respectively being measured (similar to Plate 7: B-357). Porosities associated with porotic hyperostosis are very tiny over the parietals with one section (16.94x7.75mm) on the right being heavier. This is also evident on the left side with a more involved section (18.6x27.76mm) near the junction of the lambdoid and sagittal sutures.

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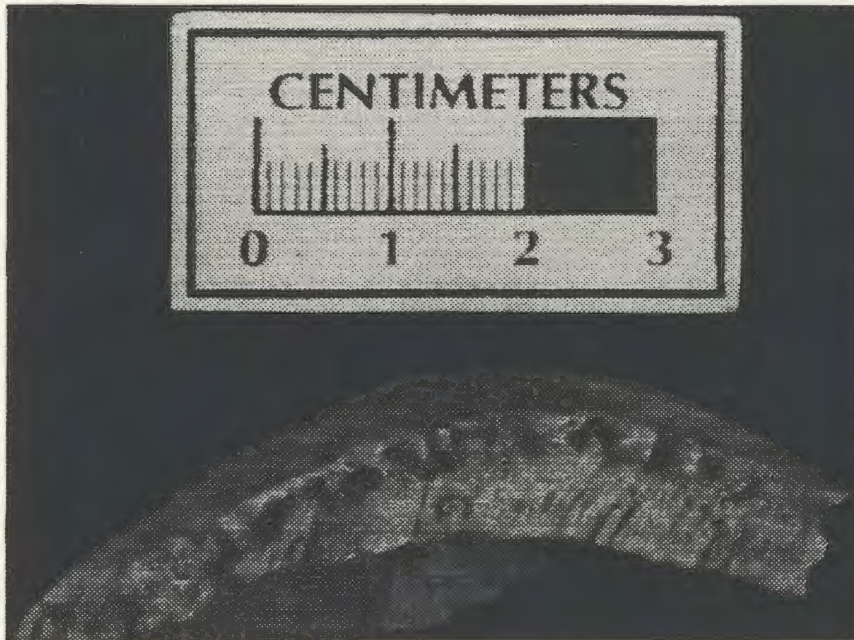
Plate 14: B-383
Diploïc Expansion of Right Parietal Along Sagittal Suture: Female, 21-24 years of age.

The right parietal also has an ectocranial lesion that is approximately 35.36mm internal to the suture that can best be described as a discoloured circular lesion with rough periostitis-like borders with areas of smooth bone 'lumps' underneath. The lesion measures 11.96x14.4mm (Plate 15).
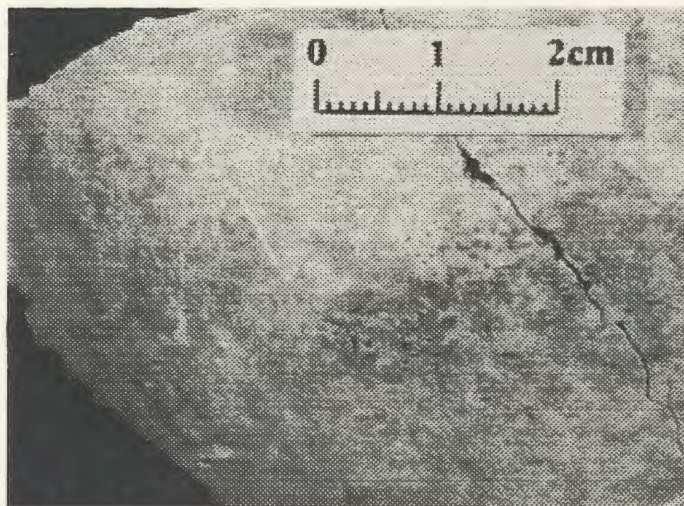
Plate 15: B-383
Ectocranial Lesion of Right Parietal: Female, 21-24 years of age. Note the presence of periostitis with the lesion.

The left parietal also presents an unusual lesion (48.59x45.7mm) of unknown etiology. It is located on the endocranial surface along the sagittal suture and ending just 16mm away from the coronal suture (Plates 16 and 17). The lesion has a spongy interior with bone spicules or islands that are smooth and irregularly spaced throughout. The outer table of the parietal in this area is very thin and essentially no inner table or diploïc area exists, but only thick, dense cortical bone can be seen.
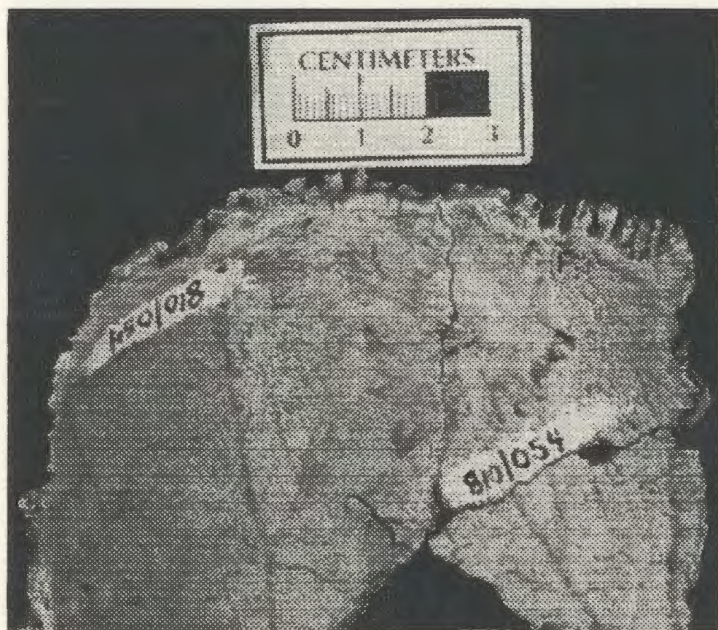
Plate 16: B-383
Endocranial Lesion of Unknown Etiology on Left Parietal: Female, 21-24 years of age.

Only a few apertures are present ectocranially in the region of this lesion. A raised area of bone can be seen on the left frontal. A section of the superior posterior area of the greater wing of the sphenoid is engulfed with smooth 'caverns' that are most likely the result of osteomyelitis (Plate 18). On the anterior side of the greater wing of the sphenoid is an area of light periostitis.

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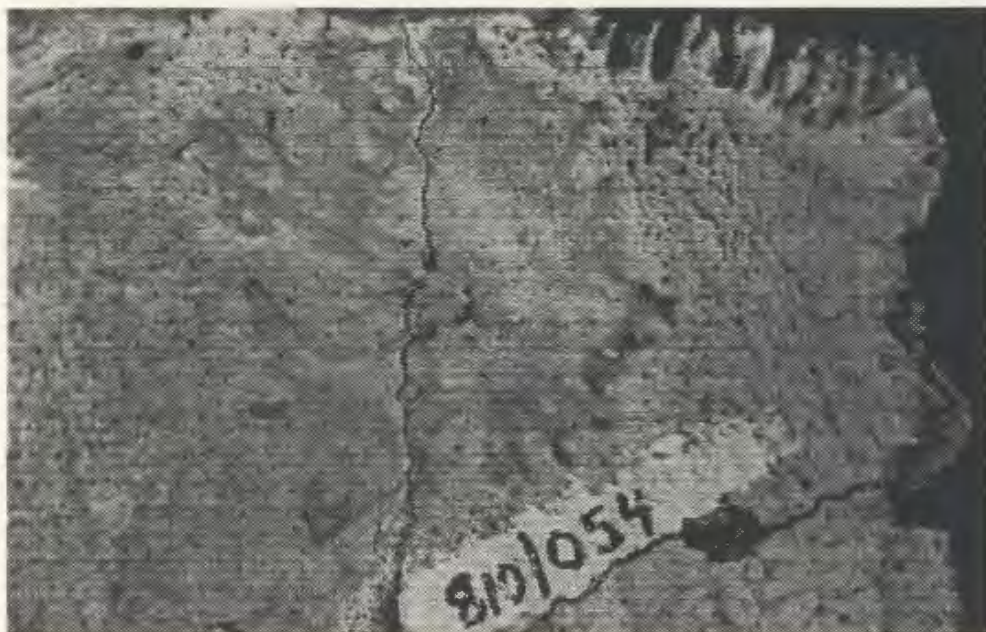
Plate 17: B-383
Close up of Plate 16:
B-383.

Plate 18: B-383
Osteomyelitis of Sphenoid:
Female, 21-24 years of age.

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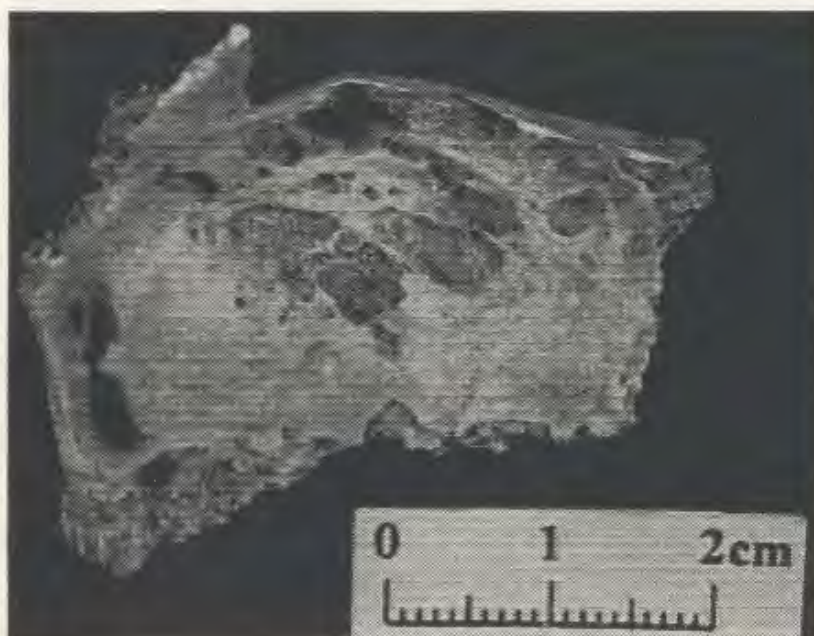
Osteoarthritis is present on the left and right lunates. Medial-lateral indentation of the vertebral bodies of the lumbar is evident. Vertebrae T10-12 have massive loss of cortical bone via lytic lesions of the centra, whereby a large proportion of the margin is missing. These pathologies may be evidence of tuberculosis-like disease (Plate 19).
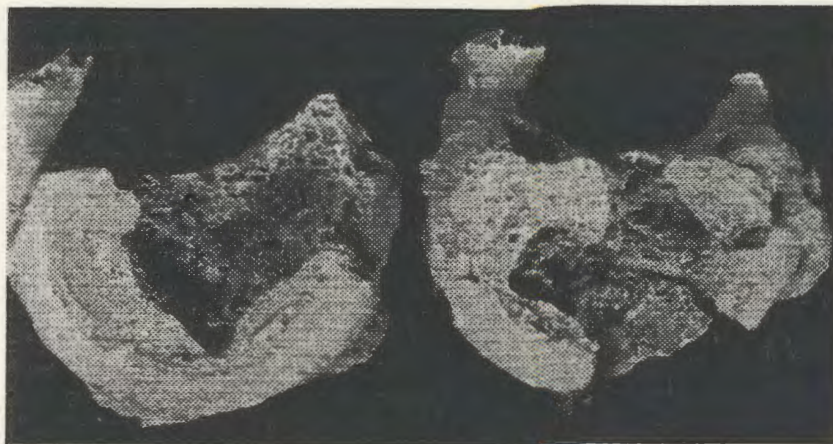
Plate 19: B-383
Possible Tuberculosis-like Lesions of Vertebrae T10-12. Female, 21-24 years of age.

Four right and three left ribs exhibit active, spongy and porous periostitis on the pleural side by the vertebral end of the bodies (Plate 20 illustrates examples). However, some are more extensive than others (e.g., R=24.58x8mm; L=33.46x9.42mm). Unfortunately, some of the periosteal lesions have been broken off which may decrease the apparent severity of them.

Three ribs show the possible evidence of ossified hemorrhages as 'shell-like' bone
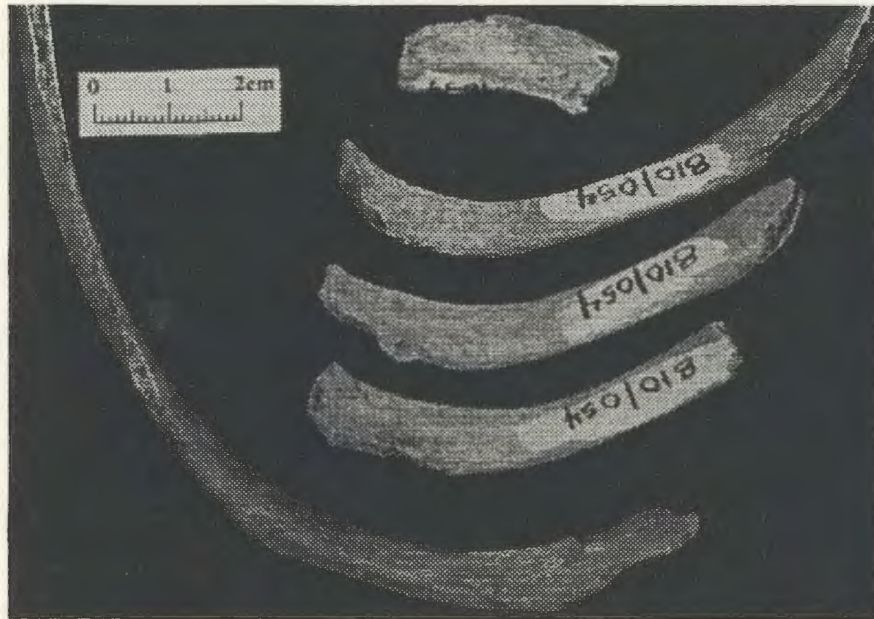
Plate 20: B-383
Periostitis on Several Ribs: Female, 21-24 years of age.

growth is noted (Plate 21 for close up of two of these ribs). One has a deep cavern/hole with smooth edges and some thin bone traversing across a section of it (upper rib shown in Plate 21); the second one (lower rib piece in Plate 21) only has the rim left of a possible ossified hemorrhage; the final ossified hemorrhage (not shown in Plate 21: B-383) can be located at the end of a broken piece of rib and unfortunately, paint from the ID# is covering most of the pathology, therefore it is hard to observe properly. Associated with all of these possible ossified hemorrhages is periostitis.

Periostitis is found on the left maxilla just posterior and superior to the third molar which is peg-shaped. The right maxilla houses an irregular PM2 socket in which the socket is rotated perpendicular to the norm.

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Plate 21: B-383
Ossified Hemorrhages on Ribs: Female, 21-24 years of age.

Severe and active periostitis is also associated with many of the long bones in this individual. For example, at the proximal end of the right ulna, on the lateral side by the radial notch, a section of periostitis is noted. Osteitis and periostitis are observed on the lateral side of the right ulna near the proximal end by the corner of the coronoid process. These two pathologies (38.71x34mm in area) are also noted on the distal lateral side of the right humerus. This lesion begins on the superior side and encircles the inferior side, lateral and superior to the capitulum, with essentially three lytic core areas (two on the front and one on the back).

The left humerus has a large depression (9.72x8.02mm) located just inferior to the medial epicondyle close to the posterior side. It is circumscribed by periostitis and woven cancellous bone is observed. The left ulna is discoloured which makes differentiating between the normal and the pathological very difficult. However, it appears that some evidence of periostitis is found at the proximal end just posterior to the radial notch and also on the medial side. The left radius has an area located just superior to the ulnar notch

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measuring 11.53x14.18mm. Some striations and pitting, evidence of periostitis, can be observed along the front of the right and left femoral and tibial shafts. This is similarly presented on the right fibula. The left fibula has a reactive area (length of shaft involved measures 79.34mm) of periostitis located closer to the proximal end of the shaft that circumscribes the entire shaft in this area.

Burial # 391: Burial Number 391 includes an infant approximately 0-2 months of age. Cribra orbitalia is medium and active in both orbits (7.04x6.42mm; R=7.32x8.42mm) (similar to Plate 42: B-464). There is no porotic hyperostosis recorded. A lytic lesion with periostitis associated with it is found on the proximal end of the right radius. A very large lytic lesion is located on the lateral end of the left clavicle. A femoral fragment of unknown side presents an interesting case. The shaft is engulfed with extremely porous periostitis and appears to be growing irregularly with concentric layers like a tree and the ends of this long bone are widening. This abnormality may have to do with the irregular deposition of new pathological bone to the periosteum, or inflammation of the periosteum due to chronic hemorrhaging.

Burial # 399: Burial Number 399 is a male individual approximately 35-40 years of age. Cribra orbitalia is bilaterally presented as light and healing (similar to Plate 11: B-381), but the left orbit is broken and cannot be measured (R=9.48x21.58mm). Porotic hyperostosis is light and healing (similar to Plate 10: B-379) on the parietals, especially centered along the sagittal suture (L=92.22x39.54mm; R=95.7x32.28mm). Porosities are also found in this same area endocranially. Osteitis is present on the alveolus of the right maxilla just superior

to M1 (Plate 22).
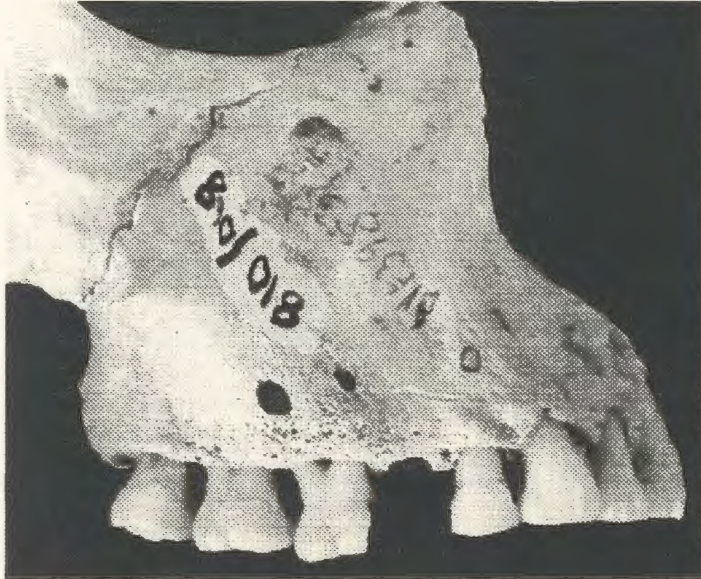
Plate 22: B-399
Osteitis of Right Maxilla Alveolus and Possible Fistula or Abscess Drainage Holes of Right M1: Male, 35-40 years of age.

Also associated with this tooth at the apex of its roots are two drainage holes that could be fistula or abscess drainages (Plate 22). The holes appear to have smooth margins indicating that they were formed prior to death. It is possible that the osteitis or increased porosities observed in this area are just bony reactions to the abscess or fistula. This picture also reveals the presence of several shiny, plaque-like bony nodules situated right at the margin of the alveolus, superior to PM1 and C.

Active periostitis is located on the superior side of the first metatarsal, while healed periostitis can be found on the midshaft area of both the right fibula and tibia. Due to their positioning in the body and the location of these periosteal lesions, it might be assumed that

one lesion has affected the other and could be associated with the same etiology. There is the possibility that osteoporosis is present in the innominates because they are extremely light in weight and very porous. However, this could also be due to postmortem destruction. An osteophyte can be found on the anterior regions of both the right and the left first carpal phalanges, as well as the superior side of the left metacarpal #3. Massive marginal lipping/osteophytes are also located on the left side of vertebra L4.

Burial # 406: Burial Number 406 was a female individual aged 40 +/- 5 years. Cribra orbitalia is presented bilaterally in a medium and healing manner via a cluster of apertures (L=10.94x9.06mm; R=10.86x13.62mm). No evidence of porotic hyperostosis is noticeable due to postmortem damage, but the skull does feel heavier than usual which could indicate diploïc hyperplasia and expansion. However, an area (23.26x26.56mm) on the endosteal surface of the left parietal has pores of different sizes and a smooth indentation that may be caused via osteitis or osteomyelitis. On the left frontal bone by the frontal eminence is a slight half-moon-shaped lytic lesion (25.26x10.98mm) that is healing (Plates 23 and 24). This lesion almost penetrates into the diploë and the margins are rough and irregular with some porous areas.

A similar, but shallower lesion, is located just superior to the nasal spine and just medial to the left orbit on the frontal bone. This lesion looks superficial because it is just starting to affect the outer table. Antemortem loss of the maxillary right PM1 and 2, as well as the mandibular right M1 is observed.

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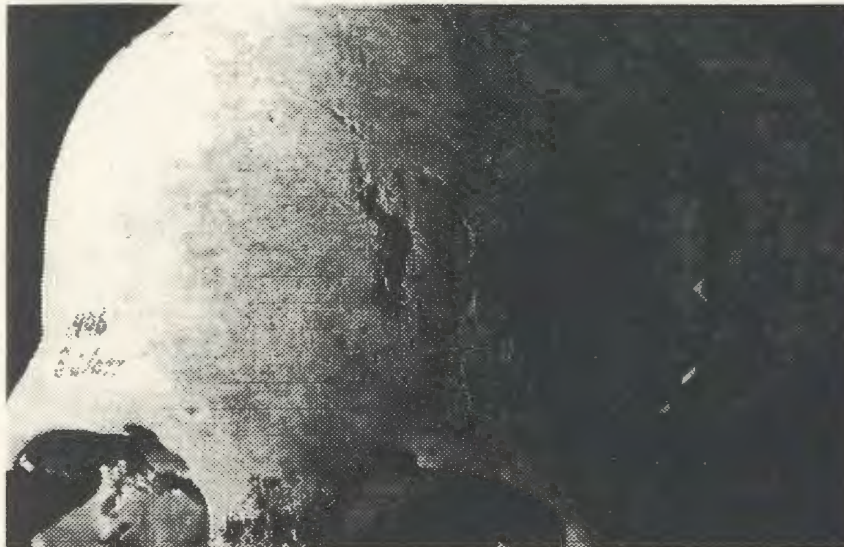
Plate 23: B-406
Healing Lytic Lesion of Left
Frontal: Female, 40 +/-5 years
of age.

Plate 24: B-406
Close up of Plate 23: B-406.
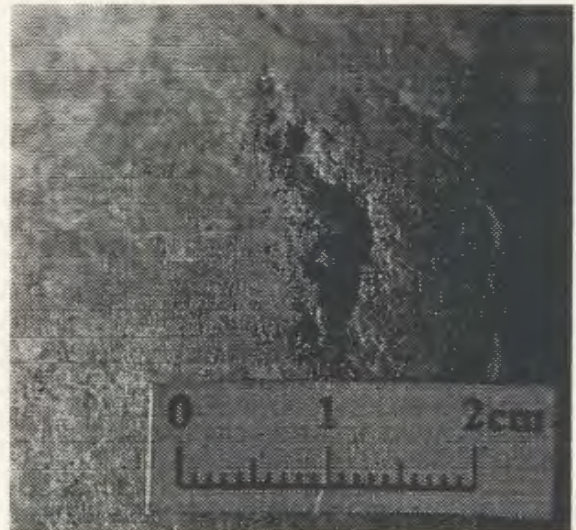
The right navicular has a lytic lesion on the plantar side approximately midway on

the bone that may be due to postmortem destruction. A section of spongy periostitis is

located on the left talus between the two calcaneal articular facets on the plantar side. This

section is active, but rather localized. The ventral side of the axis body has excessive bone

reaction resembling periostitis because of the spongy and porous nature of this area.

However, this could be attributable to degenerative arthritis. Extra bone growth, or osteophyte-like lesions/outgrowths are present in several cervical vertebrae, which extend along C3-5, but decreases in severity inferiorly (Plate 25) along the column. The appearance of these bones is almost like dripping 'candle wax' which is indicative of the pathology called flowing hyperostosis or melorrheostosis. This also possibly resembles actinomycosis (Ortner & Putschar 1997:220; Figure 350) or excessive degenerative arthritis. Some thoracic vertebrae also have the same excess of bone in this region, but postmortem processes have eroded the exterior of the centrum bone, thus making it difficult to see the whole extent of this pathology.
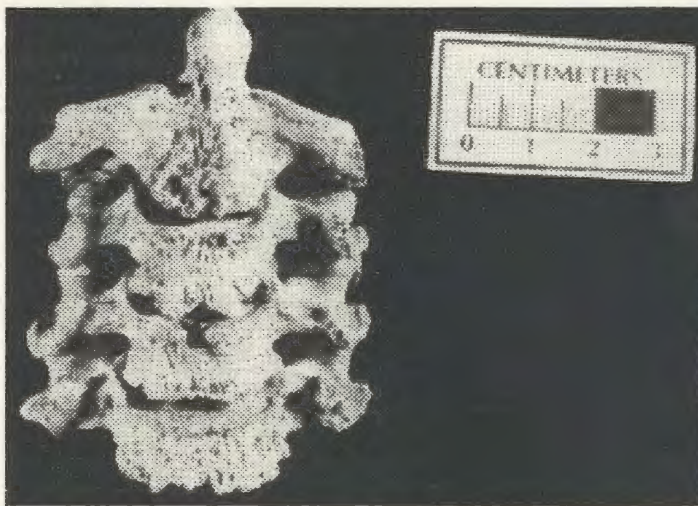
Plate 25: B-406
Actinomycosis, Degenerative Arthritis, Flowing Hyperostosis or Melorrheostosis of Vertebrae?: Female, 40 +/-5 years of age.

Many of the rib facets associated with these vertebrae have osteoarthritic lesions. Vertebrae C4 has a lytic lesion on the superior side of the centrum and the C7 spine has excess endosteal bone growth. Osteophytes are also noticeable on the internal neural arches of the thoracic and these increase in severity proceeding inferiorly along the spinal column. These bone alterations are not pathological, but are a factor of the ossification of the

ligament of flava that occurs with age. The superior articular processes around the facets of vertebrae T12 and the lumbar also have osteophytes. Most likely these osteophytes are a condition of aging and/or possibly a result of the pathological state of the cervicals.

Left rib #11 has striated and porous periostitis along the posterior side of the shaft, while an unknown numbered right rib (Plates 26 and 27) has some areas of spongy periostitis along the pleural side of the shaft. Due to the fact the bone is broken in one of the areas, an expansion in spongy bone is noticeable which has increased the thickness of the shaft (possibly a form of marrow hyperplasia).
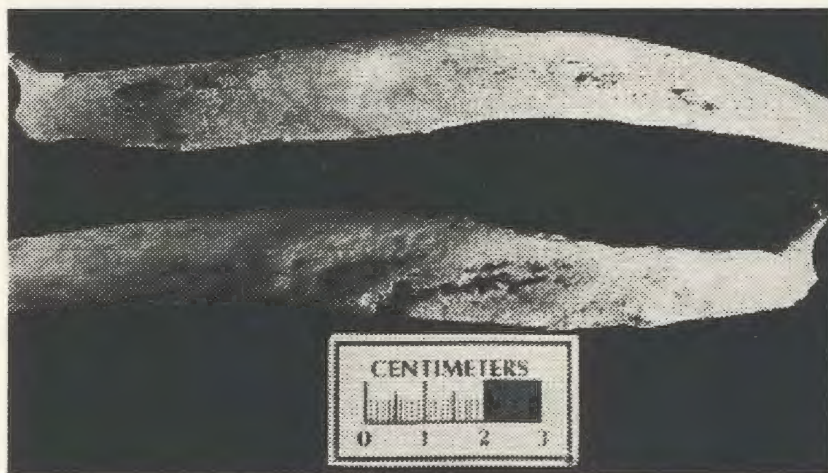
Plate 26: B-406
Periostitis of Ribs: Active and Porous. Upper bone in the picture is a right rib, while the lower bone is a left rib. Female, 40 +/-5 years of age.

Plate 27: B-406
Close up of Plate 26: B-406.

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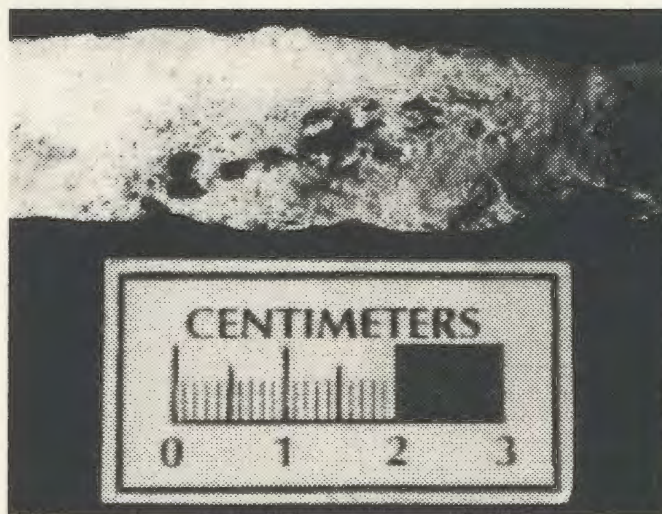
Two lower left ribs are engulfed, from just past the vertebral facets along the entire length and width of the shafts, with active spongy and porous periostitis and osteitis which have totally increased the thickness of the bone. Three lower right ribs exhibit similar pathologies to those mentioned above, but several large-sized foramina can be found within the periostitis that might be for drainage purposes.

The manubrium is also engulfed with periostitis and osteitis to such an extent that the bone is much thicker and larger as spicule-like smooth protrusions on the superior surface of the body. However, the inferior side of the manubrium does not appear to be similarly affected as the superior. Due to this pathology the manubrium feels extremely heavy when normally this bone is not. Because of the involvement of the manubrium, the clavicles are also affected. The clavicles are both increased in size due to extensive active periostitis that is spongy and porous (Plate 28). Many layers of appositional bone have been laid down and many large foramina were produced via lytic processes. However, it is possible that these lesions were caused by postmortem processes as the edges are rough and irregular. The right clavicle is more affected than the left as it starts at the sternal end and goes along the entire shaft. The scapula was not affected as there was no evidence of this pathology on the acromion process.

Except for the left humerus, radius and ulna, all of the long bones are affected with periostitis. A very porous and spongy section (32.34x11.36mm) is located on the right ulna just inferior to the supinator crest and another area is located just inferior to the coronoid process where the bone looks irregular and smooth. Unfortunately, the left radius and ulna
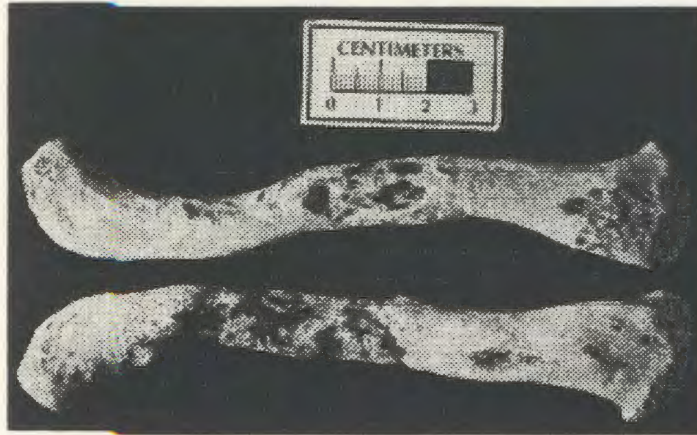
Plate 28: B-406
Periostitis of Clavicles: Active and Porous. Female, 40 +/-5 years of age.

have been affected by postmortem processes and are discoloured, which has not allowed for the evaluation of these two bones. The right humerus has a thick, spongy and porous section extending from the midshaft to the distal end of the posterior side of the shaft (110.4x43mm). Another area of periostitis can be found on the posterior lateral side of the epicondyle. It is categorized as a light, spongy layer in which some has been lost postmortem. Also, periostitis is located on the anterior medial side of the epicondyle. There is no evidence of this pathology on the left humerus, but the bone does feel light and may have been affected by osteoporosis. However, this bone has also been affected by postmortem processes.

The right femoral head appears porous, but may have been affected by postmortem means. Massive periostitis and osteitis circumscribes the distal end of this shaft and extends to the midshaft (135.38mm in length) (Plate 29). The areas vary in thickness and density. Also found in this region are large apertures and 'islands' of excess bone in which some areas are porous and others exhibit striations, especially near the extremities of the lesion. A spongy section can be found on the front of the shaft where it appears as a depression in

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the lesion which extends half way around the shaft. It is possible that something was pressing on this area inhibiting the growth of the pathological lesion.
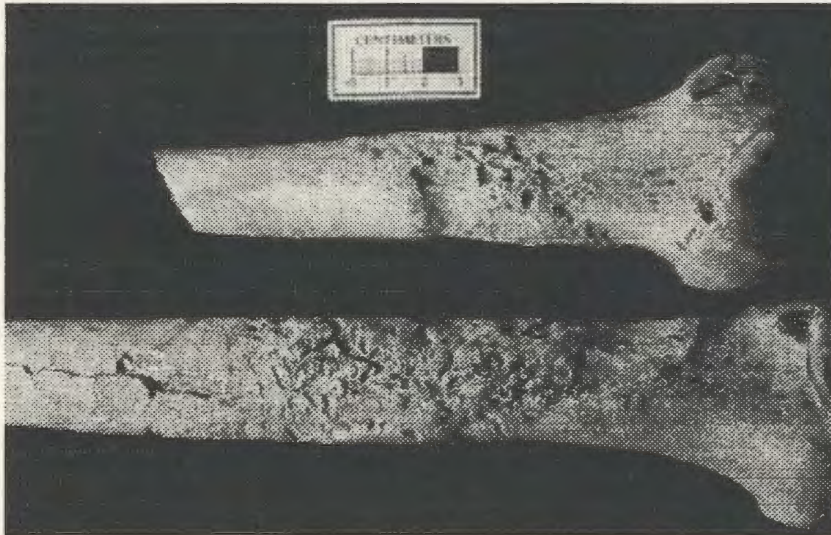
Plate 29: B-406
Periostitis and Osteitis of the Right (upper bone in picture) and Left (lower bone in picture) Femora: Active. Female, 40 +/-5 years of age.

The left femur is similar to the right, but the pathology engulfs more of the length of the shaft (~233mm) (Plates 29 and 30). Some areas of periostitis on this bone, especially near the proximal end, appear to be remodeled as the bone texture is smooth. Fortunately, postmortem processes have chipped a section of the periostitis and thus, the layering characteristic of this pathology can be observed. Also, this allowed for the thickness of the layer to be measured (3.22mm thick). There are a few areas where the bone is depressed, but no evidence of it extending into the medullary cavity is noticeable. This bone is very heavy due to the excessive dense, pathological appositional bone deposition.

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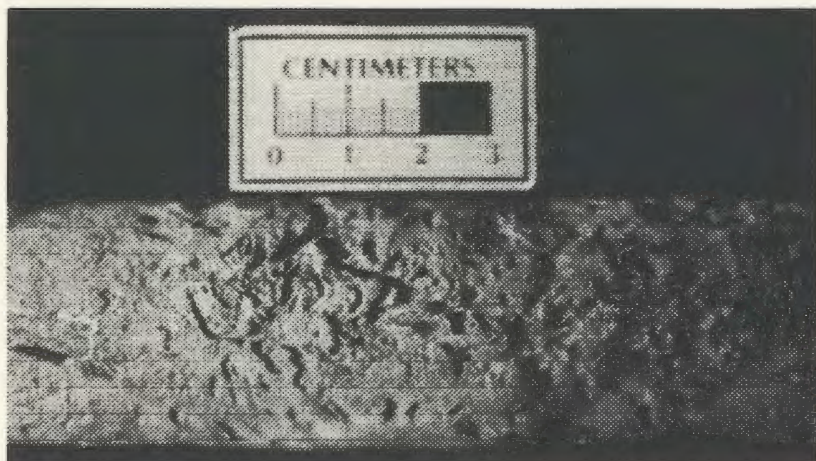
Plate 30: B-406
Close up of Periostitis and Osteitis of Left Femur: Female, 40 +/-5 years of age.

The right fibula has areas of increased thickness and thus, they may be possibly exhibiting osteitis or healed periostitis. The bone also appears to be very light and thus, may be affected by osteoporosis. The left fibula also has a large bulbous section (62.26mm in length) of periostitis located approximately in the mid to distal end of the shaft that circumscribes the shaft. This pathology is a mixture of thick striations and very spongy areas with large foramina. Due to the fact that the shaft is broken, it was possible to note that the cortex was considerably thickened and new layers of pathological bone could be seen.

There is a severe case of periostitis on the right tibia located at approximately the tibial tuberosity and extends to the distal one quarter of the shaft (212mm in length) (Plate 31). Therefore, this lesion covers approximately three-quarters of the shaft. The lesion circumscribes the entire shaft, but the posterior side is much more severe than the anterior. Some areas of this pathology look smooth and possibly remodeling, while others are spongy with thick and large striations, others are very porous while other areas are thicker and look

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like fibrous 'islands' of bone. There is a possible cloaca, or sequestrum, located on the medial side of the tibial tuberosity, but this may have been formed during postmortem. Approximately two-thirds of the left tibia is engulfed with periostitis, but it is not as severe as the right tibia. The pathology extends from about the tibial tuberosity to one-third before the distal end (180mm in length) and circumscribes the shaft. However, it is much more extensive on the medial and lateral sides. There are sections of smooth and remodeled periostitis along with areas of striations and porosities. Two cloacae are located lateral to the tibial tuberosity. One measures 7.22x3.5mm while the smaller is 3.76x1.76mm in area. Both of these cloacae reach the medullary cavity and spongy and porous periostitis is associated with them, while other areas have thin striations.
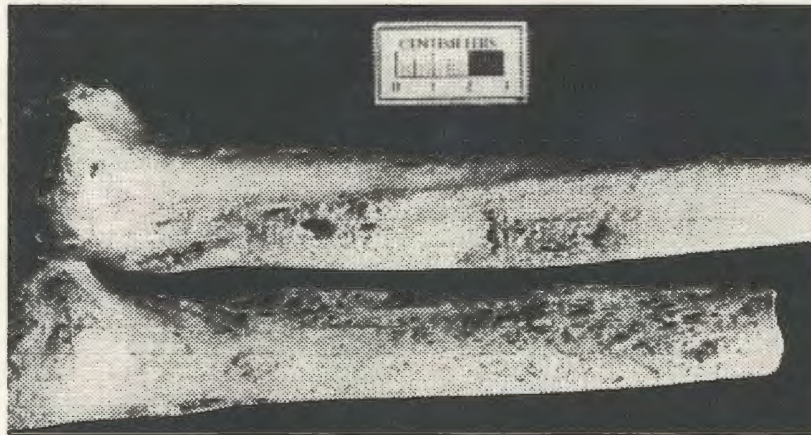
Plate 31: B-406
Periostitis of Left (upper bone in picture, lateral view) and Right (lower bone in picture, medial view) Tibiae: Severe Case. Female, 40 +/-5 years of age. Note presence of cloacae in both right and left shafts.

Burial # 411: Burial Number 411 was categorized as a female? individual approximately

30-34 years of age. Cribra orbitalia is presented bilaterally in a medium and healing manner, but only a very small section of the right orbit exists (L=24x7.78mm). The skull is very weathered and thus, it is very difficult to observe any evidence of porotic hyperostosis. However, some porosities are recognizable on the occipital and endocranially on both parietals. Antemortem tooth loss of the mandibular left and right M1's is recorded. The third and fourth cervicals are ankylosed posteriorly and on the left they are fused anteriorly.

Burial # 432: The individual associated with this burial was a male aged 30 +/- 5 years. Light and healing cribra orbitalia is bilaterally presented in both orbits (L=19.96x16.92mm; R=10.96x15.24mm) (similar to Plate 11: B-381). Light and healing porotic hyperostosis is also demonstrated on the frontal (56.94x53.9mm), the parietals around the confluence of the sagittal and lambdoidal sutures (L=67.9x49.22mm; R=67.1x38.72mm), and the occipital (38x40.38mm) (similar to Plate 10: B-379).

Superior-inferior condensation of vertebra T12, along with lateral-medial indentation of the lumbar are noted. The distal end of the right ulna for this individual illustrates a bulbous section of bone that could either be the result of a healed fracture, healed periostitis or an ossified hemorrhage. A radiograph is needed to verify this. Adjacent to the right ulna, the right radius presents healed periostitis near the interosseous crest.

Burial # 435: Burial Number 435 was a subadult individual approximately 12 +/- 6 months of age. Due to the fact that only the left orbit is present, cribra orbitalia is only observed for this orbit. It is severe and active (similar to Plate 32: B-442) to such an extent that it extends outward from the orbital wall and measures 17.49x21mm in area. Both the left and right

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parietals reveal no signs of porotic hyperostosis. However, endocranially there appears to be a modification of the inner table as it looks 'wormy' whereby many striations and pores cover a large section of the parietals and thickening of the cranium is evident (similar to Burial #442, but not as severe: see Plate 35: B-442). The sphenoid is porous just anterior to the temporal bone. The maxilla presents osteitis on both the right and left sides superior and posterior to PM1. A small section of periostitis can also be found just posterior to the molar crypt on the right side of the mandible. Periostitis and increased porosities are also evident on the frontal medial side of both the zygomatic bones.

Burial # 442: The individual associated with this burial was a subadult aged 18 +/- 6 months. Cribra orbitalia is bilaterally evident in both orbits as very severe and active and covers most of the orbital walls (L=29.74x27.26mm; R=29.36x23.66mm) (Plates 32 and 33). However, the medial aspect of the left orbital wall is broken and thus a more extensive involvement could have been observed.
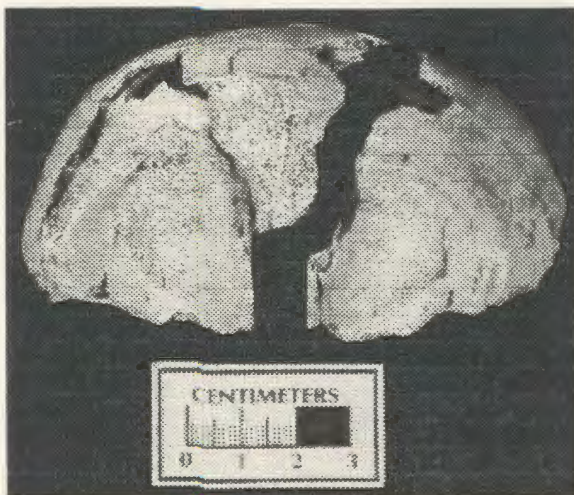
Plate 32: B-442
Cribra Orbitalia: Severe and Active. Subadult, 18 +/- 6 months of age.

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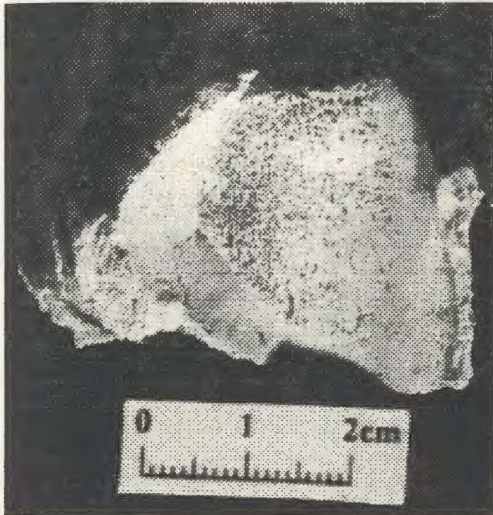
Plate 33: B-442
Close up of Plate 33: B-442.

Porotic hyperostosis is very severe and active on both the parietals centered around the lambdoid (Plate 34). Portions of the right parietal are missing, but the lesion on one of the fragments measures 28.88x16.2mm in area. The left parietal is also in pieces, but a measurement of 13.38x23.66mm for the lesion is produced.
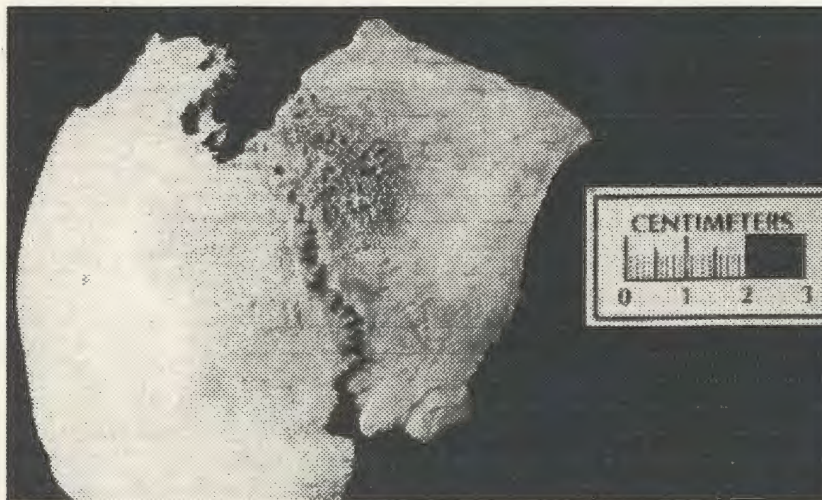
Plate 34: B-442
Porotic Hyperostosis: Active and Severe. Subadult, 18 +/- 6 months of age.

The inner table of the parietals and some of the frontal is completely nonexistent and replaced by striated, wormy and porous trabecular outgrowth of bone (Plate 35; similar to previous Burial #435).
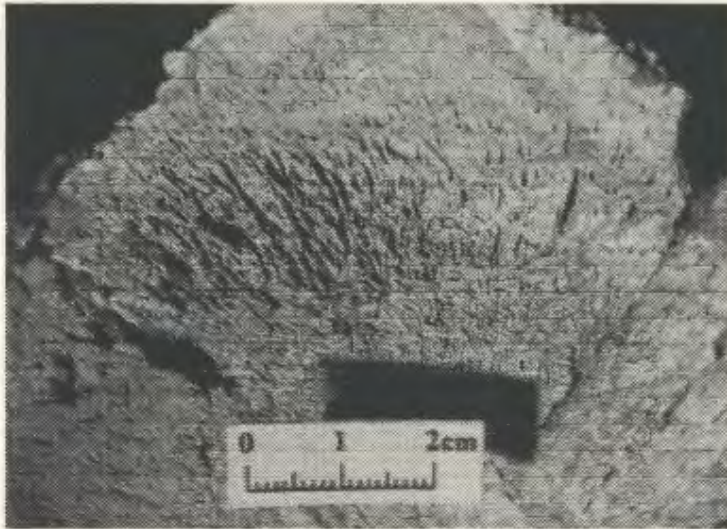
Plate 35: B-442
Trabecular Outgrowth of Inner Table of Parietals: Subadult, 18 +/- 6 months of age.

Both the left and right temporal bones include more porosities than normally expected. Severe osteitis and porosities are illustrated on the left maxilla alveolar bone mostly centered around the infraorbital foramen (Plate 36) and on the posterior section just superior to the last PM crypt (Plate 37).
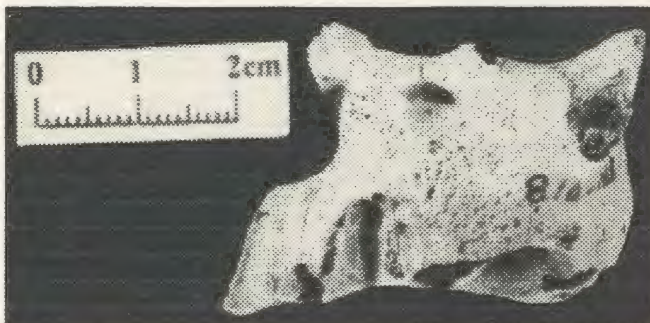
Plate 36: B-442
Osteitis and Hyperporosities of the Left Maxilla Alveolus, Centered Around the Infraorbital Foramen: Subadult, 18 +/- 6 months of age.

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Plate 37: B-442
Osteitis and Hyperporosities of the Left Posterior Maxilla Alveolus, Superior to the Final PM Crypt. Subadult, 18 +/- 6 months of age.
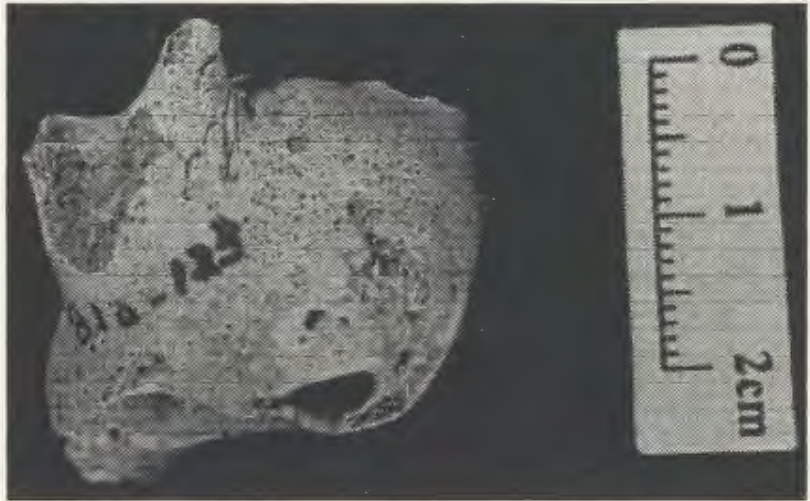
These same two pathologies are also demonstrated along both the right and left mandibular bodies, especially localized inferior to the mental foramen (Plate 38). The anterior sides of both the left and right greater wing of the sphenoid are extremely porous.
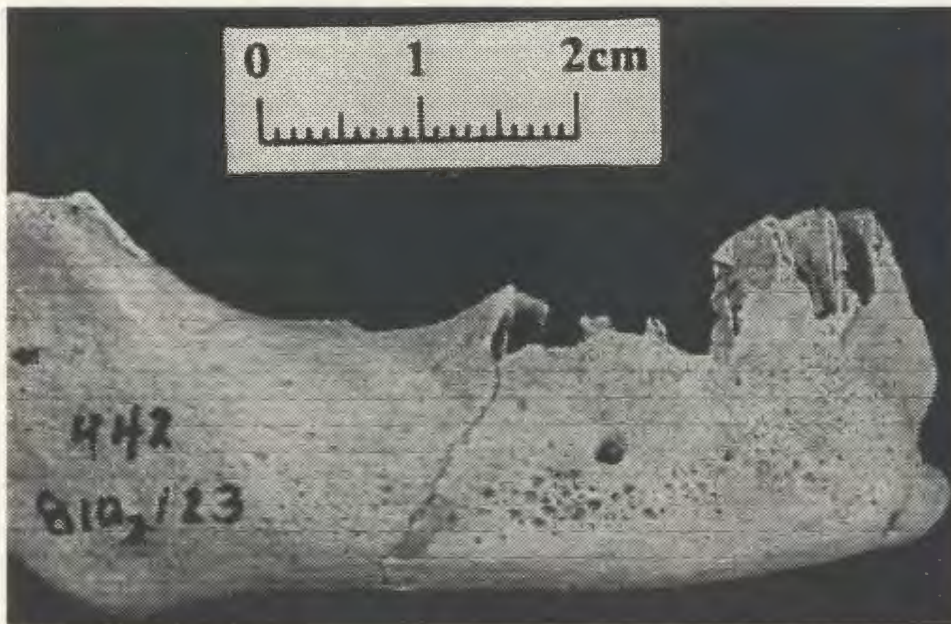
Plate 38: B-442 Osteitis of the Right Mandibular Body: Subadult, 18 +/- 6 months of age.

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Increased porosities are also acknowledged on the transverse processes of several vertebrae. The dorsal side of a left rib shaft possesses an area of depression in the bone whereby the medullary cavity is exposed and the margins of the pathology are smooth, thus suggesting the presence of necrotic action at some point antemortem.

Burial # 443: This burial presents a male? specimen approximately 17 +/- 2 years of age. Active and severe cribra orbitalia (similar to Plate 32: B-442), especially on the left orbit (L=14.72x8.6mm; R=26.24x9.96mm) is noted bilaterally. Light and active porotic hyperostosis is localized around the coronal and sagittal confluence (frontal: 23.62x43.76mm; parietals: L=40.62x21.56mm; R=64.24x36.04mm) (Plate 39).
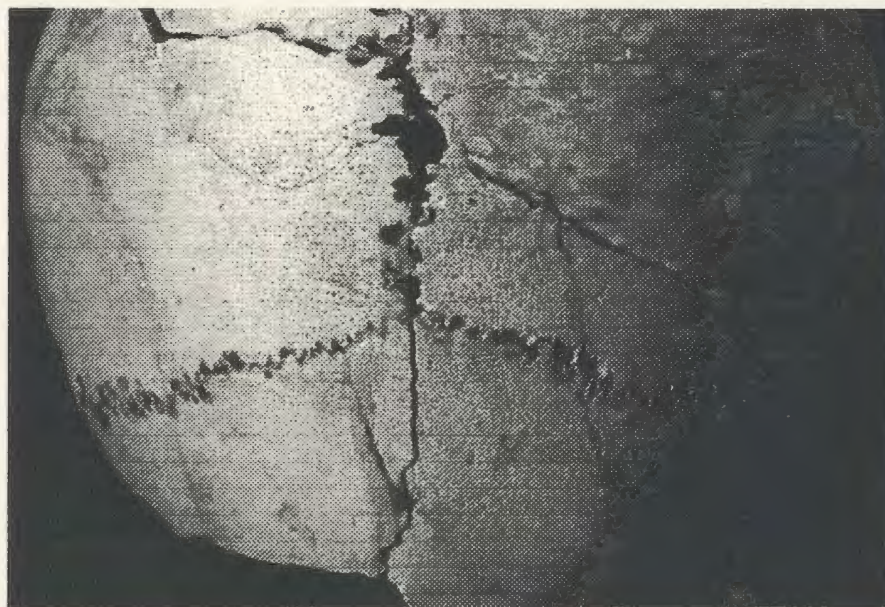
Plate 39: B-443
Porotic Hyperostosis: Active and Light. Possible Male Specimen, 17 +/- 2 years of age.

Lateral-medial indentation of the lumbar vertebrae is apparent (Plate 40).

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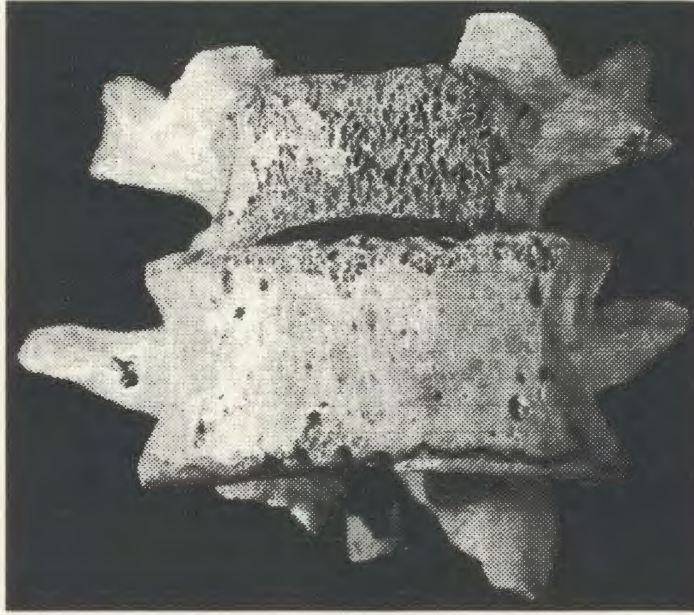
Plate 40: B-443
Lateral-Medial Indentation of Lumbar Vertebrae: Possible Male Specimen, 17 +/- 2 years of age.

The right posterior maxilla includes an area of increased porosity and unfortunately the most dense area is altered by postmortem processes (Plate 41).

Burial # 447: The individual from this burial was a subadult of 6 +/- 2 years of age. A large proportion of the right orbit is missing, but the left presents a mixture of small and large porosities, thus exhibiting medium and active cribra orbitalia (L=28.46x16.86mm) (similar to Plate 42: B-464). Light porotic hyperostosis is isolated on the occipital near the lambdoid suture (24.4x52.26mm) and the left parietal near the lambdoid suture (51.24x10.24mm). It is interesting to note that porotic hyperostosis is not observed bilaterally on the parietals as is usually the case. Therefore, it is possible that some other etiology may be causing this pathology. An increase in perforations can be observed on the left temporal bone and on the sphenoids. Osteitis is centered along the alveolar bone of both the maxilla and the mandible. A light section of periostitis can be found on a fibular shaft of unknown side. A possible

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necrotic lesion can be seen on a right rib and the medullary cavity is exposed with the margins being relatively smooth.

Plate 41: B-443
Hyperporosity of Right Posterior Maxilla: Possible Male Specimen, 17 +/- 2 years of age. This may be a factor of scurvy whereby the upper head of the lateral pterygoid muscle has undergone repeated hemorrhaging and dislocation/separation from the bone.

Burial # 464: Burial Number 464 is a female 40 +/- 5 years of age. Cribra orbitalia is medium and active in both orbits (R=8.96x16.5mm; L=9.96x22.5mm) (Plate 42).
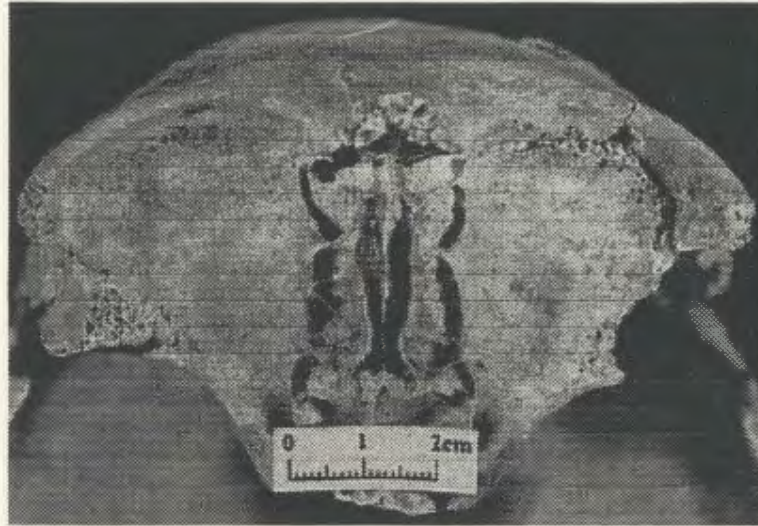
Plate 42: B-464
Cribra Orbitalia: Active and Medium. Female, 40 +/- 5 years of age.

Light porotic hyperostosis is presented on the frontal (25.26x27.92mm) bone around the coronal suture and the parietals by the coronal and sagittal confluence (L=38.1x15.14mm; R=35.14x19.06mm). Marginal lipping of vertebra L4 is observed, along with a small deposition of subperiosteal bone on the frontal wall of the centrum. Osteophytes are also recognized on the internal neural arches of the thoracic. Osteoarthritis is found on several vertebral ends of left and right ribs as well as rib facets on several vertebrae and the superior facets of the atlas for the occipital. Marginal lipping is also present on the left lunate and scaphoid.

Burial # 475: This burial presents a male approximately 45 +/- 5 years of age. Poor preservation of this skeleton has limited the amount of information available. No evidence of cribra orbitalia can be noticed, but light and healing sections of porotic hyperostosis are

present (similar to Plate 10: B-379). Due to the fact that the sagittal suture is completely fused, only one measurement (80.44x77mm) of the lesion was taken from the combined parietals. The occipital bone also exhibits porotic hyperostosis (37.5x72.76mm). It is quite possible that these areas of hyperporosities are a factor of aging due to the fact that most times cribra orbitalia and porotic hyperostosis usually occur in tandem. Other pathologies consistent with aging present on this individual are an osteophyte protrusion on the dens of the atlas, marginal lipping on vertebrae L3 and 5, osteoarthritis on L5 and on the proximal radius facet. Circular lesions are found on the inferior and superior surfaces of two lumbar vertebral centra. Antemortem loss of the mandibular left and right M1-3 teeth and the maxillary right PM1 and 2 are evident. An abscess/fistula is also noted in the region of the left canine of the maxilla.

Burial # 496: Burial Number 496 was a female individual approximately 50+ years of age. Cribra orbitalia is found bilaterally in a medium and active manner (L=8.8x11.1mm; R=10.62x11.98) (similar to Plate 42: B-464). Light and healing porotic hyperostosis is located on the parietals (L=62.12x56.7mm; R=61.7x48.92mm) and on the occipital bone (40.4x43.14mm) (similar to Plate 10: B-379).

Several different pathologies normally associated with age are also noticeable on this individual. For example, lipping at the proximal end of the left proximal tarsal phalanx #1, marginal lipping on the vertebral ends of the left ribs from #2-9, osteoarthritis at the vertebral end of right rib #10, massive osteoarthritis and marginal lipping throughout the vertebral column, especially C5-7, T9-12 and all of the lumbar, osteoarthritis on both the left

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and right clavicles at the acromion end, and the left proximal humerus and vertebra T10 exhibits frontal compression of the centrum. The thoracic vertebrae 7 and 8 are ankylosed on the left side of the neural arch. The mandible is completely edentulous, while in the maxilla only the right PM2 and possibly the left PM2 remain. Periostitis and osteitis are both found on the right femoral neck on the ventral side.

Burial # 500: This burial was a male individual around 50+ years of age. Cribra orbitalia is healing and light and can be found bilaterally (R=11.34x12.54mm) (similar to Plate 11: B-381). Light and healing porotic hyperostosis (similar to Plate 10: B-379) is present on both the parietals (L=80.18x46.3mm; R=108.26x49.3mm), the occipital (53.3x46.56mm), which has several slightly larger apertures and the frontal bone (40.42x67.02mm). Antemortem loss of the mandibular right and left molars and maxillary left PM1 and molars, the right PM's and molars, as well as abscessing of the left C and PM2 are found. Possibly attributable to this is the fact that massive attrition is noted on all of the remaining teeth.

Once again, pathologies associated with aging are found on this individual: right carpal distal phalanx has osteophytes on the medial and lateral sides of the proximal end of the bone, osteophytes on the right trapezoid and the neural arches of the vertebral column, lipping in the glenoid fossa of both scapulae, and osteoarthritis on the proximal end of an unknown sided tibia. Lytic holes are present on the superior side of C7 near the margin of the centrum (Plate 43), several other cervical vertebrae and the inferior side of a thoracic vertebra have margins that are relatively smooth and exposure of cancellous bone is noted within these lesions. Poor preservation may have accentuated the possible case of superior-

inferior condensation or a collapsed lumbar centrum.

Plate 43: B-500
Lytic Lesions of Cervical #7 Vertebra: Male, 50+ years of age.

Several button osteomata that are smooth and shiny can be found on the pleural side of several right rib shafts, which may be attributable to the presence of a respiratory disease. Periostitis (67.76mm in length) can be seen on the midshaft of the anterior crest of the right tibia, healed sections at the distal end of the right fibula (small section=22.18mm; large section=51.88mm) and at the distal end of the broken left fibula, and an active striated region, with some healing of the shaft, at the distal end of the left ulna. Two areas of possible necrotic bone (12.36mm and 10.26mm) are located on the midshaft of the anterior crest of the right tibia, which are partly associated with the areas of periostitis.

Burial # 552: Burial Number 552 represents a subadult aged 2.5 years +/- 1 year. Cribra orbitalia is presented bilaterally in a light and healing manner, but some postmortem destruction has occurred on the left anterior wall of the orbit, therefore a smaller measurement is obtained (L=7.64x16.28mm; 7.98x15.14mm) (similar to Plate 11: B-381).

Burial # 560: The individual in this burial was a female? of 45 +/- 5 years of age. Cribra

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orbitalia is bilaterally presented as active and medium (similar to Plate 42: B-464), but both orbits have been broken posteriorly, therefore only widths of the lesions could be measured (L=24.84mm; R=9.24mm). Porotic hyperostosis is found as light and healing (similar to Plate 10: B-379) on the frontal bone, especially around the sagittal and coronal confluence (29.2x53.42mm), the parietals near the sagittal suture (L=51.26x33.54mm; R=61.92x41mm) and the occipital around the lambdoidal suture with slightly larger perforations than the rest of the cranium (13.8x23.28mm).

This individual also had a healed traumatic blow, possibly a pressure fracture, to the left parietal and temporal bones in which the skull in this area is depressed and smoothing of the margins has occurred. All mandibular molars have been lost antemortem and a lytic spongy lesion exists on the right condyle, but does not exist on the respective temporal mandibular joint. Periostitis can be found on the pleural side at the vertebral end of two right ribs. This may be the result of a respiratory infection or a localized infection of some other kind.

Marginal osteophytes are present on several lumbar vertebrae (L3 and 4 on the left side; and L1 and 2 on the ventral right and left sides). Osteophytes are also found on two medial tarsal phalanges at their distal ends. Several ribs have extraneous bone growth on their inferior side of the shafts, as well as the presence of button osteoma. Many of these pathologies are probably factors of the aging process.

Burial # 612: This burial had an infant about the age of 9 +/- 3 months. Cribra orbitalia is active but light in both orbits (L=8.2x16.72mm; R=8.38x13.48mm) (similar to Plate 5: B-

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310). Medium and active porotic hyperostosis (similar to Plate 1: B-236) is noted on the occipital, however it is in too many pieces to accurately measure the extent. It is unusual that the cranial lesions are more severe than the orbital, but this could be a factor of preservation or different etiologies. However, the right and left temporal bones, the right petrous portion of the temporal and the sphenoid are extremely spongy and porotic. Two lower right ribs are fractured which have not begun to heal yet as no closure is noticeable. Periostitis is associated with the fractured region. Spongy periostitis is found along the medial sides of both the left and right tibial shafts and also on the lateral side of the distal end of the right humerus.

Burial # 638: This individual was an infant approximately 2.5 years +/- 1 year. Cribra orbitalia is light and active in both orbits (similar to Plate 5: B-310), but both orbits are broken and thus the lesions could be more extensive (L=8.64x18.52mm; R=13.02x18.62). No porotic hyperostosis is observed on this individual. A lytic hole is found on both the right and left temporal bone and also on the external wall of the external auditory meatus. A lytic lesion is also located on the neural arch of the atlas and between the transverse process and superior articular processes of a thoracic whereby spicules and remodeling of the edges have occurred. Two mandibular incisors share the same root presenting a dental irregularity that may be associated with scurvy. It is also possible that this dental irregularity may be related to either genetic or developmental determinants. Periostitis is illustrated on the right side of the maxilla just posterior to the last premolar, as well as superior to this tooth on the alveolus.

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Burial # 639: Burial Number 639 was an infant approximately 18 +/- 6 months of age. Cribra orbitalia could only be observed on the right orbit because the left is absent. However, the right orbit only presents the most superior portion, i.e., medial section that is closest to the nasal spine, which is considered to be of light severity. Active or healing characteristics could not be determined. Periostitis is found on the right femur approximately midshaft on the dorsal side (22x5.86mm), as well as the pleural side of four left ribs (#2–4 and one lower down). The lumbar neural arches are also very porous.

Burial # 663: Burial Number 663 was a subadult approximately 8.5 years +/- 1 year of age. Cribra orbitalia is observed as active and medium in what remains of the left and right orbits (similar to Plate 42: B-464). Porotic hyperostosis is active and medium along the sagittal and lambdoidal sutures of both parietals (L=67.06x29.53mm; R=71.41x20.74mm) (similar to Plate 1: B-236) and is present in a lighter manner on the occipital bone around the lambdoid suture (69.59x19.41mm). Increased areas of porosities can be found around the juncture of the left sphenoid and the temporal bones. Osteitis is demonstrated on both the left and right alveolar bone around the nasal aperture of the maxilla and also on the left body of the mandible just inferior to PM1 and 2. Reactive bone is also found on the maxilla around the socket of the right PM2.

Striated periostitis is located on the shafts of both the ulna and the radius, striations and pores are also located on the medial side of the right tibial shaft, which is also found in a less extensive manner on the left tibia in the same position, and striations and porosities can be seen covering the majority of the mid to distal end of the right and left fibulae. The

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right fibular shaft is broken which allows for the observation of the spongy appositional growth characteristic of periostitis and the lack of an outer cortex on the posterior side of the shaft. Patches of spongy periostitis are also found on the endocranial surface of the frontal bone in several of the indentations and just superior to the left PM2 on the maxilla. Deposition of extraneous bone 'bumps' of lighter colouration are observed on the inferior neural arches of T3 and 4 that almost resemble button osteoma.

Burial # 667: This burial was a subadult approximately 13 years +/- 1 year of age. Only the left orbit is present and thus only cribra orbitalia could be acknowledged for this orbit. However, cribra orbitalia is usually presented bilaterally and thus, we can assume that it would have been present on the right orbit as well. Cribra orbitalia in the left orbit is light and active and measures 6.65x12.91mm in area (similar to Plate 5: B-310). No porotic hyperostosis is observed on the calvarium. The right and left sphenoid bones are covered with many porosities that harbour spicules within them. The teeth are carious, and in consequence, an abscess is located on the right maxillary I1 and 2 root crypts that extends to the lateral side of the palatine process. The bone found inside the abscess is reactive in nature. A lytic lesion is located on the lateral condyle of the left femur epiphysis whereby discolouration of the spongy bone has occurred. This may indicate that the lesion was produced in the postmortem environment. The distal left femur metaphysis has a hole on the lateral side which could be connected with the epiphysis just previously mentioned.

Burial # 669: Burial Number 669 was a male individual approximately 50+ years of age. There is no cribra orbitalia in this individual. Only light porosities are seen on the frontal,

parietals and occipital. However, an increase in diploë is manifested (similar to Plate 7: B-357). Periostitis and osteitis (26.5x5.26mm) are found on the right fibular shaft at the distal end on the medial side and is presented in a striated and active form. Also, porosities and striations on the distal end of the left fibula shaft just opposite to the crest can be seen.

Many age-related pathologies are also exhibited on this individual in the form of osteoarthritis on the medial condyle surface of the left tibia, the distal end of the left ulnar projection and at the acromion ends of the right and left clavicles. Lipping of bone can also be seen on all carpal phalanges (proximal, distal and shafts), distal ends and inferior sides of the right and left metatarsals #1, the left calcaneus body and the proximal end of the right fibula. Osteoarthritic pitting and marginal lipping are also located on the cervicals and thoracic vertebrae, the dens and the atlas facet. Antemortem loss of the right mandibular PM2 and M1-3 teeth is also observed.

Burial # 689: The individual from Burial Number 689 was a subadult aged 24 +/- 12 months. Cribra orbitalia is demonstrated as being medium and active in both orbits (similar to Plate 42: B-464). However, both of the orbits are broken which prohibits any measurements. No porotic hyperostosis is observed. Although, the frontal bone has several raised areas with porosities and striations and an increase in diploë associated with it. Perhaps this is a variant of the hyperostosis, or just a completely different pathology all together.

Several areas of periostitis are demonstrated on several different bones: the distal end of the dorsal lateral side of the right humerus, the central area of the frontal bone just

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posterior to the nasal spine and the right and left ulnae and radii shafts at their medial distal ends. Some areas of periostitis are broken off on these two latter shafts, which has allowed for the observation of the multiple layering often associated with this pathology and the original shaft is visible. Both the right and left humeri at their distal frontal shaft areas are thickened with some pores, which is distinctive of osteomyelitis and osteitis. Multiple layering, as in concentric rings, is noticeable on both the right and left tibiae and fibulae shafts to such an extent that the shafts have been distorted from their original shape (similar to Burial 442 and 435). However, this alteration could have been produced via postmortem processes.

Burial # 774: Burial Number 774 was an infant aged approximately 0-6 months of age. Cribra orbitalia is light, but active and bilaterally demonstrated (L=19.76x15.32mm; R=19.04x15.94mm) (similar to Plate 5: B-310). Porotic hyperostosis is active and medium (similar to Plate 1: B-236) on both of the parietals situated close to the squamosal suture (R=14.7x12.54mm; L=15.12x11.52mm) and another region is located closer to the lambdoid suture (R=35.2x17.88mm; L=23.42x13.76mm). Hyperporosity and very 'spongy' bone is observed on all of the cranial bones, especially along the squamosal and lambdoid sutures on the parietals and around the periphery of the occipital bone, the left and right temporal bones, the left and right internal posterior sides of the mandible and the left posterior region of the scapular body. All of these areas of hyperporosities may be the result of the postmortem environment, or the affects of the age of this individual and the malleable nature of neonatal bone development (or perhaps both of these). Periosteal reaction (periostitis)

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is found on the right tibia on the medial side of the shaft. A possible necrotic area of bone is noted on the neural arch of a thoracic vertebra.

Burial # 833: Burial Number 833 was an infant aged 18 +/- 12 months. Cribra orbitalia is presented as light, but active in both orbits (R=6.69x16.9mm; L=10.3x16.58mm) (similar to Plate 5: B-310). The preservation of this cranium is very poor, which has hindered any observation of porotic hyperostosis. Periostitis can be found on two left ribs on their dorsal sides whereby one is near the sternal end and the other is located by the vertebral end. Periostitis is also located at the proximal end of the left fibula shaft that runs to the midshaft, the medial sides of the left and right tibial shafts with it being more extensive on the right than the left, on the interior side of the mandible on the coronoid process, the frontal side of the left zygomatic and the dorsal side of the maxilla just posterior to the premolar sockets. The mandible demonstrates osteitis, especially centered around the mental symphysis. This pathology is also found on the maxilla by the nasal aperture. The palatine bone is very porous which could be due to several factors, such as being a normal variant, a condition associated with scurvy, or postmortem destruction.

Burial # 895: The individual from this burial was a subadult approximately 12 +/- 2.5 years of age. Cribra orbitalia is bilaterally presented as active and severe (L=14.96x22.5mm; R=12.18x22.52mm) (similar to Plate 32: B-442). Active and medium porotic hyperostosis (similar to Plate 1: B-236) is located on the parietals centered around the lambdoid and sagittal confluence (R=39.51x30.59mm; L=27.18x29.11mm). Hyperporosities are located on the maxillary bone just dorsal to the zygomatic and maxilla suture, on the left and right

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sphenoid bones ectocranially and on the temporal bones, especially by the zygomatic process and toward the sphenoid bone. A section of periostitis is found on the right maxilla just posterior to the molars and also on the left mandible superior to the mandibular foramen and anterior to the coronoid process. It appears that the sacral canal has not fused yet or this individual has Spina bifida.

Burial # 934: This burial was an infant approximately 0-6 months of age. Active and severe cribra orbitalia is presented in both orbits (similar to Plate 32: B-442), but there is only enough of the right to measure (11.72x19.27mm). Possible porotic hyperostosis is exhibited on the endocranial side of the frontal bone by extreme spongy bone and porosities, along the sutures of the parietals and around the lambdoidal suture of the occipital. The mandible is extremely spongy on the inferior side of the body around the mental symphysis. The dorsal sides of the left and right scapulae, the dorsal side of all ribs and the dorsal side of the right tibia are very porous and spongy. Porosities are also visible on some of the thoracic neural arches. These pathologies or bone alterations could be the result of age and bone development and/or the postmortem environment affecting the bone. Most of the lower ribs at the vertebral end on the ventral side show multiple foramina that could be lytic lesions.

These descriptions of the manifestations of skeletal alterations observed in the Dickson Mounds Mississippian Period sample provides the reader with an idea of those pathologies present. By illustrating and describing these lesions, a better understanding has been attained. The 40 burials outlined here are the prime sources of data used to discern percentages and relationships which are located in the proceeding chapter.

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CHAPTER 6: RESULTS

Observation of the Dickson Mounds skeletal population from the Mississippian Period has provided a wealth of information pertaining to the demographic profile and the presence of pathological lesions attributable to nutritional anaemias. From an overall population profile, to the larger population broken down into different cohorts, trends in the effects of different variables for the manifestation of nutritional anaemia were apparent. The results provide evidence of the interrelationships among several variables that is important to the skeletal alterations previously outlined. These factors may include age, sex and/or the presence of other bony manifestations observed on individual skeletons from Dickson Mounds.

6.1 AGE AND SEX

Of the 110 skeletal remains analysed for this study, an approximate equal ratio of males to females was presented: 20% (N=22) to 24.55% (N=27), respectively. The rest of the population consists of 3.64% Male?, 4.55% Female? and 47.27% No Gender or Subadults. Therefore, almost half of the population analysed for this study were subadults.

When the age distribution is calculated, approximately 30% of the population was less than 2 years of age. This corresponds with a high infant mortality rate that is well known for this time period. The next largest proportion involves the age group of 50+ (10.91%). Table 3 presents the age distribution in its entirety.

Table 3: Age Distribution for Dickson Mounds

Age Cohorts(years)	Number	Percent
less than 2	33	30.00
2 to 5	9	8.18
6 to 10	6	5.45
11 to 15	5	4.55
16 to 20	6	5.45
20 +/- 5	9	8.18
25 +/- 5	2	1.82
30 +/- 5	9	8.18
35 +/- 5	7	6.36
40 +/- 5	6	5.45
45 +/- 5	6	5.45
50+	12	10.91
TOTAL	110	100.00

The sex distribution for those individuals above the age of 16 is presented in Graph 2 below.

This graph illustrates that more males lived longer (16.33%), i.e., the 50+ age category,

while more females died younger (28.56%), i.e., from ages 16-30 +/- 5 years.

Graph 2: Age Distribution by Sex for the Dickson Mounds Collection
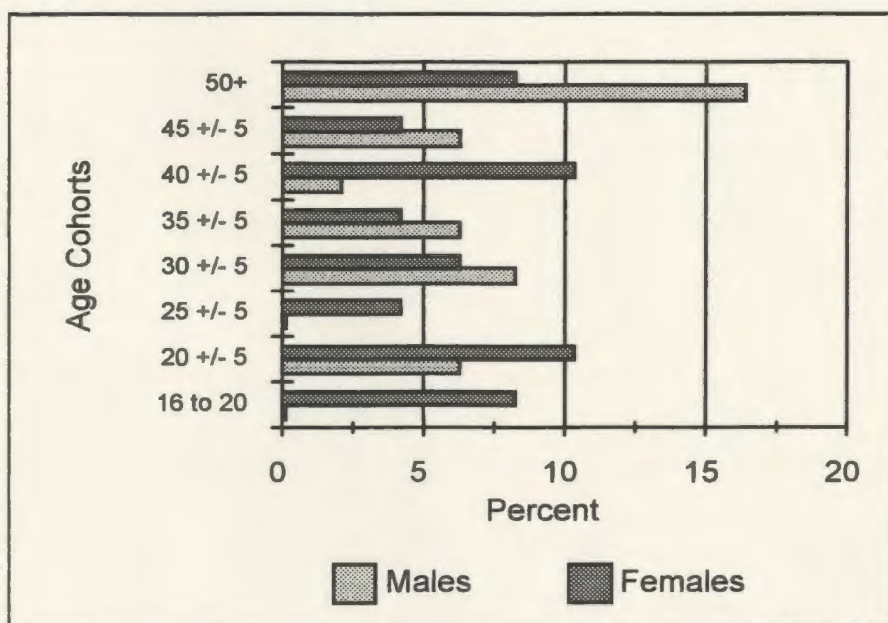
6.2 CRIBRA CRANII POPULATION

Of the 110 individuals observed, only thirty-nine (35.45%) were diagnosed with some form of cribra orbitalia, while one individual (0.91%) had porotic hyperostosis with no cribra orbitalia. The sex distribution for those with cribra orbitalia is presented below in Table 4. This information reveals that an approximate equal distribution exists between males and females (7:10), as no statistically significant difference existed when the two sexes were compared by Chi-Square analysis.

Table 4: <u>Sex Distribution of Those With Cribra Orbitalia</u>

Sex	Number	Percent
Male	7	17.95
Female	10	25.64
Subadult	19	48.72
Male?	1	2.56
Female?	2	5.13
TOTAL	**39**	**100**

These data also illustrate that almost half of this segment of the population were composed of subadults (48.72%). When the information is broken down by age cohorts, this same trend is noted. However, a large percentage is below the age of 2 and between 2 and 5 years, 25.64% and 12.82%, respectively (refer to Table 5 and Graph 3). The next age cohort with a relatively large percentage is that above the age of 50 years (12.82%).

Table 5: <u>Age Distribution of Those With Cribra Orbitalia</u>

Age	Number	Percent
less than 2	10	25.64
2 to 5	5	12.82
6 to 10	2	5.13
11 to 15	2	5.13
16 to 20	3	7.69
20 +/- 5	2	5.13
25 +/- 5	1	2.56
30 +/- 5	2	5.13
35 +/- 5	2	5.13
40 +/- 5	3	7.69
45 +/- 5	2	5.13
50+	5	12.82
TOTAL	39	100

Graph 3: <u>Age Distribution of Those With Cribra Orbitalia</u>
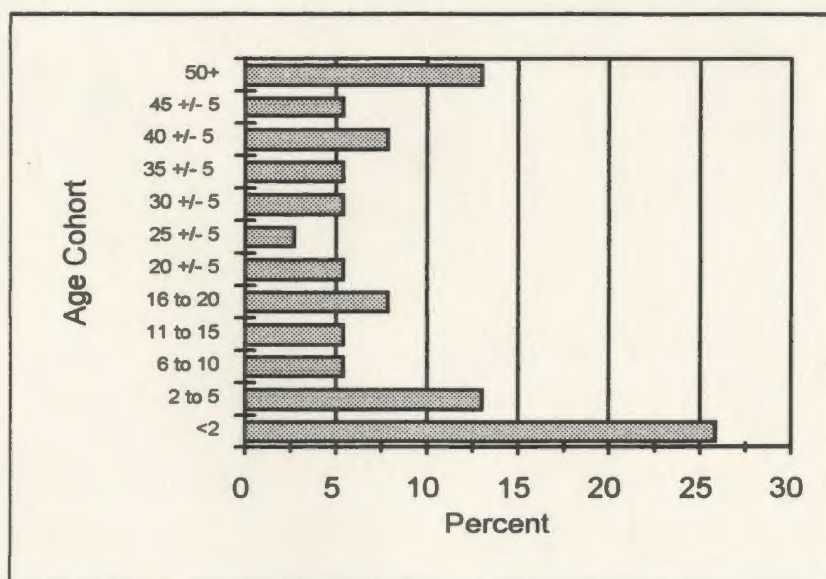
When these data are broken down to include information about age and sex distributions,

a pattern emerges. It appears that more females at younger ages, i.e., 16-25 +/- 5 years, have

cribra orbitalia while males at older ages, i.e., 45+, are diagnosed with these lesions (Graph

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4). This difference in age distribution is significant ($X^2 = 5.517$, $p < 0.02$) when the population is divided into age cohorts of 16-25 and 30-50+ years of age for males and females. This function was executed in order to see which sex at which age would more predominantly exhibit manifestations of these lesions. Therefore, these results reveal that females of childbearing age and elderly males were more susceptible to developing cribra orbitalia than any other sexed and aged cohort.

Graph 4: <u>Age and Sex Distribution for Those With Cribra Orbitalia</u>
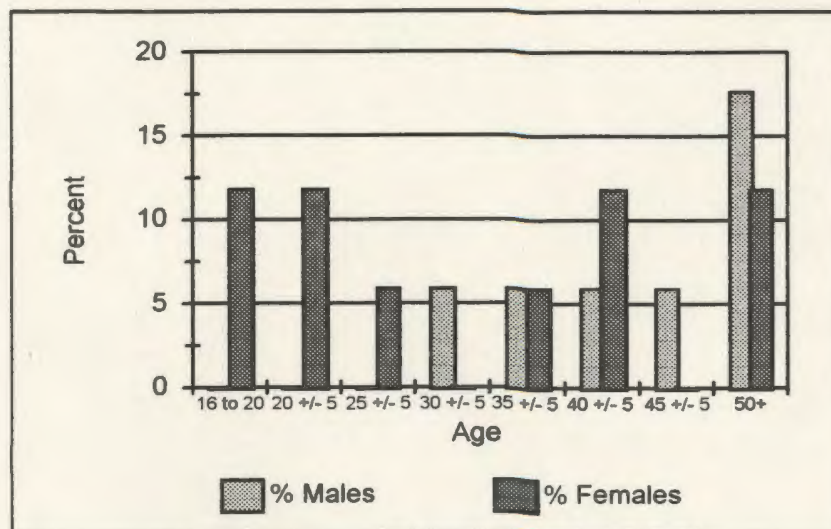
When the severity of the lesions was assessed, 63.64% (N=21) were active and 36.36% (N=12) were healing forms of cribra orbitalia. More females (12.12%, N=4) had active lesions than males (0%). However, this difference was not statistically significant. This was also evident for healing lesions, where 18.18% (N=6) of the females and 9.09% (N=3) of the males had this form of lesion (refer to Graph 5). A statistically significant relationship ($X^2 = 7.107$, $p < 0.01$) was presented whereby more subadults from the age of

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0-15 years (45.45%; N=15) had active lesions than adults (N=6) (Table 6).

Graph 5: Severity of Cribra Orbitalia for Adults and Subadults
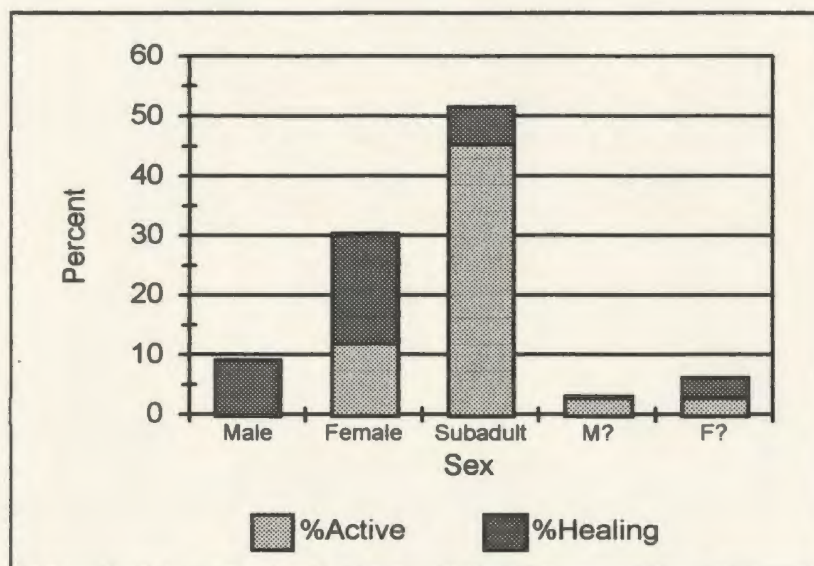
Table 6: Age Distribution of Severity for Cribra Orbitalia

Age	Active	Healing	%Active	%Healing
less than 2	9	0	27.27	0.00
2 to 5	2	2	6.06	6.06
6 to 10	2	0	6.06	0.00
11 to 15	2	0	6.06	0.00
16 to 20	2	1	6.06	3.03
20 +/- 5	1	1	3.03	3.03
25 +/- 5	0	1	0.00	3.03
30 +/- 5	0	2	0.00	6.06
35 +/- 5	0	2	0.00	6.06
40 +/- 5	1	1	3.03	3.03
45 +/- 5	1	0	3.03	0.00
50+	1	2	3.03	6.06
TOTAL	21	12	63.64	36.36

Active and healing lesions can be categorized as either light, medium or severe.

When this is performed more medium severity lesions (24.24%; N=8) were found for the

active form of cribra orbitalia while light severity lesions (27.27%; N=9) were found for the

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healing form (Table 7).

Table 7: Severity Categories for Cribra Orbitalia

Type	Number	Percent
Active & Light	6	18.18
Active & Medium	8	24.24
Active & Severe	7	21.21
Healing & Light	9	27.27
Healing & Medium	3	9.09
TOTAL	33	100

When the presence of cribra orbitalia and porotic hyperostosis were combined it became apparent that a large percentage of the lesions were centered on the orbit (30%) and on all of the cranial bones, i.e., orbits, frontal, parietals and occipital, (22.5%) (Table 8). This information also illustrates, as have many other studies, that a large percentage (12.5%) of the lesions were found on the orbits, parietals and the occipital. This helps cement the relationship between these two types of pathologies and their geography on the skull.

Porotic hyperostosic lesions were analysed separately from cribra orbitalia in order to understand this pathology on its own. Analysis first separated all of the bones of the skull instead of looking at the complete cranium. As a result, 25.76% (N=17) of these types of lesions were active and 74.24% (N=49) were healing. The type of severity and their distribution for each cranial bone; the frontal, parietals and occipital are presented in Table 9.

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Table 8: <u>Presence of Cribra Orbitalia and Porotic Hyperostosis</u>

Presence	Number	Percent
Orbits only	12	30.00
Frontal only	0	0.00
Left Parietal only	0	0.00
Right Parietal only	0	0.00
Occipital only	0	0.00
Orbits & Frontal	0	0.00
Orbits & Parietals	4	10.00
Orbits & Occipital	3	7.50
Orbits + Frontal + Parietals	4	10.00
Orbits + Frontal + Occipital	0	0.00
Orbits + Parietals + Occipital	5	12.50
All	9	22.50
Parietals + Occipital	2	5.00
Frontal + Parietals + Occipital	1	2.50
TOTAL	**40**	**100**

Table 9: <u>Type of Lesion on Individual Cranial Bones</u>

Cranial Bone	# Active	# Healing	% Active	% Healing
Frontal	2	7	3.03	10.61
L. Parietal	6	14	9.09	21.21
R. Parietal	6	14	9.09	21.21
Occipital	3	14	4.55	21.21
TOTAL	**17**	**49**	**25.76**	**74.24**

As mentioned earlier, active and healing lesions are also broken down to further subcategories of severe, medium and light. These data are presented in Table 10. This information reveals that the most active and severe lesions were found on the parietals (6.06%), while healing and light lesions were more apt to be observed on the parietals (16.67%) as well. Although this appears to contradict itself, it demonstrates that the parietals are usually involved in the presentation of porotic hyperostosis.

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Table 10: Severity of Porotic Hyperostosis

Severity	Frontal	Left Parietal	Right Parietal	Occipital	% Frontal	% Left Parietal	% Right Parietal	% Occipital
Active & Severe	0	1	1	0	0.00	1.52	1.52	0.00
Active & Medium	1	4	4	2	1.52	6.06	6.06	3.03
Active & Light	1	1	1	1	1.52	1.52	1.52	1.52
Healing & Medium	0	3	3	6	0.00	4.55	4.55	9.09
Healing & Light	7	11	11	8	10.61	16.67	16.67	12.12
TOTAL	9	20	20	17	13.64	30.30	30.30	25.76

When sex is factored into the equation, these data reveal that both males and females had healing lesions in approximately equal distributions, 35% (N=7) and 30% (N=6), respectively. In fact, approximately 75% (N=15) of the collection had the healing form of porotic hyperostosis, while only 25% (N=5) had active. All the active forms of porotic hyperostosis found on skulls from this collection were present on Subadults. This becomes especially evident when the information is broken down by age cohorts (Table 11). Once again, a large majority of the active lesions were diagnosed on individuals below the age of 2 (19.05%; N=4). In fact, all active lesions that are present in this collection are found on those individuals below the age of 16-20 years. As a result, a statistically significant relationship exists for those individuals below the age of 15 years who have active lesions

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and for those who are above this age and have healing lesions (X^2 = 9.669, p < 0.01).

Table 11: **Severity of Porotic Hyperostosis Lesions for Dickson Mounds**

Age	Active	Healing	%Active	%Healing
less than 2	4	0	19.05	0.00
2 to 5	0	1	0.00	4.76
6 to 10	1	0	4.76	0.00
11 to 15	1	0	4.76	0.00
16 to 20	1	1	4.76	4.76
20 +/- 5	0	1	0.00	4.76
25 +/- 5	0	1	0.00	4.76
30 +/- 5	0	1	0.00	4.76
35 +/- 5	0	2	0.00	9.52
40 +/- 5	0	1	0.00	4.76
45 +/- 5	0	2	0.00	9.52
50+	0	4	0.00	19.05
TOTAL	7	14	33.33	66.67

6.3 PERIOSTITIS AND/OR OSTEITIS AND OTHER PATHOLOGIES IN THE POPULATION

When the entire population of 110 individuals was assessed for the presence of periostitis and/or osteitis, 64.55% (N=71) of the individuals manifested these pathologies. As in other populations, this pathology centers on the long bones with a predisposition of the lower limb rather than the upper (Table 12). Periostitis was also prevalent on the ribs. Osteitis is found predominantly on the maxilla (36.8%) and the mandible (52.6%). Fractures are more apparent on the ulnae (15.8%) and the ribs (73.7%). Areas of necrosis were found more on the ribs (50%), tibiae (25%) and the vertebrae (25%), while lytic lesions were mostly found on the vertebrae (56.6%). Areas of ossified hemorrhages were located predominantly on the ribs (85.7%) and the fibulae (14.3%). Therefore, the majority of the pathologies are present on the ribs.

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Table 12: Periostitis and Other Nutritionally-Related Pathologies for Each Bone Element

Type	Max	Mand	Hum	Rad	Ulna	Fem	Tib	Fib	Ribs	Vert	C/T/MC/MT	Skull	Other	TOTAL
Peri	6	7	16	13	19	25	65	32	38	1	16	0	33	271
Peri/Ost	6	4	1	2	1	3	5	3	2	0	0	40	0	67
Ost	7	10	1	0	0	0	1	0	0	0	0	0	0	19
Fracture	0	0	0	0	3	0	0	0	14	0	0	0	2	19
Hemo	0	0	0	0	0	0	0	1	6	0	0	0	0	7
Necro	0	0	0	0	0	0	1	0	2	1	0	0	0	4
Lat-Med	0	0	0	0	0	0	0	0	0	28	0	0	0	28
Comp	0	0	0	0	0	0	0	0	0	2	0	0	0	2
Lytic	0	0	2	3	0	7	1	0	0	30	0	7	3	53

Percent:

Type	Max	Mand	Hum	Rad	Ulna	Fem	Tib	Fib	Ribs	Vert	C/T/MC/MT	Skull	Other	TOTAL
Peri	2.2	2.6	5.9	4.8	7.0	9.2	24.0	11.8	14.0	0.4	5.9	0.0	12.2	100
Peri/Ost	9.0	6.0	1.5	3.0	1.5	4.5	7.5	4.5	3.0	0.0	0.0	59.7	0.0	100
Ost	36.8	52.6	5.3	0.0	0.0	0.0	5.3	0.0	0.0	0.0	0.0	0.0	0.0	100
Fracture	0.0	0.0	0.0	0.0	15.8	0.0	0.0	0.0	73.7	0.0	0.0	0.0	10.5	100
Hemo	0.0	0.0	0.0	0.0	0.0	0.0	0.0	14.3	85.7	0.0	0.0	0.0	0.0	100
Necro	0.0	0.0	0.0	0.0	0.0	0.0	25.0	0.0	50.0	25.0	0.0	0.0	0.0	100
Lat-Med	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	100.0	0.0	0.0	0.0	100
Comp	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	100.0	0.0	0.0	0.0	100
Lytic	0.0	0.0	3.8	5.7	0.0	13.2	1.9	0.0	0.0	56.6	0.0	13.2	5.7	100

Peri = periostitis, Ost = osteitis; Hemo = hemorrhage; Necro = necrosis; Lat-Med = lateral-medial indentation of the vertebra; Comp = central compression of the vertebra; Lytic = lytic lesions;   Max = maxilla; Mand = mandible; Hum = humerus; Rad = radius; Fem = femur; Tib = tibia; Fib = fibula; Vert = vertebra; C = carpal; T = tarsal, MC = metacarpal; MT = metatarsal; Other = scapula, innominate, clavicle, manubrium

When the sex distribution for the presence of periostitis and/or osteitis was calculated, females (26.76%;N=19) displayed a slightly larger proportion than males (22.54%;N=16), which was not significant. When age was factored in, a similar trend found with the presence of cribra cranii was observed for these lesions (compare Table 13 with Table 5). For example, infants under the age of 2 years made up a large proportion (36.62%) of those with periostitis and/or osteitis as well as individuals over the age of 50+ (11.27%). A statistically significant relationship exists whereby more individuals under the age of 2 years possessed periosteal lesions than those individuals between the ages of 2-15 years (X^2 = 4.076, p < 0.05). However, no significant difference was calculated when subadults (0-15 years of age) were compared to adults (16-50+ years of age).

Table 13: Age Distribution for All the Population with Periostitis and/or Osteitis

Age	Number	Percent
less than 2	26	36.62
2 to 5	3	4.23
6 to 10	3	4.23
11 to 15	2	2.82
16 to 20	3	4.23
20 +/- 5	5	7.04
25 +/- 5	2	2.82
30 +/- 5	6	8.45
35 +/- 5	6	8.45
40 +/- 5	4	5.63
45 +/- 5	3	4.23
50+	8	11.27
TOTAL	71	100

Frequencies of the presence of periostitis and/or osteitis, along with other possible nutritionally-related pathologies, were first calculated to produce an overall understanding of the population as a whole. The results presented above suggest that, in general, this population had a large involvement of periostitis and/or osteitis. Once this general picture was generated, the smaller population that exhibited cribra cranii was analysed. Of the forty individuals with this pathology, 72.50% (N=29) presented periostitis and/or osteitis. Although this is an overwhelming proportion, it is not significant when compared to the whole population that included individuals with and without cribra cranii.

When the presence of periostitis and/or osteitis, in conjunction with other possible nutritionally-related pathologies and cribra cranii are categorized by bone element, a similar tendency toward certain bones being affected as noted in the entire population, was also observed for this smaller population. The calculations reveal that long bones, especially lower limbs, were more commonly affected by periostitis (Table 14). Once again, osteitis was observed more on the maxilla (44.4%) and the mandible (33.3%) than other skeletal elements. Fractures were noted more often in the ribs (62.5%) and the ulnae (25%), while evidence of ossified hemorrhages was observed only on the ribs for those individuals first diagnosed with cribra cranii. Areas of necrosis were observed in equal frequencies on the tibiae and the ribs (40%), while the vertebrae only had 20% of this form of pathology. Lytic lesions were also more centralized on the vertebrae (63.6%) and the skull (18.2%) than other areas of the skeleton.

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Table 14: Periostitis and/or Osteitis and Other Nutritionally-Related Pathologies for Each Bone Element for Those

With Cribra Cranii

Type	Max	Mand	Hum	Rad	Ulna	Fem	Tib	Fib	Ribs	Vert	C/T/MC/MT	Skull	Other	TOTAL
Peri	3	2	5	8	9	9	27	16	28	0	9	22	8	146
Peri/Ost	3	1	0	0	0	2	1	0	2	3	0	0	0	12
Ost	4	3	0	0	0	0	0	1	0	0	0	1	0	9
Fracture	0	0	0	0	2	0	0	0	5	0	0	0	1	8
Hemo	0	0	0	0	0	0	0	0	3	0	0	0	0	3
Necro	0	0	0	0	0	0	2	0	2	1	0	0	0	5
Lat-Med	0	0	0	0	0	0	0	0	0	13	0	0	0	13
Comp	0	0	0	0	0	0	0	0	0	3	0	0	0	3
Lytic	0	0	1	1	0	2	0	0	0	14	0	4	0	22

Percent:

Type	Max	Mand	Hum	Rad	Ulna	Fem	Tib	Fib	Ribs	Vert	C/T/MC/MT	Skull	Other	TOTAL
Peri	2.1	1.4	3.4	5.5	6.2	6.2	18.5	11.0	19.2	0.0	6.2	15.1	5.5	100
Peri/Ost	25.0	8.3	0.0	0.0	0.0	16.7	8.3	0.0	16.7	25.0	0.0	0.0	0.0	100
Ost	44.4	33.3	0.0	0.0	0.0	0.0	0.0	11.1	0.0	0.0	0.0	11.1	0.0	100
Fracture	0.0	0.0	0.0	0.0	25.0	0.0	0.0	0.0	62.5	0.0	0.0	0.0	12.5	100
Hemo	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	100.0	0.0	0.0	0.0	0.0	100
Necro	0.0	0.0	0.0	0.0	0.0	0.0	40.0	0.0	40.0	20.0	0.0	0.0	0.0	100
Lat-Med	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	100.0	0.0	0.0	0.0	100
Comp	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	100.0	0.0	0.0	0.0	100
Lytic	0.0	0.0	4.5	4.5	0.0	9.1	0.0	0.0	0.0	63.6	0.0	18.2	0.0	100

See Table 12 for meaning of abbreviations.

When this segment of the population is segregated by sex, females make up 64.28%

(N=9) of those individuals who have both cribra cranii and periostitis and/or osteitis.

However, this large percentage did not prove to be significant, even though numerically the

occurrence of females having these pathologies was almost in a ratio of 2:1 (N for Females

= 9; N for Males = 5). Once again, a large percentage of the population with both of these

pathologies were individuals under the age of 2 years (31.03%) and above the age of 50+

(13.79%) (Table 15; Graph 6). Unlike earlier calculations, this segregation of more

Subadults presenting these pathologies did not prove to be significant.

Table 15: Age Distribution of Those Individuals With Both Cribra Cranii and

Periostitis and/or Osteitis

Age	Number	Percent
less than 2	9	31.03
2 to 5	3	10.34
6 to 10	2	6.90
11 to 15	1	3.45
16 to 20	2	6.90
20 +/- 5	2	6.90
25 +/- 5	1	3.45
30 +/- 5	1	3.45
35 +/- 5	2	6.90
40 +/- 5	2	6.90
45 +/- 5	0	0.00
50+	4	13.79
TOTAL	29	100

Graph 6: Age Distribution of Those With Both Cribra Cranii and Periostitis and/or

Osteitis
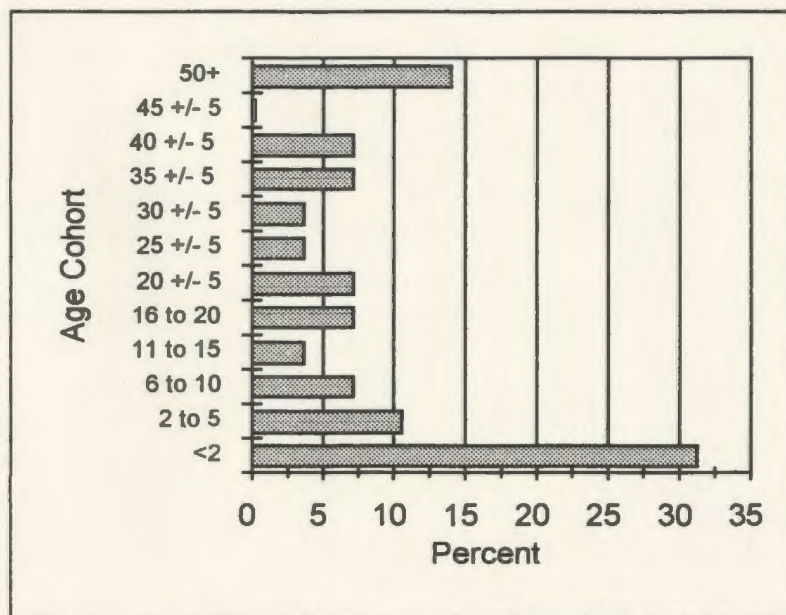
6.4 ANTEMORTEM TOOTH LOSS

Antemortem tooth loss was first calculated for the entire adult (15+ years of age) population of 110 individuals in order to produce a generalized view of this pathology and to see if it was normal for this population or a result of nutritional deficiencies. A generalized trend is noticed for both the upper and lower jaws in which molars tend to have a predisposition of being lost antemortem than other teeth (Table 16; Graph 7). A percentage was calculated first for the mandibular teeth, then for the maxillary teeth, and then finally these results were amalgamated to produce a total percentage for both upper and lower jaws. Out of 1597 available tooth sockets, 253 or 15.84%, were lost antemortem. Of these 253 teeth lost antemortem, 42 or 16.60%, were single-rooted teeth.

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Table 16: **Teeth Lost Antemortem for All Adults From Dickson Mounds**

Tooth	Mand. Amt	Max. Amt	Total	Mand. %	Max. %	Total %
LM3	13	11	24	8.72	10.58	9.49
LM2	19	11	30	12.75	10.58	11.86
LM1	20	8	28	13.42	7.69	11.07
LPM2	6	3	9	4.03	2.88	3.56
LPM1	4	4	8	2.68	3.85	3.16
LC	3	2	5	2.01	1.92	1.98
LI2	4	2	6	2.68	1.92	2.37
LI1	6	4	10	4.03	3.85	3.95
RI1	3	5	8	2.01	4.81	3.16
RI2	3	5	8	2.01	4.81	3.16
RC	1	4	5	0.67	3.85	1.98
RPM1	3	8	11	2.01	7.69	4.35
RPM2	9	8	17	6.04	7.69	6.72
RM1	22	12	34	14.77	11.54	13.44
RM2	20	9	29	13.42	8.65	11.46
RM3	13	8	21	8.72	7.69	8.30
TOTAL	149	104	253	100	100	100

Graph 7: **Teeth Lost Antemortem for All Adults From Dickson Mounds**

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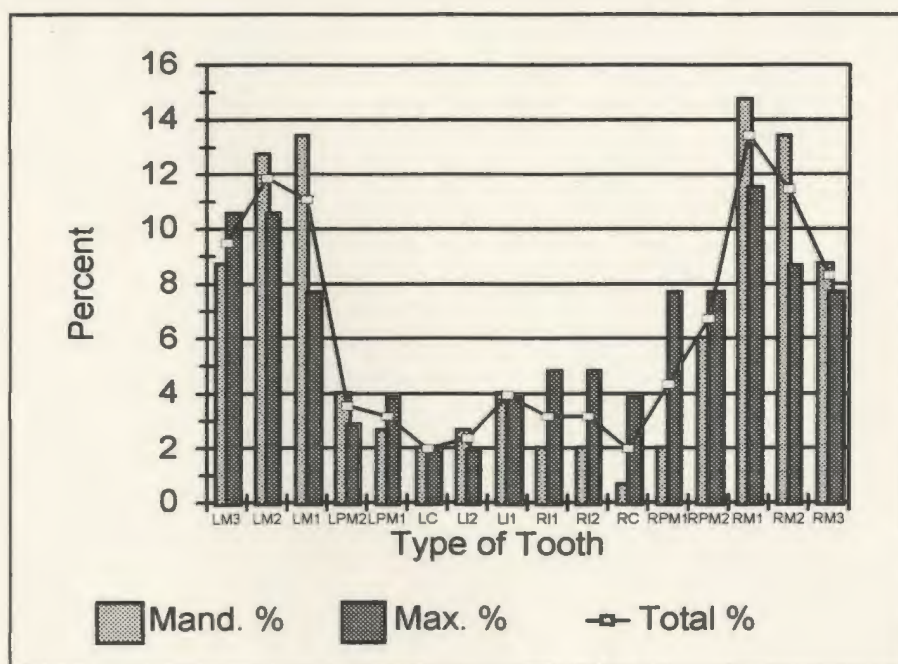
Antemortem tooth loss was also calculated for those individuals with just cribra cranii and a similar result that was noted for the entire population was produced for this segregated population as well (Table 17; Graph 8). However, only a slight difference can be seen when comparing the percentage of loss for premolars. A slightly higher percentage of right maxillary premolars are lost to those with cribra cranii than for the entire population. Out of 494 available tooth sockets for this population, only 93 or 18.83%, were lost antemortem. This percentage is quite comparable to that calculated for the entire population. Of the 93 teeth lost antemortem, 15 or 16.13%, were single-rooted teeth. This percentage is also very similar to that which was calculated for the entire population. As a result, no significant difference was detected between those with antemortem tooth loss who had cribra cranii when compared to the rest of the population.

Table 17: Antemortem Tooth Loss for the Adults with Cribra Cranii

Tooth	# Mand	# Max	Total	% Mand	% Max	Total %
LM3	5	3	8	8.47	8.82	8.60
LM2	6	3	9	10.17	8.82	9.68
LM1	8	2	10	13.56	5.88	10.75
LPM2	2	1	3	3.39	2.94	3.23
LPM1	1	2	3	1.69	5.88	3.23
LC	1	1	2	1.69	2.94	2.15
LI2	1	1	2	1.69	2.94	2.15
LI1	2	1	3	3.39	2.94	3.23
RI1	2	1	3	3.39	2.94	3.23
RI2	2	1	3	3.39	2.94	3.23
RC	1	1	2	1.69	2.94	2.15
RPM1	1	4	5	1.69	11.76	5.38
RPM2	3	4	7	5.08	11.76	7.53
RM1	9	4	13	15.25	11.76	13.98
RM2	9	3	12	15.25	8.82	12.90
RM3	6	2	8	10.17	5.88	8.60
TOTAL	59	34	93	100	100	100

Graph 8:Percent of Teeth Lost Antemortem for Those Individuals with Cribra Cranii
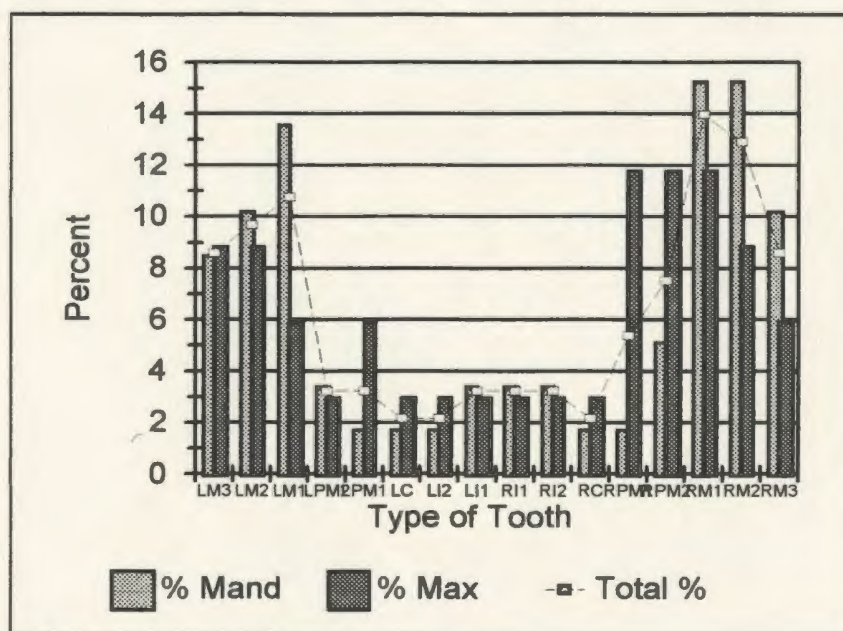
A similar distribution of type of teeth lost antemortem was also observed for those individuals of the population who had periostitis and/or osteitis when compared to the entire population (Table 18; Graph 9). Out of 1101 available tooth sockets for this population, only 174 or 15.80%, were lost antemortem. Of these 174 lost teeth, 29 or 16.67%, were single-rooted teeth. All of these percentages are comparable to those calculated for the entire population, as well as those who had cribra cranii. As a result, it was not significant to have antemortem tooth loss and periostitis and/or osteitis when compared to the rest of the population.

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Table 18: <u>Antemortem Tooth Loss for The Adults with Periostitis and/or Osteitis</u>

Tooth	# Mand	# Max	Total	% Mand	% Max	Total %
LM3	8	9	17	8.79	10.84	9.77
LM2	12	9	21	13.19	10.84	12.07
LM1	14	7	21	15.38	8.43	12.07
LPM2	3	2	5	3.30	2.41	2.87
LPM1	2	3	5	2.20	3.61	2.87
LC	2	1	3	2.20	1.20	1.72
LI2	3	1	4	3.30	1.20	2.30
LI1	3	3	6	3.30	3.61	3.45
RI1	2	4	6	2.20	4.82	3.45
RI2	2	4	6	2.20	4.82	3.45
RC	1	3	4	1.10	3.61	2.30
RPM1	2	6	8	2.20	7.23	4.60
RPM2	5	6	11	5.49	7.23	6.32
RM1	14	11	25	15.38	13.25	14.37
RM2	12	8	20	13.19	9.64	11.49
RM3	6	6	12	6.59	7.23	6.90
TOTAL	91	83	174	100	100	100

Graph 9: <u>Antemortem Tooth Loss for Those With Periostitis and/or Osteitis</u>

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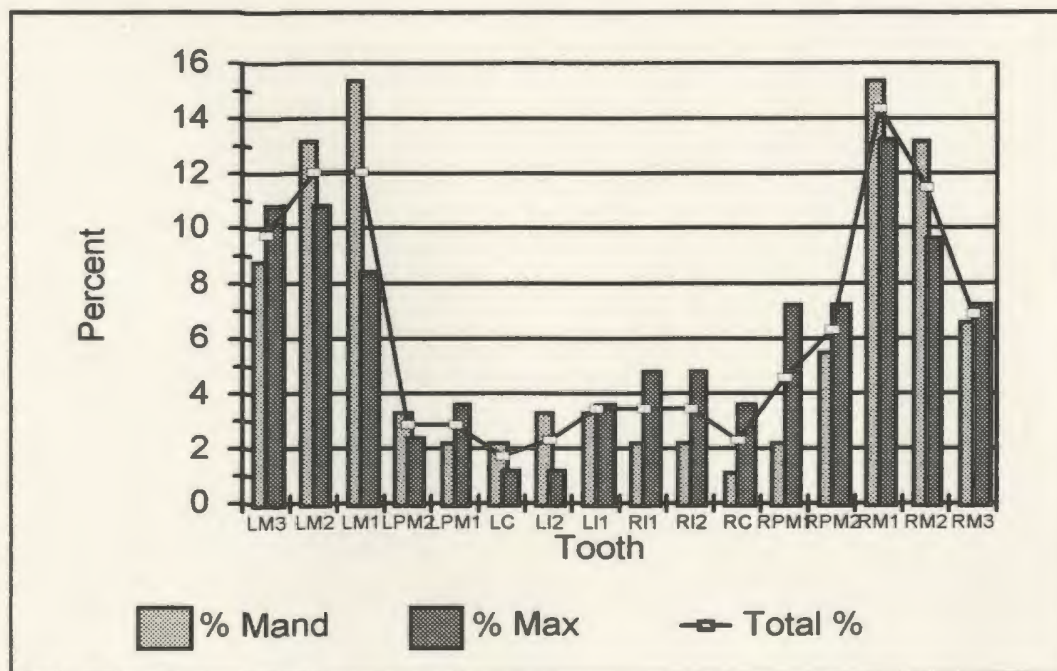
Finally, a population of individuals who presented both cribra cranii and periostitis and/or osteitis in conjunction with antemortem tooth loss was assessed. A very similar distribution of previous results found for the entire population pertaining to the type of tooth lost antemortem was noted for this category (Table 19; Graph 10). Out of 320 available tooth sockets observed in this population, only 62 or 19.38%, were lost antemortem. Of these 62 teeth lost antemortem, 12 or 19.35%, were single-rooted teeth. Although slightly higher results were obtained for this population concerning the overall frequency of teeth lost antemortem, no significance was noted when compared to the rest of the population who were not diagnosed with these same pathological criteria.

Table 19: Antemortem Tooth Loss for Those Adults With Both Cribra Cranii and Periostitis and/or Osteitis

Tooth	# Mand	# Max	Total	% Mand	% Max	Total %
LM3	3	3	6	8.82	10.71	9.68
LM2	3	3	6	8.82	10.71	9.68
LM1	4	2	6	11.76	7.14	9.68
LPM2	1	1	2	2.94	3.57	3.23
LPM1	1	2	3	2.94	7.14	4.84
LC	1	1	2	2.94	3.57	3.23
LI2	1	1	2	2.94	3.57	3.23
LI1	1	1	2	2.94	3.57	3.23
RI1	1	1	2	2.94	3.57	3.23
RI2	1	1	2	2.94	3.57	3.23
RC	1	1	2	2.94	3.57	3.23
RPM1	1	2	3	2.94	7.14	4.84
RPM2	2	2	4	5.88	7.14	6.45
RM1	4	3	7	11.76	10.71	11.29
RM2	6	2	8	17.65	7.14	12.90
RM3	3	2	5	8.82	7.14	8.06
TOTAL	34	28	62	100	100	100

Graph 10: <u>Antemortem Tooth Loss for Those Adults With Both Cribra Cranii and Periostitis and/or Osteitis</u>
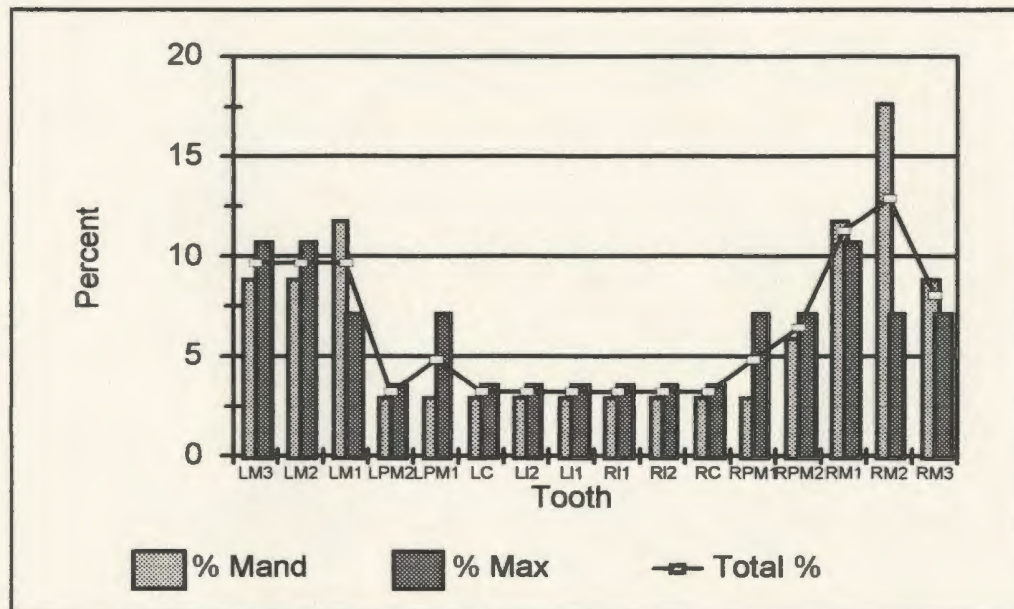
6.5 SUMMARY OF INDIVIDUALS WHO POSSESSED MULTIPLE DEFICIENCIES IN IRON, VITAMIN C, FOLIC ACID AND VITAMIN B_{12}

The forty individuals who were described earlier in Chapter 5.5, all possess either cribra orbitalia, porotic hyperostosis or both. Therefore, the possibility exists that all of these burials exhibited nutritional anaemia caused by deficiencies in iron, vitamin C, folic acid and vitamin B_{12} as it has been hypothesized that deficient nutrients causing anaemia will manifest on the skeleton as cribra cranii. However, in order to be more confident in this statement, it would be advisable to also provide isolated evidence of skeletal alterations characteristic of each of these nutrients when they are lacking in the diet.

When this is performed, a different result is found. Only seven of these forty individuals, or 17.5%, possessed evidence of multiple nutrient deficiencies of all of the forementioned nutrients. When the entire population of 110 is considered, this percentage decreases to 6.4%. This percentage includes Burial Numbers 381, 442, 443, 447, 663, 774 and 895 (refer to descriptions previously presented in this Chapter 5.5). As a result, only 6.4% presented at least one skeletal alteration indicative of iron deficiency anaemia, scurvy and megaloblastic anaemia. These skeletal alterations may include cribra orbitalia and/or porotic hyperostosis; osteitis or hyper-porosities of the mandible, maxilla and/or sphenoid; periosteal remodeling; evidence of necrosis; and osteoporosis of the vertebrae.

In summary, these seven burials contained both cribra orbitalia and porotic hyperostosis. With the presence of these two pathologies it can be certain that these individuals were suffering from anaemia, which was possibly caused or related to (a) nutritional deficiency, i.e., iron, vitamin C, folic acid or vitamin B_{12}, or all of these combined. All of these burials also possessed evidence of periostitis and subperiosteal new bone growth, except for Burial Numbers 442 and 443 which were void of these pathologies. The presence of this nonspecific pathology suggests infection and possibly a reaction to insufficient nutrients in the diet, such as vitamin C, folic acid and vitamin B_{12}. Osteitis or an abnormal increase in porosities, especially located on the maxilla, mandible or sphenoid, was also expressed in each of these seven burials. This type of lesion is most indicative of scorbutic episodes that entail low grade continuous hemorrhaging and bleeding of the muscle attachments.

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The presence of scurvy and possibly megaloblastic anaemia were also exemplified via the presence of a possible ossified hemorrhage on the left femur of Burial Number 381. This individual also possessed a healed fracture of the left clavicle. Fracturing of bones can be a sign of irregular collagen formation that occurs during bouts of scurvy. Necrosis was also observed in Burial Numbers 442, 447 and 774 which possibly suggested the presence of megaloblastic anaemia. Osteoporotic vertebrae, either in the form of central compression or lateral-medial indentation, reveal possible signs of iron deficiency and/or vitamin C deficiency. This form of pathology was evident in Burial Numbers 381 and 443. By identifying pathologies that are indicative of a deficiency in each of the four nutrients of iron, vitamin C, folic acid and vitamin B_{12}, it provides evidence that more than one nutrient can be deficient in one individual and thus, it illustrates skeletal evidence of their existence.

Although four nutrients were concentrated on for this study, skeletal evidence of deficiencies in at least two of them also provide proof that multiple deficiencies can exist in one individual when concerned with the etiologies of anaemia. The number of individuals possessing evidence of skeletal manifestations of at least two deficiencies within the segregated population of forty is greatly increased when these new parameters are constructed. When this was performed, sixteen additional individuals illustrated signs of deficiencies in both iron and vitamin C. Therefore, an additional 40% of the forty, or 14.5% of the 110, possess multiple deficiencies. In total, this suggests that over half of the population with cribra cranii (N=40) or just more than 20% of the entire population utilized for this study possibly suffered from multiple deficiencies. These included Burial Numbers

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283, 348, 357, 366, 391, 399, 406, 432, 435, 475, 496, 500, 612, 669, 833 and 934. All of these individuals possessed either cribra orbitalia or porotic hyperostosis, which is usually indicative of both iron deficiency anaemia and sometimes scurvy. Periostitis was identified in all of these burials as well, except for Burial Numbers 357, 432, 475 and 935. Osteitis was also present in Burial Numbers 283, 399, 435, 612, 833 and 934 which possibly demonstrates the presence of vitamin C deficiency. Several other pathologies such as fracturing (366 and 612), evidence of ossified hemorrhages (as in 391 and 432) and fistulae (357, 399 and 475) also possibly point to vitamin C deficiency.

Of these sixteen individuals, six (Burial Numbers 357, 366, 475, 496, 500 and 669) may be tenuous because of their age, which is predominantly over 40 years. It is possible that those pathologies identified as deficiency-related may in fact have to do with age-related processes. For example, fractures, osteoporotic vertebrae and hyperostosis in these individuals may be a fact of aging rather than of insufficiencies in the diet. Another tenuous multiple deficiency was Burial Number 406 as this individual also had evidence of actinomycosis, melorrheostosis or flowing hyperostosis of cervicals 3-5. It is possible that these other pathologies may be influencing the manifestation of cribra orbitalia, porotic hyperostosis, widespread periostitis and a possible cloaca on the right tibia (refer to descriptions in Chapter 5.5 for a more thorough explanation of these pathologies).

Several other burials not included in the sixteen above also showed signs of multiple deficiencies, but they also entailed the possibility of tuberculosis-like lesions. Burial Numbers 270 and 383 show signs of lesions generally associated with tuberculosis. The

most prevalent evidence was the presence of lytic lesions/holes found in the centrum of several vertebrae of both of these individuals (refer to descriptions in Chapter 5.5). Burial Number 383 also illustrated destructive and lytic, possibly osteomyelitic, lesion/area of the right distal humeral joint. This individual also possessed a very strange and localized lesion of unknown etiology on the endocranial surface of the left parietal. Due to the ambiguity in the etiology of this lesion, it is hard to know how it may affect the rest of the skeleton. The presence of tuberculosis-like disease may have produced the lesions identified as cribra cranii and possible deficiency lesions, or the tuberculosis-like lesions may be related to the concomitant presence of deficiencies which have resulted in these skeletal manifestations.

Burial Number 270 who presented with cribra orbitalia, exhibited a possible sign of a nutritional deficiency. However, this individual also illustrated a possible hemangioma of the left ilium (refer to Plate 4: B-270), according to Lallo's (1973) study of this population. A hemangioma is a benign blood tumor that is rarely found in the skeleton, but can normally be found in the vertebral region and the cranial vault (Ortner and Putschar 1997:376-378). However, differential diagnoses suggest that this pathology may also be caused by metastatic carcinoma, lymphoma, multiple myeloma, Paget's disease or several other blood-borne diseases (Ortner and Putschar 1997).

As was discussed in Chapter 2.2, the presence of infection and some diseases can lead to nutritional deficiencies. Also, the presence of parasites or microorganisms in the body can exacerbate a sub-optimal nutritional status of an individual. In any respect, the illustration of what appeared to be a nutritional deficiency pathology, in conjunction with

certain diseases or infections, may in fact be evidence that something more than just an infection has affected an individual and that possibly a nutritional deficiency may also have been present. Therefore, it should be apparent that single etiologies used by paleopathologists are inappropriate and a more encompassing viewpoint should be established.

CHAPTER 7: DISCUSSION

The Dickson Mounds skeletal collection has been used by many researchers for different reasons, but as each study is performed, more results are being reported. It is through these diverse approaches that a better understanding will be attained of the population who inhabited the Spoon River area of Central Illinois for several centuries. This particular study examined the nutritional and health statuses of the Mississippian Period skeletal complex. Biogeographical, subsistence, demographic and cultural factors of the Dickson Mounds Mississippian Period population were taken into consideration for this analysis. Also, medical, clinical and nutritional literature presented the basis for the possibilities of the manifestation of multiple nutritional deficiencies in one human individual. By observing skeletal evidence of pathological lesions of anaemia and specific nutritional deficiencies within this population, a more encompassing hypothesis pertaining to nutritional anaemia has been formulated.

7.1 A GENERALIZED OVERVIEW

By analyzing the Dickson Mounds Mississippian population as a whole a better demographic profile was discerned. As a result, each smaller population that was ascertained from the larger, allowed for comparisons between the two, as well as pointing out anomalies or regular trends. Results presented in Chapter 6 reveal that an approximate equal ratio of males to females (22:27) exists. The greater female to male ratio is unusual since, in most populations males are usually more prevalent. This is noted at least for

historic populations in Canada (McVey and Kalbach 1995). This slight tendency for females over males at Dickson Mounds may have to do with a bias imparted by the observer toward identifying female attributes more than male. However, this should have been avoided via the use of metric methods to determine sex. Also, there is usually a tendency to identify more males than females (Weiss 1972) due to the ease in identifying robust qualities as male-derived and that males usually live to an older age than females in prehistoric populations (Walker 1995).

The age distribution of the Mississippian Period population strongly indicates that subadults make up almost 50% of the whole population. This is interesting to note since subadults are usually under-represented due to a bias in excavation techniques, smallness of the bones, poor preservation and differential burial practices (e.g., anatomical position of individual and placement within the cemetery) (Saunders 1992 and those authors cited within).

When specific age groups were determined, it became apparent that a large proportion of the subadult cohort was made up of individuals less than two years of age (30%), which indicated a very high infant mortality rate. Such a high infant mortality rate is in accordance with other studies performed on this time period and this type of population (Clarke 1977). The next largest age cohort represented in this population belonged to those above the age of fifty years. Therefore, a U-shaped curve was constructed illustrating a large percentage at each end of the age spectrum used to define this population. When age groups were broken down by sex, this information revealed that more males were living longer and

more females were dying younger. The majority of females died by the age of 25 +/- 5 years of age with a peak around 20 +/- 5 years of age. A large percentage of females dying young is most likely due to death occurring during childbirth or poor nutrient status following pregnancies and lactation. However, Graph 2 also illustrates that a peak of female age at death exists at 40 +/- 5 years of age. It is unknown at this moment why such a peak exists for this population. It may reflect a bias toward identifying female attributes for older skeletal remains or this age group could represent the most prevalent age at which females died in this population.

When the Mississippian Period population of Dickson Mounds was scrutinized for the presence of cribra orbitalia and porotic hyperostosis, it was noticed that approximately 35% (N=39) of the individuals had cribra orbitalia only while only one individual presented with porotic hyperostosis alone. The presentation of approximately one third of the population having cribra orbitalia and/or porotic hyperostosis is within the range of other studies concentrating on these two pathologies and the Dickson Mounds Mississippian population (El-Najjar *et al.* 1975; Goodman *et al.* 1984; Lallo and Rose 1979; Lallo *et al.* 1977; Mensforth 1991). The sex distribution of cribra cranii revealed that an approximate equal ratio of males to females (7:10) existed with a slight preponderance of females exhibiting these pathologies. However, no statistically significant difference existed between males and females and the presence of cribra cranii.

Age distribution of individuals with cribra cranii illustrates that individuals under the age of five years and over the age of fifty years, were more often affected by this pathology.

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Once again this was illustrated by a U-shaped curve found in Graph 3. By presenting a similar distribution as the original demographic profile, it possibly illustrates that the morbidity (i.e., cribra orbitalia and porotic hyperostosis, or anaemia) of these particular individuals may be associated with their mortality. To see if a relationship existed between specific sex and age cohorts for the Dickson Mounds population with cribra cranii, several categories were collapsed: 16-25 +/- 5 years of age and 50+ years of age for both males and females. A statistically significant relationship ($X^2 = 5.517$, $p < 0.02$) was observed between those females in the collapsed age cohort of 16-25 +/- 5 years and those males over the age of fifty. This relationship obviously illustrated that younger females, in the childbearing age range, and males over the age of fifty had a prevalence of manifesting this pathology more so than other cohorts.

Such a segmentation of the population into specific age and sex cohorts is most likely related to the loss of nutrients experienced by females because of pregnancy and/or multiparity, lactation and menstruation; and the age-related decrease in mobility and access to food, and a decrease in the metabolism, absorption and consumption of nutrients. It is also quite possible that those pathologies identified as cribra cranii for the elderly cohort may have been related to the aging process and possibly could have been misidentified as anaemia-related, thus providing a larger percentage. The preponderance of females over males, subadults under the age of five, and particularly less than two years of age, and the elderly cohort exhibiting cribra cranii was an expected demographic profile for this form of pathology (Carlson *et al.* 1974; El-Najjar *et al.* 1975; El-Najjar *et al.* 1976; Fornaciari *et*

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al. 1982; Goodman *et al.* 1984; Hengen 1971; Lallo and Rose 1979; Lallo *et al.* 1977; Mensforth 1991; Stuart-Macadam 1985, 1987b, 1988, and 1989a; Walker 1986). It suggests that these cohorts are more susceptible to the manifestation of anaemia than other cohorts presented in this population.

Susceptibility relates to the discussion in Chapter 3, i.e., in that pregnancy and/or multiparity, lactation and menstruation for females; premature birth, plasticity of the skeleton, early weaning and poor maternal diets for infants; immobility and a decrease in consumption, absorption and utilization of nutrients for the elderly all factor in the production of nutrient deficiencies and their concomitant skeletal manifestations. Also factored into this equation is the presence of parasites and/or microorganisms that can drain their host of nutrients, along with infections which can also alter the nutritional status of an individual.

Severity of the cribra orbitalia lesions for this population indicates that almost 64% of them were considered active, with the remaining 36% healing. This means that more individuals were dying with active lesions than healing lesions which could translate into a correlation between morbidity and mortality. Although more females manifested these lesions than males in both the active and healing categories (see Graph 5), no statistically significant difference was calculated. However, a statistically significant relationship (X^2 = 7.107, p < 0.01) was calculated when subadults under the age of fifteen years were compared to adults over the age of fifteen years for the manifestation of active lesions. This means that subadults had a stronger prevalence toward exemplifying and suffering from

active forms of cribra orbitalia than adults. This is in accordance with other studies concerning the manifestation of cribra orbitalia (e.g., Goodman *et al.* 1984; Lallo and Rose 1979; Lallo *et al.* 1977; Mensforth 1991; Stuart-Macadam 1985, 1987b, 1988, and 1989a). Analysis also revealed that the severity of the active lesions was predominantly of the medium (approximately 24%) category and those lesions that were healing were of a light nature (approximately 27%).

By segregating the active and healing lesions into their respective forms of severity, it demonstrated that healing cribra orbitalia was usually light and the active lesions were normally medium to severe in nature, thus providing a more comprehensive view of this pathology. However, one must keep in mind that healing could entail lesions which may have been active within the light, medium or severe type at one time since it is difficult to determine how the lesions originated. It also showed that individuals suffering from cribra orbitalia in the Dickson Mounds Mississippian population were likely more susceptible to the harsher forms of this pathology. However, the larger percentage of active versus healing lesions may be related to the relative ease of identifying the presence of spicules within the lesions than their absence.

The distribution of cribra orbitalia and porotic hyperostosis revealed that the majority of these anaemia-related pathologies were centered on the anterior portion of the orbit and on the frontal, parietals and the occipital bones. The preponderance of lesions being located on these areas of the skull have been demonstrated in other studies (Stuart-Macadam 1987a and b, 1988, 1989a and b) and thus, these results help to cement the relationship between

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cribra orbitalia and porotic hyperostosis and their distribution on the skull. Evidence of more orbital lesions possibly illustrates that this population was suffering from the less severe form of anaemia because it is believed that cribra orbitalia represents the beginning stage of skeletal involvement, while the presentation of anaemia on the remaining cranial bones indicates a more severe or drawn out form (Stuart-Macadam 1989b). As a result, it might be suggested that the Mississippian Period population at Dickson Mounds was suffering from a less severe or more acute form of anaemia.

As stated previously, with the majority of cribra orbitalia lesions being active and severe, it possibly indicated that these individuals suffered from a more severe form of anaemia. However, when the cribra orbitalia lesions were compared to the porotic hyperostosic lesions, literature indicates that if both orbital and calvarial lesions are found together they then signify a greater severity or prolonged occurrence of anaemia rather than orbital lesions alone. Therefore, when more information is gathered and brought together, more can be said about the severity of the anaemia at Dickson Mounds: anaemia was relatively severe, but more acute cases were noted than prolonged cases. This preponderance toward cribra orbitalia may be related to the relative ease of identifying it in comparison to porotic hyperostosis.

When porotic hyperostosis was considered separately from cribra orbitalia, a different trend was noticed. A distribution approximately equal between males and females (7:6) was observed for healing lesions. When age was factored into the equation, a statistically significant ($X^2 = 9.669$, $p < 0.01$) relationship existed whereby subadults under

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the age of fifteen years had a higher tendency of being diagnosed with active porotic hyperostosis and those above this age had a higher tendency of having healing lesions. This once again cements the fact that subadults tend to manifest active forms of anaemia more often than adults, as adults tend to be illustrating healing forms of anaemia possibly resulting from childhood episodes (Stuart-Macadam 1985).

The presence of periostitis and/or osteitis in the entire Mississippian Period population of Dickson Mounds observed in this thesis was high. Approximately 65% of the population was diagnosed with both lesions, with a preponderance on the lower limbs, and the maxilla and mandible being more involved (Table 12). This revealed that the presence of periostitis affected a large percentage of the population and thus, infection rates were considered high. This translates into the possibility of the lack of a relationship between periostitis and cribra cranii as such a large proportion of the population manifested these nonspecific infection pathologies without the presence of cribra cranii. However, it does provide evidence that infection was quite common at Dickson Mounds and thus, the possibility of these infections precipitating or exacerbating nutritional deficiencies (refer to Chapter 2.2) that can cause anaemia might be well presented.

Females had a slightly higher prevalence toward the identification of periostitis and/or osteitis (27% compared to 23% for males), which was not statistically significant. The age distribution of the presentation of these two pathologies was similar (i.e., U-shaped curve) to other trends already noted for this population, with subadults under the age of two and adults fifty years and older possessing more of these pathologies than other age groups.

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This was also exemplified by a statistically significant relationship ($X^2 = 4.076$, $p < 0.05$) between those individuals under the age of two possessing periosteal reactions than those individuals between the ages of two to fifteen years. This revealed that once again, subadults under the age of two years have a higher incidence of being diagnosed with pathological lesions that could be related to the causes of their mortality. It also demonstrates that this section of the population was more susceptible to infections and possibly nutritional deficiencies which may be related to early weaning.

The predominant forms of pathologies other than cribra orbitalia, porotic hyperostosis and periostitis and/or osteitis were fractures in the ribs, necrosis in the ribs, tibiae and vertebrae, lytic lesions in the vertebrae and ossified hemorrhages in the ribs and fibulae (refer to Table 12). As a result, a majority of the pathologies were located on the ribs for the entire population. The presence of these pathologies may be linked to deficiencies in iron, vitamin C, folic acid and vitamin B_{12}. For example, necrotic action can be related to atherosclerosis and blood clotting noted in megaloblastic anaemia; fracturing of bones may be related to the abnormal formation of collagen noted in scurvy; and ossified hemorrhages may be related to lack of vascular integrity in both scurvy and megaloblastic anaemia.

The presence of periostitis and/or osteitis in conjunction with cribra cranii was also analysed for this population. As expected, a very large proportion, almost 75% of the population that had cribra cranii (N=40), also presented periostitis and/or osteitis. However, as already alluded to, it may not be significant to find both of these pathologies together

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because of the relative predominance of periostitis and/or osteitis in the whole population (N=110). One possible explanation is that the Mississippian Period population from Dickson Mounds suffered from various forms of infection that may have precipitated nutritional deficiencies, possibly causing anaemia, especially in those individuals with already sub-optimal levels of iron, vitamin C, folic acid and vitamin B_{12}. The synergism and antagonism of these four nutrients and the immune system were discussed in great detail in Chapter 2.2.

As in the larger population, similar trends in the skeletal location of periostitis and/or osteitis (lower limbs), fractures (ribs and ulnae), ossified hemorrhages (ribs), necrosis (ribs, tibiae and vertebrae) and lytic lesions (vertebrae and skull) were observed for the condensed population that only possessed cribra cranii (compare Table 12 with Table 14). Although females represented a larger segment of the population with periostitis and cribra cranii (almost a 2:1 ratio), no significant relationship was encountered possibly because of the small sample size. It may be that females had a higher chance of contracting infections than males in this population. This may be related to a decrease in nutritional status due to the drain put on females during times of pregnancy and lactation, which can predispose these females to more virulent and protracted infections. A U-shaped distribution was produced with a large proportion of individuals under the age of two (31%) and those over the age of fifty years (14%) being diagnosed with both cribra cranii and periostitis and/or osteitis (refer to Table 15 and Graph 6). Once again, this may represent the disadvantaged outlook these age cohorts must face, even in today's societies. Also, demonstration of infants and the

elderly exhibiting both of these pathologies may suggest a relationship between cribra cranii and periostitis that has been exhibited in nutritional and immunological research.

The similarity in distribution of the age at death and sex for the presence of cribra cranii, periostitis and/or osteitis and the presence of both cribra cranii and periostitis and/or osteitis, suggests a correlation. One cannot determine absolutely if each of these forms of morbidity caused mortality in this population, but a relationship is definitely involved. There is the possibility that the interrelationships presented in earlier chapters on nutrition for the four proposed nutrients and their skeletal alterations are indeed being exemplified in this population as similar trends and distributions were discovered for each independent analysis performed for this study.

A generalized trend of antemortem tooth loss for the whole population was also present in the segregated populations, i.e., those with cribra cranii, those with just periostitis and/or osteitis and those who possessed both cribra cranii and periostitis and/or osteitis. These trends entail more upper and lower molars being lost antemortem than other teeth in either the maxilla or the mandible (compare Graphs 7-10) and a generalized similar percentage (16-19%) of single rooted teeth being lost antemortem. However, a slightly higher percentage of single rooted antemortem tooth loss was exhibited when the two populations of cribra cranii and periostitis and/or osteitis were collapsed together as opposed to when they were separated (compare 19% to 16% and 17%, respectively). Due to the fact that antemortem tooth loss of single rooted teeth is a known sequella of scurvy (Ortner 1984), it was hypothesized that an increased percentage should have been observed when

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the population was broken down into different pathological cohorts. This was in fact observed, but the difference is not as significant as would have been expected in order to provide a direct and positive link. There is the possibility that loss of single-rooted teeth has occurred via decomposition of the periodontal ligament just after death. Therefore, only a generalized statement can be made: slightly more single rooted teeth were lost antemortem for those possessing a combination of both cribra cranii and periostitis and/or osteitis than those with only one of these pathologies. As a result, tooth loss could not be used as a signal to denote the presence of scurvy for this population. It is possible that other underlying factors may be involved in explaining antemortem tooth loss.

7.2 INDIVIDUALS POSSESSING MULTIPLE DEFICIENCIES

Only seven individuals within the Mississippian Period population from Dickson Mounds exhibited skeletal alterations that may be indicative of a combination of multiple deficiencies in iron, vitamin C, folic acid and vitamin B_{12}. These seven individuals included Burial Numbers 381, 442, 443, 447, 663, 774 and 895 (refer to Chapter 6). Each of these burials possessed evidence of both cribra orbitalia and porotic hyperostosis. Therefore, in the realm of nutritional deficiencies, this demonstrates that all of the aforementioned nutrients could have been lacking in their diets as it was hypothesized that hyperostosis can be caused by deficiencies in iron, vitamin C, folic acid and vitamin B_{12} because they all cause some form of anaemia.

Periostitis and/or osteitis was also manifested in all of these burials, except for

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Numbers 442 and 443. This suggested that infection could have been present in these individuals. This may be due to the presence of parasites and/or microorganisms, or nutritional deficiencies in vitamin C, folic acid or vitamin B_{12}, or a combination of both in which each condition may have exacerbated the other. Osteitis was present in all of the burials leading to the conclusion that those lesions located on the skull, specifically the mandible, maxilla or sphenoid, may likely be related to a deficiency in vitamin C, or scurvy. Increased porosities in these areas may be indicative of the low grade hemorrhaging and chronic inflammation associated with bleeding resulting from abnormal blood vessel formation of the maxillary artery and its tributaries during scorbutic episodes and minor mechanical trauma from muscle contraction of the temporalis and pterygoid muscles (Ortner and Ericksen 1997). The possibility of an ossified hemorrhage on the left femur of the individual in Burial Number 381, also points to scurvy and/or megaloblastic anaemia due to the role of vitamin C in vascular integrity and folic acid and vitamin B_{12} deficiencies in blood clotting.

Necrosis was found on individuals from Burial Numbers 442, 447 and 774 which indicated a prevention of nutrients entering these areas possibly due to a deficiency in folic acid or vitamin B_{12}. Although other factors unrelated to nutrition may have caused a fractured left clavicle in Burial Number 381, it also suggests a possible relation to scurvy as a lack in vitamin C can cause bone to be brittle due to the malformation or lack of proper collagen. Osteoporotic vertebrae of Burial Numbers 381 and 443 possibly represented a lack in both iron and vitamin C (and possibly calcium) in the diet as these two nutrients are

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responsible for proper bone development. Burial Number 895 possessed spina bifida which relates to a deficiency in his/her mother and may be translated to a folic acid deficiency in both of these individuals. In any case, it does represent a folic acid deficiency in at least one person: the mother, and thus it provides possible evidence of its existence within this population. However, genetic predispositions are also a possible etiology for this form of a neural tube defect.

Of the remaining thirty-three burials, many exhibited signs of deficiencies in at least two nutrients. These included Burial Numbers 283, 348, **357**, **366**, 391,399, 406, 432, 435, 475, 496, **500**, 612, **669**, 833 and 934 which all exhibited possible signs of vitamin C and iron deficiencies (Burial Numbers in bold are discussed below). This was exemplified by the presence of cribra orbitalia and/or porotic hyperostosis in all of them, along with evidence of scurvy via either periostitis, osteitis, the presence of fistulae and ossified hemorrhages or fractures. By presenting evidence of pathologies that are indicative of iron *and* vitamin C deficiencies, it provides skeletal evidence that more than one nutritional deficiency can exist in one individual at a time. As a result, this ability to identify multiple deficiencies that cause anaemia should be acknowledged by other paleopathologists when they encounter similar skeletal manifestations.

The six burials listed in bold above are only possibilities of multiple deficiencies as each individual was old (post 40 years of age) and aging processes may be accountable for the pathologies that were identified (e.g., loss of bone or teeth and fractures). Therefore, their inclusion within this list, while tenuous, are nonetheless intriguing.

Several other burials presented possible signs of multiple deficiencies, but these may be related to the identification and presence of other diseases. For example, Burial Numbers 256 and 383 both possess pathologies that may be indicative of tuberculosis-like disease. The most characteristic pathology identified was the presence of tuberculosis-like lesions in several vertebrae. However, the presence of lytic lesions in the vertebrae which are normally classified as tuberculosis-related, could also be manifested in osteomyelitis, or neoplasm of metastatic carcinoma or multiple myeloma (Ortner and Putschar 1997). Several small button osteoma-like lesions were also identified on the pleural surface of several ribs for the individual associated with Burial Number 383, which resemble lesions associated with pulmonary tuberculosis. These forms of lesions were also identified in several other individuals from Dickson Mounds. The presence of tuberculosis-like lesions has been identified in the Illinois area and in relatively the same time period by other researchers (Buikstra 1992; Milner 1992).

The presence of both tuberculosis-like lesions and cribra cranii (along with evidence of possible ossified hemorrhages on several ribs of Burial Number 383), hence possible nutritional deficiencies, poses the potential of the existence of the interrelationship between these two circumstances. In fact, according to Lindenbaum (1979:23) an association exists between inflammatory diseases and folate deficiency as the infection seems to precipitate megaloblastic anaemia. A study performed by Roberts *et al.* (cited by Lindenbaum 1979:23) reported that serum folate concentrations were depressed in patients with tuberculosis, which may be related to a decrease in folate intake often associated with inflammatory

illnesses, or as a result of an actual increase in folate requirements during infection. McMurray *et al.* (1990) also suggest that a strong association exists between malnutrition and infection with *Mycobacterium tuberculosis* in humans.

According to these authors, a dietary deficiency in vitamin C, along with protein and vitamin A, have been implicated in impaired responses to tuberculosis. This is most likely due to vitamin C's importance in cell-mediated immunity which is known to be crucial in fending off infection with the tubercle bacillus (McMurray *et al.* 1990:59). Even animal models, such as guinea pigs, had a greater susceptibility to tuberculosis (and diphtheria, pneumococci, anthrax bacilli, salmonella, rickettsia and typhus fever) when placed on scorbutic diets (Scrimshaw *et al.* 1968:97). Also, a diet deficient in vitamin B_{12}, as in strict vegetarians, was associated with a higher incidence of tuberculosis. This relationship has been linked to the impairment of bacterial killing in phagocytes demonstrated in vitamin B_{12} deficiencies (McMurray *et al.* 1990:67).

Burial Number 270 exhibits a possible hemangioma of the left ilium that may or may not be related to the manifestation of cribra orbitalia. The identification of a hemangioma in this individual was first performed by Lallo (1973) in his Ph.D. thesis. However, differential diagnosis of this pathology could also provide metastatic carcinoma, lymphoma, multiple myeloma, Paget's disease or several other blood-borne diseases as etiologies (Ortner and Putschar 1997). Therefore, the presence of any one of these diseases may have exacerbated the sub-optimal nutritional status of this individual, thereby causing the manifestation of cribra orbitalia, or it was possible that these two were separate entities.

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If carcinoma is the etiology, it is possible that a deficiency in vitamin C may have intensified the state of this individual. Apparently, optimal levels of vitamin C have been demonstrated to afford some protection/resistance against a variety of chemical and physical carcinogens, as well as oncogenic viruses (Siegel 1993). This has been exhibited via supplementation of vitamin C and the concomitant increase in T lymphocyte activity, monocyte and macrophage functions including increased neutrophil chemotaxis and phagocytosis, and delayed hypersensitivity. Also, the role of vitamin C as an antioxidant may help to contribute significantly to its anticancer activity (Ito and Hirose, and Block *et al.* cited by Siegel 1993:184).

A nutritional deficiency may also have been displayed in this individual because of the pain that may have been associated with the ilium lesion and ambulation, thus decreasing access to food or it increases the reliance on others to obtain his/her food, thus leading to a possibly insufficient diet. Due to the incompleteness of this burial, it may be impossible to narrow down the exact etiology.

Actinomycosis, melorrheostosis or flowing hyperostosis of cervical vertebrae 3-5 was possibly identified in Burial Number 406. This pathology may have some relation to the presence of cribra orbitalia, porotic hyperostosis, wide-spread periostitis, and a possible cloaca of the right tibia. Although different etiologies may exist for these individuals, it may still be feasible to suggest that nutritional deficiencies may have been present as well, as infection and cancer are known to alter nutritional status (Siegel 1993).

By identifying pathologies characteristic of the lack of each of the four nutrients

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utilized in this study in each of the seven individuals, it is possible to present a multiple nutrient hypothesis. Due to the fact that each of these four nutrients can cause anaemia, then essentially paleopathological evidence has been presented for a potential multiple nutrient hypothesis for anaemia. In those individuals in which at least one other pathology, other than cribra orbitalia and porotic hyperostosis, was identified, a multiple nutrient hypothesis can still be advanced as more than one manifestation is being exemplified. Therefore, paleopathologists should no longer be concentrating on a single etiology when it comes to anaemia as identification of multiple nutritional deficiencies from the Dickson Mounds Mississippian Period population provides this evidence. Finally, nutritional deficiencies may be related to the presence of infections and this interrelationship should not be ignored.

7.3 DIFFERENTIAL DIAGNOSES

It is quite plausible that the pathologies described for those individuals with possible multiple nutritional anaemias in this collection may not have been caused by dietary deficiencies, but may be the result of other etiologies. However, the cause may still be nutritional, but due to a lack of other nutrients other than the four concentrated on in this thesis (e.g., copper). There is the possibility of not only genetic, i.e., sickle cell anaemia, thalassemia, but also environmental factors causing anaemia that may be involved. The likelihood that cribra orbitalia and porotic hyperostosis may be caused by the presence of genetic anaemias is highly unlikely for this area of the world as no evidence has been found

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for its existence here. The infracranial lesions characteristic for genetic anaemias were not found in the Dickson Mounds Mississippian Period skeletal collection. As a result, a genetic etiology can be ruled out.

Environmental factors are likely prospects as an explanation for the presence of cribra orbitalia and porotic hyperostosis in this collection as an increase in population within the area and sedentism has been demonstrated. An increase in population and sedentism drains the natural resources of the surrounding environment as *more* people are relying on the same amount of flora and fauna, and therefore the limited supply becomes exhausted and renewal becomes cumbersome. With a decline in some natural resources, others such as maize, may be relied on too extensively and nutritional deficiencies become inevitable due to such a focused diet.

This maize dependence has been exhibited not only via an increase in archaeological implements associated with agriculture (Harn 1971), but also with stable isotope analysis. Buikstra (1992) provides delta ^{13}C values from -12.6 ‰ (Early Mississippian: A.D. 1000-1150), -10.2 ‰ (A.D. 1150-1250) and -11.2 ‰ (A.D. 1250-1350) for the Dickson Mounds area. As carbon extracted from human bone normally ranges from -21.5 to -7.5 parts per mil (Chisolm cited by Buikstra 1992:87), the more reliance on C_4 plants such as maize, means that smaller negative numbers will ensue. Such high or "heavy" values suggest that maize, with its characteristic $^{13}C/^{12}C$ ratios, was relied on quite extensively by those in the Dickson Mounds area during the Mississippian Period. This adds credence to the presentation of cribra orbitalia, porotic hyperostosis and other nutritionally related pathologies observed in

this collection as maize is not composed of valuable levels of either iron, vitamin C, folic acid or vitamin B_{12} that were useable to the consumer. It also suggests that those flora and fauna (as presented in Tables 1 and 2 of Chapter 4) which were available to the people of Dickson Mounds were not necessarily being relied upon to supplement their diet.

With an increase in both sedentism and population levels comes a proportional flux in the decrease in hygienic conditions and consequently, a concomitant appearance of bacterial, parasitic and viral diseases within the population (Kent 1986). Therefore to some degree, demography of the Dickson Mounds population may have influenced the presence or predisposition of hyperostosis as well.

The presence of hookworms and/or tapeworms could also be a factor via the ingestion of infested fish and other foods. For example, the fish tapeworm, *Diphyllobothrium latum*, acquired from eating raw fresh water fish in northern Europe, the U.S.S.R., and sometimes in the northern United Stated has a strong appetite for vitamin B_{12} and thus, can cause megaloblastic anaemia for those individuals who contain the worm (Scrimshaw 1977:1537; Scrimshaw *et al.* 1959; Scrimshaw *et al.* 1968:43). Recovery from megaloblastic anaemia occurs only with the expulsion of these worms. Iron loss is also noted in several common infections such as hookworm and schistosomiasis to such an extent that as the number of worms increases, a similar concomitant loss of iron also occurs (Layrisse and Roche 1964). As a result, these environmental factors also help to contribute to nutrient depletion and anaemia, especially during times of infection via reducing the absorption of essential nutrients. Literature research to date has not provided any evidence

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of the presence of hookworms or tapeworms for the Dickson Mounds area and population. However, due to their reliance on fresh water fish and increase in population, which may have debilitated sanitary waste facilities, the presence of hookworm and tapeworm organisms may be warranted in the possible production of skeletal manifestations noted for this population.

The newest trend in paleopathological research pertaining to the manifestation of cribra orbitalia and porotic hyperostosis is that it has less to do with nutrition, particularly iron deficiency, and more to do with an 'adaptational' response of the body to invading microorganisms and/or parasites. By becoming hypoferremic, a mild state of iron deficiency, the body is essentially withholding iron from the pathogen invaders so that they cannot grow and proliferate. In essence, this has been termed a form of 'nutritional immunity' (Weinberg 1974, 1978 and 1984). Stuart-Macadam (1988, 1991, 1992) and others (e.g., see various chapters in Stuart-Macadam and Kent 1992 and those references cited within) proclaim that these lesions should no longer be used as an indicator of nutritional stress, but as an indicator of the attempt to adapt to the pathogen load in one's environment. As a result, cribra orbitalia and porotic hyperostosis are viewed as signs of successful adaptation rather than as an indicator of maladaptation (Holland and O'Brien 1997).

Those who support the 'nutritional immunity' hypothesis believe that the manifestation of these pathologies is a form of adaptation in which the body is able to withhold iron from the invading microorganisms and/or parasites. It has been demonstrated

that microbial growth is dependent upon the assimilation of free ionic iron from its host as these organisms are not equipped with their own reserves. As a result, they must compete with their hosts in order to obtain this iron so that they can grow and proliferate. In retaliation, the body tries to remove all obtainable free ionic iron from the serum by placing it in storage proteins, such as transferrin and lactoferrin, and the reticuloendothelial tissues. These proteins help to sequester iron from the body fluids and redistribute it into cellular storage sites. A corollary of this action is that many microbes produce a class of phenolate or hydroxymate compounds known as siderophores, which can chelate iron and make it accessible to the microbe (Bhaskaram 1988). However, the net result of these actions depends on the critical balance between the circumstances that control the concentration of free ionic iron in the body.

Those advocates who believe that diet is probably not a major factor in the manifestation of cribra orbitalia and porotic hyperostosis, state that iron is a ubiquitous trace element and it could take several years to become deficient in this one element. Therefore, they believe that the presence of parasites and/or microorganisms, and not a diet deficient in iron, are more important in the etiology of these pathological lesions. The evidence of the presence of pathogens within the Dickson Mounds environment has not yet been discovered. However, with such a dense population and sedentary lifestyle it would be most likely that some forms did exist. Also, with the presence of agricultural fields in the nearby vicinity, contact with possible hosts of carriers of infection, i.e., rats, could have occurred, thus increasing the likelihood of transmission.

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The large proportion of nonspecific periostitis found in this population may be indicative of the presence of infectious disease and thus, its presence may support the nutritional immunity and environmental hypotheses. However, due to the association of periostitis and the pathologies identified as nutritionally related for this thesis, it seems more appropriate not to label the etiology as solely pertaining to parasitism and/or microorganisms, but to also include, and rely more heavily on, a multiple nutrient hypothesis for this population. Unfortunately, no studies have been performed on coprolites or other archaeological remains that may have provided evidence for the existence of parasites and/or microorganisms at Dickson Mounds.

Pathogen invasions in the human body may be warranted as a plausible explanation for the decrease in circulating nutrients, namely iron, and their concomitant inaccessibility to parasites and/or microorganisms, but this can only be an acute physiological response by the body. This reaction by the human body is "adaptational" only when it is encountered for short periods of time. In no uncertain terms is it *adaptational* when the body chronically reacts to a pathogen load in this manner, as death would be inevitable if stored nutrients are withheld for important biological processes in the body or if bone becomes so degraded that physical fitness is affected. Also, acute episodes of parasitism would not be manifested in bones, as only chronic diseases have the ability to do so. As stated by Brown and Black (1981:471) "although such responses [the hypoferremic response] may represent an adaptive response in the defense against microbial invasion, they nevertheless create a *temporary* state of nutritional insufficiency" (added emphasis). The key word in this statement is

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'temporary' because the body can only withstand a short-lived state of hypoferremia before it becomes maladaptive. Since the only information essentially apparent about iron deficiency is that it has a cost, anaemia, it becomes problematic in understanding that long-term benefits exist with the state of hypoferremia. As a result, it would appear to be more disadvantageous to be suffering from anaemia, rather than it being a sign that an individual is adapting. Thus, something other than pathogen load must be causing the presentation of cribra orbitalia and porotic hyperostosis.

One is left to question the actual validity of the nutritional immunity hypothesis when studies have illustrated that anaemic episodes have decreased immunity to parasites. For example, Duncombe *et al.* (study cited by Sherman and Helyar 1988:179; study also performed by Bolin *et al.* cited by Sherman 1984:257) observed that young iron-deficient rats took longer to expel the intestinal parasite *Nippostrongylus brasiliensis* because of impaired immunological rejection. It was also discovered that iron deficiency diminished the acquired resistance to this parasite and reinfected iron deficient animals had higher worm counts than iron-sufficient. With the presence of invading organisms, iron absorption from the diet is inhibited and iron excretion is slightly increased so that access to this trace element is reduced. These circumstances are reversed during iron deficiency anaemia which entails a distinguishing trademark that can be identified in living patients, but unfortunately cannot be translated into skeletonized individuals.

Although the iron nutritional immunity hypothesis is attractive and many *in vitro* studies exist to provide positive evidence,

"... clinical data does not support the suggestion that iron deficiency protects against infection or that correction of iron deficiency, particularly if it is achieved gradually by oral iron therapy, increases incidence or severity of infectious disease in man... On the contrary, attempts to prevent development of iron deficiency in young children are associated with reduced infection-related morbidity...this may be due to the *essential role of iron in the optimum activity of enzymes* such as ribonucleotidyl reductase and myeloperoxidase, which play an essential role in functions of lymphocytes and neutrophils respectively" (emphasis mine Chandra 1990:14).

In fact, Chandra (1990) also states that chronic iron deficiency in young children has been associated with a slight increase in the incidence of common infections, such as upper respiratory illness or diarrhea. Also, according to Chandra, Woodford and Hyam (1977:260), the abnormalities in immunocompetence observed in individuals with iron deficiency can be easily and completely reversed via iron therapy, which possibly supports a cause-and-effect relationship between iron deficiency and reduced immune system functions.

According to Goodman (1994), Stuart-Macadam and other authors who embrace the nutritional immunity hypothesis are vulgarizing the concept of adaptation, or otherwise utilizing 'Cartesian Reductionism'. He proposes that "signs of stress are seen [by these researchers] as adaptations for no other reason than that they exist in stressed but surviving organisms" (Goodman 1994:164). By hypothesizing that iron deficiency is an adaptive response to stress suggests that a narrow and simplistic view of adaptation is being held. Such a view has serious implications because the relationship between the human body and the invader is just assumed to lead to adaptive readjustments with little comprehension of

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the underlying conditions being explored.

A major underlying condition that is important to understand is the nutritional status of an individual prior to the onset of disease as it can affect the virulence of the invading pathogen. It is believed by this author and Goodman (1994) that Stuart-Macadam misinterprets the concept of 'nutritional status'. By definition, nutritional status is "the state resulting from the balance between the supply of nutrients on the one hand and the expenditure of the organism on the other" (McLaren cited by Goodman 1994:166). Therefore, numerous factors can affect the accessibility and utilization of nutrients which may not be realized by those proponents of the nutritional immunity model.

Holland and O'Brien (1997) also take Goodman's point of view that the adaptational response of hypoferremia toward parasites and/or microorganisms presents a simplistic approach to evolution. They believe by placing cribra orbitalia and porotic hyperostosis under the 'umbrella of evolutionary theory', possibilities will arise with the misuse of the nutritional immunity model toward parasites that will evidently lead to a misunderstanding of the archaeological record. These authors, as well as this one, believe that it is erroneous to cite that the presence of hyperostosis is a sign of evolutionary success, because in fact, it is a sign that something is definitely wrong.

If hyperostosis is viewed as a form of successful evolution of the adaptation to pathogens, then it would be inferred that those who presented evidence of it skeletally would be the healthiest individuals or the cohorts with the best survival potential in the population (Holland and O'Brien 1997). This would then mean that females and children, along with

a small proportion of elderly, would be the healthiest cohorts of their community. This seems to be very ironic because these cohorts are viewed as usually being the most unhealthy and the most predisposed to the insult of disease. It then becomes hard to understand that Stuart-Macadam and others are implicitly proposing that these cohorts are showing signs of success, when in fact women, children and the elderly are often the more nutritionally disadvantaged in a population.

Goodman (1994:167) also presents a very important and influential passage taken from Dubo in 1978 stating that iron withholding should be viewed more as an adjustment rather than as an adaptation as,

> "There is *no clear evidence* that hypoferremia increases the long-term adaptations of the organism, and there is not a clear relationship between iron withholding, chronic iron status, and porotic hyperostosis . . . It is as if iron can be sequestered from microorganisms without affecting the need for iron in the host" (emphasis mine).

Although this passage maybe outdated due to the publication year of 1978, it still presents the impact on the human body if it were to withhold iron from microorganisms for extended periods. No studies have proven that by placing an individual in a hypoferremic state, does the incidence of infection from invading pathogens decrease. However according to Sherman (1984:254), anaemia of infection does afford the host the advantage of competition between itself and the microbe for iron, and ultimately for survival. But by becoming hypoferremic for long periods of time many biological consequences can occur: iron deficiency can affect resistance to disease, work capacity or activity and cognition and behaviour. With these devastating costs in mind, it becomes possible that deficiencies can

be related to deleterious social and political changes that can consequently influence nutritional adequacy and disease load in a population.

The connection between deficiencies in iron, vitamin C, folic acid and vitamin B_{12} with respect to abnormal functioning of the immune system were discussed in Chapter 2.2. Although some evidence exists against the deleterious effects of nutritional deficiencies on immunity, that is, deficiencies do not alter the system, a substantial amount of information does support the fact that without these nutrients, problems in proper defense against microbes and parasites will ensue. Therefore, it becomes clear that iron, vitamins C and B_{12}, as well as folic acid are critical to the immune system. As a result, this information should not be ignored and should be taken into consideration when observing populations exposed to certain infections.

This discussion has brought together many different possible etiologies regarding the presentation of certain pathologies identified within the Dickson Mounds Mississippian Period skeletal collection. One should recognize that in general, paleopathological diagnoses are extremely complicated, but when nutritional deficiencies are added, it becomes even more difficult to assess the skeletal alterations. This is due to the fact that human nutrition and metabolism are severely abstract. Although any of the etiologies put forth in this discussion are plausible, I am suggesting that within the realm of nutritional deficiencies a new perspective is needed. With the evidence presented from this Dickson Mounds sample it should be acknowledged that paleopathologists should no longer isolate iron deficiency as the sole cause for the presentation of nutritional anaemia, but that other

nutritional deficiencies, perhaps multiple nutrients, should be included within the etiology

if it is warranted.

CHAPTER 8: CONCLUSION

Evidence of skeletal manifestations of anaemia in the anthropological literature is abundant. Although a substantial amount of information exists regarding the possible etiologies: genetic, environmental (parasite and microorganisms included) and nutritional, more research is needed. This is especially relevant for the realm of nutritional anaemia. Many researchers have moved away from nutritional hypotheses concerning anaemia because they believe that iron is a ubiquitous element in nature and thus, deficiencies should be rare. As a result, nutritional etiologies have digressed in order to concentrate on new trends that may explain the presentation of cribra orbitalia and porotic hyperostosis. Although these new areas of research are warranted and provide substantial evidence, other areas have been ignored.

This thesis has attempted to again focus the study of anaemia in past populations on nutrition by putting forth a different approach to nutritional hypotheses. The iron deficiency anaemia hypothesis has been generally dismissed because it does not provide a full explanation for the presence of anaemia, especially in those populations which consumed sufficient iron. Therefore, other factors must have been present to alter skeletal material characteristic of anaemia. Since anaemia is the result of a disturbance in hemoglobin synthesis, *any* nutrient that is involved in hematopoiesis and/or any nutrient required during the absorption, transportation, activation and storage of these nutrients will consequently be affected by deficiencies. Therefore, because past studies have isolated iron as the only proponent in a nutritional etiology for anaemia, the nutritional basis for this condition has

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lost credibility.

Medical and clinical research has provided the evidence that many nutrients can be responsible for developing anaemia. This research has also illustrated the fact that it is quite rare to be deficient in only one nutrient at a time, as nutrients are interdependent within the human body. Within the realm of the four nutrients isolated for this study, it has become apparent that they interact substantially. As examples, iron depends on vitamin C for proper absorption from the plasma to the liver; both iron and ascorbic acid are required during the proline hydroxylation reaction whereby normal formation of collagen occurs; conversion of folate to the circulating form of folic acid via reductions and substitutions in the pteridine ring is dependent on ascorbate and vitamin B_{12}; and both folate and a vitamin B_{12}-containing enzyme are needed for the synthesis of thymidylate and thus, DNA synthesis. As a result, each of these four nutrients is dependent on another and therefore, a synergistic interrelationship exists whereby a deficiency in one can cause metabolic problems associated with another. This becomes most important in the maintenance of erythropoiesis. When iron, vitamin C, folic acid and vitamin B_{12} are lacking in the diet, the many maturational stages of an RBC are affected causing alterations in the quality and quantity of mature erythrocytes.

Due to the interrelationship between these four nutrients and their role in hematopoiesis, it is proposed that when iron, vitamin C, folic acid and vitamin B_{12} are deficient in the diet, thus causing anaemia, similar skeletal alterations are manifested. These bony changes are in the form of cribra orbitalia and porotic hyperostosis. By presenting one

or both of these pathologies, anaemia in the Dickson Mounds skeletal population was diagnosed. At the same time, characteristic skeletal changes for single nutrient deficiencies of iron, vitamin C, folic acid and vitamin B_{12} were also identified on the same skeletons. As a result, similar bony alterations tied these four nutritional factors together within the realm of anaemia, but in return, their differences allowed for the identification of each single deficient nutrient.

By using this approach, the presentation of deficiencies in iron, vitamin C, folic acid and vitamin B_{12} were identified in several individuals from the Mississippian Period skeletal collection from Dickson Mounds. After a macroscopic assessment of the sample, seven individuals possessed bony alterations that were indicative of deficiencies in all of these nutritional factors. Sixteen additional individuals from Dickson Mounds presented signs of deficiencies in at least two of these nutrients. Therefore, more than 20% of the 110 individuals from the Mississippian Period skeletal sample present with multiple deficiencies and demonstrate the possibilities of observing multiple nutrient deficiencies that are linked to anaemia.

Nutritional deficiencies in the Dickson Mounds population may have been caused by several factors. One important reason stems from the regular consumption of a diet lacking in these four nutrients. Biogeographical information of the Spoon River area reveals that an abundant and diverse assortment of fauna and flora were available to the Dickson Mounds people. The nutritional compositions of these foods suggest that sufficient nutrient consumption should have been attained if *all* of the resources were being utilized. However,

archaeological evidence and stable isotope analysis suggests that a heavy reliance on maize occurred in this time period. An increase in population and sedentism during the Mississippian Period at Dickson Mounds may have drained the resources to such an extent that little dietary variety was available. Also, it has been proposed that a large proportion of the fauna available to these people were hunted and traded to larger populated centers in the South in exchange for more prestigious nonedible food items such as rare shells. It should also be kept in mind that cooking and storage practices employed by these people may have ultimately decreased the amount of nutrients available in the foods that were eaten. Therefore, it becomes highly possible that nutritional deficiencies in iron, vitamin C, folic acid and vitamin B_{12} were plausible, especially if these people relied heavily on nutrient-poor maize.

Skeletal evidence of the appearance of nutritional deficiencies may also have been caused by the presence of diseases. Infections can debilitate an individual's nutritional status to such an extent that levels of nutrients can become critical. Deficiencies can result if a person begins with relatively low levels and contracts a parasite or microbe, or deficiencies can occur if the invasion on the immune system is prolonged. This can arise because of a decrease in food consumption during periods of illness, depressed absorption of nutrients and an increase in catabolic reactions during infection. An individual's nutritional status can also dictate the severity of the infection, as those with improper nutrient levels tend to suffer from more illnesses in general, which are usually more serious and protracted. Therefore, loss of nutrients during infections can place an individual under

extreme nutritional and immunological stress.

Analysis of the Dickson Mounds sample provided evidence that diseases were affecting the Mississippian Period population resulting in a high prevalence of periostitis and/or osteitis discovered in the entire sample. Such high rates of nonspecific infections within this population may be related to the inability of individuals to defend against parasite and/or microorganism invasions because of insufficient nutrient stores. A deficiency in vitamin C may have been related to severe forms of tuberculosis-like lesions in several adults and a hemangioma in a subadult, as insufficient levels of this vitamin can impair immunologic responses and intensify carcinomic entities. There is also the possibility that the presence of these two diseases, along with other nonspecific infections in this population, could have altered the nutritional status and consequently, the survivorship of the people buried at Dickson Mounds.

8.1 FUTURE RESEARCH

This thesis has illustrated the possibilities of a multiple nutrient hypothesis for an etiology of anaemia. However, a macroscopic analysis of these skeletal remains can be limiting as the naked eye can only observe so much. Therefore, other alternatives, i.e., radiographic and histological methods, should be entertained for future research in this area. A histological approach would have been most beneficial for the identification of megaloblastic anaemia as this disease affects all cells within the human body. Therefore with good preservation of bone, hypersegmented nuclei may have been observable in

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megaloblastic individuals due to the insufficient production of amino acids and defective DNA synthesis. Unfortunately, no study of this sort has been performed on skeletal material, but it does leave the door open for future research.

Another test that researchers employ now to identify folic acid deficiency in living humans that paleopathologists may be able to utilize in the future, is the deoxyuridine (dU) suppression test. A dU suppression test allows for the analysis of DNA synthesis in patients who are folate deficient. This test measures the specific deficiency of folic acid in the bone marrow or lymphocytes by the *in vitro* suppression of incorporation of [^{3}H] thymidine into DNA when non radioactive dU plus the deficient vitamin is added (Herbert 1989:52). Apparently, the megaloblastic hematopoiesis found in folate deficiency is caused by the impaired methylation of deoxyuridine monophosphate (dUMP) to thymidine monophosphate (dTMP), which in turn leads to a reduced supply of thymidine triphosphate (dTTP) at the DNA replication fork and a reduced rate of DNA strand replication (Gross *et al.* 1975:229; Wickramasinghe and Fida 1994:1656). As such, the composition of the DNA synthesized in deficiency states may be abnormal as a result of misincorporation into DNA of uracil in lieu of thymine (Wickramasinghe and Fida 1994:1656; also cited by Gross *et al.* 1975:229). Perhaps it could be possible to use dU suppression tests that these authors employed on living subjects and apply it to DNA extracted from archaeological human remains. However, this form of analysis should only be utilized when actual skeletal evidence of folate deficiency is observed first, due to the high cost of this experiment and the destructive nature of DNA extraction procedures.

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Analysis of the Late Woodland sample from Dickson Mounds would have allowed for a comparison between two different time periods. In effect, this could substantiate the claims made in this thesis as different rates of cribra orbitalia and porotic hyperostosis have been found between these two populations. For example, differences in population numbers, habitation, presence/absence of parasites and/or microorganisms, diet and subsistence would have added credence to the possible underlying factors that can lead to nutritional deficiencies within the Mississippian Period skeletal sample. However, comparisons between these two time periods, as well as other populations have already been performed in the literature (e.g., Lallo *et al.* 1977). As a result, it is known that differences *do* exist and that an increase in maize subsistence, a decrease in faunal subsistence, with the possibilities of sedentism and population expansion have elevated the rates of nutritional deficiencies and the presence of concomitant anaemic skeletal alterations in this population.

A major area that must be placed under consideration for not only this study, but for all those that follow which concentrate on nutrition in past populations, is that researchers must be wary in assuming that prehistoric health can be measured using modern Western standards. Many anthropologists employ standards based on estimates from U.S. government studies of modern Americans on those populations that they are examining. However, if geographic and temporal differences are taken into consideration, as well as adaptations to diverse environments through both biological and cultural means, then a better assessment will be produced. As a result, anthropologists can never assume that evolution has afforded humans with a shared nutritional requirement. Compositions have

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changed in order to adapt to different environments, populations, habitations and subsistence practices.

As a result of these dietary differences, differential presentation of nutritional anaemia in independent time periods, geographic locations and subsistence practices substantiates the nutritional hypothesis for anaemia. This becomes evident when considering a question posed by Holland and O'Brien (1997:190) that "if hunter-gatherer groups also lose blood and acquire parasites, why then do bioarchaeologists report that the skulls of earlier hunter-gatherer groups typically do not exhibit the evidence of anemia that the later agriculturist groups do?" In effect, this fascinating question appears to refute the parasitism model and suggests that in any other model dealing with the presentation of hyperostosis, diet <u>must</u> be an included factor.

It is interesting/ironic to note that Holland and O'Brien (1997:184) agree that it is time to reevaluate thoughts about maize and health as well as other factors intertwined within the origins and the evolution of agriculture, but they "question the need to replace one overly simplistic model with another". While this thesis does not present a model, per se, it puts forth a view that nutrition should not be deemed as simplistic, but rather very complicated. If anything, this thesis has provided evidence that paleopathologists can no longer utilize simplistic models, especially pertaining to possible nutritional pathologies, as only complicated relationships exist within the human body. Therefore, simplistic views should no be longer formulated as diet contains a *multitude* of factors. As an entity, diet *is* a single factor, but it comprises many different levels, such as availability, use and nutrient

composition of foods, as well as absorption, utilization, storage and relationships of nutrients that ultimately interact with one another in a sophisticated and synergistic manner. Therefore, deficiencies and their impact on the human skeleton must be examined in a very arduous fashion within the contexts of environment and nutritional status of a population. Without such a disposition, researchers will lose sight of the complex nature of nutrition and eventually revert back to single nutrient hypotheses that have plagued the paleopathological literature in the past. The time has come to present an improved view regarding a nutritional etiology for anaemia: more than one nutrient may be the cause.

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APPENDIX

SEX DETERMINATION RECORD

Inventory #:   Burial #:   Catalogue #:

SEX:	MALE	FEMALE	INDETERMINATE

PELVIC OBSERVATIONS:	MALE	FEMALE
1. Subpubic Concavity:	V shape	U shaped
2. Ischiopubic Ramus:	no ridge	ridge
3. Ventral Arc:	absent	present
4. Pubic Bone Shape:	narrow	wide;rectangular
5. Dorsal Pitting:	absent	present
6. Sciatic Notch:	sm;acute;deep	lrg;wide;shallow
7. Auricular Surface:	not raised	raised margin
8. Pre-auricular Sulcus:	absent	present
9. Ilium Shape:	high;vert. trend	lower;lat. divergent
10. Pelvic Inlet:	heart shaped	elliptical
11. True Pelvis:	rel. small	rel. wide;shallow
12. Obturator Foramen:	large;ovoid	sm;triangular
13. Acetabulum:	lrg;lat. directed	sm;ant-lat directed
14. Sacrum Shape:	long;narrow	short;broad
15. # Sacral Elements:	5+	5
16. Sacral Curve (lat):	even gentle curve	sharp S1-S2 & S3-S5
17. Muscle Marking:	marked;rugged	faint;smooth

CRANIAL OBSERVATIONS:	MALE	FEMALE
1. Rel. Size; Build	lrg;rugged	sm;smooth
2. Forehead:	steep;vert.	full;rounded
3. Frontal Eminences:	small	large
4. Supraorbital Tori:	medium-large	small-medium
5. Orbits & Margins:	low;sq & sm;round	high;rnd;lrg & sharp
6. Nasal Aper. & Margin:	high;nrw & sharp	low;wide & sharp
7. Nasal Bones:	lrg;sharp > midline	sm;wide > at junct
8. Zygomatic Bones:	hvy;ends post E.A.M.	grac;ends pre E.A.M.
9. Parietal Eminences:	small	large
10. Mastoid Process:	medium-large	small-medium
11. Occipital Area:	pro. muscle lines	protub not marked
12. Occipital Condyles:	large	small
13. Palate Shape:	lrg;broad;U shaped	sm;parabolic
14. Rel. Tooth Size:	lrg;5 cusp M1	rel. small

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(continued)

	MALE	FEMALE
15. Mandible Size:	lrg;high symp.	sm;lower corp. & ramus dimensions
16. Gonial Angle:	<125;flaring	>125;no flare
17. Chin Form:	square	rounded

METRICS (Arikara Aboriginals):
1. Prox. Breadth: >74.56mm = MALES
2. Distal Breadth: >50.88 = MALES
3. Circ. Nut. For.: >91.21mm = MALES

AGE DETERMINATION RECORD

Inventory #:   Burial #:   Catalogue #:

AGE: +/-   yrs./months

SUBADULT:

TOOTH ERUPTION: AGE =

DIAPHYSIS LENGTH: Humerus =   mm   Radius =   mm

                             Ulna =   mm   Femur =   mm

                             Tibia =   mm   Fibula =   mm

                             Ilium Breadth =   mm   **AGE =**

EPIPHYSEAL FUSION: **AGE =**

ADULT:

CLAVICLE: Phase 1 / 2 / 3   **AGE =** 18-20 / 21-25 / 26-30

PELVIC DATA: 1. Pubic Symphyseal Face: **AGE =**

                 2. Auricular Surface: **AGE =**

STERNAL RIB ENDS (4TH rib):   **AGE =**

Male:		Female:	
Phase 0: <16.5		Phase 0:	
Phase 1: 16.5-18		Phase 1: <15.5	
Phase 2: 24.1-27.7		Phase 2: 15.5-19.3	
Phase 3: 24.1-27.7		Phase 3: 20.5-24.7	
Phase 4: 25.7-30.6		Phase 4: 24.4-31	
Phase 5: 34.4-42.3		Phase 5: 33.7-46.3	
Phase 6: 44.3-55.7		Phase 6: 43.3-58.1	
Phase 7: 54.3-64.1		Phase 7: 59.2-71.2	
Phase 8: 65-78		Phase 8: 70.4-82.3	

ECTOCRANIAL SUTURES: **AGE =**

    Pterion: 0 / 1 / 2 / 3   Midlambdoid: 0 / 1 / 2 / 3

    Sphenofrontal: 0 / 1 / 2 / 3   Lambdoid: 0 / 1 / 2 / 3

DENTAL WEAR: **AGE =**

Maxilla:		Mandible:	
Phase A: 12-18		Phase A: 12-18	
Phase B1: 16-20		Phase B1: 16-20	
Phase B2: 16-20		Phase B2: 16-20	
Phase C: 18-22		Phase C: 18-22	
Phase D: 20-24		Phase D: 20-24	
Phase E: 24-30		Phase E: 24-30	
Phase F: 30-35		Phase F: 30-35	
Phase G: 35-40		Phase G: 35-40	
Phase H: 40-50		Phase H: 40-50	
		Phase I: 45-55	

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